Chapter 5: Obesity

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Background

The prevalence of overweight and obesity has been increasing in many countries and has been recognised as a global cause for concern (World Health Organization, 2007). Excess body weight has been associated with several adverse health outcomes and with important consequences for cardiovascular health in particular. Estimates suggest 35% of ischaemic heart disease (IHD) cases are attributable to overweight and obesity, as well as 80% of type 2 diabetes cases and 55% of hypertension cases (World Health Organization, 2007).

In England, 2009 data suggest 33% of women and 44% of men were overweight, with a further 24% of women and 22% of men classified as obese (NHS Information Centre, 2010), a pattern consistent with other nations of the UK (Bates *et al.*, 2009). If trends are to continue, it has been predicted that, by 2050, 60% of men and 50% of women will be obese (Butland *et al.*, 2007). A similar pattern has been observed in children and adolescents in the UK, with around 25% of four- to 10-year-olds and 36% of 11- to 18-year-olds reported as overweight (Bates *et al.*, 2009). It is thought that one quarter of children may be obese by 2050 (Butland *et al.*, 2007). With the considerable health costs associated with the co-morbidities, this represents a significant burden for health care systems.

Overweight and obesity are international classifications of body fatness, and are defined as having a body mass index (BMI) of ≥25kg/m^2 and of ≥30kg/m^2 respectively (World Health Organisation, 1995). Although a crude weight-for-height measure, BMI provides a useful tool for the assessment of health risk. At present, WHO does not recommend different, population-specific BMI cut points for determining overweight and obesity, despite evidence that adiposity-related disease risks may be higher at lower BMI levels in certain populations (WHO expert consultation, 2004). Rather, according to the WHO Expert Consultation, intermediate cut-off points of 23.0, 27.5, 32.5 and 37.5 kg/m^2 should be used, where necessary, as trigger points for public health action.

In children, where the interpretation of BMI is limited by growth trajectories, it is common practice to interpret BMI in relation to an age-specific reference population mean. The International Obesity Task Force (IOTF) thus defines obesity in childhood as the BMI Z-score (or standard deviation score) that, if maintained through to adulthood (18 years), would equate to a classification of overweight and obese (Cole *et al.*, 2000). Other classifications are also found in the literature: for example, adopting cut-off points at the extremes of distribution, such as above the 90th centile. In these methods, the mean, standard deviation and centiles will vary according to the reference population or growth charts used. The studies included in this review employ different reference sources and a variety of cut-off points for describing incident overweight and obesity in children. As well as incident overweight and obesity, many of the studies in the review report continuous measures of or changes in body weight (in kg or lb).

Body fatness can also be estimated using a number of other methods. Skinfold thickness measurements can be taken using callipers at the biceps, triceps, subscapular and supra-iliac sites, to give an indication of subcutaneous fat. Waist circumference is a crude measure of intra-abdominal fat, which has been associated with increased risk of cardio-metabolic disorders: a circumference of 102cm or more in men and 88cm in women constitutes one of the defining facets of metabolic syndrome (Scottish Intercollegiate Guidelines Network, 2007). Waist-to-hip ratio is also thought to give an indication of body fat distribution. Body fat distribution can be assessed using dual-energy X-ray absorptiometry (DEXA) when applied to specific areas of the body or combined with further measurements of body dimensions such as sagittal diameter and/or waist circumference.

The role of carbohydrates in weight gain or incidence of obesity is unclear. The theoretical basis for the purported carbohydrate-body weight link lies in the glycaemic response elicited by carbohydrates, with the suggestion that foods with a high glycaemic index or load (GI or GL) may interfere with hunger and satiety signalling, and consequently lead to overconsumption (Bornet *et al.*, 2007;Bellisle and Drewnowski, 2007;Van Dam and Seidell, 2007). There has also been the suggestion that liquid sources of carbohydrates may interfere with appetite regulatory systems to a greater extent than solid energy sources (Van Dam and Seidell, 2007;Bellisle and Drewnowski, 2007). Existing reviews in the field, however, are cautious in their conclusions, citing inconsistencies in the literature (Van Dam and Seidell, 2007;Gibson, 2008;Vega-Lopez and Mayol-Kreiser, 2009). Many reviews focus on a specific type of carbohydrate, such as added sugars, or sugar-sweetened beverages (Gibson, 2008;Malik *et al.*, 2006). Some authors assert that increased intakes of carbohydrates, as indicated by dietary surveys, have occurred concurrently with the rising rates of overweight and obesity (Dyson, 2008;Malik *et al.*, 2006). An early report by the Committee on Medical Aspects of Food Policy (Committee on Medical Aspects of Food Policy, 1989) reported evidence of an inverse relationship between reported sugar intake and body weight (Richardson, 1972;Keen *et al.*, 1979). On the other hand, the same publication reported evidence of weight loss prompted by sugar restriction (Mann *et al.*, 1970;Rifkind *et al.*, 1966;Werner *et al.*, 1984;Thornton *et al.*, 1983).

The focus of this chapter is to review literature investigating the prevention of weight gain or increased body fatness in relation to dietary carbohydrate in healthy individuals (or those with minimally raised markers of cardiometabolic disease). Cohort studies were required to have a minimum duration of follow-up of at least 3 years for inclusion. Additionally, cohort results in which a change in intake *and* change in outcome are reported over the same time period were excluded. This decision was taken so that results were included only where a direction of effect could be identified. Intervention trials were only included that were at least one year in duration.

Previous studies in COMA reports

The two tables below list studies included in previously published reports from the Committee of Medical Aspects of Food Policy (Committee on Medical Aspects of Food Policy, 1989;Committee on Medical Aspects of Food Policy, 1994;Lewington *et al.*, 2007) that concerned the relationship between dietary carbohydrates and obesity.

Excluded studies

Articles listed in Table 5.1 were not eligible for inclusion in this review for the reasons listed.

Table 5.1 Previous studies in COMA reports*: excluded studies

Authors, Year	Intervention description	Intervention duration/ follow up	Exclusion code that would be applied in this review	Exclusion detail
(Burr *et al.*, 1989)	1) Fat advice 2) Fish advice 3) Fibre advice	2 years	6	Subjects did not fit the definition of 'healthy' – all were diagnosed with acute myocardial infarction.
(Dreon *et al.*, 1988)	Not applicable	Not applicable	2	The study was not a randomised trial or cohort/prospective study (cross-sectional survey).
(Kanders *et al.*, 1988)	1) Balanced deficit diet 2) Balanced deficit diet supplemented with aspartame	12 weeks	3	No carbohydrate difference between groups was reported.
(Keen *et al.*, 1979)	Not applicable	Not applicable	2	The study was not a randomised trial or cohort/prospective study (cross-sectional survey).
(Kromhout, 1983)	Not applicable	Not applicable	2	The study was not a randomised trial or cohort/prospective study (cross-sectional survey).
(Lissner *et al.*, 1987)	1) Low fat, high carbohydrate 2) Medium fat, medium carbohydrate 3) High fat, low carbohydrate	2 weeks	2	Subjects were not reported to be randomly allocated to groups.
(Lock *et al.*, 1980)	1) Usual diet + sucrose 2) Usual diet + dried glucose syrup	2 years	2	Subjects were not reported to be randomly allocated to groups.
(Macdonald, 1967)	1) Sucrose-cream diet 2) Sucrose-sunflower oil diet 3) Glucose-cream diet 4) Glucose-sunflower oil diet	5 days	2	Subjects were not reported to be randomly allocated to groups.
(Mann and Truswell, 1972)	1) Basal diet 2) Basal + starch diet 3) Basal + sucrose diet	14 days	6	Subjects did not fit the definition of 'healthy' – all had been admitted to hospital with non-metabolic conditions such as cerebral vascular accident and nerve palsy.
(Mann *et al.*, 1974)	1) Basal diet 2) Basal diet + sucrose replaced by starch 3) Basal diet + starch removed	14 days	2	Subjects were not reported to be randomly allocated to groups.
(Porikos *et al.*, 1977)	1) Aspartame sweetened products	15 days	2	Subjects were not reported to be randomly allocated to groups.
(Porikos *et al.*, 1982)	1) High sucrose diet 2) Aspartame diet	24 days	2	Subjects were not reported to be randomly allocated to groups.
(Reiser *et al.*, 1979a)	1) Sucrose diet 2) Starch diet	6 weeks	2	Subjects were not reported to be randomly allocated to groups.
(Reiser *et al.*, 1979b)	1) Diet comprised 30% of calories from sucrose 2) Diet comprised 30% of calories from wheat starch	6 weeks	2	Subjects were not reported to be randomly allocated to groups.
(Richardson, 1972)	Not applicable	Not applicable	2	The study was not a randomised trial or cohort/prospective study (cross-sectional survey).
(Rifkind *et al.*, 1966)	1) Sucrose-restricted diet	10 weeks	2	Subjects were not reported to be randomly allocated to groups.
(Romieu *et al.*, 1988)	Not applicable	1 year	5	Data not reported on relevant outcome in relation to relevant carbohydrate.
(Rosenthal *et al.*, 1985)	1) High-complex-carbohydrate, high-fibre, low-fat, low-cholesterol diet	26 days	2	The study did not have a 'control' group - all subjects received the same intervention.
(Thornton *et al.*, 1983)	1) Usual diet + refined carbohydrate foods 2) Usual diet + wholegrain foods	6 weeks	6	Subjects did not fit the definition of 'healthy' – all had radiolucent gall stones.
(Werner *et al.*,	1) Usual diet + sucrose	6 weeks	6	Subjects did not fit the definition of 'healthy' – all

Authors, Year	Intervention description	Intervention duration/ follow up	Exclusion code that would be applied in this review	Exclusion detail
1984)	2) Usual diet + saccharine			had radiolucent gall stones.

Included studies

The following paper would have been eligible for inclusion in this review had it been published after the 1990 cut point.

Table 5.2 Previous RCT in COMA reports*: included study

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial Design (washout duration)	Intervention duration	Intervention Style	Total number of participants	Intervention description
(Mann *et al.*, 1970)	Generally healthy	Office workers 100% Male Age: 35-53	Parallel Group	22 weeks	Substitution	51	1) Low sugar diet – foods containing sucrose were cut out and replaced with substitutes to maintain weight. 2) Reduced starch diet – starchy foods were halved and replaced with substitutes to maintain weight. 3) Usual diet.

*(Committee on Medical Aspects of Food Policy, 1989;Committee on Medical Aspects of Food Policy, 1994;Lewington *et al.*, 2007)

This randomised controlled trial assessed serum-lipids and weight loss in men (n=51) over a 22-week period (Mann *et al.*, 1970). Subjects were randomised to either a low sugar diet, a reduced starch diet or were requested to continue with their usual diet. Men in the low sugar diet group tended to experience greater weight loss compared to the reduced starch group, in which weight loss was minimal. The statistical significance of the differences however was not reported.

Summary of the evidence base

Cohort Studies

An overview of each cohort study that provides data for this chapter may be seen in Table 5.3. **No meta-analyses of cohort data were possible using this evidence base. This was generally due either to variation in detail of outcome or exposure within a limited number of studies, although insufficient information concerning the variance around the risk estimates was also occasionally an issue. Essentially, studies were not similar enough to be pooled.**

In total, 37 papers provided data on 29 studies. Of these, 13 studies followed cohorts of children or adolescents, and the remainder studied adults. Data on children younger than age 5 were not included in this review.

These cohort studies were conducted in the USA (14), Denmark (3), Australia (3), the UK (2), Germany (1), the Netherlands (1), Norway (1), Finland (2), and Europe (EPIC) (1). Most included both male and female participants, but 7 cohorts had only females (Palmer *et al.*, 2008;Hays *et al.*, 2006;Phillips *et al.*, 2004;Albertson *et al.*, 2009;Colditz *et al.*, 1990;Sammel *et al.*, 2003;Fiorito *et al.*, 2009) and 2 included men only (Koh-Banerjee *et al.*, 2003;Bazzano *et al.*, 2005).

Dietary assessment was mostly achieved through food frequency questionnaires (FFQ), but a smaller number of studies employed food diaries (Magarey *et al.*, 2001;Johnson *et al.*, 2007;Albertson *et al.*, 2009;Tam *et al.*, 2006;Cheng *et al.*, 2009), dietary recall (Fiorito *et al.*, 2009;Haffner *et al.*, 1991) or the dietary history method (Boreham *et al.*, 1999;Rissanen *et al.*, 1991;Koppes *et al.*, 2009).

Length of follow-up ranged from the minimum for inclusion (3 years) in the National Weight Control Registry study (Phelan *et al.*, 2007) to a maximum of 23 years in the Amsterdam Growth and Health Study (Koppes *et al.*, 2009). The average duration of follow-up was 8 years (taking longest follow-up for multiple papers).

The size of each cohort study in terms of participant numbers at baseline varied markedly. No restriction was placed on size of cohort with regard to inclusion in the review. The smallest cohort study of healthy postmenopausal women had just 67 participants (Hays *et al.*, 2006), and the largest was the multicentre European study (EPIC) with in excess of 146,000 participants (Du *et al.*, 2009).

Please interpret observational data with caution: With observational studies, especially in the field of diet and nutrition, there is substantial potential for biases caused by incomplete adjustment for confounding, measurement error in the exposure estimate, and other biases in participant selection or data collection. The bias could be large in size, and act in either direction, either towards or away from the null.

Trial Design

Thirty one publications provided information on the relationship between body weight, weight change, markers of adiposity and aspects of dietary carbohydrate.

Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4. These papers provided data from 26 studies of adults and 4 of children or adolescents (Abrams *et al.*, 2007;Ebbeling *et al.*, 2003;Demol *et al.*, 2009;James *et al.*, 2004). None employed a cross-over design. There are two papers from the Women's Health Initiative Dietary Modification Trial, which provided data at different periods of follow-up (Tinker *et al.*, 2008;Howard *et al.*, 2006). Most studies used men and women, but 10 included only women (Bhargava, 2006;Clifton *et al.*, 2008;Dale *et al.*, 2009;Gardner *et al.*, 2007;Howard *et al.*, 2006;Pasman *et al.*, 1997a;Pasman *et al.*, 1997b;Shah *et al.*, 1996;Sheppard *et al.*, 1991;Sichieri *et al.*, 2007a;Tinker *et al.*, 2008). With the exception of the Women's Health Initiative Dietary Modification Trial (Tinker *et al.*, 2008;Howard *et al.*, 2006) and the feasibility study for this trial (Sheppard *et al.*, 1991) in which a large proportion of participants were less than BMI 25, all trials of adults used overweight or obese participants only. No trials of exclusively lean individuals met the criteria for inclusion in this review. The evidence base is therefore mainly reliant on studies that have explored the impact of dietary carbohydrate on individuals with pre-existing excess adiposity.

Trials were conducted in the USA (16), Denmark (1), Australia (3), Spain (1), New Zealand (2), the UK (1), Germany (1), the Netherlands (2), Brazil (1) and Israel (2).

The duration of interventions are detailed in the Trials Characteristics Table, and ranged from 12 months to 6 years in the Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006).

The average number of participants across all trials, except the very large Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006;Tinker *et al.*, 2008), was 230. Thirteen trials were rather small, with less than 100 participants in total.

Certain studies stated that diets were *ad libitum* in nature (Due *et al.*, 2004;Ebbeling *et al.*, 2003;Ebbeling *et al.*, 2007;Ebbeling *et al.*, 2005;Shah *et al.*, 1996;Swinburn *et al.*, 2001), that is, the participants were not guided to consume a specific quantity of food or to achieve a certain level of energy intake.

Risk of bias

A summary of the risk of bias assessment is provided in Table 5.5. Criteria for judging whether a risk of bias was evident were based on the Cochrane Handbook. A judgement of ‘unclear’ was provided if there was insufficient evidence within the paper to make a clear judgement. Judgements concerning whether there was evidence of a risk of bias in terms of outcome assessment (the experimenters involved in assessing the outcome were aware which intervention had been followed by each participant) are reported as the final column in each of the specific results tables.

All trials included were randomised controlled trials. All were judged to be either ‘unbiased’ or ‘unclear’ (method of random allocation to groups not reported in paper) in terms of allocation sequence generation or allocation concealment. None were judged to be ‘biased’ in these aspects of trial design. Blinding of participants and researchers to the various dietary approaches was more difficult to achieve, as might be anticipated with dietary intervention trials. Only 1 trial (of children) was judged as ‘unbiased’ in respect of participants awareness of the dietary intervention (Abrams *et al.*, 2007), but 6 trials were judged to have ‘no bias’ in respect of researchers awareness (Ebbeling *et al.*, 2007;Howard *et al.*, 2006;Gardner *et al.*, 2007;Pasman *et al.*, 1997b;Sacks *et al.*, 2009).

There was some evidence of incomplete (Abrams *et al.*, 2007;Abete *et al.*, 2008;Carels *et al.*, 2005;Delbridge *et al.*, 2009;Keogh *et al.*, 2007;McManus *et al.*, 2001;Pasman *et al.*, 1997a;Pasman *et al.*, 1997b;Sheppard *et al.*, 1991;Sichieri *et al.*, 2007a) or selective outcome reporting in certain publications (Das *et al.*, 2007;Demol *et al.*, 2009;Ebbeling *et al.*, 2003;Greenberg *et al.*, 2009;Pasman *et al.*, 1997a;Abrams *et al.*, 2007).

Table 5.3 Description of cohort studies providing outcome data on body weight, body mass index, body fat and fat distribution

Study name	Reference	Country	Subjects age	Length of follow-up (years)	Gender	Dietary assessment method	Method of body weight assessment	Total number of cohort participants at baseline*	Loss of cohort members to follow-up %
				Studies of children and adolescents					
Adelaide Longitudinal Study of Growth & Nutrition	(Magarey *et al.*, 2001)	Australia	Children (2-15 yr)	13	M&F	3-d and 4-d food diary	Measured	500	Not reported
ALSPAC	(Johnson *et al.*, 2007)	UK	Children (5+ yr)	4	M&F	3-d food diary	Measured	692	25
	(Johnson *et al.*, 2008)	UK	Children (5+ yr)	4	M&F	3-d food diary	Measured	682	25
Amsterdam Growth and Health Study	(Koppes *et al.*, 2009)	The Netherlands	Adolescents (12-15 yr)	23	M&F	Diet history	Measured	698	50
	(Twisk *et al.*, 1998)	The Netherlands	Adolescents (12-15 yr)	14	M&F	Diet history	Measured	233	22
	(Van Lenthe *et al.*, 1998)	The Netherlands	Adolescents (12-15 yr)	14	M&F	Diet history	Measured	233	22
Cardiovascular Risk in Young Finns Study	(Nissinen *et al.*, 2009)	Finland	Children and adolescents (3-18 yr)	21	M&F	FFQ	Measured	3596	33
Copenhagen Childrens' Study	(Lissau *et al.*, 1993)	Denmark	Children (9-10 yr)	12	M&F	General Questionnaire	Measured	881	Not reported
Massachusetts Institute of Technology Growth and Development Study	(Phillips *et al.*, 2004)	USA	Children (8-12 yr)	6.9	F	FFQ	Measured	196	28
National Heart, Lung, and Blood Institute Growth and Health Study	(Albertson *et al.*, 2009)	USA	Children (9-10 yr)	7	F	3-d food diary	Measured	2379	Not reported
	(Barton *et al.*, 2005)	USA	Children (9-10 yr)	10	F	3-d food diary	Measured	2379	10
	(Striegel-Moore *et al.*, 2006)	USA	Children (9-10 yr)	10	F	3-d food diary	Measured	2379	11
Nepean study	(Tam *et al.*, 2006)	Australia	Children (8 yr)	5.4	M&F	3-d food diary	Measured	268	Not reported

Study name	Reference	Country	Subjects age	Length of follow-up (years)	Gender	Dietary assessment method	Method of body weight assessment	Total number of cohort participants at baseline*	Loss of cohort members to follow-up %
Oslo Youth Study	(Kvaavik *et al.*, 2005)	Norway	Adolescents (13 yr)	19	M&F	General questionnaire (used at baseline, in 1981 and 1991) and FFQ (used in 1999)	Self-reported	1086	15.7
Pediatric Bone Mineral Accrual Study	(Mundt *et al.*, 2006)	Australia	Children and adolescents (8-19 yr)	5	M&F	Food recall	Measured	208	Not reported
Pennsylvania Study of Health and Development of Young girls	(Fiorito *et al.*, 2009)	USA	Children (mean 5 yr)	10	F	3x 24-hr recalls	Measured	170	15
The DONALD Study	(Cheng *et al.*, 2009)	Germany	Children and adolescents (9-18 yr)	4	M&F	3-d food diary	Measured	376	Not reported
	(Libuda *et al.*, 2008)	Germany		5	M&F	3-d food diary	Measured	1170	Not reported
The Northern Ireland Young Hearts Project	(Boreham *et al.*, 1999)	UK	Adolescents (12-15 yr)	4	M&F	Diet history	Measured	509	1.7
				Studies of adults					
Black Women's Health Study	(Palmer *et al.*, 2008)	USA	Adults (21-69 yr)	10	F	FFQ	Self-reported	59000	20
Danish Diet, Cancer and Health Study**	(Halkjaer *et al.*, 2009)	Denmark	Adults (50-64 yr)	5	M&F	FFQ	Measured and self-reported	56506	21
	(Halkjaer *et al.*, 2006)	Denmark	Adults (50-64 yr)	5	M&F	FFQ	Measured	56506	21
Eating Behaviour and Body Weight in Women	(Hays *et al.*, 2006)	USA	Adults (55-65 yr)	4.4	F	FFQ	Measured	67	Not reported
EPIC Denmark, Germany, Italy, The Netherlands and the United Kingdom**	(Du *et al.*, 2009)	Europe	Adults (20-78 yr)	6.5	M&F	FFQ	Measured and self-reported	146543	30
Finnish Mobile Clinic Health	(Rissanen *et al.*,	Finland	Adults	5.7	M&F	Diet history	Measured	6102	7

Study name	Reference	Country	Subjects age	Length of follow-up (years)	Gender	Dietary assessment method	Method of body weight assessment	Total number of cohort participants at baseline*	Loss of cohort members to follow-up %
Surveys	1991)		(30-64 yr)						
Health Professionals' Follow-Up Study	(Koh-Banerjee *et al.*, 2003)	USA	Adults (40-75 yr)	9	M	FFQ	Self-reported	51529	35
MONICA	(Hare-Bruun *et al.*, 2006)	Denmark	Adults (30-60 yr)	6	M&F	Diet history	Measured	Subsample of 552	Not reported
	(Iqbal *et al.*, 2006)			5	M&F	7-d food diary	Measured	2025	Not reported
MONICA I	(Halkjaer *et al.*, 2004)	Denmark	Adults (30-60 yr)	6	M&F	FFQ	Measured	2436	Not reported
National Weight Control Registry	(Phelan *et al.*, 2007)	USA	Adults (Average 50 yr)	3	M&F	FFQ	Self-reported	891	Not reported
Nurses' Health Study	(Colditz *et al.*, 1990)	USA	Adults (30-55 yr)	4	F	FFQ	Self-reported	121700	Not reported
Pawtucket Heart Health Program	(Parker *et al.*, 1997)	USA	Adults (18-64 yr)	4	M&F	FFQ	Measured	556	Not reported
Penn Study of Ovarian Aging	(Sammel *et al.*, 2003)	USA	Adults (35-47 yr)	4	F	FFQ	Measured	436	Not reported
Physicians' Health Study I	(Bazzano *et al.*, 2005)	USA	Adults (40-84 yr)	13	M	FFQ	Self-reported	22071	Not reported
San Antonio Heart Study	(Haffner *et al.*, 1991)	USA	Adults (25-64 yr)	8	M&F	Food recall	Measured	2217	Not reported
The CARDIA Study	(Ludwig *et al.*, 1999)	USA	Young adults (18-30 yr)	10	M&F	FFQ	Measured	5115	Not reported
The Framingham Heart Study	(Dhingra *et al.*, 2007)	USA	Adults (mean 53 yr)	4	M&F	General questionnaire	Measured	8997	Not reported

*Numbers vary between publications of the same study (some are sub-cohorts)

** The Danish 'Diet Cancer and Health' study is affiliated with EPIC

Table 5.4 Trial characteristics table (studies with grey shading are on children and adolescents)

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consump tion data provided ?	Funding Source
(Abete *et al.*, 2008)	No medical conditions which influence outcomes No medication Weight stable	Spain 56% Male Age: (36) BMI: (32)	Parallel Group	8 weeks Energy-restricted, plus 1 yr mainten-ance	Free living diet plan Yes	32	1. Higher GI diet 2. Lower GI diet	1. Individually prescribed diet within a strict dietary frame-work repeated on a 3 day rotation basis. 84% of CHO provided by rice and potatoes. 2. Individually prescribed diet within a strict dietary frame-work repeated on a 3 day rotation basis. 84% of CHO provided by pasta and legumes.	1. %E: C 47.8 P 19.6 F 32.6 Fibre g/d:18.5 GI 60-65 units 2. %E: C 50.2 P 18.3 F 31.5 Fibre g/d:24.9 GI 40-45 units	yes	Government funding
(Abrams *et al.*, 2007)	Tanner stage 2 or 3 or premenarcheal Healthy, non-obese	USA 50% Male Age: 9-13 yr BMI: non-obese	Parallel Group	1 year	Free living diet plan No	97	1. Prebiotic supplement 2. Maltodextrin control supplement	1: 8g/d of a 1:1 mix of oligofructose and long-chain inulin. Mixed with calcium-fortified orange juice and drunk with breakfast 2: 8g/d maltodextrin consumed as above	Supplements provided: 1. 12 kcal/d 2. 32 kcal/d	no	USDA, NIH, NCRR, NIDDK
(Bhargava, 2006) The Women's Health Trial: Feasibility Study in Minority Populations	Age 50-80y Post-menopausal Women	UK and USA 0% Male Age: 50 - 79 BMI: 29	Parallel Group	12 months	Free living diet plan No	2208	1. Low fat 2. Control	1. Reduce fat intake to 20% and increase fruit, vegetable and grain consumption. 2. No intervention	1. 5430 kJ, E%: F 20, 13g/d sat fat, 13g/d fibre 2. 6149 kJ, 20g/d sat fat, 12g/d fibre	yes	National Cancer Institute
(Carels *et al.*, 2005)	BMI >30 No CHD, T2DM or HTN Sedentary only	USA 56% Male Age: (43) BMI: (38)	Parallel Group	Duration of treatment program unclear. 12 months follow-up	Free living diet plan Yes	53	1. Weight loss program 2. Weight loss program + low GI education	1. LEARN program only. Advice: increase activity, reduce energy and fat. . GI=56 pre and 57 post intervention. 2. LEARN program + Low GI dietary advice. Advice: increase activity, reduce energy and fat GI= 55 pre and 52 post intervention. GI values from Brand-Miller tables.	1. %E: C 53 P 17 F 32 Energy: 1659kJ/d 2. %E: C 54 P 18 F 29 Energy: 1674kJ/d	yes	Not reported

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consumption data provided ?	Funding Source
(Clifton *et al.*, 2008) Australian Protein Study	27-40 Female adults	Australia 0% Male Age: (49) BMI: (33)	Parallel Group	12 weeks intensive, plus 12 mo follow up	Free living diet plan Yes, aim for 5600 kJ each group	119	1. High protein diet 2. High carbohydrate diet	1. 46% CHO, 34% PRO, 20% FAT 2. 64% CHO, 17% PRO, 20% FAT	1. %E: C 46.4 P 23.2 F 28.5 g/d: C 179 P 94.6 F 51.4 Energy: 6583kJ/d Fibre g/d:3.9 2. %E: C 50.8 P 19.6 F 27.5 g/d: C 189.5 P 77 F 48.4 Energy: 6391kJ/d Fibre g/d:4.3	yes	Meat and Livestock Australia
(Dale *et al.*, 2009)	BMI >27.5	New Zealand 0% Male Age: (45) BMI: (32)	Factorial	2 years	Free living diet plan Unclear if energy restricted	200	1. High MUFA diet 2. High carbohydrate diet	1. 40%CHO, 25%PRO, 21%MUFA 2. 55%CHO, 15-20%PRO, 25-30%FAT	1. %E: C 43 P 22 F 31 g/d: C 185 P 88 F 61 Energy: 6985kJ/d Fibre g/d:23 2. %E: C 47 P 22 F 27 g/d: C 183 P 77 F 46 Energy: 6192kJ/d Fibre g/d:23	yes	Health Research Council of New Zealand
(Dansinger *et al.*, 2005)	≥1 cardiac risk factor BMI 27-42 Free of chronic disease No insulin therapy No medications which influence outcomes	USA 49% Male Age: (49) BMI: (35)	Parallel Group	12 months	Free living diet plan Weight Watchers energy restricted- other diets not	160	1. Atkins 2. Zone 3. Weight watchers 4. Ornish	1. Carbohydrate restriction (%E 41 CHO). 2. Macronutrient balance (%E 42 CHO). 3. Calorie restriction (%E 46 CHO). 4. Fat restriction. (%E 55 CHO) For all participants dietary advice was strictly followed for the first 2 months. Participants then selected their own adherence levels.	1. g/d: C 190 P 82 F 80.5 Energy 1846 kcal/d Fibre g/d:13 2. g/d: C 198 P 90.4 F 66 Energy 1886 kcal/d Fibre g/d:17.4 3. g/d: C 202 P 80 F 58 Energy 1755 kcal/d Fibre g/d:14 4. g/d: C 237 P 74 F 54.5 Energy 1711 kcal/d Fibre g/d:14.5	yes	NIH
(Das *et al.*, 2007) CALERIE	BMI 25-30 Generally healthy No medications which influence outcomes Not extremely athletic/active Weight stable	USA % Male: not reported Age: (35) BMI: (28)	Parallel Group	12 months	All food provided Yes	34	1. Energy restricted high GL diet 2. Energy restricted low GL diet	1. 30% calorie restriction. fibre 15 g/1000kcal. Estimated GI=86, GL=116 g/1000 kcal 2. 30% calorie restriction. fibre 15 g/1000 kcal. Estimated GI=53, GL=45 g/1000kcal	1. %E: C 60 P 20 F 20 2. %E: C 40 P 30 F 30	yes	NIH and Government funding

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consump tion data provided ?	Funding Source
(Delbridge *et al.*, 2009)	Age 18-70y BMI >27 Generally healthy	Australia 50% Male Age: 44 BMI: 39	Parallel Group	12 months Weight maintenan ce plan following 3 month weight loss	Free living diet plan First 3 months energy restricted. Unrestricted during 12 month weight maintenance phase	141	1. Low fat, high protein weight maintenance diet 2. Low fat, high carbohydrate weight maintenance diet	1. Low fat, high protein (30%) diet prescribed for weight maintenance 2. Low fat, high carbohydrate diet prescribed for weight maintenance Diets isocaloric	1. %E: C 40 P 30 F 30 2. %E: C 55 P 15 F 30	Intended diet	Meat and Livestock Australia
(Demol *et al.*, 2009)	BMI >95th centile No medications which influence outcomes No recent weight loss program Without chronic disease	Israel 38% Male Age: 12 - 18(14) BMI: mean not reported	Parallel Group	12 weeks intensive, 9 mo maintenan ce plan	Free living diet plan Yes, aim for 1200- 1500 kcal/day for each group	55	1. Low carbohydrate, high protein 2. Low carbohydrate, high fat 3. High carbohydrate, low fat	All groups prescribed energy restriction to 1200-1500 kcal/d 1. Low-carbohydrate, low-fat, protein-rich diet containing 60 g carbohydrate (up to 20%), 30% fat and 50% protein. 2. Low-carbohydrate, high-fat diet containing: 60 g carbohydrate (up to 20%), 60% fat and 20% protein 3. High-carbohydrate, low-fat diet containing: 50–60% carbohydrate, 30% fat and 20% protein	1. %E: C 20 P 50 F 30 g/d: C 60 2. %E: C 20 P 20 F 60 g/d: C 60 3. %E: C 50 P 20 F 30	Intended diet	Not reported
(Due *et al.*, 2004) The Danish Protein Swap Study	Previously overweight/obese	Denmark 24% Male Age: (40) BMI: (30)	Parallel Group	6 months strict, 6-12 mo less strict, plus 24 mo follow up	All food provided No, *ad libitum*	50	1. High protein 2. Moderate protein	1. 25%PRO, <30%FAT 2. 12%PRO, <30%FAT	1. %E: C 48.9 P 21.2 F 30 Energy: 8400kJ/d 2. %E: C 54.7 P 13.9 F 31.4 Energy: 8200kJ/d	yes	Research institute funding, The Federation of Danish Pig Producers and Slaughterhouse and The Danish Livestock and Meat Board
(Ebbeling *et al.*, 2003)	BMI >95th centile Generally healthy	USA 28% Male Age: 13 - 21(16) BMI: (36)	Parallel Group	6 months strict, 6-12 mo less strict	Free living diet plan 1. Low GL diet – no, *ad libitum* 2. Low fat	16	1. Low GL diet 2. Low fat diet	1. Low to moderate GL foods (non-starchy vegetables, fruits, legumes, nuts, dairy). Target 45-50%CHO, 30-35%FAT. Ad lib diet. GL (g/1000kcal) was 86, 6 at baseline, 68 at 6 months and 69 at 12 months 2. Conventional low fat diet. Increase grains, vegetables &	1. %E: C 51 P 19 F 31 Energy 1522 kcal/d 2. %E: C 55 P 18 F 28 Energy 1604 kcal/d	yes	National Institute of Diabetes & Digestive & Kidney Diseases, Charles H. Hood Foundation and NIH

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consumption data provided?	Funding Source
					diet – yes			fruit. Target energy reduction 250-500kcal/d. Targets:55-60%CHO, 25-30%FAT. GL (g/1000kcal) was 79 at baseline, 77 at 6 months and 79 at 12 months			
(Ebbeling *et al.*, 2005)	Age 18-35y BMI >27.5 Healthy	USA 12% Male Age: 28 BMI: obese	Parallel Group	6 months strict, 6-12 mo less strict	Free living diet plan 1. Low GL diet – no, *ad libitum* 2. Low fat diet – yes	34	1. Low GI diet 2. Low fat diet	1. Ad lib low GI food, 45-50% CHO, 30-35%FAT. GL 53 g/1000kcal 2. Meal plans based on an exchange system, energy deficit of 250-500kcal/d. GL 77 g/1000 kcal	1. %E: C 47.2 P 21.1 F 33 Energy 1391 kcal/d Fibre g/d:20.7 2. %E: C 59.4 P 18.7 F 23.4 Energy 1409 kcal/d Fibre g/d:17.8	yes	National Institute of Diabetes & Digestive & Kidney Diseases, Charles H. Hood Foundation and research institute funding
(Ebbeling *et al.*, 2007)	Age 18-35y BMI >30 Generally healthy No medication No recent weight loss program Non smokers No T2DM	USA 21% Male Age: 18 - 35(27) BMI: mean not reported	Parallel Group	6 months intensive, 12 mo follow up. Monthly group workshops through-out 18 mo	Free living diet plan No	73	1. Low GL diet 2. Low fat diet	1. *Ad libitum* low GL foods. Target: 40% CHO, 25% PRO, 35% FAT. 2. General healthy eating advice. Target: 55% CHO, 25% PRO, 20% FAT. *Ad libitum* consumption.	Approx from figures: 1. %E: C 40 P 21 F 36 Energy 1600 kcal/d Fibre g/d:12 2. %E: C 53 P 21 F 25 Energy 1500 kcal/d Fibre g/d:10	yes	National Institute of Diabetes & Digestive & Kidney Diseases, Charles H. Hood Foundation and research institute funding
(Foster *et al.*, 2003)	No medications which influence outcomes Without chronic disease	USA 32% Male Age: (44) BMI: (34)	Parallel Group	12 months	Free living diet plan Yes, for LEARN weight management diet only	63	1. Low carbohydrate diet 2. Conventional diet plan	1. Atkins diet book provided. Low CHO, high FAT, high PRO 2. LEARN weight management diet. High CHO, low FAT, energy restricted diet (1200-1500kcal/d for women and 1500-1800kcal/d for men).	1. <20g CHO for 1st 2 wks, rising until desired wt. achieved. 60% participants ketotic in first 8 wks, falling to 20% at 1 yr 2. %E: C 60 P 15 F 25	Intended diet	NIH
(Frisch *et al.*, 2009)	Age 18-70y BMI 25-30 Generally healthy	Germany 31% Male Age: (47) BMI: (34)	Parallel Group	6 months, plus 6 mo follow up Weekly phone contact 1st 6 mo, then continue diet for next 6 mo	Free living diet plan Yes	200	1. Moderate carbohydrate diet 2. High carbohydrate diet	1. Prescribed diet: <40% CHO, 25% PRO, >35% FAT. Energy deficit >500kcal/d. 2. Conventional low fat diet. Prescribed diet: >55% CHO, 15% PRO, <30% FAT. Energy deficit >500kcal/d.	1. %E: C 40.9 P 19.3 F 36.5 Energy 1742 kcal/d 2. %E: C 49.5 P 17.7 F 29.7 Energy 1783 kcal/d	yes	German Health Insurances and the Institute for Applied Telemedicine

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consumption data provided?	Funding Source
(Gardner *et al.*, 2007) A to Z Weight Loss Study	Generally healthy Moderate alcohol intake No T2DM Pre-menopausal Weight stable	USA 0% Male Age: (41) BMI:27 - 40(32)	Parallel Group	12 months 8 wks intensive weekly sessions, continue diets w. email and telephone contact until 12mo post random-isation	Free living diet plan Zone and LEARN diets energy restricted- other diets not	311	1. Atkins: low carbohydrate 2. Zone: moderate carbohydrate 3. Ornish: high carbohydrate	1. Atkins diet: very low in carbohydrate 2. Zone: reduced carbohydrate 3. Ornish: high carbohydrate intake 4. LEARN program (data not extracted) – lifestyle, exercise, attitudes, relationships, nutrition	1. %E: C 17.7 P 27.7 F 54.7 Energy: 5781.97kJ/d Fibre g/d:11 2. %E: C 42 P 23.7 F 34.8 Energy: 6091.8kJ/d Fibre g/d:16.9 3. %E: C 63.1 P 16.9 F 21.1 Energy: 5895kJ/d Fibre g/d:22.1	yes	NIH
(Greenberg *et al.*, 2009) DIRECT	Age 40-65y BMI >27	Israel 86% Male Age: (52) BMI: (31)	Parallel Group	2 years 18 classes attended over 2 yr, at wks 1, 3, 5, 7, then each 6th week	Free living diet plan Yes, for diet (1)	322	1. Low fat 2. Med-iterranean 3. Low carbohydrate	1. Diet based on the American Heart Association guidelines. 2. Mediterranean group not relevant for this review – data not extracted 3. Based on the Atkins diet.	1. %E: C 50.2 P 23.8 F 28 Energy 1261 kcal/d Fibre g/d:22.5 2. %E: C 44.9 P 24 F 32.9 Energy 1393 kcal/d Fibre g/d:22.6 3. %E: C 37.6 P 26.4 F 36.4 Energy 1323 kcal/d Fibre g/d:16	yes	Research institute funding, Atkins Research Foundation and University funding
(Howard *et al.*, 2006) The Women's Health Initiative Dietary Modification Trial	Age 50-79y Fat intake >32% Post-menopausal	USA 0% Male Age: (62) BMI: (29)	Parallel Group	8 years 18 group sessions in 1st yr, 4 per yr thereafter	Free living diet plan No	48835	1. Low fat 2. Control	1. Advice: reduce fat intake to 20%, increase fruit, vegetables and wholegrain 2. Received information relating to health and healthy diets	1. %E: C 52.7 F 29.8 Energy 1446 kcal/d Fibre g/d:16.9 2. %E: C 44.7 F 38.1 Energy 1564 kcal/d Fibre g/d:14.4	yes	National Heart, Lung, and Blood Institute
(James *et al.*, 2004) CHOPPS	Children aged 7-11 yr Attending one of 6 junior schools in Dorset	UK (England) 50% Male Age: 7 - 11 BMI: 10-12% obese in each group	Parallel Group Cluster Random-ised Trial	12 months 1 hr session per term, over 4 terms	Free living diet plan No	644	1. Control 2. Reduced carbonated beverages	1. No intervention 2. Educational intervention discouraging the consumption of any 'fizzy' drinks and encouraging a healthy diet.	3d drinks diaries completed – no other dietary data available Total carbonated drinks w sugar at 12 mo over 3d 1. 1.2 glasses 2. 0.9 glasses	no	Educational grants and Bournemouth Diabetes and Endocrine Centre
(Keogh *et al.*, 2007)	Age 20-65y BMI 27-40 Moderate alcohol intake	Australia 32% Male	Parallel Group	12 weeks Active weight	Free living diet plan Yes, aim for	44	1. Low carbohydrate diet	1. Energy restricted, low CHO diet, low in saturated fat.	1. %E: C 33 P 40 F 27 Fibre g/d:26	Intended diet only	Research institute funding

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consump tion data provided ?	Funding Source
	No HTN or T2DM No medications which influence outcomes	Age: (49) BMI: (33)		loss phase 1-12 wk, monthly dietician meeting until wk 52	~6000 kJ for each group		2. High carbohydrate diet	2. Energy restricted, high CHO diet, low in saturated fat.	2. %E: C 60 P 20 F 20 Fibre g/d:40		
(Layman *et al.*, 2009)	BMI >25 Non-smokers No lipid-lowering medication/ steroids/ antidepressants	USA 45% Male Age: 40 - 56(45) BMI: (33)	Parallel Group	12 months 4 months weight loss followed by 8 mo weight main-tenance. Weekly meetings for 12 mo	Free living diet plan Yes	130	1. High carbohydrate, low protein diet 2. Low carbohydrate, high protein diet	1. Carbohydrate energy 55%. Protein provided ~15% energy intake, with carbohydrate:protein ratio >3.2 and lipids ~30% energy intake. Protein provided 0.8g.kg/d. Kcal and fibre were similar between groups 2. Carbohydrate energy 37% Protein provided ~30% energy intake, with carbohydrate:protein ratio <1.5 and lipids ~30% energy intake. Protein provided 1.6g.kg/d. Kcal and fibre similar between groups	1. g/d: C 232 P 70 F 51 Energy: 6800kJ/d Fibre g/d:25 2. g/d: C 168 P 116 F 67 Energy: 7180kJ/d Fibre g/d:20	yes	The National Cattlemen's Beef Association, Beef Checkoff, and Kraft Foods
(McManus *et al.*, 2001)	Age 18-70y BMI >25 Free of chronic disease	USA 10% Male Age: (44) BMI:27 - 46(34)	Parallel Group	18 months	Free living diet plan Yes	101	1. Moderate fat diet 2. Low fat diet	1. Intended diet: 45-50%CHO, 30%FAT. Energy limit of 1200kcal/d (women) or 1500kcal/d (men). 2. Intended diet:60-65%CHO, 20%FAT. Energy limit of 1200kcal/d (women) or 1500kcal/d (men)	1. %E: C 47 P 19 F 35 Energy: 1877kJ/d Fibre g/d:25 2. %E: C 50 P 19 F 30 Energy: 1697kJ/d Fibre g/d:19	yes	The Peanut Institute, The International Olive Oil Council and The International Tree Nut Council
(Pasman *et al.*, 1997a)	BMI >30 Energy restriction during trial run-in Weight loss >5kg during run-in	The Netherlands 0% Male Age: (41) BMI: (33)	Parallel Group	14 months	Supplement Energy restricted for 2 months but no energy restriction thereafter for 14 months	39	1. Guar gum - High compliance 2. Control 3. Guar gum - Low compliance	1. 20g partially hydrolysed guar gum in 2x10g doses daily to be consumed in afternoon and evening. Dissolved in 200ml water/coffee/orange juice. High compliance - consumed >80% supplements 2. Nothing was provided as placebo to the control group 3. 20g partially hydrolysed guar gum in 2x10g dose. 50-80% compliant	Nb. groups 1 and 3 are post-hoc defined – subjects not randomised to these groups initially 1. 5.8 MJ/d 2. 6.6 MJ/d 3. 7.0 MJ/d	yes	Sandoz Nutrition Ltd (Novartis Nutrition)

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consumption data provided?	Funding Source
(Pasman *et al.*, 1997b)	BMI >30 Energy restriction during trial run-in Good compliance during run-in	The Netherlands 0% Male Age: (35) BMI: (31)	Parallel Group	14 months	Supplement No	33	1. CHO/Cr-Pic (Chromium III)/Fibre/ Caffeine 2. Carbohydrate supplement 3. Control	1. Group not comparable, multi-ingredient supplement. Data not extracted 2. 50g carbohydrate daily, dissolved in 250ml water (42% glucose, 58% maltodextrin) 3. No supplement	1. data not extracted 2. %E: C 50 P 13 F 36 Energy: 8100kJ/d Fibre g/d:12 3. %E: C 42 P 15 F 37 Energy: 7600kJ/d Fibre g/d:15	yes	Novartis Nutrition Ltd
(Sacks *et al.*, 2009)	Age 30-70y BMI 25-40 No CVD or T2DM	USA 36% Male Age: (51) BMI: (33)	Parallel Group	2 years Contact through-out 2 yrs	Free living diet plan Yes	811	1. Low-fat, average-protein 2. Low-fat, high-protein 3. High-fat, average-protein 4. High-fat, high-protein	ALL DIETS: energy deficit 750kcal/d 1. 20% fat, 15% protein and 65% CHO. 2. 20% fat, 25% protein and 55% CHO. 3. 40% fat, 15% protein and 45% CHO 4. 40% fat, 25% protein and 35% CHO	1. %E: C 57.5 P 17.6 F 26.2 Energy: 1636 kcal/d 2. %E: C 53.4 P 21.8 F 25.9 Energy: 1572 kcal/d 3. %E: C 49.1 P 18.4 F 33.9 Energy: 1607 kcal/d 4. %E: C 43 P 22.6 F 24.3 Energy: 1624 kcal/d	yes	NIH
(Shah *et al.*, 1996) American Low-fat Study	20-40% above ideal weight Age 25-45y Generally healthy Moderate alcohol intake Non smokers Without chronic disease Women	USA 0% Male Age: 25 - 45(36) BMI: (30)	Parallel Group	6 months 26 wk phase w 16 group sessions Stated goal was 12 mo inter-vention	Free living diet plan Yes	122	1. Low fat, high complex carbohydrate diet 2. Energy restricted diet	1. Limit fat to 20g/d, *ad libitum* complex carbohydrates 2. Energy restriction to 4184 or 5021kj/d (based on weight). Reduce fat intake to <30% total energy. Increase complex carbohydrates such as grains, legumes, fruits & vegetables. Reduce simple sugar consumption	1. %E: C 61 P 16 F 22 Energy: 1617 kcal/d Fibre g/d:17.4 2. %E: C 54 P 16 F 30 Energy: 1531 kcal/d Fibre g/d:16.7	yes	NIH grant
(Sheppard *et al.*, 1991) The Women's Health Trial Feasibility Study	<150% of ideal weight for frame Age 45-69y At risk of breast cancer Not on low fat diet	USA 0% Male Age: (56) BMI: (26)	Parallel Group	24 months Meetings: Weekly for 2 mo. Biweekly for 2 mo., monthly thereafter	Free living diet plan No	303	1. Low fat diet 2. Control	1. Dietary advice aimed to decrease fat intake: 20% FAT (from 39%). No emphasis on weight loss. 2. No intervention	1. %E: C 58.7 P 19.1 F 22.8 Energy: 5640kJ/d 15% E CHO increase at 2 years 2. %E: C 46.9 P 16.1 F 36.5 Energy: 6748kJ/d	yes	National Cancer Institute

Authors, Study Name	Subject Inclusion Criteria	Characteristics of Participants	Trial Design (washout duration)	Inter-vention Duration	Intervention style/ Energy restriction goal?	Total No. Subjects	Intervention Group Names	Intervention Description	Diet/Supplement Nutritional Characteristics	Consumption data provided ?	Funding Source
(Sichieri *et al.*, 2007a)	Age 25-45y BMI 23-30 Generally healthy No T2DM Parity ≥1 Pre-menopausal	Brazil 0% Male Age: (37) BMI: (27)	Parallel Group	18 months Monthly contact	Substitution Yes	203	1. Low GI/GL diet 2. High GI/GL diet	1. Energy restriction 100-300kcal/d. Staple foods provided. At 18m, GI=30, GL=104 2. Energy restriction 100-300kcal/d. Staple foods provided. At 18m, GI=72, GL=280	1. %E: C 60 P F 27 Energy: 11200kJ/d Fibre g/d:36 2. %E: C 62 P F 26 Energy: 14000kJ/d Fibre g/d:45	yes	NIH and research institute funding
(Swinburn *et al.*, 2001) New Zealand Diabetic Workforce Study	Age >40y Impaired glucose tolerance (2-h blood glucose 7.8-11.0 mmol/l) or high normal blood glucose (7.0-7.8 mmol/l)	New Zealand 74% Male Age: >40 - (53) BMI: mean not reported	Parallel Group	12 months With 5 yr follow up	Free living diet plan No	176	1. Low fat 2. Control	1. Reduced fat, *ad libitum* E diet 2. No intervention – usual diet	1. %E: C 54.5 P 18.6 F 25.9 Energy: 1832 kcal/d Fibre g/d:20.5 2. %E: C 45.6 P 16.5 F 33.8 Energy: 2307 kcal/d Fibre g/d:20.3	yes	Auckland Medical Research Foundation, National Heart Foundation of New Zealand, and the Lotteries Medical Board
(Tinker *et al.*, 2008) The Women's Health Initiative Dietary Modification Trial	Age 50-79y Fat intake >32% Post-menopausal No type 2DM No cancer	USA 0% Male Age: (62) BMI: (29)	Parallel Group	12 mo intensive 8 years follow up	Free living diet plan No	48835	1. Control 2. Low fat	1. Received information relating to health and healthy diets 2. Advice: reduce fat intake to 20%, increase fruit, vegetables and grains	At 1 yr post randomisation: 1. %E: C 48 P 16.8 F 35 Energy: 1594 kcal/d Fibre g/d:15.5 2. %E: C 58.5 P 17.6 F 24.2 Energy: 1502 kcal/d Fibre g/d:18.5	yes	NIH

Table 5.5 Bias assessment

Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Abete *et al.*, 2008)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Abrams *et al.*, 2007)	Unclear	Unclear	No bias	No bias	Bias	Bias	No Bias
(Bhargava, 2006)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Carels *et al.*, 2005)	Unclear	Unclear	Bias	Bias	Bias	No Bias	No Bias
(Clifton *et al.*, 2008)	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Dale *et al.*, 2009)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Dansinger *et al.*, 2005)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Das *et al.*, 2007)	No Bias	Unclear	Bias	Unclear	No Bias	Bias	No Bias
(Delbridge *et al.*, 2009)	No Bias	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Demol *et al.*, 2009)	Unclear	Unclear	Bias	Bias	No Bias	Bias	No Bias
(Due *et al.*, 2004)	No Bias	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Ebbeling *et al.*, 2003)	Unclear	Unclear	Bias	Bias	No Bias	Bias	Unclear
(Ebbeling *et al.*, 2005)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Ebbeling *et al.*, 2007)	No Bias	No Bias	Bias	No Bias	No Bias	No Bias	No Bias
(Foster *et al.*, 2003)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Frisch *et al.*, 2009)	No Bias	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Gardner *et al.*, 2007)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias
(Greenberg *et al.*, 2009)	No Bias	Unclear	Unclear	Unclear	No Bias	Bias	Bias
(Howard *et al.*, 2006)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias
(James *et al.*, 2004)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Keogh *et al.*, 2007)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Layman *et al.*, 2009)	Unclear	Unclear	Bias	Unclear	No Bias	Unclear	Unclear
(McManus *et al.*, 2001)	No Bias	No Bias	Bias	Bias	Bias	No Bias	No Bias
(Pasman *et al.*, 1997a)	Unclear	Unclear	Bias	Unclear	Bias	Bias	Bias
(Pasman *et al.*, 1997b)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Sacks *et al.*, 2009)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Shah *et al.*, 1996)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Sheppard *et al.*, 1991)	Unclear	Unclear	Bias	Bias	Bias	No Bias	No Bias
(Sichieri *et al.*, 2007a)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Swinburn *et al.*, 2001)	No Bias	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Tinker *et al.*, 2008)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias

Body weight and body mass index

Results reported in this section include only continuous measures of body weight and body mass index (BMI).

Body weight, body mass index and total carbohydrates

Summary of cohort results in adults

Data were extracted from six publications reporting results from 1 Danish study: MONICA (Iqbal *et al.*, 2006) and 5 US studies: the National Weight Control Registry (Phelan *et al.*, 2007;Colditz *et al.*, 1990;Hays *et al.*, 2006;Ludwig *et al.*, 1999;Parker *et al.*, 1997). The NHS and the Pawtucket Heart Health Program studies estimated consumption of total carbohydrates as grams per day, and four expressed carbohydrate intakes as percentage contribution to energy (see Table 5.6). Both the National Weight Control Registry (Phelan *et al.*, 2007) and the NHS (Colditz *et al.*, 1990) relied upon self-reported weight measurements. All other studies used body weight measurements taken by a clinician or professional. All but one of the studies reported change in body weight from baseline to follow-up as the outcome.

Due to variation in presentation of the exposure (grams or percentage energy) and outcome (mean follow-up body weight or change in weight), it was not possible to combine these studies in a meta-analysis.

The two studies that reported baseline carbohydrate intake expressed as grams per day and change in body weight observed inconsistent results. The Nurses' Health Study (Colditz *et al.*, 1990) reported an inverse association, indicating decreasing body weight changes for each unit increase in carbohydrate at study baseline. However, the coefficient was very small. The much smaller Pawtucket Heart Health Program cohort (Parker *et al.*, 1997) reported a positive direction of association, with increasing body weight gains for each additional unit of baseline carbohydrate consumed. However, this was not statistically significant.

Four studies expressed baseline carbohydrate intake as the percentage of energy and explored the relationship with changes in body weight over time. Most of these studies described negative beta coefficients, suggestive of a decrease in body weight over the period of follow-up with increasing baseline carbohydrate intake, however the majority of the analyses were not found to be statistically significant and the effects described were generally small. An exception to this was the Eating Behaviour and Body Weight in Women study (Hays *et al.*, 2006), in which the negative estimate achieved statistical significance at the 1% level. In this small study of postmenopausal women, each one percentage increase in carbohydrate energy at baseline was associated with a decrease in weight per year of 0.12 kg ($p<0.05$).

One study was particularly unusual in that participants were part of a National Weight Control Registry, a Registry of those who had lost ≥ 13.6kg and maintained this weight loss for ≥ 1 year (Phelan *et al.*, 2007). Phelan *et al.* reported mean body weight change in cohort members who stated that they followed a low carbohydrate diet or not. The low carbohydrate diet members gained 7kg (SD 7.1) compared to a gain of 5.7kg (SD 8.7) experienced by the other members.

The CARDIA study of black and white participants aged 18-30 years at baseline, reported adjusted mean body weight at 10 year follow up as opposed to weight change (Ludwig *et al.*, 1999). Mean body weight of participants in the lowest quintile of carbohydrate intake was significantly higher (by approx. 3kg) than for those in the highest quintile of intake (p=0.04). However, this study found that the association between body weight and carbohydrate was attenuated by additional adjustment for dietary fibre.

In summary, these studies provide some evidence of smaller body weight gains or lower final BMI in individuals consuming a higher percentage of energy from carbohydrate, but the associations are not entirely consistent, tend to be weak and generally do not achieve statistical significance.

Exposure definition and assessment

The MONICA study (Iqbal *et al.*, 2006) collected dietary data using food diaries. All other studies used FFQs.

Adjustment for appropriate confounders

Most studies included important covariates in their analyses (age, gender where appropriate, baseline weight or BMI, energy intake, smoking). The Eating Behaviour and Body Weight in Women Study and the National Weight Control Registry study were less fully adjusted. The CARDIA study found that the inverse association between body weight and carbohydrate was attenuated by additional adjustment for dietary fibre (Ludwig *et al.*, 1999). However, it should be recognised that as expressed by Colditz *et al.* (Colditz *et al.*, 1990) one of the strongest predictors of weight gain is likely to be prior experience of weight change and that regardless of the covariates included in a model, the influence of nutrients is likely to be rather weak relative to this effect. With regard to energy adjustment, there is considerable debate about the merits of adjustment, with no clear consensus. It may be considered appropriate to adjust for energy in terms of minimising the impact of dietary under reporting and the effects of body size (bigger size equating to higher energy intake), whilst others consider that (depending on the exposure) energy intake is on the causal pathway and therefore adjustment would ‘remove’ the effect of the exposure and may therefore be inappropriate. Different methods of energy adjustment (nutrient density approach, residuals approach etc.) may also potentially have different effects on the outcome (Sichieri *et al.*, 2007b;Willett *et al.*, 1997).

Summary of cohort results in children

Data were extracted from three cohort studies: the Amsterdam Growth and Health Study, The Northern Ireland Young Hearts Project and the Adelaide Longitudinal Study of Growth and Nutrition conducted in The Netherlands, Northern Ireland, and Australia respectively (Twisk *et al.*, 1998;Boreham *et al.*, 1999;Magarey *et al.*, 2001). Body weight was professionally measured in all 3 studies. There was no evidence of an association between total carbohydrate intake and follow-up BMI in any of these studies of children and adolescents (see Table 5.7).

Exposure definition and assessment

All three studies reported carbohydrate intake as percent energy, with the Amsterdam Growth and Health Study (Twisk *et al.*, 1998) additionally reporting grams per day. This was measured by dietary history in both the Amsterdam Growth and Health Study and the Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) and using food diaries in the Adelaide Longitudinal Study of Growth and Nutrition (Magarey *et al.*, 2001).

Adjustment for appropriate confounders

The Northern Ireland Young Hearts Project adjusted for social class and sexual maturity. The Adelaide Longitudinal Study of Growth and Nutrition (Magarey *et al.*, 2001) included gender, energy intake, age and parental BMI as covariates in the model. Adjustments applied in the Amsterdam Growth and Health Study were not clearly reported (Twisk *et al.*, 1998).

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Weight and weight change

Twenty one papers provided data on 20 trials that compared the effects on body weight of diets higher or lower in dietary carbohydrate. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.8. All studies were conducted on adults.

Data from three studies; Ebbeling *et al.* (Ebbeling *et al.*, 2007), Gardner *et al.* (Gardner *et al.*, 2007) and Sacks *et al.* (Sacks *et al.*, 2009) could not be included in the meta-analysis.

Data on body weight change in the study by Ebbeling *et al.* (Ebbeling *et al.*, 2007) were published only in a figure, from which it was inappropriate to accurately extract numerical values. This study compared the effects of a low fat (20% energy), high carbohydrate (55% energy) diet with a moderate fat (35% energy), lower carbohydrate (40% energy, low glycaemic load) diet in 73 obese young adults (18-35 yr) with a 6-month intensive intervention period and a 12-month follow-up period (group workshop sessions continued throughout, as did the diet). The low carbohydrate diet was not energy-restricted, but the high carbohydrate diet was designed to create an energy deficit in the region of 250-500 kcal per day. However, there was no difference in body weights in the 2 diet groups at the 6, 12 and 18-month follow-up points ($p=0.99$ for the group x time interaction). Overall, both groups lost weight equally. However, there were some differences between subjects, with those assessed as having an elevated post-load insulin concentration being more responsive to the low carbohydrate/low GL diet. This group of participants lost more weight initially, and over the full course of the study (-5.8kg vs. -1.2kg for low carb vs. high carb, at 18 months $p<0.04$). The low insulin responders were not differentially affected by the carbohydrate content of the diets.

The A to Z weight loss study reported by Gardner *et al.* compared weight losses on 4 popular weight loss diets that differed in carbohydrate content in 311 overweight or obese non diabetic, premenopausal women (Gardner *et al.*, 2007). Data from three groups were extracted for this review. These compared the Atkins (low carbohydrate), Zone (moderate carbohydrate) and Ornish (high carbohydrate) diets over the period of one year (for details of the macronutrient composition of these diets see Table 5.4). The group following the Atkins diet approach (low carbohydrate) lost the most weight at 12 months, and this was significantly different from the loss in the Zone group participants ($p<0.05$). There was no difference in weight loss between the Zone and Ornish diet groups.

Sacks *et al.* (Sacks *et al.*, 2009) allocated 811 overweight adults to 4 diets that differed in the proportion of energy derived from the macronutrients in a 2x2 factorial design (high vs. low fat and average vs. high protein), which also permitted a dose response analysis of the effect of carbohydrate percentage (ranging from 35 to 65% energy). Participants were given group and individual guidance throughout the 2 years of the study. At the end of the study, weight losses were similar in the groups that were assigned to diets with 65% or 35% carbohydrates (2.9 and 3.4 kg, respectively, $p>0.2$). Additionally, there was no impact of the fat or protein content on this finding.

An additional three studies could not be included in the meta-analysis as the % energy from carbohydrate difference between groups was less than 5%.

In the trial conducted by McManus *et al.* (McManus *et al.*, 2001) 101 overweight men and women were allocated to a lower carbohydrate, moderate-fat diet (35% of energy) or a higher carbohydrate, low-fat diet (20% of energy). After 18 months, 31/50 subjects in the moderate-fat group, and 30/51 in the low fat group were available for measurements. In the lower carbohydrate, moderate-fat group, there was a mean decrease in body weight of 4.1 kg, compared to an increase in the low-fat group of 2.9 kg ($p<0.001$ between the groups). However, at 18 months, the difference between groups in percentage energy from carbohydrate was less than 5%.

Dale and colleagues randomly assigned 200 women who had previously lost 5% or more of their initial body weight to groups that were advised to follow a high carbohydrate or a relatively high monounsaturated fat diet (Dale *et al.*, 2009). These groups were also divided into those receiving either an intensive or less intensive form of support. After 2 years of follow-up, data were available from 174 (87%) participants. Weight losses did not vary significantly between diet groups and the authors concluded that 'diets of different macronutrient composition produced comparable beneficial effects in terms of weight loss maintenance'. However, achieved differences in percentage energy from carbohydrate between groups were much less than intended (43 vs. 47% energy) and since this was less than our 5% cut-off, this study was not included in the meta-analysis.

Clifton *et al.* (Clifton *et al.*, 2008) studied the effect of lower carbohydrate, higher protein intakes on the maintenance of weight loss in 79 healthy obese women after a 12 week intensive weight loss phase. After one year of follow-up, weight loss was similar in the high-protein (34% of energy) and the high-carbohydrate diet groups (64% of energy).

Overall, the findings from the studies not included in the meta-analysis are inconsistent, but generally do not indicate a differential benefit in terms of weight loss with either higher or lower carbohydrate diets.

Trials were separated into 2 main types on the basis of proportion of energy derived from percentage macronutrients. For inclusion in meta-analysis there was a difference of energy from carbohydrate between trial groups of 5% or more. Actual consumption was used rather than intended diet unless otherwise stated – see trial characteristics table.

Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat was 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein was 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more but protein intakes were also different by more than 2%.

Fourteen studies provided sufficient data for inclusion in the meta-analyses comparing different carbohydrate intakes and changes in weight (in kg) (see Table 5.8). Data from the Women's Health Initiative Dietary Modification Trial are provided in 2 publications which report outcomes at different time points post randomisation (1 yr, 3 years and 90 months) (Tinker *et al.*, 2008;Howard *et al.*, 2006). Data from the 90 months follow-up only were included in this meta-analysis as the intervention was judged to still be 'ongoing' at that point and provides information on the long term impact of dietary change on body weight (Howard *et al.*, 2006). One study reported results from four groups (Dansinger *et al.*, 2005) and one study reported results from three groups (Delbridge *et al.*, 2009). For both of these studies the group with the lowest carbohydrate intake was compared with the group with highest carbohydrate intake. There were insufficient studies to stratify by protein shift only, or by a shift in both fat and protein, so these results were combined into one plot.

These remaining 14 studies were stratified into the two categories based on reported differences (≥2%) in macronutrients. Eight studies reported changes in percentage fat with less than 2% change in protein. Six studies reported changes in protein of at least 2% with or without changes in fat.

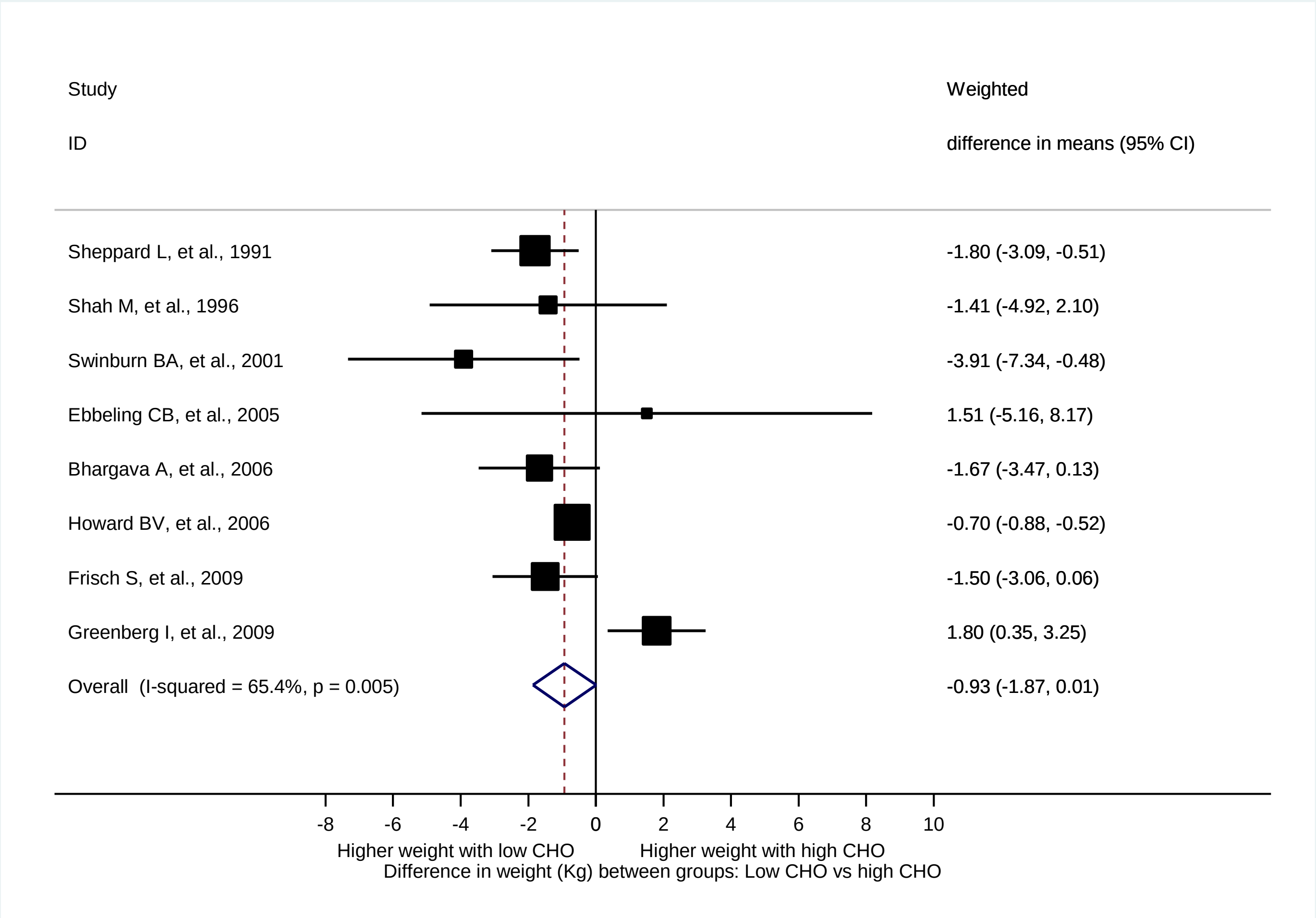
All studies included adults as participants. Definitions of different levels of carbohydrate, fat and protein are reported in the trial characteristics table (Table 5.4). Studies where weight was presented as pounds were converted to kilograms. The first follow up reported at the end of the intervention and over 12 months was used. This varied from 12 months to 6 years.

There were insufficient studies to create a funnel plot for examining possible publication bias.

Carbohydrate swapped with fat

The pooled estimate indicated that weight was 0.93kg lower (95% CI, -0.01 to 1.87kg) with consumption of a high carbohydrate, low fat diet compared with a low carbohydrate and high fat diet. This was not significantly different from zero (p=0.05). Heterogeneity denoted by I^2 was 65% (95% CI, 26 to 84%). Statistically, there was no evidence that high carbohydrate, low fat diets result in differences in weight after 12 months.

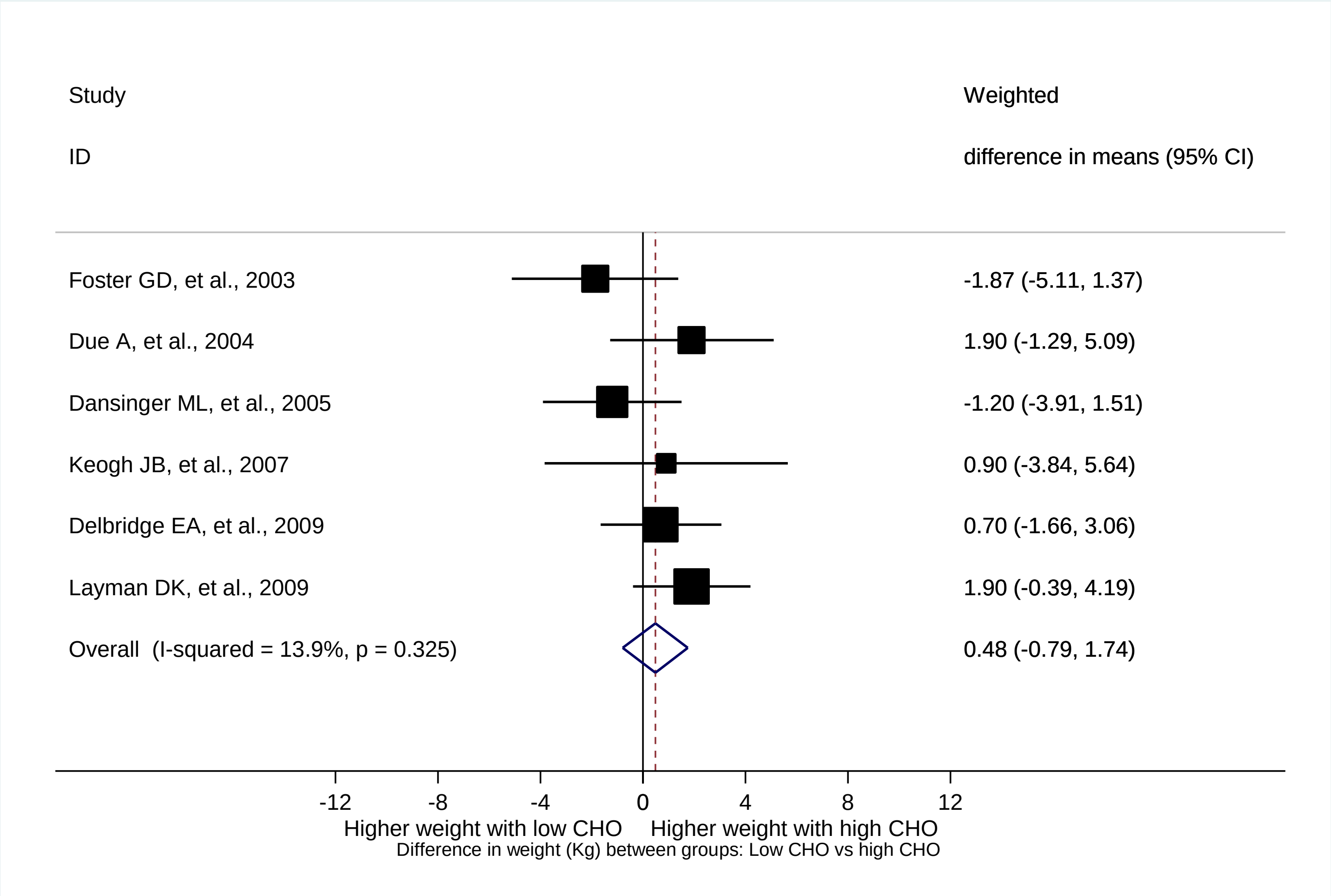
Figure 5.1 Forest plot for high carbohydrate, lower fat vs. lower carbohydrate, higher fat diets and body weight (kg)



Carbohydrate swapped with protein and swapped with both protein and fat

The pooled estimate for studies with a change in protein or fat and protein indicated that weight was 0.48kg higher (95% CI, -0.97 to 1.74kg) with consumption of a higher carbohydrate, lower protein and fat diet compared with a lower carbohydrate, higher protein and fat diet. This was not significantly different from zero (p=0.46). Heterogeneity denoted by I^2 was 14% (95% CI, 0 to 78%). Statistically, there was no evidence that a diet higher in carbohydrate and lower in fat and protein is associated with differences in body weight.

Figure 5.2 Forest plot for high carbohydrate diets swapped with protein only or fat and protein and body weight (kg)



Due to high levels of unexplained heterogeneity, four continuous variables were analysed using metaregression to determine if they had an effect on the pooled estimates. These included between-group percentage difference in fat, percentage difference in protein, percentage difference in carbohydrate and initial BMI of participants. None were statistically significant. The difference in the influence of percentage dietary fat was of borderline statistical significance (p=0.08). A metaregression analysis using difference in fat only decreased the pooled estimate for all studies of -0.38kg by 0.24kg (95%CI -0.03 to 0.51) for each 1% increase in fat. This means that if the difference in carbohydrate was due to the carbohydrate being replaced with fat (rather than protein) the pooled estimate is pulled to the left resulting in higher body weight with a lower carbohydrate diet.

Body mass index

Thirteen papers on 12 trials provided data on the effects of high or low carbohydrate diets on BMI. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.9.

Two studies were conducted on obese adolescents (Ebbeling *et al.*, 2003;Demol *et al.*, 2009), and were therefore not included in the meta-analysis of adult studies. The study reported by Demol *et al.* compared the effects of a high carbohydrate low fat diet, with lower carbohydrate diets that varied in the proportion of energy derived from fat or protein on body weight and the metabolic profile of obese adolescents (Demol *et al.*, 2009). Both low carbohydrate diets aimed to deliver up to 20% of energy from carbohydrate (60g/d), which was compared with 50-60% of energy from carbohydrate in the high carbohydrate diet. The participants were free-living and were instructed to follow a pre-set rotating menu, which aimed to deliver 1200-1500 kcal per day. After one year, all groups had lost weight, however the changes in BMI and BMI-percentile within each diet group did not differ. All diets were equally effective in reducing weight.

Ebbeling 2003 (Ebbeling *et al.*, 2003) compared the effects of a conventional reduced fat (30-35% energy), high carbohydrate (55-60% energy) diet with a moderate fat (30-35% energy), lower carbohydrate (45-50% energy, low/moderate GI) diet in 16 obese adolescents (13-21 yr) over 12 months. Fourteen subjects completed the full trial. There was a decrease in BMI at 12 months in the lower carbohydrate group (-1.2 SE 0.7), but a small increase in the high carbohydrate group (+0.6 SE 0.5), and the difference between groups was statistically significant in the analysis of completers and using the intention-to-treat approach (p=0.02).

These 2 studies of adolescents provide contradictory evidence concerning the benefits of high carbohydrate, low fat diets on BMI change.

Two studies were excluded from the meta-analysis since the difference in carbohydrate between the intervention groups was less than 5% energy in the main analysis (McManus *et al.*, 2001;Dale *et al.*, 2009). These excluded studies of adults provide contradictory evidence concerning the benefits of higher carbohydrate, low fat diets on BMI change, but the differences in carbohydrate content are small.

In the trial conducted by McManus *et al.* (McManus *et al.*, 2001) 101 overweight men and women were allocated to a lower carbohydrate, moderate-fat diet (35% of energy) or a higher carbohydrate, low-fat diet (20% of energy). After 18 months, 31/50 subjects in the moderate-fat group, and 30/51 in the low fat group were available for measurements. In the lower carbohydrate, moderate-fat group, BMI decreased by 1.6 kg/m^2, compared to an increase in the low-fat group of 1.4 kg/m^2 (p<0.001). However, at 18 months, the difference in percentage energy from carbohydrate was less than 5% between groups.

Dale and colleagues randomly assigned 200 women who had previously lost 5% or more of their initial body weight to groups that were advised to follow a high carbohydrate or a relatively high monounsaturated fat diet (Dale *et al.*, 2009). These groups were also divided into those receiving either an intensive or less intensive form of support. After 2 years of follow-up, data were available from 174 (87%) participants. BMI change did not vary significantly between diet groups and the authors concluded that 'diets of different macronutrient composition produced comparable beneficial effects in terms of weight loss maintenance'.

Eight studies of adults therefore remained for inclusion in the meta-analyses comparing different carbohydrate intakes and changes in BMI (weight/height^2) reported as kg/m^2. One study reported results from four groups (Dansinger *et al.*, 2005) and one study reported results from three groups (Gardner *et al.*, 2007). For both of these studies the group with the lowest carbohydrate intake was compared with the group with highest carbohydrate intake. Data from the Women's Health Initiative Dietary Modification Trial are provided in 2 publications which report outcomes at different time points post randomisation (1 yr, 3 years and 90 months) (Tinker *et al.*, 2008;Howard *et al.*, 2006). Data from the 90 months follow-up only were included in this meta-analysis as the intervention was judged to still be 'ongoing' at that point and provides information on the long term impact of dietary change on BMI (Howard *et al.*, 2006).

Trials were separated into 3 main types on the basis of proportion of energy derived from percentage macronutrients. For inclusion in meta-analysis there a difference of energy from carbohydrate between trial groups of 5% or more was considered meaningful. Actual consumption was used rather than intended diet unless otherwise stated – see trial characteristics table.

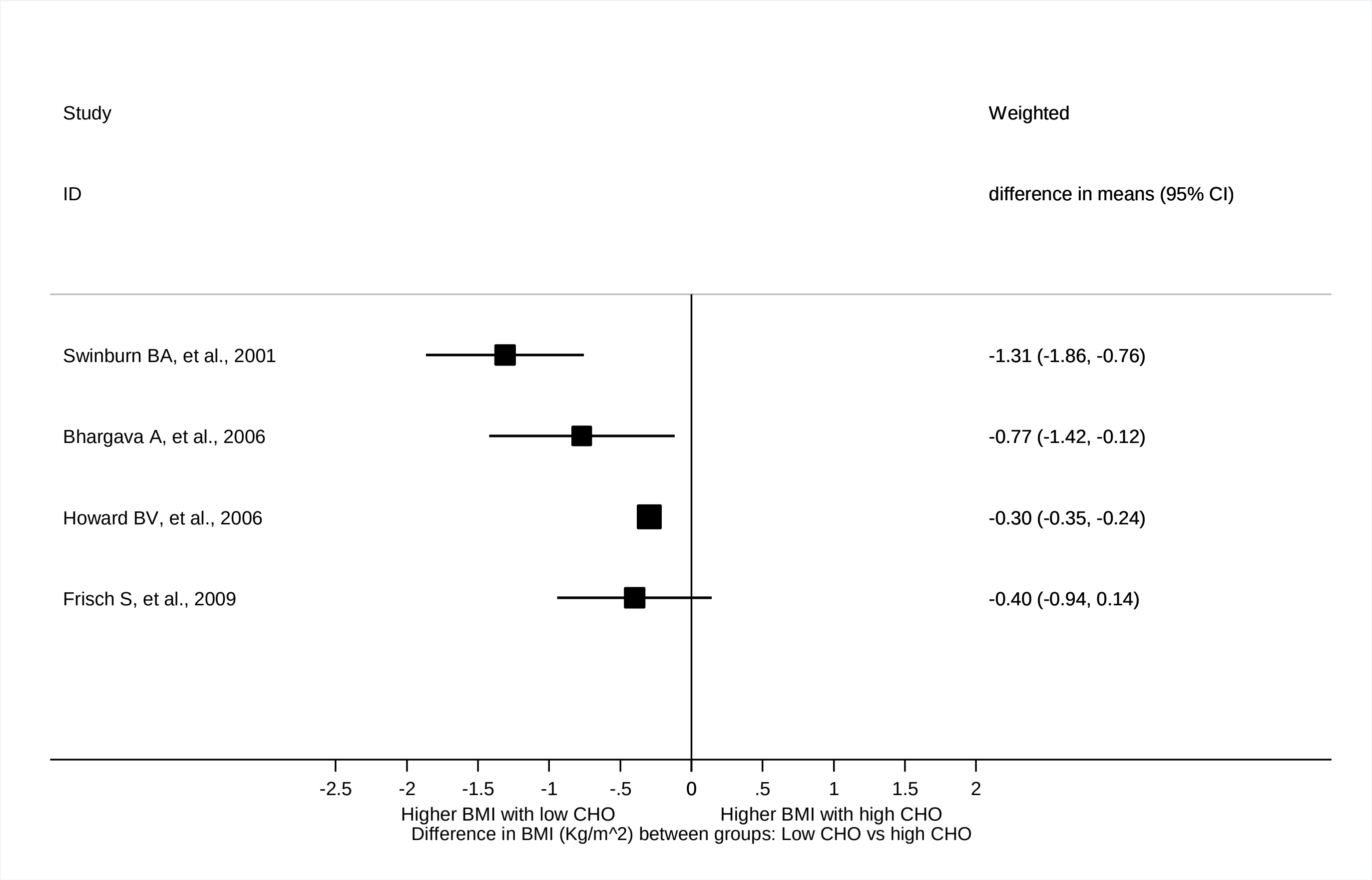
Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat was 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein was 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more but protein intakes were also different by more than 2%.

The studies were stratified according to whether fat only was exchanged with carbohydrate or whether protein only or both protein and fat were exchanged with carbohydrate. There were insufficient studies to stratify by protein shift only, or by a shift in both fat and protein, so these results were combined into one plot. All studies included adults as participants. Definitions of different levels of carbohydrate, fat and protein are reported in the trial characteristics table (Table 5.4). The higher carbohydrate diets varied in percentage energy from carbohydrate between 47 and 63%, and the lower carbohydrate diets between 18 and 49%. The first follow up reported at the end of the intervention and over 12 months was used. This varied from 12 months to 7 years.

Higher carbohydrate, low fat diets compared with lower carbohydrate, higher fat diets

No pooled estimate was reported for studies reporting differences in fat and carbohydrate as heterogeneity was high at 80%. Statistically there was no evidence that BMI was different for high carbohydrate low fat diets.

*Figure 5.3 Forest plot for higher carbohydrate, lower fat vs. lower carbohydrate, higher fat diets and BMI (*kg/m^2)



Higher carbohydrate and lower protein or higher carbohydrate and lower protein and fat diets

The pooled estimate indicated that BMI was 0.26 kg/m^2 (95% CI, -0.46 to 0.98) different with consumption of a high carbohydrate lower fat lower protein diet or high carbohydrate lower protein diet. This was not significantly different from zero (p=0.48). Heterogeneity for studies with change in fat and protein and carbohydrate was 59% (95% CI, 0 to 86%). A funnel plot was not carried out due to the small number of studies included in the analysis. Statistically, there was no evidence that a diet higher in carbohydrate and lower in protein and fat or protein only is associated with differences in BMI.

*Figure 5.4 Forest plot for higher carbohydrate diets swapped with protein only or fat and protein and BMI (*kg/m^2)

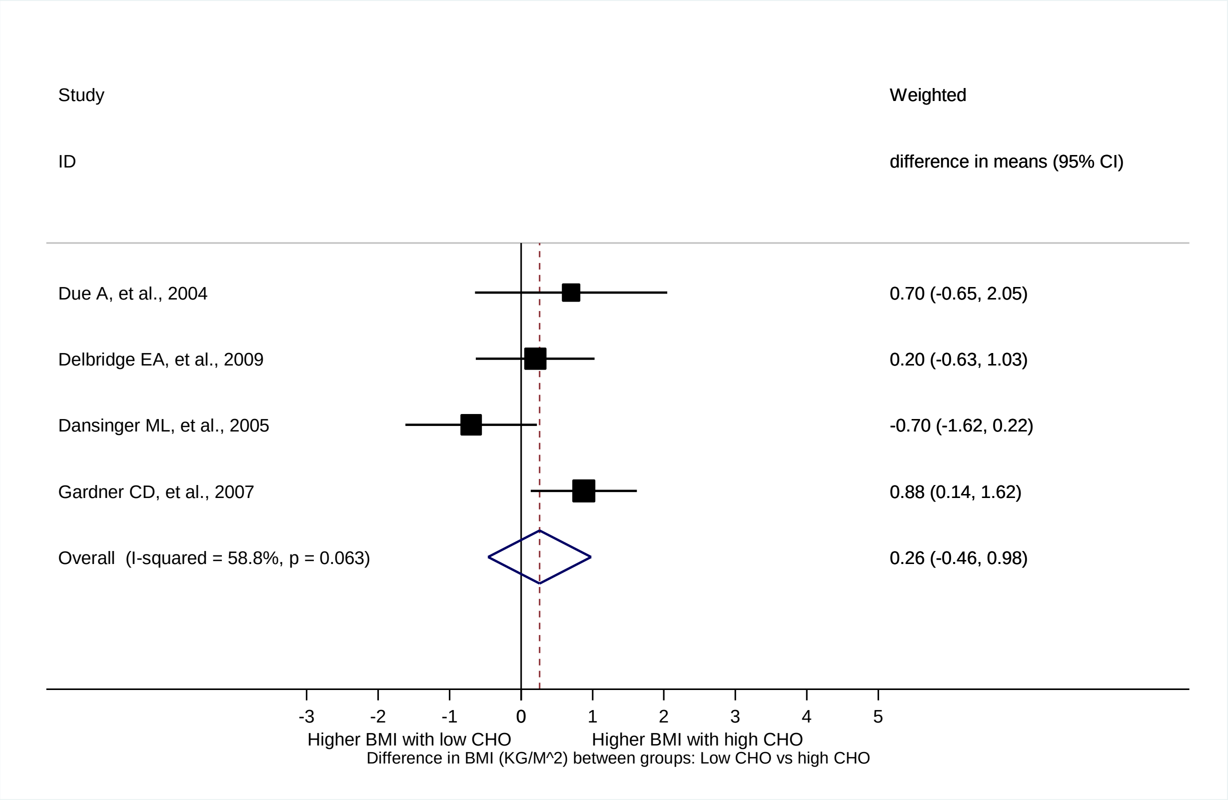


Table 5.6 Body weight and total carbohydrate: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p tren d	Adjustments
13625 (Colditz *et al.*, 1990) NHS	USA, Primarily White, Cancer free, No heart disease, Not diabetic	30-55 %M 0	31940	4 years	FFQ (61)	Carbohydrate, total (grams/day)	Change in weight (kg) Baseline to Follow-up Self-reported			1 g/day		-0.001 (0.000714)			Age, BMI, Energy intake
14120 (Parker *et al.*, 1997) Pawtucket Heart Health Program	USA, Primarily White, Not diabetic	18-64 %M 38	556	4 years	FFQ	Carbohydrate, total (grams/day)	Change in weight (kg) Baseline to Follow-up Measured by clinician/ professional			1 g/day		0.5988 (7.7676)	0.94		Age, BMI, energy intake, physical activity, Smoking
13668 (Ludwig *et al.*, 1999) The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, Not diabetic	18-30 %M 45.9	5115	10 years	FFQ (700)	Carbohydrate, total (% energy)	Body weight (kg) Measured by clinician/ professional	Race - White	(51.9) vs (33.5)		kg: 75.2 vs. 76.6			0.04	Age, Alcohol, Weight, Centre, Education, energy intake, physical activity, Gender, Smoking, Vitamin intake
13669 The CARDIA Study								Race - Black	(51.9) vs (33.5)		kg: 82.6 vs. 84.1			0.03	As above
13809 (Iqbal *et al.*, 2006) MONICA	Denmark, Primarily White	30-60 (45) %M 48.9	2025	5 years	Food diary	Carbohydrate, total (% energy)	Change in weight (kg) Baseline to Follow-up Measured by clinician/ professional	Men		1 % Energy		-0.77 (0.67)	0.25		Age, BMI, Cohort, Education, Energy intake, Food volume, Physical activity, Smoking
14195 MONICA								Women				-1.2 (0.95)	0.19		As above
14271 MONICA								Men, BMI <25				-0.2 (0.9)			As above
14272								Men, BMI 25-30				-1 (1)			As above

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
MONICA															
14273 MONICA								Men, BMI >30				-4.7 (2.7)			As above
14277 MONICA								Women, BMI <25				-0.9 (1.1)			As above
14278 MONICA								Women, BMI 25-30				-2.1 (2.1)			As above
14279 MONICA								Women, BMI >30				-5.5 (5)			As above
14296 MONICA								Obese Men with familial obesity				-4.5 (2.32)	0.05		As above
14297 MONICA								Obese Men without familial obesity				-3.1 (2.3)	0.17		As above
14298 MONICA								Men, Normal weight + familial obesity				-1.1 (1.5)	0.44		As above
14299 MONICA								Men, Normal weight without familial obesity				0.2 (0.8)	0.86		As above
14300 MONICA								Obese Women with familial obesity				-3.8 (4)	0.34		As above
14301 MONICA								Obese Women without familial obesity				-3.2 (5)	0.52		As above
14302 MONICA								Women, Normal weight with familial obesity				-1.2 (1.9)	0.55		As above
14303 MONICA								Women, Normal weight without familial obesity				-1.2 (1.2)	0.31		As above
14690 (Hays *et al.*, 2006) Eating Behaviour and Body Weight in Women	USA, Primarily White, BMI <30, Generally healthy, No medications which influence outcomes, Post-menopausal	55-65 (61) %M 0	67	4.4 years	FFQ	Carbohydrate, total (% energy)	Rate of weight change (kg/yr) Measured by clinician/ professional			1 % Energy		-0.123 (0.041)	0.008		Fibre, Hunger Score, Protein

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14757 (Phelan *et al.*, 2007) National Weight Control Registry	USA, Previous weight loss programme participants	(50) %M 29	891	3 years	FFQ	Carbohydrate, total (% energy)	Change in weight (kg) Baseline to Follow-up Self-reported				Low carbohydrate diet group: 7kg (SD 7.1) Not low carbohydrate diet: 5.7kg (SD 8.7)			0.16	Gender, Baseline weight, Previous weight loss

Table 5.7 BMI and total carbohydrate: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	P trend	Adjustments
14165 (Boreham *et al.*, 1999) The Northern Ireland Young Hearts Project	Northern Ireland	12-15 %M 49.3	509	3 years (11)	Dietary history	Carbohydrate, total (% energy)	BMI Measured by clinician/ professional	Male	1 % Energy	Beta coefficient not reported		NS	Social Class, Genderual maturity
14213								Female	1 % Energy	Beta coefficient not reported		NS	Social Class, Sexual maturity
14612 (Magarey *et al.*, 2001) Adelaide Longitudinal Study of Growth & Nutrition	Australia	2-15 %M 58	500	13 years	Food diary	Carbohydrate, total (% energy)	BMI Measured by clinician/ professional		1 % Energy	0 (0.001)	0.77		Energy Intake, gender, parental BMI, BMI at previous age
13312 (Twisk *et al.*, 1998) Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 46	233	14 years (22)	Dietary history	Carbohydrate, total (grams/day)	BMI Measured by clinician/ professional		g/day	0.02 (-0.02, 0.05)	0.33		Time, gender
13311						Carbohydrate, total (% energy)	BMI Measured by clinician/ professional		1 % Energy	-0.01 (-0.04, 0.02)	0.53		As above

Table 5.8 Body weight and high carbohydrate diets: RCT data

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
*16851 (Bhargava, 2006)		Control	379/ allocated not reported	75.92 (SD 12.56)	75.53 (SD 12.62)	-0.39	0.05				Body weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Low fat	615/ allocated not reported	75.95 (SD 12.43)	73.86 (SD 12.6)	- 2.09	0.05	0.05						Decrease	
17430 (Clifton *et al.*, 2008)		High carbohydrate diet	38/38			4.4 (SD 6.1)					Body weight	Measured by clinician/ professional (kg)	1.25 years	Decrease	Unclear
		High protein diet	40/41			4.6 (SD 5.5)								Decrease	
17383 (Dale *et al.*, 2009)		High carbohydrate diet	89/100	85.5 (SD 15.6)	83.5 (SD 15.9)						Body weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		High MUFA diet	85/100	86.2 (SD 13.6)	84.8 (SD 14.7)									Decrease	
17361		High carbohydrate diet	89/100	85.5 (SD 15.6)	83.0 (SD 15.2)						Body weight	(kg)	2 years	Decrease	Unclear
		High MUFA diet	85/100	86.2 (SD 13.6)	84.3 (SD 14.3)									Decrease	
15978		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100							0.7 (CI -1.1, 2.4)	Body weight	(kg)	2 years	Decrease in both	Unclear
**15692 (Dansinger *et al.*, 2005)		Atkins	40/40			-2.1 (SD 4.8)	0.01				Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	No bias
		Ornish	40/40			-3.3 (SD 7.3)	0.01							Decrease	
		Weight watchers	40/40			-3 (SD 4.9)	0.01							Decrease	
		Zone	40/40			-3.2 (SD 6)	0.01							Decrease	
**15307 (Delbridg		Low fat, high carbohydrate	70/70			3.8 (SE 0.9)					Body weight	Measured by clinician/	1 year	Increase	Unclear

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
e *et al.*, 2009)		weight maintenance diet										professional (kg)			
		Low fat, high protein weight maintenance diet	68/71			3.1 (SE 0.8)		0.544						Increase	
**16026 (Due *et al.*, 2004)		High protein	23/23			-6.2 (CI -8.6, -3.8)					Body weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Moderate protein	18/18			-4.3(CI -6.4, -2.2)								Decrease	
15464 (Ebbeling *et al.*, 2007)		Low fat diet	ITT: 37/37				<0.05				Body weight change	Measured by clinician/ professional	1 year	Decrease	No bias
		Low GL diet	ITT: 36/36				<0.05	NS						Decrease	
15465		Low fat diet	ITT: 37/37								Body weight change	Measured by clinician/ professional	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36					NS						Decrease	
15467	Insulin (30m post 75gdose) <57.5µUI /ml	Low fat diet	ITT: 37/37				<0.05				Body weight change	Measured by clinician/ professional	1 year	Decrease	No bias
		Low GL diet	ITT: 36/36				NS	NS						Decrease	
15468	Insulin (30m post 75gdose) <57.5µUI /ml	Low fat diet	ITT: 37/37				NS				Body weight change	Measured by clinician/ professional	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36				NS	NS						Decrease	
15470	Insulin (30m post	Low fat diet	ITT: 37/37		No loss		NS				Change in weight	Measured by clinician/ professional	1 year	Decrease	No bias

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
	75gdose) >57.5µUI /ml	Low GL diet	ITT: 36/36		Greater loss		<0.05	NS						Decrease	
15471	Insulin (30m post 75gdose) >57.5µUI /ml	Low fat diet	ITT: 37/37			-1.2	NS				Body weight change	Measured by clinician/ professional (kg)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-5.8	<0.05	0.004						Decrease	
*15420 (Ebbeling *et al.*, 2005)		Low fat diet	12/17	83.2 (SE 3.3)		-6.1% (CI -11.2, -0.7)					Body weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Low GI diet	11/17	93.9 (SE 5.3)		-7.8% (CI -13, -2.2)								Decrease	
**15194 (Foster *et al.*, 2003)		Conventional diet plan	30/30			-2.5 (SD 6.3)	<0.05				% change in body weight	Measured by clinician/ professional	1 year	Decrease	Unclear
		Low carbohydrate diet	33/33			-4.4 (SD 6.7)	<0.05	0.26						Decrease	
*15149 (Frisch *et al.*, 2009)		High carbohydrate diet	100/100			-4.3 (SE 5.1)					Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Moderate carbohydrate diet	100/100			-5.8 (SE 6.1)		0.065						Decrease	
15133 (Gardner *et al.*, 2007)		Atkins: low carbohydrate Zone: moderate carbohydrate Ornish: high carbohydrate	Atkins: 77/77 Zone: 79/79 Ornish: 76/76			-4.7 (-6.3, -0.8) -1.6 (-2.8, -0.4) -2.2 (-3.6, -0.8)			Atkins different from Zone, P<0.05		Body weight change (kg) Mean (95% CI)	Measured by clinician/ professional	1 year	Decrease	No bias
15136		Ornish: high carbohydrate minus Zone: moderate carbohydrate	Zone: 79/79 Ornish: 76/76						No significant difference		Body weight change	Measured by clinician/ professional	1 year	Decrease in both	No bias

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
15139		Atkins: low carbohydrate minus Ornish: high carbohydrate	Atkins: 77/77 Ornish: 76/76						No significant difference		Body weight change	Measured by clinician/ professional	1 year	Decrease in both	No bias
*15661 (Greenberg *et al.*, 2009)		Low carbohydrate	109/109			-4.7 (SD 6.5)		NS			Body weight change	Measured by clinician/ professional (kg)	24 months	Decrease	Unclear
		Low fat	104/104			-2.9 (SD 4.2)								Decrease	
(Howard *et al.*, 2006) *16240		Control	25056/ 29294	76.7 (SD 16.5)	76.1 (SD 16.9)	-0.1 (SD 10.1)					Body weight	Measured by clinician/ professional (kg)	90 months	No change	No bias
		Low fat diet	16297/ 19541	76.8 (SD 16.6)	75.7 (SD 17.1)	-0.8 (SD 10.1)		<0.001						Decrease	
**15597 (Keogh *et al.*, 2007)		High carbohydrate diet	completers not reported/ 12			-4.6 (SE 2.1)	0.01				Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Low carbohydrate diet	completers not reported/ 13			-5.5 (SE 1.2)	0.01							Decrease	
**14950 (Layman *et al.*, 2009)		High carbohydrate, low protein diet	51/66			-7.4 (SE 0.6)	NS				Change in weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Low carbohydrate, high protein diet	52/64			-9.3 (SE 1)	NS							Decrease	
14952		High carbohydrate, low protein diet	51/66			-8.0 (SE 0.7)	NS				% change in body weight	Measured by clinician/ professional	1 year	Decrease	Unclear
		Low carbohydrate, high protein diet	52/64			-9.6 (SE 0.9)	NS							Decrease	

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
14885 (McManus *et al.*, 2001)		Low fat diet	13/51			-5.0 (SD 7.3)	<0.05				Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Moderate fat diet	27/50			-4.8 (SD 5.2)	<0.001							Decrease	
14886		Low fat diet	30/51			2.9 (SD 7.7)	NS				Body weight change	Measured by clinician/ professional	18 months	Increase	Unclear
		Moderate fat diet	31/50			-4.1 (SD 6.5)	<0.01							Decrease	
14888		Low fat diet	13/51			-8.0 (SD 13)	<0.05				Change in Ideal body weight (%)	Measured by clinician/ professional	1 year	Decrease	Unclear
		Moderate fat diet	27/50			-8.0 (SD 9.0)	<0.001							Decrease	
14889		Low fat diet	30/51			5 (SD 13)	NS				Change in Ideal body weight (%)	Measured by clinician/ professional	18 months	Increase	Unclear
		Moderate fat diet	31/50			-6 (SD 11)	<0.01	0.001						Decrease	
16014 (Sacks *et al.*, 2009)		High-fat, average-protein	ITT: /204					NS			Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	No bias
		High-fat, high-protein	ITT: /201					NS						Decrease	
		Low-fat, average-protein	ITT: /204					NS						Decrease	
		Low-fat, high-protein	ITT: /202					NS						Decrease	
16015		High-fat, average-protein	ITT: /204					NS			Body weight change	Measured by clinician/ professional (kg)	18 months	Decrease	No bias
		High-fat, high-protein	ITT: /201					NS						Decrease	
		Low-fat, average-protein	ITT: /204					NS						Decrease	
		Low-fat, high-protein	ITT: /202					NS						Decrease	
16012		High-fat,	ITT:					NS			Body	Measured by	2 years	Decrease	No bias

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
		average-protein	/204								weight change	clinician/ professional (kg)			
		High-fat, high-protein	ITT: /201					NS						Decrease	
		Low-fat, average-protein	ITT: /204					NS						Decrease	
		Low-fat, high-protein	ITT: /202					NS						Decrease	
*15417 (Shah *et al.*, 1996)		Energy restricted diet	36/36	175.7 (SD 9.7)	173.9 (SD 19)						Body weight	Assessment method not reported (lb)	1 year	Decrease	Unclear
		Low fat, high complex CHO	39/39	176.2 (SD 9.8)	170.8 (SD 14.1)									Decrease	
15710 (Sheppard *et al.*, 1991)		Control	105/119	66.0 (SD 9.8)		-0.4 (SD 3.5)					Body weight	Measured by clinician/ professional (kg)	1 year	No change	Unclear
		Low fat diet	171/184	68.9 (SD 10.8)		-3.0 (SD 4.8)								Decrease	
*15711		Control	94/119	66.0 (SD 9.8)		-0.1 (SD 4.1)					Body weight	Measured by clinician/ professional (kg)	24 months	No change	Unclear
		Low fat diet	158/184	68.9 (SD 10.8)		-1.9 (SD 4.9)								Decrease	
*15835 (Swinburn *et al.*, 2001)		Control diet	70/70			0.59 (SE 1.61)					Body weight change	Measured by clinician/ professional (kg)	1 year	No change	Unclear
		Low fat	66/66			-3.32 (SE 0.68)								Decrease	
15856		Control diet	57/70			1.06 (SE 0.46)					Body weight change	Measured by clinician/ professional (kg)	2 years	No change	Unclear
		Low fat	47/66			-3.15 (SE 0.78)								Decrease	
15857		Control diet	51/70			2.13 (SE 0.7)					Body weight change	Measured by clinician/ professional	3 years	No change	Unclear

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from base-line	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
												(kg)			
		Low fat	48/66			-1.6 (SE 0.78)								Decrease	
15858		Control diet	52/70			1.26 (SE 0.68)					Body weight change	Measured by clinician/ professional (kg)	5 years	No change	Unclear
		Low fat	51/66			1.06 (SE 0.64)								Decrease	
15354 (Tinker *et al.*, 2008)		Control	24977/29294	76.2 (SD 16.3)	75.9 (SD 16.5)						Body weight	Not reported (kg)	1 year	No change	Unclear
		Low fat diet	17026/19541	76.4 (SD 16.5)	74 (SD 16.5)			0.001						Decrease	
15362		Control	22321/29294	76.2 (SD 16.3)	76.2 (SD 16.6)						Body weight	Not reported (kg)	6 years	No change	Unclear
		Low fat diet	14409/19541	76.4 (SD 16.5)	75.6 (SD 16.8)			0.001						Decrease	

*This result was used in the meta-analysis for carbohydrate/fat swap and body weight (Figure 5.1)

**This results was used in the meta-analysis for carbohydrate/protein and carbohydrate/fat and protein swap and body weight (Figure 5.2)

Table 5.9 BMI and high carbohydrate diets: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
Adolescent studies													
15392 (Demol *et al.*, 2009)	High carbohydrate, low fat	20/20	33.8 (SD 1.5)	31.1 (SD 1.6)					BMI	Measured by clinician/ professional	1 year	Decrease	Bias
	Low carbohydrate, high fat	17/17	33.7 (SD 1.6)	32.6 (SD 1.7)			NS					Decrease	
	Low carbohydrate, high protein	18/18	35.2 (SD 1.6)	32.4 (SD 1.6)			NS					Decrease	
15394	High carbohydrate, low fat	20/20	3.3 (SD 0.3)	2.5 (SD 0.3)					BMI SDS	Measured by clinician/ professional	1 year	Decrease	Bias
	Low carbohydrate, high fat	17/17	3.1 (SD 0.3)	2.7 (SD 0.4)			NS					Decrease	
	Low carbohydrate, high protein	18/18	3.4 (SD 0.3)	2.7 (SD 0.4)			NS					Decrease	
15019 (Ebbeling *et al.*, 2003)	Low fat diet	8/8	37.1 (SE 1.2)		0.6 (SE 0.5)				BMI	Measured by clinician/ professional	1 year	No change	Bias
	Low GL diet	8/8	34.9 (SE 1.0)		-1.2 (SE 0.7)		0.02					Decrease	
Adult studies													
*16865 (Bhargava, 2006)	Control	379/allocated not reported	28.9 (SD 4.65)	28.75 (SD 4.68)		0.05			BMI	Measured by clinician/ professional	1 year	Decrease	Unclear
	Low fat	615/allocated not reported	28.77 (SD 4.43)	27.98 (SD 4.47)		0.05	0.05					Decrease	
15977 (Dale *et al.*, 2009)	High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.2 (CI -0.4, 0.9)	BMI	Measured by clinician/ professional	2 years	Decrease in both	Unclear
17362	High carbohydrate diet	89/100	31.8 (SD 5.2)	30.8 (SD 5.1)					BMI	Measured by clinician/ professional	2 years	Decrease	Unclear
	High MUFA diet	85/100	31.9 (SD 4.9)	31.2 (SD 5.1)								Decrease	
17384	High carbohydrate	89/100	31.8 (SD 5.2)	31.0 (SD 5.4)					BMI	Measured by clinician/ professional	1 year	Decrease	Unclear

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
	diet												
	High MUFA diet	85/100	31.9 (SD 4.9)	31.4 (SD 5.3)								Decrease	
**15693 (Dansinger *et al.*, 2005)	Atkins	40/40			-0.7 (SD 1.6)	0.01			BMI	Measured by clinician/ professional	1 year	Decrease	No bias
	Ornish	40/40			-1.4 (SD 2.5)	0.05						Decrease	
	Weight watchers	40/40			-1.1 (SD 1.7)	0.01						Decrease	
	Zone	40/40			-1.1 (SD 2)	0.01						Decrease	
**15316 (Delbridge *et al.*, 2009)	Low fat, high carbohydrate weight maintenance diet	70/70			1.3 (SE 0.3)				BMI	Measured by clinician/ professional	1 year	Increase	Unclear
	Low fat, high protein weight maintenance diet	68/71			1.1 (SE 0.3)		0.624					Increase	
**16028 (Due *et al.*, 2004)	High protein	18/23			-1.5 (CI -2.8, -0.8)				BMI	Measured by clinician/ professional	1 year	Decrease	Unclear
	Moderate protein	23/18			-2.2 (CI -3, -1.2)							Decrease	
*15156 (Frisch *et al.*, 2009)	High carbohydrate diet	100/100			-1.5 (SD 1.8)	0.05			BMI	Measured by clinician/ professional	1 year	Decrease	Unclear
	Moderate carbohydrate diet	100/100			-1.9 (SD 2.1)	0.05	0.11					Decrease	
**14058 (Gardner *et al.*, 2007)	Atkins: low carbohydrate	77/77			-1.65 (SD 2.54)		0.05		BMI	Measured by clinician/ professional	1 year	Decrease	No bias
	Ornish: high carbohydrate	76/76			-0.77 (SD 2.14)		NS					Decrease	
	Zone: moderate carbohydrate	79/79			-0.53 (SD 2)							Decrease	
	Low fat diet	380/19541	26.2 (SD 4.9)	26.1 (SD 4.8)	0.1 (SD 2.8)		0.67					Decrease	
(Howard *et al.*, 2006) *16241	Control	24943/ 29294	29.1 (SD 5.9)	29.2 (SD 5.9)	0.3 (SD 3.1)				BMI	Measured by clinician/ professional	90 months	No change	No bias
	Low fat diet	16230/ 19541	29.1 (SD 5.9)	29.0 (SD 6.1)	0.003 (SD 3.2)		<0.001					Decrease	

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
14891 (McManus *et al.*, 2001)	Low fat diet	13/51			-1.8 (SD 2.9)	<0.05			BMI	Measured by clinician/ professional	1 year	Decrease	Unclear
	Moderate fat diet	27/50			-2.0 (SD 2.1)	<0.001						Decrease	
14892	Low fat diet	30/51			1.4 (SD 3.3)	<0.05			BMI	Measured by clinician/ professional	18 months	Increase	Unclear
	Moderate fat diet	31/50			-1.6 (SD 2.5)	<0.01	<0.001					Decrease	
*15859 (Swinburn *et al.*, 2001)	Control diet	70/70			0.22 (SE 0.15)				BMI	Measured by clinician/ professional	1 year	No change	Unclear
	Low fat	66/66			-1.09 (SE 0.24)							Decrease	
15860	Control diet	57/70			0.38 (SE 0.15)				BMI	Measured by clinician/ professional	2 years	No change	Unclear
	Low fat	47/66			-1.01 (SE 0.28)							Decrease	
15861	Control diet	51/70			0.75 (SE 0.24)				BMI	Measured by clinician/ professional	3 years	No change	Unclear
	Low fat	48/66			-0.46 (SE 0.28)							Decrease	
15862	Control diet	52/70			0.59 (SE 0.27)				BMI	Measured by clinician/ professional	5 years	No change	Unclear
	Low fat	51/66			0.72 (SE 0.28)							Decrease	
15363 (Tinker *et al.*, 2008)	Control	24977/ 29294	28.9 (SD 5.8)	28.7 (SD 5.7)					BMI	Not reported	1 year	No change	Unclear
	Low fat diet	17026/ 19541	28.9 (SD 5.8)	28 (SD 5.8)			0.001					Decrease	
15364	Control	22321/ 29294	28.9 (SD 5.8)	29.1 (SD 5.8)					BMI	Not reported	6 year	No change	Unclear
	Low fat diet	14409/ 19541	28.9 (SD 5.8)	28.8 (SD 5.9)			0.001					Decrease	

*This result was used in the meta-analysis for carbohydrate/fat swap and BMI (Figure 5.3)

**This results was used in the meta-analysis for carbohydrate/protein and carbohydrate/fat and protein swap and BMI (Figure 5.4)

Body weight and carbohydrate supplements

No cohort studies reported results concerning carbohydrate supplements and body composition.

Summary of RCT data

One randomised double blind trial explored the effects of a 50g carbohydrate supplement (42% glucose, 58% maltodextrins) compared with a control (do nothing) intervention on body weight over 16 months (Pasman *et al.*, 1997b). An initial 2 month weight loss phase with a very low energy diet was employed, and then the trajectory of weight gain was compared in the 2 groups over a further 14 months in the carbohydrate and control groups. A further group was also followed which consumed a combination supplement of carbohydrate, chromium-picolinate, caffeine and soluble fibre. These data were not extracted. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.10. While there was somewhat less weight regain in the group that consumed a daily carbohydrate supplement, this was not statistically different from the regain in the control group.

nb. while the authors claim the study was double-blind, it is unclear how this was achieved without a placebo product.

Table 5.10 Body weight and carbohydrate supplements: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Follow-up	p-value difference between groups	Outcome/Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15499 (Pasman *et al.*, 1997b)	Carbohydrate	11/10	68.1 ±55.2%	NS	Percent weight lost in very low energy diet phase regained	Measured by clinician/ professional	14 months	Increase	unclear
	Control	9/9	85.5 ±55.8%					Increase	

Body weight and sucrose

Summary of cohort results in adults

The NHS and the Pawtucket Heart Health Program provided data on sucrose intake and change in body weight (Colditz *et al.*, 1990;Parker *et al.*, 1997), neither of which showed evidence of a statistically significant relationship (see Table 5.11). Both estimated baseline sucrose intake from FFQs, in grams per day. One relied upon self-reports of weight change (Colditz *et al.*, 1990). Both studies adjusted for important confounders (age, baseline BMI, energy intake).

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs reported data on sucrose and body weight change.

Table 5.11 Body weight and sucrose: cohort studies in adults

Result ID/ Reference / Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14174 (Colditz *et al.*, 1990) NHS	USA, Primarily White, Cancer free, No heart disease, Not diabetic	30-55 %M 0	121700	4 years	FFQ (61)	Sucrose	Change in weight (kg) Baseline to Follow-up Self-reported	1 g/day	-0.0024 (0.0012)		Age, BMI, EI
14121 (Parker *et al.*, 1997) Pawtucket Heart Health Program	USA, Primarily White, Not diabetic	18-64 %M 38	556	4 years	FFQ	Sucrose	Change in weight (kg) Baseline to Follow-up Measured by clinician/ professional	1 g/day	2.5961 (3.1911)	0.41	Age, BMI, EI, PA, Smoking

Body weight or BMI and dietary fibre

Summary of cohort results in adults

Data were extracted from four publications presenting data from the following four cohort studies: NHS (Colditz *et al.*, 1990); MONICA (Iqbal *et al.*, 2006); the CARDIA Study (Ludwig *et al.*, 1999); and the Eating Behaviour and Body Weight in Women study (Hays *et al.*, 2006). The NHS and MONICA reported data on dietary fibre in grams per day in relation to weight change (Colditz *et al.*, 1990;Iqbal *et al.*, 2006). No statistically significant beta coefficients were found, nor did there appear to be a consistent direction of effect in these two studies.

The two studies that calculated fibre density, on the other hand, both reported statistically significant results: the CARDIA study (Ludwig *et al.*, 1999) found mean body weight at follow up, adjusted for covariates, to be significantly lower among high fibre-density consumers than low fibre-density consumers; whilst the Eating Behaviour and Body Weight in Women study (Hays *et al.*, 2006) found fibre density to be positively associated with weight change, suggesting an increase in body weight with increasing fibre density. In this small study of 36 postmenopausal women, each one gram fibre per 1000 kcal increase at baseline was associated with an increase in weight per year of 0.38 kg (p=0.005) over the 4 year follow-up period.

The majority of the studies gathered anthropometric measures using clinicians or professionals; the NHS (Colditz *et al.*, 1990), however, relied upon self-reported weight.

In summary, these cohort studies provide inconsistent evidence concerning the association between dietary fibre intake and BMI or body weight.

Exposure definition and assessment

With the exception of MONICA (Iqbal *et al.*, 2006), which collected food diaries, all studies measured dietary intake via FFQ. Varying methods were used to calculate fibre from dietary data.

Adjustment for appropriate confounders

The Eating Behaviour and Body Weight in Women study (Hays *et al.*, 2006) adjusted only for carbohydrate intake, hunger score and protein intake. All other studies included the important covariates of age, weight and energy intake (Colditz *et al.*, 1990) as well as other appropriate lifestyle or demographic variables (Iqbal *et al.*, 2006;Ludwig *et al.*, 1999). In the CARDIA study dietary fibre remained independently associated with body weight after additional adjustment for carbohydrate intake, and the relationship between dietary fibre and body weight was generally more robust than between dietary carbohydrate and body weight (Ludwig *et al.*, 1999).

Summary of cohort results in children

Only The DONALD Study reported on the relationship between dietary fibre and change in body weight in children (Cheng *et al.*, 2009). This study found no association between dietary fibre intake assessed by food diaries and BMI change over 4 years in 376 German girls and boys.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Two studies provided data on the effects of dietary fibre supplementation on body weight and BMI changes in children and adults respectively (Abrams *et al.*, 2007;Pasman *et al.*, 1997a).

Abrams *et al.* (Abrams *et al.*, 2007) conducted an exploratory study of the effects of a prebiotic supplement containing oligofructose and inulin and habitual calcium intake on changes in body composition of pre-pubertal children. This randomised, double-blind trial included 97 children initially aged 9-13 years who were allocated to consume either an 8g/d prebiotic supplement or an equivalent 8g/d maltodextrin control supplement for one year. The prebiotic supplement group exhibited a significantly smaller increase in body weight, BMI and BMI Z-score than the control group ($p<0.05$ for all) after one year. The control group tended towards an exaggerated increase in adiposity, whereas the prebiotic group grew along recommended lines.

One study of adults provided data on the effects of partially hydrolysed guar gum supplements on body weight and BMI maintenance in weight-reduced subjects over 16 months (Pasman *et al.*, 1997a). Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.14. The consumption of 20g of water soluble fibre (guar gum) per day for 14 months, following a 2 month very low calorie diet weight loss phase, did not prevent weight regain compared with the non-treated control condition (no placebo used).

nb. this is a rather curious design – the paper does not clarify whether the decision to split into high and low compliance groups was made *a priori*. Furthermore, the paper does not report the degree of hydrolysis, molecular weight or viscosity of the guar gum, so difference from native guar gum cannot be assessed.

Table 5.12 Body weight and dietary fibre and fibre density: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14173 (Colditz *et al.*, 1990) NHS	USA, Primarily White, Cancer free, No heart disease, Not diabetic	30-55 %M 0	121700	4 years	FFQ (61)	Dietary Fibre, g/d (Southgate method)	Change in weight (kg) Baseline to Follow-up Self-reported			1 g/day		0.0055 (0.003)			Age, BMI, energy intake
14194 (Iqbal *et al.*, 2006) MONICA	Denmark, Primarily White	30-60 (45) %M 48.9	2025	5 years	Food diary	Dietary Fibre, g/d	Change in weight (kg) Baseline to Follow-up Measured by clinician/ professional	Men		1 g/day		9.6 (6.9)	0.17		Age, BMI, Cohort, Education, energy intake, Food volume, Physical activity, Smoking
14197								Women		1 g/day		-22.3 (13.4)	0.1		As above
14274								Men, BMI <25		1 g/day		10.5 (8.2)			As above
14275								Men, BMI 25-30		1 g/day		9.7 (11)			As above
14276								Men, BMI >30		1 g/day		-7 (23.2)			As above
14280								Women, BMI <25		1 g/day		-31.5 (14.7)			As above
14281								Women, BMI 25-30		1 g/day		27.5 (32.3)			As above
14282								Women, BMI >30		1 g/day		-59.4 (47.2)			As above
13666 (Ludwig *et al.*, 1999) The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, Not diabetic	18-30 %M 45.9	5115	10 years	FFQ (700)	Fibre density (g/unit energy. AOAC method)	Body weight (kg) Measured by clinician/ professional	Race - White	Q5 vs Q1 (12.3) vs (5.2)	g/4184kJ /day	kg: 75.01 vs. 78.66			<0.001	Age, Alcohol, Weight, Centre, Education, Energy intake, physical activity, Gender, Smoking, Vitamin intake
13667								Race - Black	Q5 vs Q1 (12.3) vs (5.2)	g/4184kJ /day	kg: 79.92 vs. 83.52			0.001	As above
14691 (Hays *et al.*, 2006) Eating Behaviour and Body Weight in Women	USA, Primarily White, BMI <30, Generally healthy, No medications which influence outcomes, Post-menopausal	55-65 (61) %M 0	67	4.4 years	FFQ	Fibre density (g/unit energy)	Rate of weight change (kg/yr) Measured by clinician/ professional			1 g/ 1000 kcal		0.384 (0.117)	0.005		CHO, Hunger Score, Protein

Table 5.13 BMI and dietary fibre: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14006 (Cheng *et al.*, 2009) The DONALD Study	Germany	(9) %M46	376	4 years	Food diary	Dietary fibre, g/d (The dietary fibre content for foods was determined by different enzymatic methods as defined in the respective food tables)	Change in BMI SD score Measured by clinician/ professional	1 g/day	-0.007 (0.01)	0.5	Age, Breastfed, EI, GI, Parental overweight, Gender, Birth year

Table 5.14 Body weight and BMI and dietary fibre: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
Children											
(Abrams *et al.*, 2007)	Prebiotic supplement	48/50	42.6 (SE 1.3)	47.7 (SE 0.4)		0.048	Body weight	Measured by clinician/ professional (kg)	12 months	Increase	unclear
	Control – maltodextrin supplement	49/50	41.3 (SE 1.3)	49.0 (SE 0.4)						Increase	
	Prebiotic supplement	48/50	0.26 (SE 0.18)	0.25 (SE 0.04)	0.30	0.048	BMI Z-score	Measured by clinician/ professional	12 months	Increase	unclear
	Control – maltodextrin supplement	49/50	0.20 (SE 0.18)	0.38 (SE 0.04)	0.001					Increase	
	Prebiotic supplement	48/50	18.9 (SE 0.37)	19.5 (SE 0.15)		0.016	BMI	Measured by clinician/ professional	12 months	Increase	unclear
	Control – maltodextrin supplement	49/50	18.6 (SE 0.37)	20.0 (SE 0.15)						Increase	
Adults											
15531 (Pasman *et al.*, 1997a)	Control	10/14	78.3 (SD 10.6)	85.0 (SD 12.0)	<0.05		Body weight maintenance after loss	Measured by clinician/ professional (kg)	14 months	Increase	unclear
	Guar gum - High compliance	10/10	78.6 (SD 10.2)	85.6 (SD 13.1)	<0.05	NS				Increase	
	Guar Gum - Low compliance	10/10	77.3 (SD 6.1)	88.4 (SD 10.1)	<0.05	NS				Increase	
15534	Control	11/14	28.8 (SD 3.8)	31.4 (SD 5.1)	<0.05		BMI maintenance after weight loss	Measured by clinician/ professional	14 months	Increase	Unclear
	Guar gum - High compliance	10/10	29.7 (SD 2.3)	32.3 (SD 3.7)	<0.05	NS				Increase	
	Guar gum - Low compliance	10/10	29.0 (SD 3.3)	33.2 (SD 5.0)	<0.05	NS				Increase	

Body weight and BMI and carbohydrate rich food groups

Summary of cohort results in adults

One cohort study of adult men and women from south eastern New England (USA) provided data. The Pawtucket Heart Health Program investigated the link between various baseline nutrients and food groups, including certain carbohydrate-rich foods and change in body weight after 4 years (Parker *et al.*, 1997). In this study, the consumption of 'sweets' was measured via FFQ. However, a precise definition was not provided in the paper. These foods were not significantly associated with weight change. Analyses included appropriate adjustments for confounders (Table 5.15).

Summary of cohort results in children

Data were extracted from three publications, reporting results from three cohort studies in children (Table 5.16). These included the Cardiovascular Risk in Young Finns Study, the National Heart, Lung and Blood Institute Growth and Health Study and the MIT Growth and Development Study (Nissinen *et al.*, 2009;Barton *et al.*, 2005;Phillips *et al.*, 2004). All three studies used measured, rather than self-reported weight to calculate BMI. The food groups studied were 'sweets' and 'candy' (Nissinen *et al.*, 2009;Phillips *et al.*, 2004), breakfast cereals (Barton *et al.*, 2005), potato or corn crisps and baked goods (Phillips *et al.*, 2004). Consumption of 'sweets' or 'candy' was not significantly associated with BMI in either study. Results were also non-significant for potato and corn crisps and for baked goods (Phillips *et al.*, 2004). However, a significant negative association was reported between frequency of breakfast cereal consumption and BMI z-score (Barton *et al.*, 2005), indicating a lower BMI with increasing breakfast cereal consumption.

Exposure definition and assessment

The Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009) and the MIT Growth and Development Study (Phillips *et al.*, 2004) both employed FFQs to collect dietary data, whilst the National Heart, Lung and Blood Institute Growth and Health Study (Barton *et al.*, 2005) used three-day food diaries. Food group consumption was recorded as units per month (Nissinen *et al.*, 2009), frequency over three days (Barton *et al.*, 2005) or per cent contribution to energy intake (Phillips *et al.*, 2004).

Adjustment for appropriate confounders

All three studies included some covariates for adjustment, including age, weight, and some parental characteristics. The National Heart, Lung and Blood Institute Growth and Health Study additionally adjusted for study site, physical activity and energy intake; and the MIT Growth and Development Study for age at menarche and fruit and vegetable intake.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs reported data concerning carbohydrate rich foods and body weight or BMI.

Table 5.15 Body weight and carbohydrate rich food groups: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14122 (Parker *et al.*, 1997) Pawtucket Heart Health Program	USA, Primarily White, Not diabetic	18-64 %M 38	556	4 years	FFQ	'Sweets' – not defined in paper	Change in weight (kg) Baseline to Follow-up Measured by clinician/ professional	Servings / week	-0.3057 (0.4751)	0.52	Age, BMI, Energy intake, physical activity, Smoking

Table 5.16 BMI and carbohydrate rich food groups: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Beta coefficient (SE)/(CI)	p	P trend	Adjustments
14183 (Barton *et al.*, 2005) National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	10 years (10)	Food diary	Breakfast cereal consumption frequency (number of days consuming out of 3 days recorded, 0, 1, 2 or 3)	BMI z-score Measured by clinician/ professional				-0.15 (0.005)		<0.001	Study site, Parental education, number of parents, physical activity, energy intake
13924 (Nissinen *et al.*, 2009) Cardiovascular Risk in Young Finns Study	Finland, Primarily White	3-18 (11) %M 45.2	3596	21 years (33)	FFQ (19)	'Sweets' - confectionery	BMI Measured by clinician/ professional	Male		10 Units/ month	0.01 (0.14)	0.98		Age, Weight, Parental education
14633								Female		10 Units/ month	0.03 (0.14)	0.85		As above
14185 (Phillips *et al.*, 2004) Massachusetts Institute of Technology Growth and Development Study	USA, Primarily White, BMI <30, Generally healthy	8-12 %M 0	196	6.9 years (28)	FFQ (116)	Percent of calories from chips (potato and corn crisps)	BMI z-score Measured by clinician/ professional		1. <1 2. 1 to 1.4 3. 1.5 to 2.4 4. ≥2.5	% Energy	1. Referent 2. 0.032 3. 0.030 4. 0.047	1. N/A 2. 0.39 3. 0.39 4. 0.26	0.24	Age at menarche, Fruit and veg, Parental overweight
14184						Percent of calories from candy (confectionery)			1. <1.5 2. 1.5 to 24 3. 2.5 to 3.9 4. ≥4	% Energy	1. Referent 2. 0.021 3. 0.005 4. 0.082	1. N/A 2. 0.52 3. 0.9 4. 0.066	0.09	As above
14186						Percent of calories from baked goods (buns, pastries, cookies, cakes, pies			1. <2 2. 2 to 3.4 3. 3.5 to 4.4 4. ≥4.5	% Energy	1. Referent 2. -0.029 3. -0.03 4. -0.027	1. N/A 2. 0.31 3. 0.45 4. 0.42	0.33	As above

	and brownies)

Body weight and BMI and whole grains

Summary of cohort results in children

The literature search found no cohort studies in adults investigating wholegrain intake and body weight or BMI. Only one cohort study in children was found: the DONALD Study (Cheng *et al.*, 2009) (see Table 5.17). No association was found between total whole grain foods and change in BMI SD score in this study, adjusting for several appropriate covariates.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Data from one intervention, the Women's Health Initiative Dietary Modification Trial are tabulated in Tables 5.18 and 5.19 using two publications with different follow-up points (Howard *et al.*, 2006;Tinker *et al.*, 2008). Details concerning the design, participants, duration and nature of the intervention are included in Table 5.4.

The Women's Health Initiative Randomized Controlled Dietary Modification Trial was designed to test the hypothesis that a low fat, high fruit and vegetable (F&V), high grain diet would reduce the risk of cardiovascular disease in middle-aged and older women. The goal of the dietary intervention was to decrease total fat to 20% of energy intake, to increase F&V portions to 5 or more per day and to increase servings of grains to a minimum of 6 per day. Changes in outcome may therefore not be attributed solely to the increase in whole grain intake. There were no weight loss or energy restriction goals for either group.

Dietary change was implemented through a behavioural modification program that ran intensively throughout the first year of the trial and then less intensively thereafter. At the year 1 assessment, wholegrain consumption in the intervention group had increased by one third of a serving, whilst the comparison group remained unchanged (Tinker *et al.*, 2008).

The intervention group experienced greater weight loss than the control group at 1 year (difference between groups 1.9kg, $p<0.001$) and maintained some of this weight loss difference at 7.5 years (difference between groups of 0.6 kg after 7.5 years ($p<0.001$).

Similarly, mean BMI was lower at 1 year and at 7.5 years in the intervention group compared with the control group (both time points $p<0.001$). Actual body weight and BMI changes in this study were small, but this was a very large trial and 27% of the women had an initial BMI less than 25kg/m^2. These data provide some evidence that a lower fat, higher fruit, vegetable and whole grain diet may be helpful in the prevention of weight gain in the long term, at least in postmenopausal women.

Table 5.17 BMI and whole grains: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)	p	Adjustments
14007 (Cheng *et al.*, 2009) The DONALD Study	Germany	(9) %M 46	376	4 years	Food diary	Total wholegrain foods (Amaranth, whole buckwheat, bulgar, spelt, spelt flakes, barley flakes, whole barley, unripe spelt, whole and rolled oats, oat flakes, oat flour, millet, millet flakes, whole kamut wheat, corn bran, unpolished rice, popcorn, puffed rice, puffed wheat, puffed spelt, whole rice flour, rice flakes, wholemeal rye, rye flour, whole triticale, whole wheat, wheat germ, wheat bran, whole-wheat flakes, and whole-wheat flour)	Change in BMI SD score Measured by clinician/ professional	1 g/day	0.005 (0.008)	0.5	Age, Breastfed, energy intake, GI, Parental overweight, Gender, Birth year

Table 5.18 Body weight and whole grains: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
16240 (Howard *et al.*, 2006)	Control	25056/29294	76.7 (SD 16.5)	76.1 (SD 16.9)	-0.1 (SD 10.1)		Body weight	Measured by clinician/ professional (kg)	7.5 years	No change	No bias
	Low fat diet	16297/19541	76.8 (SD 16.6)	75.7 (SD 17.1)	-0.8 (SD 10.1)	<0.001				Decrease	
15354 (Tinker *et al.*, 2008)	Control	24977/29294	76.2 (SD 16.3)	75.9 (SD 16.5)			Body weight	Assessment method not reported (kg)	1 year	No change	unclear
	Low fat diet	17026/19541	76.4 (SD 16.5)	74 (SD 16.5)		0.001				Decrease	

Table 5.19 BMI and whole grains: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
16241 (Howard *et al.*, 2006)	Control	24943/29294	29.1 (SD 5.9)	29.2 (SD 5.9)	0.3 (SD 3.1)		BMI	Measured by clinician/ professional	7.5 years	No change	No bias
	Low fat diet	16230/19541	29.1 (SD 5.9)	29.0 (SD 6.1)	0.003 (SD 3.2)	<0.001				Decrease	
15363 (Tinker *et al.*, 2008)	Control	24977/29294	28.9 (SD 5.8)	28.7 (SD 5.7)			BMI	Assessment method not reported	1 year	No change	unclear
	Low fat diet	17026/19541	28.9 (SD 5.8)	28 (SD 5.8)		0.001				Decrease	

Body weight and BMI and glycaemic index and load

Summary of cohort results in adults

Two publications from two cohort studies: the MONICA study and the EPIC study reported change in body weight during follow-up in relation to baseline dietary glycaemic index (GI) (Hare-Bruun *et al.*, 2006;Du *et al.*, 2009) The MONICA study also investigated body weight change in relation to glycaemic load (GL) (Hare-Bruun *et al.*, 2006) (see Table 5.20).

Anthropometry was professionally measured in the MONICA study, with a combination of direct measures and self-reported data in the EPIC study. The EPIC study (Du *et al.*, 2009) reported a beta coefficient suggestive of increases in weight over time with each additional 10 GI units, however confidence intervals were wide and the beta coefficient was small. Statistical significance was not reported separately by subgroup for the quintile comparisons in the MONICA study (Hare-Bruun *et al.*, 2006), however none of the p values for men and women combined indicated statistical significance. Only one of the beta coefficients in the MONICA study (Hare-Bruun *et al.*, 2006) revealed a statistically significant result, indicative of greater weight change with increasing GI for women, but not men. No statistically significant differences in body weight change were found between high GL and low GL quintiles.

Exposure definition and assessment

The glycaemic index is a relative measure of the plasma glucose response induced by a specific food, as compared to the response induced by the same amount of carbohydrate from a reference source, such as white bread or pure glucose (Liu *et al.*, 2000). Similarly, the glycaemic load is the product of a specific food's GI and its carbohydrate content (Liu *et al.*, 2000), therefore taking into account both the quality and quantity of carbohydrate consumed. This may be interpreted as a measure of diet-induced insulin demand (Stevens *et al.*, 2002).

Intake was assessed using a dietary history in the MONICA study (Hare-Bruun *et al.*, 2006) and FFQ in EPIC (Du *et al.*, 2009). The studies used different reference sources to calculate GI values: white bread in the MONICA study (Hare-Bruun *et al.*, 2006) and glucose was used for the EPIC study (Du *et al.*, 2009).

Dietary GI and GL were calculated by summing the products of the GI for each food multiplied by its carbohydrate content per serving multiplied by the average number of servings of that food per day (to give dietary GL), then dividing by the average daily carbohydrate intake to give dietary GI:

Dietary GI = {∑[(servings of food per day) x (CHO content) x GI)]}/total CHO (Meyer *et al.*, 2000).

The glycaemic index (and thus also GL) is determined not only by the nature of the carbohydrate component of a food or diet, but also by the types and amounts of protein, fat and dietary fibre, as well food processing and storage (Venn and Green, 2007). Unless tightly controlled in an experimental situation, in most cases high and low GI/GL diets differ in many ways other than the carbohydrate fraction, including dietary fibre content, energy density and sensory quality.

Adjustment for appropriate confounders

Both studies included important covariates in their adjustments.

Summary of cohort results in children

One cohort study in children, the DONALD study (Cheng *et al.*, 2009), reported the change in BMI SD score in relation to both glycaemic index and glycaemic load. GI and GL were calculated from food diaries, using glucose as the reference food. None of the associations achieved statistical significance. All analyses were adjusted for appropriate confounders, including age, gender, energy intake, fibre intake, parental overweight and whether breastfed (Table 5.21).

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Five studies provided data on the effects of diets high or low in glycaemic index or load on body weight, and four of these provided sufficient information to be included in a meta-analysis. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.22. Data on body weight change in the study by Ebbeling *et al.* (Ebbeling *et al.*, 2007) were published only in a figure, from which it was inappropriate to extract numerical values, and so this could not be included in the meta-analysis. This study compared the effects of a low fat (20% energy), high carbohydrate (55% energy) diet with a moderate fat (35% energy), lower carbohydrate (40% energy, low glycaemic load) diet in 73 obese young adults (18-35 years) with a 6-month intensive intervention period and a 12-month follow-up period (group workshop sessions continued throughout, as did the diet). The low carbohydrate diet was not energy-restricted, but the high carbohydrate diet was designed to create an energy deficit in the region of 250-500 kcal per day. However, there was no difference in body weights in the two diet groups at the 6, 12 and 18-months follow-up points ($p=0.99$ for the group x time interaction). Overall, both groups lost weight equally. However, there were some differences between subjects, with those assessed as having an elevated post-load insulin concentration being more responsive to the low carbohydrate/low GL diet. This group of participants lost more weight initially, and over the full course of the study (-5.8kg vs. -1.2kg for low vs. high carbohydrate diets, at 18 months $p<0.04$). The low insulin responders were not differentially affected by the carbohydrate content of the diets.

Four studies providing dietary differences in glycaemic index or glycaemic load between groups were included in a meta-analysis (Carels *et al.*, 2005;Das *et al.*, 2007;Ebbeling *et al.*, 2005;Sichieri *et al.*, 2007a). All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from 12 to 18 months.

The study by Carels *et al.* (Carels *et al.*, 2005) was a comparison between the LEARN weight loss program (low energy and fat diet with enhanced physical activity) with and without low GI education. The difference in average dietary glycaemic index and load between the two groups was small, and total carbohydrate and energy intakes were similar (GL in higher vs. lower group respectively 70.9 g/1000kcal and 67.0 g/1000kcal). The study by Ebbeling *et al.* (Ebbeling *et al.*, 2005) compared an *ad libitum* low GL diet (53g/1000 kcal) with an energy restricted, higher GL diet (77g/1000 kcal). Reported energy and fibre intakes were similar. The CALERIE study (Das *et al.*, 2007) and the study reported by Sichieri *et al.* (Sichieri *et al.*, 2007a) both compared high GI/GL and low GI/GL diets that were equally energy restricted.

The overall pooled estimate indicated that body weight was 0.08kg (95% CI, -0.96 to 1.13) higher with consumption of a high dietary glycaemic index or load diet but this was not significantly different from zero (p=0.88) (Figure 5.5). Heterogeneity denoted by I^2 was 0% (95% CI, 0 to 8). Statistically, there was no evidence of a difference in body weight with differences in dietary glycaemic index or glycaemic load.

Figure 5.5 Forest plot for high glycaemic index or glycaemic load diets and body weight (kg)

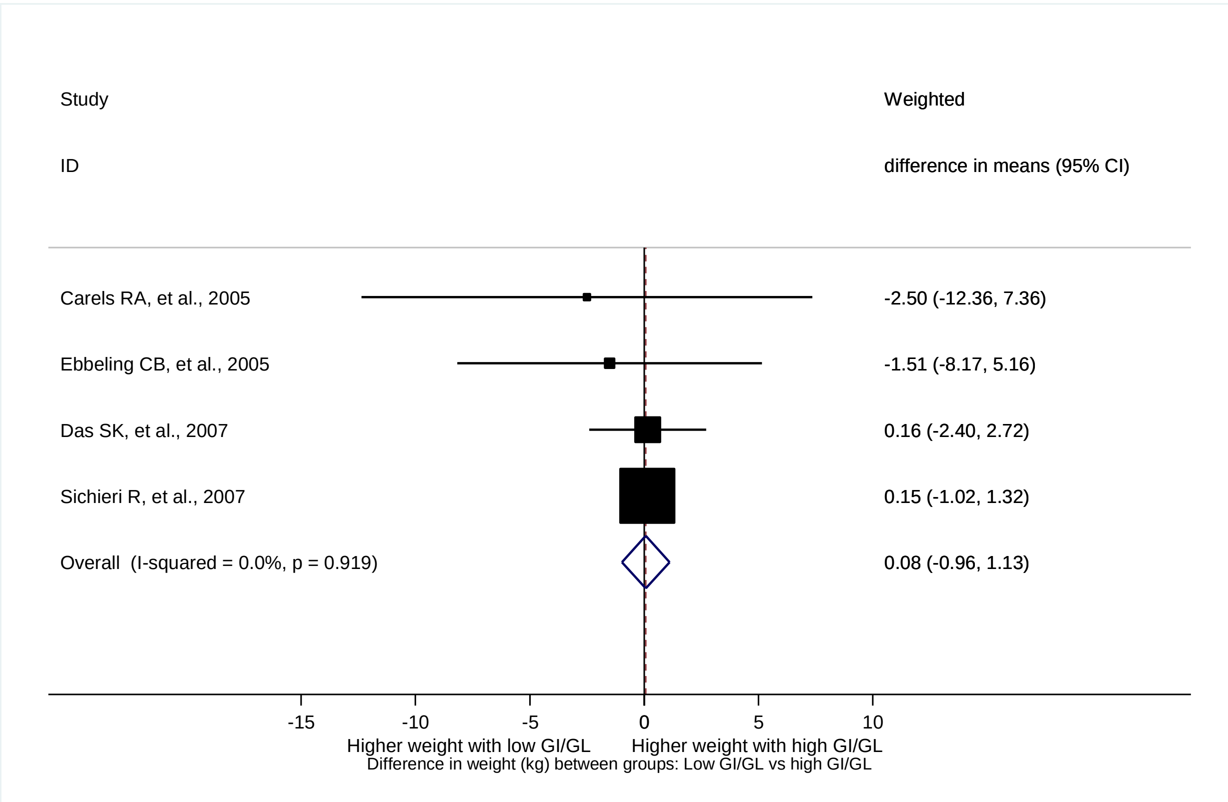


Table 5.20 Body weight and glycaemic index and load: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	Adjustments
13657 (Du *et al.*, 2009) EPIC Denmark, Germany, Italy, The Netherlands and UK	Europe, Cancer free, No heart disease, Not diabetic	20-78 (53) %M 40	146543	6.5 years (30)	FFQ	Glycaemic index	Body weight change (g) Both self reported and direct measures			10 Units/ day		84 (-5, 172)		Age, Alcohol, Baseline weight, carbohydrate intake, Smoking, Education, fat intake, Fibre, Height, Menopausal Status, Physical activity, Protein, Gender
13825 (Hare-Bruun *et al.*, 2006) MONICA	Denmark, Primarily White, Not diabetic	30-60 (45) %M 48.9	552	6 years	Dietary history	Glycaemic load	Change in weight (kg) Baseline to Follow-up Measured by clinician/ professional	Men	Q5 vs Q1		Mean: 2.2 SD: 4.6 vs.Mean: 1.8 SD: 6.6			
13829 MONICA						Glycaemic load		Women	Q5 vs Q1		Mean: 3.4 SD: 4.4 vs.Mean: 3.2 SD: 5.2			
13807 MONICA						Glycaemic index		Men	Q5 vs Q1		Mean: 1.9 SD: 4.5 vs.Mean: 1.9 SD: 5.7			
13821 MONICA						Glycaemic index		Women	Q5 vs Q1		Mean: 2.2 SD: 6 vs.Mean: 1.9 SD: 5.9			
13833 MONICA						Glycaemic index		Men		1 Unit		-0.0002 (-0.002, 0.002)	NS	Age, Weight, Education, fat intake, Protein, energy intake, Fibre, physical activity, Smoking
13837 MONICA						Glycaemic index		Women		1 Unit		0.002 (0.0001, 0.004)	<0.05	As above

Table 5.21 BMI and glycaemic index and load: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14005 (Cheng *et al.*, 2009) The DONALD Study	Germany	(9) %M46	376	4 years	Food diary	Glycaemic load	Change in BMI SD score Measured by clinician/ professional		1 g/day	-0.017 (0.01)	0.08	Age, Breastfed, energy intake, Fibre, Parental overweight, Gender, Birth year
14004 The DONALD Study						Glycaemic index	Change in BMI SD score Measured by clinician/ professional		1 Unit/day	-0.014 (0.01)	0.2	As above
14012 The DONALD Study						Glycaemic index	Change in BMI SD score Measured by clinician/ professional	BMI <25	1 Unit/day	-0.017 (0.011)	0.1	As above
14017 The DONALD Study						Glycaemic index	Change in BMI SD score Measured by clinician/ professional	BMI >25	1 Unit/day	-0.0001 (0.021)	1	As above

Table 5.22 Body weight and BMI and glycaemic index and load: RCT data

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
*14908 (Carels *et al.*, 2005)		Weight loss program	~19/~26	104.8 (SD 21.2)	96.6 (SD 15.9)			NS	Body Weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Weight loss program + low GI education	~19/~26	101.2 (SD 16.3)	94.1 (SD 15.3)							Decrease	
14909		Weight loss program	~19/~26	37.2 (SD 5.1)	34.4 (SD 4.3)			NS	**BMI**	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Weight loss program + low GI education	~19/~26	38.0 (SD 13.4)	35.5 (SD 13.4)							Decrease	
*15224 (Das *et al.*, 2007)		Energy restricted high GL diet	15/17	78.5 (SD 12.3)		-8% (SD 4.1%)			Body weight	Measured by clinician/ professional (kg)	1 year	Decrease	No bias
		Energy restricted low GL diet	14/17	78 (SD 9.3)		-7.8% (SD 5%)		NS				Decrease	

Result ID/ Author	Subgroup detail	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15464 (Ebbeling *et al.*, 2007)		Low fat diet	ITT: 37/37				<0.05		Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	No bias
		Low GL diet	ITT: 36/36				<0.05	NS				Decrease	
15465		Low fat diet	ITT: 37/37						Body weight change	Measured by clinician/ professional (kg)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36					NS				Decrease	
15467	Insulin (30m post 75gdose) <57.5µUI/ml	Low fat diet	ITT: 37/37				<0.05		Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	No bias
		Low GL diet	ITT: 36/36				NS	NS				Decrease	
15468	Insulin (30m post 75gdose) <57.5µUI/ml	Low fat diet	ITT: 37/37				NS		Body weight change	Measured by clinician/ professional (kg)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36				NS	NS				Decrease	
15470	Insulin (30m post 75gdose) >57.5µUI/ml	Low fat diet	ITT: 37/37		No loss		NS		Body weight change	Measured by clinician/ professional (kg)	1 year	Decrease	No bias
		Low GL diet	ITT: 36/36		Greater loss		<0.05	NS				Decrease	
15471	Insulin (30m post 75gdose) >57.5µUI/ml	Low fat diet	ITT: 37/37			-1.2	NS		Body weight change	Measured by clinician/ professional (kg)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-5.8	<0.05	0.004				Decrease	
*15420 (Ebbeling *et al.*, 2005)		Low fat diet	12/17	83.2 (SE 3.3)		-6.1% (CI -11.2, -0.7)			Body weight	Measured by clinician/ professional (kg)	1 year	Decrease	Unclear
		Low GI diet	11/17	93.9 (SE 5.3)		-7.8% (CI -13, -2.2)						Decrease	
15775 (Sichieri *et al.*, 2007a)		High GI/GL diet	46/102			-1.25 (SD 3.2)			Body weight change	Measured by clinician/ professional (kg)	1 year	No change	Unclear
		Low GI/GL diet	44/101			-1.0 (SD 2.4)						No change	
15776		High GI/GL diet	44/102			-0.95 (SD 3.2)			Body weight change	Measured by clinician/ professional (kg)	15 months	No change	Unclear
		Low GI/GL diet	49/101			-1.04 (SD 3)						No change	
*15777		High GI/GL diet	60/102			-0.26 (SD 3.6)			Body weight change	Measured by clinician/ professional (kg)	18 months	No change	Unclear
		Low GI/GL diet	63/101			-0.41 (SD 2.9)						No change	

*This result was used in the meta-analysis for glycaemic index or glycaemic load diets and body weight change (Figure 5.5)

Body weight or BMI and sweetened beverages

[Fruit juice data were only extracted from cohort studies which were already captured in the review. In accord with our original protocol, a specific search for studies reporting fruit juice consumption has not been undertaken.]

Summary of cohort results in adults

Results from one publication concerning the Black Women's Health Study (Palmer *et al.*, 2008), were extracted (see Table 5.23). Beverage intake was measured by FFQ, and body weight change was self-reported. The results of this study are difficult to interpret: firstly, no statistical comparisons were performed; and secondly, group means refer to weight gain only. Weight decrease was also observed at follow-up, however, whilst the proportion of the cohort reporting weight loss is given, figures for mean weight lost are missing.

Summary of cohort results in children

Data were extracted from six publications presenting results from the following six cohort studies (Kvaavik *et al.*, 2005;Nissinen *et al.*, 2009;Libuda *et al.*, 2008;Striegel-Moore *et al.*, 2006;Phillips *et al.*, 2004;Fiorito *et al.*, 2009). Clinic measures of weight and height were used to estimate BMI in all studies except the Oslo Youth Study (Kvaavik *et al.*, 2005) which relied upon self-reported data. Three European cohort studies reported no association between sweetened beverage consumption and BMI (Kvaavik *et al.*, 2005;Nissinen *et al.*, 2009;Libuda *et al.*, 2008). Three US cohorts reported associations that indicate increasing BMI with increasing consumption of sweetened beverages.

The National Heart, Lung, and Blood Institute Growth and Health Study (Striegel-Moore *et al.*, 2006) found that in a large cohort of black and white US girls, BMI increased by 0.01 unit for every 100 g of regular soda consumed. The MIT Growth and Development Study (Phillips *et al.*, 2004) also showed a statistically significant increase in BMI z-score with increasing intake of soda (sugar-sweetened only). The Pennsylvania Study of Health and Development of Young Girls (Fiorito *et al.*, 2009) also reported a statistically significant relationship between sugar-sweetened beverage consumption at age five and difference in BMI-for-age-percentile at ages 5-15 years (see Table 5.24). Girls drinking two or more servings per day (serving defined as 8oz) at age 5 years had higher BMI-for-age-percentiles at all subsequent ages compared with lower consumers ($p<0.05$).

The DONALD study, National Heart, Lung and Blood Institute Growth and Health study and the Pennsylvania Study of Health and Development of Young Girls (Libuda *et al.*, 2008;Striegel-Moore *et al.*, 2006;Fiorito *et al.*, 2009) included fruit juices in their study. None of these found a statistically significant association between baseline fruit juice consumption and BMI at follow-up.

Collectively, these studies provide conflicting evidence concerning the relationship between sweetened beverages and BMI, with the US studies tending to find small but positive associations and the European studies tending to report no evidence of a statistical association.

Exposure definition and assessment

Dietary data were collected by diet recall (Fiorito *et al.*, 2009), food diaries (Libuda *et al.*, 2008;Striegel-Moore *et al.*, 2006) and FFQ (Nissinen *et al.*, 2009;Phillips *et al.*, 2004). One study assessed intake using a general questionnaire (Kvaavik *et al.*, 2005). One study distinguished between 'soda' and 'fruit drinks' (Striegel-Moore *et al.*, 2006); others considered all forms of sugar-containing beverages together.

Adjustment for appropriate confounders

Each study included a different set of covariates in their analyses. The DONALD study (Libuda *et al.*, 2008) adjusted for more confounders than the other studies.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

One UK study of primary school children provided data on carbonated beverage consumption and body mass index (see Table 5.25). The cluster randomised controlled trial reported by James *et al.* (James *et al.*, 2004) recorded both BMI and BMI z-score at baseline and one year after an intervention study which aimed to assess the effects of an educational programme designed to reduce the consumption of carbonated beverages in children. A total of 644 children aged 7-11 years were randomised to intervention or control groups. Drinks diaries completed over 3 days revealed that carbonated drinks consumption decreased by 0.6 glasses in the intervention group (half of these were carbonated drinks with sugar) but increased by 0.2 glasses in the control group (mean difference 0.7, 95% confidence interval 0.1 to 1.3). *nb. carbonated beverages here included all types of carbonated beverage, whether non-calorically- or sugar-sweetened.*

The intervention and control group children increased BMI and z-score similarly, with no significant between-group difference in the changes. However, interpretation of this study with regard to the effects of carbohydrate on body weight change is hampered by the fact that the intervention concerned carbonated beverages, which were not exclusively sugar-sweetened. Additionally, the relatively low number of children consenting to participate (36% of children provided drinks diaries at the start and end) and subsequent drop-out of approximately 10% of children means that the study population is not necessarily representative of UK school children in general. A follow up 2 years later with data from 434 of the original 644 children found no differences between the groups in terms of the prevalence of overweight(James *et al.*, 2007).

Table 5.23 Body weight and sweetened beverages: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Contrast (mean)	Exposure Units	Mean outcome (SD)	Adjustments
14319 (Palmer *et al.*, 2008) Black Women's Health Study	USA, Black, Cancer free, No heart disease, Not diabetic	21-69 %M 0	59000	10 years (20)	FFQ (68)	Full-calorie sugar sweetened beverages (fruit drinks, including fortified fruit drinks, kool-aid and fruit juice other than orange and grapefruit)	Body weight change (kg) Self-reported	1. Increasers: ≤1/wk to ≥1/d 2. Maintain High: ≥1/d to ≥1/d 3. Maintain Low: ≤1/wk to ≤1/wk 4. Decreases: ≥1/d to ≤1/d 5. All others	Servings (336g)/ day	Weight gain 1. 5.4 (0.26) 2. 5.5 (0.14) 3. 5.2 (0.10) 4. 4.6 (0.15) 5. 5.3 (0.07)	

Table 5.24 BMI and sweetened beverages: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14210 (Fiorito *et al.*, 2009) Pennsylvania Study of Health and Development of Young girls	USA, Primarily White, Age 5-18y	(5) %M 0	170	10 years (15)	Dietary recall	Mixed sugar and non-calorically sweetened beverage consumption at age 5 yr	BMI centile Measured by clinician/ professional	Age 9	1.<1 2.>=1<2 3.>2	Servings/ day	1. 60.8 (27.3) 2. 63 (26.8) 3. 77.1 (22.4)		Significant main effect of beverage frequency group p<0.05		
14214 Pennsylvania Study of Health and Development of Young girls							BMI centile	Age 11	1.<1 2.>=1<2 3.>2	Servings/ day	1. 60.1 (27) 2. 63.6 (29.4) 3. 75.4 (22.5)				
14215 Pennsylvania Study of Health and Development of Young girls							BMI centile	Age 13	1.<1 2.>=1<2 3.>2	Servings/ day	1. 60.1 (26.2) 2. 62.4 (25.3) 3. 70.6 (26.9)				

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14216 Pennsylvania Study of Health and Development of Young girls	USA, Primarily White, Age 5-18y	(5) %M 0	170	10 years (15)	Dietary recall	Mixed sugar and artificial sweetener beverages	BMI centile-age 15		1.<1 2.>=1<2 3.>2	Servings/ day	1. 60.3 (25.5) 2. 60.4 (23.9) 3. 66.6 (25.2)				
14524 (Kvaavik *et al.*, 2005) Oslo Youth Study	Norway, Ethnicity unknown	(13) %M 49	1086	19 years (15.7)	Question-naire (general)	Full-calorie sugar-sweetened beverages (High consumers: reported >3 times/wk (1991) and 4 times/wk (1999) Low consumers: <2 times/wk (1991) and <3 times/wk (1999). Inconsistent: changing consumption frequency)	BMI Self-reported	Female	1: Long-term low consumer 2: Inconsistent consumer 3: Long-term High Consumer		BMI 1: 23.4 (4.1) 2: 23 (3.9) 3: 24 (5.3)			0.627	
14525 Oslo Youth Study								Male			BMI 1: 25.6 (3.5) 2: 25.9 (4.2) 3: 25.3 (4.2)			0.685	
13413 (Libuda *et al.*, 2008) The DONALD Study	Germany, Age 11-18y	9-18 (12) %M 51	1170	5 years	Food diary	Energy from regular soft drinks at baseline (carbonated and non-carbonated sugar sweetened drinks such as lemonades, iced tea and both diluted and sugar sweetened fruit juice drinks)	BMI SDS Measured by clinician/ professional	Male		1 MJ/day		0.037	0.211		Age, Birthweight, EI, Maternal BMI, Maternal Education, Assessment period, Years of adolescence
13605 The DONALD Study								Female		1 MJ/day		0.005	0.877		As above
17589 The DONALD Study						Energy from 100% fruit juice at baseline		Male		1 MJ/day		0.033	0.310		As above
17594 The DONALD Study						Energy from 100% fruit juice at baseline		Female		1 MJ/day		-0.046	0.161		As above
14632 (Nissinen *et al.*, 2009) Cardiovascular Risk in Young Finns Study	Finland, Primarily White	3-18 (11) %M 45.2	3596	21 years (33)	FFQ (19)	Full-calorie sugar sweetened beverages	BMI Measured by clinician/ professional	Male		10 Units/ month		0.01 (0.1)	0.51		Age, Weight, Parental education

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14096 (Phillips *et al.*, 2004) Massachusetts Institute of Technology Growth and Development Study	USA, Primarily White, BMI <30, Generally healthy	8-12 %M 0	196	6.9 years (28)	FFQ (116)	Percent of calories from soda (sugar sweetened only)	BMI z-score Measured by clinician/ professional		1. <0.74 2. 0.75 to 1.4 3. 1.5 to 3.1 4. ≥3.2	% Energy		1. Referent 2. 0.089 3. 0.172 4. 0.178	1. N/A 2. 0.03 3. <0.001 4. 0.001	<0.001	Age at menarche, Fruit and veg, Parental Overweight
14634 Cardiovascular Risk in Young Finns Study								Female		10 Units/ month		-0.16 (0.14)	0.24		Age, Weight, Parental education
13799 (Striegel-Moore *et al.*, 2006) National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	10 years (10)	Food diary	'Regular soda' (all non-diet carbonated beverages except water)	BMI Measured by clinician/ professional			100 g/day		0.011 (0.005)	<0.05		Energy intake, Ethnicity, Other beverage, Study area, Visit
17583 National Heart, Lung, and Blood Institute Growth and Health Study						'Fruit drinks' (fruit-flavoured drinks, punches and ades that contain <100% juice)				100 g/day		0.009 (0.007)	NS		As above
17582 National Heart, Lung, and Blood Institute Growth and Health Study						'Fruit juice' (fruit and vegetable juices)				100 g/day		0.005 (0.007)	NS		As above

Table 5.25 BMI and sweetened beverages: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
Children study											
14911 (James *et al.*, 2004)	Control minus reduced carbonated beverages	Control: 279/319 Intervention: 295/325				0.1 (CI -0.1, 0.3), NS	BMI	Measured by clinician/ professional	1 year	Increase in both	unclear
14912	Control	279/319	17.6 (SD 0.7)	18.3 (SD 0.8)	0.8 (SD 0.3)		BMI	Measured by clinician/ professional	1 year	Increase	unclear
	Reduced carbonated beverages	295/325	17.4 (SD 0.6)	17.9 (SD 0.7)	0.7 (SD 0.2)					Increase	
14931	Control minus reduced carbonated beverages	Control: 279/319 Intervention: 295/325				0.04 (CI -0.04, 0.12), NS	BMI z-score	Measured by clinician/ professional	1 year	Increase in both	unclear
14932	Control	279/319	0.47 (SD 0.2)	0.6 (SD 0.19)	0.08 (SD 0.13)		BMI z-score	Measured by clinician/ professional	1 year	Increase	unclear
	Reduced carbonated beverages	295/325	0.5 (SD 0.23)	0.48 (SD 0.23)	0.04 (SD 0.07)					Increase	

Categorical Body Weight-Related Outcomes

Studies in this section of the chapter provide obesity-related data in the form of categorical variables such as risk of major weight gain, weight gain above a certain threshold, or incident obesity. Additionally, data have been reported as mean dietary intakes in subjects classified as weight gainers or non-gainers or in subjects above and below selected cut-points indicative of obesity e.g. >85th or 90th BMI centile.

Weight gain and total carbohydrate intake and high carbohydrate diets

Summary of cohort results in adults

Data were extracted from two publications (Table 5.26), presenting findings from two cohort studies: the San Antonio Heart Study and the Finnish Mobile Clinic Health Surveys conducted in the USA and Finland respectively (Haffner *et al.*, 1991;Rissanen *et al.*, 1991). No statistically significant differences in percentage energy from carbohydrate or total carbohydrate intake were observed between weight gainers and non-gainers in the San Antonio Heart Study of Mexican Americans and non-Hispanic whites. Findings from the Finnish Mobile Clinic Health Survey indicate a significantly increased risk of weight gain over 5.7 years for women in the highest quintile of carbohydrate consumption compared with women in the lowest quintile. However, this estimate is age-adjusted only, and no data are provided concerning the range of carbohydrate intakes in each quintile. No such association was reported for men in this study.

Incident weight gain was defined differently in each study: ≥ 5kg/5year in the Finnish Mobile Clinic Health Survey; and ≥ 6.8kg in the San Antonio Heart Study. In both cohort studies body weight was measured by research staff.

Exposure definition and assessment

The San Antonio Heart Study measured carbohydrate intake as both per cent energy and grams per day using dietary recall data (Haffner *et al.*, 1991); the Finnish Mobile Clinic Health Survey as per cent energy only (derived from dietary history) (Rissanen *et al.*, 1991).

Adjustment for appropriate confounders

Both cohorts adjusted for age only.

Summary of cohort results in children

No cohort studies of children reported categorical outcomes in response to carbohydrate consumption.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

One randomised controlled trial assessed the percentage of participants that gained more than 5kg or lost more than 10kg over the course of a 2 year low fat trial (Sheppard *et al.*, 1991). The women in this study (which was a feasibility study for the Women's Health Intervention Dietary Modification Trial) were randomised to either a low fat diet or to continue with their usual diet. The low fat group participants were instructed to reduce their fat intake to less than 20% of energy, replacing fat sources of energy with non-fat energy (carbohydrate energy percent increased by 15% in the low fat group, and by 2.9% in the control group over the duration of the trial). There was no energy restriction imperative and the participants were not markedly overweight, although both groups reported lower energy intakes at 1 and 2 years after baseline (-1653 kJ and -448 kJ in low fat and control groups, at 2 years).

At the 2 year follow-up, the intervention women (n=158) weighed 1.9kg less than at baseline, and the control women (n=94) weighed 0.1kg less. A higher percentage of the women in the low fat/high carbohydrate group experienced large changes (decreases) in body weight (10kg loss) compared with the control group (see Table 5.27). The statistical significance of the differences between groups was not reported. Results should be treated with caution, however, as there is likely to be severe under-reporting in this study.

Table 5.26 Weight gain and total carbohydrate: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	RR (CI)	Mean exposure (SD)	p	Adjust-ments
14068 (Haffner *et al.*, 1991) San Antonio Heart Study	USA	25-64 %M 42.3	(73) /2217	8 years	Dietary recall	Carbohydrate, total (% energy)	Weight gain ≥6.8 kg Measured by clinician/ professional	Mexican-American Men		1 % Energy		Gainers: (n: 73) 40.5 (12.2) Non-gainers: (n: 257) 41.3 (10.4)	0.45	Age
14072 San Antonio Heart Study			(35) /2217					Non-Hispanic White Men		1 % Energy		Gainers: (n: 35) 41.3 (12) Non-gainers: (n: 155) 39.3 (11.5)	0.36	Age
14077 San Antonio Heart Study			(127) /2217					Mexican-American Women		1 % Energy		Gainers: (n: 127) 44.6 (9.4) Non-gainers: (n: 322) 44.2 (11.5)	0.64	Age
14081 San Antonio Heart Study			(65) /2217					Non-Hispanic White Women		1 % Energy		Gainers: (n: 65) 39.8 (11.1) Non-gainers: (n: 195) 42 (10.6)	0.30	Age
14069 San Antonio Heart Study	USA	25-64 %M 42.3	(73) /2217	8 years	Dietary recall	Carbohydrate, total (grams/day)	Weight gain ≥6.8 kg Measured by clinician/ professional	Mexican-American Men		g/day		Gainers: (n: 73) 247 (124.1) Non-gainers: (n: 257) 245.2 (102.4)	0.52	Age
14073 San Antonio Heart Study			(35) /2217					Non-Hispanic White Men		g/day		Gainers: (n: 35) 241.9 (121.3) Non-gainers: (n: 155) 232.8 (112.9)	0.92	Age
14078 San Antonio Heart Study			(127) /2217					Mexican-American Women		g/day		Gainers: (n: 127) 185.6 (89.9) Non-gainers: (n: 322) 183.9 (82.2)	0.38	Age
14082 San Antonio Heart Study			(65) /2217					Non-Hispanic White Women		g/day		Gainers: (n: 65) 160.2 (83.6) Non-gainers: (n: 195) 170.9 (80.7)	0.38	Age
13143 (Rissanen *et al.*, 1991) Finnish Mobile Clinic Health Surveys	Finland	30-64 %M 48	(Number of cases not reported) /6102	5.7 years (7)	Dietary history	Carbohydrate, total (% energy)	Weight gain ≥ 5kg Measured by clinician/ professional	Women	Q5 vs Q1		1.7 (1, 2.6)			Age
13144 Finnish Mobile Clinic Health Surveys							Weight gain ≥ 5kg Measured by clinician/ professional	Men	Q5 vs Q1		Carbohydrate intake did not predict weight change in men (data were not reported)			Age

Table 5.27 Weight gain and loss and high carbohydrate diets: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Outcome at follow-up	Outcome/Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15717 (Sheppard *et al.*, 1991)	Control	105/119	3% of participants	weight gain ≥ 5kg	Weight measured by clinician/ professional	1 year	No change	Unclear
	Low fat diet	171/184	5% of participants				Decrease	
15715	Control	105/119	2% of participants	weight loss ≥ 10kg	Weight measured by clinician/ professional	1 year	No change	Unclear
	Low fat diet	171/184	8% of participants				Decrease	

Weight regain and carbohydrate supplement

No cohort studies reported results concerning carbohydrate supplements and weight regain.

Summary of RCT data

One small randomised double blind trial, explored the effects of a 50g carbohydrate supplement (42% glucose, 58% maltodextrins) compared with a control (do nothing) intervention on weight regain expressed as percentage of participants that regained more than 50% of the weight lost in an initial very low energy diet (VLED) weight loss phase (Pasman *et al.*, 1997b). A further group was also followed which consumed a combination supplement of carbohydrate, chromium-picolinate, caffeine and soluble fibre. These data were not extracted. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.28. The average amount of weight regain was 62% in subjects in the carbohydrate supplement group, compared with 86% in the control group. Thirty six percent of the carbohydrate group compared with 21% of the controls regained less than 50% of the weight lost earlier. Neither of these outcome differences were statistically different.

nb. while the authors claim the study was double-blind, it is unclear how this was achieved without a placebo product.

Table 5.28 Weight regain and carbohydrate supplement: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Within group Δ from baseline	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15500 (Pasman *et al.*, 1997b)	Carbohydrate	11/10	61.8 (SD 55.2)	Amount of weight regain (post VLCD) %	Measured by clinician/ professional	14 months	Increase	Unclear
	Control	9/9	85.5 (SD 55.8)				Increase	
15501	Carbohydrate	11/10	36% of participants	Weight regain <50% (of lost weight)	Measured by clinician/ professional	14 months	Increase	Unclear
	Control	9/9	21% of participants				Increase	

Weight gain and sucrose

Summary of cohort results in adults

Data were extracted from one publication, reporting results from the San Antonio Heart Study (Haffner *et al.*, 1991). No statistically significant differences in terms of sucrose intake were detected between weight gainers and non-gainers in this study. Body weight was measured by a clinician, with incident weight gain defined as ≥ 6.8kg. Sucrose intake was estimated from 24-hour dietary recalls.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs reported data on sugars and weight gain expressed as a binary outcome.

Table 5.29 Weight gain and sucrose: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Mean exposure (SD)	p	Adjustments
14070 (Haffner *et al.*, 1991) San Antonio Heart Study	USA	25-64 %M 42.3	(73) /330	8 years	Dietary recall	Sucrose g/day	Weight gain ≥6.8 kg Measured by clinician/ professional	Mexican-American Men	Gainers: (n: 73) 8.41 (7.4) Non-gainers: (n: 257) 9.17 (6.72)	0.18	Age
14074 San Antonio Heart Study			(35) /190					Non-Hispanic White Men	Gainers: (n: 35) 8.86 (8.19) Non-gainers: (n: 155) 8.33 (6.62)	0.96	Age
14079 San Antonio Heart Study			(127) /449					Mexican-American Women	Gainers:(n: 127) 10.6 (7.2) Non-gainers: (n: 322) 10.6 (7.9)	0.72	Age
14083 San Antonio Heart Study			(65) /260					Non-Hispanic White Women	Gainers: (n: 65) 9.3 (7.4) Non-gainers: (n: 195) 9.7 (7.2)	0.61	Age

Weight gain and polysaccharides

Summary of cohort results in adults

Data were extracted from one publication, reporting results from the San Antonio Heart Study (Haffner *et al.*, 1991). No statistically significant differences in terms of starch intake were detected between weight gainers and non-gainers in this study. Body weight was measured by a clinician, with incident weight gain defined as ≥ 6.8kg. Starch intake was estimated from 24-hour dietary recalls.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs reported data on polysaccharides and weight change.

Table 5.30 Weight gain and polysaccharides: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Mean exposure (SD)	p	Adjust ments
14071 (Haffner *et al.*, 1991) San Antonio Heart Study	USA	25-64 %M 42.3	(73) /330	8 years	Dietary recall	Starch, total g/day	Weight gain ≥6.8 kg Measured by clinician/ professional	Mexican-American Men	Gainers: (n: 73) 10.4 (7.6) Non-gainers: (n: 257) 10.5 (6.5)	0.87	Age
14075 San Antonio Heart Study			(35) /190					Non-Hispanic White Men	Gainers:(n: 35) 6.03 (4.32) Non-gainers: (n: 155) 7.55 (5.82)	0.15	Age
14080 San Antonio Heart Study			(127) /449					Mexican-American Women	Gainers: (n: 127) 11 (7.1) Non-gainers: (n: 322) 11.4 (6.9)	0.82	Age
14084 San Antonio Heart Study			(65) /260					Non-Hispanic White Women	Gainers: (n: 65) 8 (5.8) Non-gainers: (n: 195) 7.9 (5.7)	0.69	Age

Weight gain, overweight and obesity and confectionery and desserts

Summary of cohort results in adults

The Penn Study of Ovarian Aging provided data on weight gain and added sugars, in the form of desserts and candy (Sammel *et al.*, 2003). Consumption of desserts and candy was lower in participants who gained 10lb (4.5 kg) or more compared with those who did not (p=0.015). The study was limited to pre-menopausal females only. Diet was measured by FFQ, and weight was measured rather than self-reported. The analyses adjusted for BMI at baseline only (table 5.31).

Summary of cohort results in children

Data were extracted from two publications, presenting results from two Scandinavian cohort studies (Nissinen *et al.*, 2009;Lissau *et al.*, 1993) (see Table 5.32). The Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009) reported relative risk of overweight (BMI>25) in relation to ‘sweets’ intake: confidence intervals for both male and female subgroups straddled 1, suggesting no evidence of an increased risk following increased ‘sweets’ intake. The Copenhagen Children’s Study (Lissau *et al.*, 1993) reported an odds ratio of 0.4 for risk of overweight in young adulthood (BMI>90th centile) with frequent reported consumption of ‘sweets’ (everyday or several times per week) compared with less than once per week, however this did not achieve statistical significance. Both studies used researcher measurements of height and weight, though incidence of overweight was defined differently in each.

Exposure definition and assessment

The Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009) assessed candy intake via FFQ, whilst the Copenhagen Children’s Study (Lissau *et al.*, 1993) ascertained consumption frequency by questionnaires administered to the participants’ mothers. The Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009) was concerned with change in intake, rather than absolute intake.

Drawing on these findings, it is important to note that the study methodologies are subject to limitations and more specifically, under-reporting. Poppitt *et al.* highlight that consumption of added sugar intakes is likely to be subject to under-reporting by participants (Poppitt *et al.*, 1998). Furthermore, confectionery and desserts, whilst being generally high in carbohydrate, may also be rich sources of fats and this means that it is difficult to draw conclusions concerning the role of carbohydrates in obesity from these studies. These results should therefore be interpreted cautiously.

Adjustment for appropriate confounders

Both studies appeared to adjust appropriately for confounders, including age, gender and BMI (where appropriate). The Copenhagen Children's Study (Lissau *et al.*, 1993) additionally adjusted for occupation in adulthood, and the Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009) for education, physical activity and smoking.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs reported data on added sugars and weight gain, overweight or obesity as a binary outcome.

Table 5.31 Weight gain and sweet foods including confectionery: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Mean exposure (SD)	p	Adjustments
13922 (Sammel *et al.*, 2003) Penn Study of Ovarian Aging	USA, Cancer free, Generally healthy, Not Alcoholics, Not diabetic, Pre-menopausal, Without liver cirrhosis	35-47 (41) %M 0	(85) /436	4 years	FFQ (25)	Sweet snack foods (desserts and candy)	Weight gain ≥10lb Measured by clinician/ professional	servings/day	Gainers: 0.9 (0.9) Non-gainers: 1.5 (2.3)	0.015	BMI category at baseline

Table 5.32 Risk of overweight and confectionery: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	RR (CI)	p	Adjustments
14351 (Lissau *et al.*, 1993) Copenhagen Childrens' Study	Denmark, Primarily White	9-10 %M Not reported	(35) /881	12 years	Questionnaire (general)	Sweets/ candy (Sweet-eating habits)	BMI>90th centile in young adulthood Measured by clinician/ professional		1. Daily 2. >1	times/ week	0.4	0.1	BMI, Occupation, Gender
14701 (Nissinen *et al.*, 2009) Cardiovascular Risk in Young Finns Study	Finland, Primarily White	3-18 (11) %M 45.2	(Number of cases not reported) /3596	21 years (33)	FFQ (19)	Sweets/ candy (change in intake)	BMI >25 Measured by clinician/ professional	Male	Increased intake vs Stable low intake		1.02 (0.71, 1.46)		Age, BMI, Education, physical activity, Smoking
14704 Cardiovascular Risk in Young Finns Study								Female	Increased intake vs Stable low intake		0.87 (0.62, 1.22)		Age, BMI, Education, physical activity, Smoking

Weight gain and total cereals or high cereal foods

Summary of cohort results in adults

Data were extracted from two publications, reporting results from two US cohort studies, one including only males (Bazzano *et al.*, 2005) and one only females (Sammel *et al.*, 2003). Data on breakfast cereal consumption and the broader ‘breads and cereals’ exposure category are included here (see Table 5.33). The Penn Study of Ovarian Aging (Sammel *et al.*, 2003) used clinician or professional measurements to categorise weight gain, however the Physicians’ Health Study I (PHS I), relied upon self-reports. Findings from the PHS I, at 13 years, (Bazzano *et al.*, 2005) are suggestive of a decreased risk of weight gain of 10kg or more for those who consume more than one serving of breakfast cereal per day as compared with those who rarely consumed breakfast cereal. Results were non-significant, however, when examining weight gain of 15kg or more. The type of breakfast cereal (whole vs. refined grain) had little impact on the point estimates. The Penn Study of Ovarian Aging (Sammel *et al.*, 2003) had a shorter follow-up of four years, and studied trends for total cereal consumption and weight gain of 10lb (4.5kg) or more. Differences between consumption of total cereal foods (including bread, breakfast cereals and salted snacks) in weight gainers and non-gainers were not found to be statistically significant.

These 2 cohort studies provide some evidence of a lower risk of significant weight gain with consumption of breakfast cereal, but no overall impact of total cereal foods.

Exposure definition and assessment

Both studies employed FFQs to collect dietary data.

Adjustment for appropriate confounders

The PHS I (Bazzano *et al.*, 2005) included a suitable range of covariates in the analyses. The Penn Study of Ovarian Aging (Sammel *et al.*, 2003) adjusted only for initial BMI.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs reported data on total cereals and weight gain.

Table 5.33 Weight gain and total cereals or cereal-based foods: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Contrast (mean)	Exposure Units	RR (CI)	Mean exposure (SD)	p	p trend	Adjustments
13193 (Bazzano *et al.*, 2005) PHS I	USA	40-84 (54) %M 100	(1550) /22071	13 years	FFQ (61)	Breakfast cereals, total	Weight gain ≥10kg Self-reported	≥1 vs Rarely	servings/ day	0.71 (0.58, 0.85)			0.0003	Age, Alcohol, BMI, Hypercholesterol-aemia, hypertension, physical activity, Smoking, Supplements
13195 PHS I			(551) /22071				weight gain ≥ 15kg Self-reported	≥1 vs Rarely	servings/ day	0.74 (0.54, 1.02)			0.08	As above
13187 PHS I			(1550) /22071			Breakfast cereals, high fibre (Wholegrain types)	Weight gain ≥10kg Self-reported	≥1 vs Rarely	servings/ day	0.78 (0.64, 0.96)			0.01	As above
13188 PHS I			(551) /22071				Weight gain ≥ 15kg Self-reported	≥1 vs Rarely	servings/ day	0.82 (0.58, 1.16)			0.27	As above
13189 PHS I			(1550) /22071			Breakfast cereals, low fibre (Refined grain types)	Weight gain ≥10kg Self-reported	≥1 vs Rarely	servings/ day	0.77 (0.56, 1.06)			0.05	As above
13190 PHS I			(551) /22071				weight gain ≥ 15kg Self-reported	≥1 vs Rarely	servings/ day	0.69 (0.39, 1.22)			0.21	As above
13916 (Sammel *et al.*, 2003) Penn Study of Ovarian Aging	USA, Cancer free, Generally healthy, Not Alcoholics, Not diabetic, Pre-menopausal, Without liver cirrhosis	35-47 (41) %M 0	(85) /436	4 years	FFQ (25)	Breads and cereals = Sum of servings of bread, cereals and salty snacks	Weight gain ≥10lb Measured by clinician/ professional		servings/ day		Gainers: 1.8 (1.9) Non-gainers: 2 (1.7)	0.606		BMI

Weight regain and glycaemic index

No cohort data reported results concerning glycaemic index and weight regain after loss.

Summary of RCT data

One small study is included here that reported weight regain at 12 months post-intervention for 16 obese individuals that had been randomly allocated to either a high or low GI diet for 8 weeks (Abete *et al.*, 2008) (see Table 5.34). The extent of weight regain was only statistically significant in the group following the high GI diet (however, this re-gain assessment represented only 47% of the initial study group of 32 participants).

Table 5.34 Weight regain and glycaemic index: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Within group Δ from baseline	p-value within group Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15554 (Abete *et al.*, 2008)	Higher GI diet	8/16	5.1 (SD 5.4)	0.003	Weight regain from initial follow-up	Measured by clinician/ professional (kg)	1 year	Decrease	unclear
	Lower GI diet	7/16	4 (SD 5.5)	0.101				Decrease	

Weight gain, overweight and obesity and sweetened beverages

[Fruit juice data were only extracted from cohort studies which were already captured in the review. In accord with our original protocol, a specific search for studies reporting fruit juice consumption has not been undertaken.]

Summary of cohort results in adults

A publication from the Framingham Heart Study (Dhingra *et al.*, 2007) was the only cohort study in adults to be extracted (see Table 5.35). Dhingra *et al.* (Dhingra *et al.*, 2007) presented results on both mixed sugar- and non-calorically sweetened beverages. Being a baseline consumer of at least one 12oz can of sweetened carbonated drinks per day compared with zero drinks was found to increase the odds of being obese (BMI >30) at the 4 year follow-up by 31%. However, interpretation of this study with regard to the effects of carbohydrate on body weight change is hampered by the fact that the exposure concerned carbonated beverages, which were not exclusively sugar-sweetened. Intake was measured by a general questionnaire, and weight was assessed by clinicians. The analyses adjusted for several important confounders.

Summary of cohort results in children

Data were extracted from four publications, reporting results from the following four cohort studies: Cardiovascular Risk in Young Finns Study, The Oslo Youth Study, The Pennsylvania Study of Health and Development of Young Girls study and the Nepean study (Nissinen *et al.*, 2009;Kvaavik *et al.*, 2005;Fiorito *et al.*, 2009;Tam *et al.*, 2006). Results are included in Table 5.36. The Oslo Youth Study (Kvaavik *et al.*, 2005) was the only study to use self-reported weight to derive BMI. Of those studies which reported risk estimates (Nissinen *et al.*, 2009;Kvaavik *et al.*, 2005), the Cardiovascular Risk in Young Finns Study reported no association and the Oslo Youth Study found an increased risk of overweight for females (but not males), who reported an increase in sweetened beverages, over the 21 years of follow-up.

The Pennsylvania Study of Health and Development of Young Girls (Fiorito *et al.*, 2009) tracked 5 year old white US girls through to 15 years. At each follow-up, the percentage of girls who were greater than the 85^{th} percentile for BMI was highest in the girls who consumed sweetened beverages most frequently at age 5 (main effect of beverage frequency group $p<0.001$). This association was not observed for 100% fruit juice intake. The Nepean study (Tam *et al.*, 2006) reported a statistically significant difference in intake of soft drinks and cordial between categories of weight trends, although it is unclear where this difference lay as post-hoc analyses were not reported. The same study found intakes of carbohydrates from fruit juice and fruit drinks to not significantly differ between weight gainers, maintainers and losers.

The Cardiovascular Risk in Young Finns Study experienced a higher percentage loss to follow-up than the other studies (Nissinen *et al.*, 2009).

Overall, the four studies provide inconsistent directions of association but generally do not support an association between sweetened beverages and weight gain or obesity when expressed as a categorical outcome.

Exposure definition and assessment

Definitions of exposure varied between studies. A publication from the Framingham Heart Study (Dhingra *et al.*, 2007) presented results on both mixed sugar and non-calorically sweetened beverages. The Oslo Youth Study (Kvaavik *et al.*, 2005) measured 'full-calorie sweetened beverages', focusing on carbonated drinks. The Pennsylvania Study of Health and Development of Young Girls (Fiorito *et al.*, 2009) included carbonated beverages, as well as all sugar- or non-calorically sweetened drinks and fruit-flavoured drinks (<100% fruit). The Nepean study (Tam *et al.*, 2006) examined beverages separately as soft drinks and cordial, and fruit juices and drinks. The Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009), on the other hand, reported a change in intake of sugar-sweetened beverages as the exposure variable.

The studies also used a variety of methods to collect consumption data: FFQ, general questionnaire, dietary recall or diet diary.

Adjustment for appropriate confounders

The Cardiovascular Risk in Young Finns Study (Nissinen *et al.*, 2009) appeared to be the only study to include an appropriate range of adjustments.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Only one trial provided data on the relationship between sweetened beverages and overweight or obesity (see Table 5.37). The cluster randomised controlled trial reported by James *et al.* (James *et al.*, 2004) reported overweight and obesity defined using a range of different approaches, at baseline and one year after an intervention study, which aimed to assess the effects of an educational programme designed to reduce the consumption of carbonated beverages in children. The objective was to reduce consumption of all carbonated beverages, whether sugar-sweetened or non-calorically sweetened in 6 primary schools in southwest England.

A total of 644 children aged 7-11 years were randomised to intervention or control groups, and drinks diaries completed over 3 days revealed that carbonated drinks consumption decreased by 0.6 drinks (average drink volume 250 ml) in the intervention group but increased by 0.2 drinks in the control group (mean difference 0.7, 95% confidence interval 0.1 to 1.3). However, drinks data were available only on a sample of the whole study population (approx. 36%).

Overall, the children gained weight, as might be expected. Based on the percentage of children >91st centile (using revised 1990 growth standards), a higher proportion of children in the control group were overweight or obese at follow-up compared with the intervention group (mean difference 7.7%, (2.2% to 13.1%)). Other methods of classifying the weight status of the children are also reported and are captured in the table (Table 5.37). These include overweight or obesity defined by British waist circumference centile charts, International Obesity Task Force and by 1990 British centile charts. All show a similar trend to the proportion overweight/obese using percentage <91st centile, but statistical significance was not reported. However, interpretation of this study with regard to the effects of carbohydrate on body weight change is hampered by the fact that the intervention concerned carbonated beverages, which were not exclusively sugar-sweetened. Additionally, the relatively low number of children consenting to participate and subsequent drop-out of approximately 10% of children means that the study population is not necessarily representative of UK school children in general.

Table 5.35 Obesity and sweetened beverages: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Contrast (mean)	Exposure Units	RR (CI)	Adjustments
14233 (Dhingra *et al.*, 2007) The Framingham Heart Study	USA, No heart disease, Without metabolic syndrome	(53) %M 43	(548) /8997	4 years	Question-naire (general)	Mixed sugar and non-calorically sweetened beverages (soft drinks - number of 12oz cans)	BMI >30 Measured by clinician/ professional	≥1 vs 0	12 oz serving/day	1.31 (1.02, 1.68)	Age, BMI, Smoking, saturated fat intake, energy intake, Fibre, GI, Magnesium Intake, physical activity, Gender, trans-fatty acid

Table 5.36 Prevention of weight gain and sweetened beverages: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Subgroup	Contrast (mean)	Exposure Units	Mean outcome (SD)	Mean exposure	RR (CI)	p	Adjustments
14217 (Fiorito *et al.*, 2009) Pennsylvania Study of Health and Development of Young girls	USA, Primarily White, Age 5-18y	(5) %M 0	(Number of cases not reported) /170	10 years (15)	Dietary recall	All sugar-sweetened and non-calorically sweetened beverages including fruit flavour drinks (>100% fruit juice), sports drinks and sodas	BMI ≥ 85th centile Measured by clinician/ professional	Age 9	1.<1 2. ≥1 to 2 3.>2	Serv-ings/ day	1. 24.2% 2. 29.4% 3. 46.2%				
14229							BMI ≥ 85th centile	Age 11	1.<1 2. ≥1 to 2 3.>2	Serv-ings/ day	1. 21.7% 2. 29.4% 3. 53.9%				
14230							BMI ≥ 85th centile	Age13	1.<1 2. ≥1 to 2 3.>2	Serv-ings/ day	1. 22.2% 2. 19.6% 3. 46.2%				
14231							BMI ≥ 85th centile	Age 15	1.<1 2. ≥1 to 2 3.>2	Serv-ings/ day	1. 18.5% 2. 18.4% 3. 32%				
14522 (Kvaavik *et al.*, 2005) Oslo Youth Study	Norway, Ethnicity unknown	(13) %M 49	(Number of cases not reported) /1086	19 years (15.7)	Question-naire (general)	Full-calorie sugar sweetened beverages (High consumers: reported consuming >3 times/wk (1991)	BMI>25 Self-reported	Female	Long term high consumers vs Long term low consumers				1.57 (0.46, 5.33)		BMI
14527							BMI>25	Male	Long term				1.05		BMI

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Subgroup	Contrast (mean)	Exposure Units	Mean outcome (SD)	Mean exposure	RR (CI)	p	Adjustments
Oslo Youth Study						and 4 times/wk (1999) Low consumers: reported consuming <2 times/wk (1991) and <3 times/wk (1999). Inconsistent: changing consumption frequency)	Self-reported		high vs Long term low consumers				(0.46, 2.4)		
14528 Oslo Youth Study							BMI>30 Self-reported	Female	Long term high vs Long term low consumers				0.8 (0.09, 6.85)		BMI
14529 Oslo Youth Study							BMI>30 Self-reported	Male	Long term high vs Long term low consumers				2.29 (0.48,10.96)		BMI
14705 (Nissinen *et al.*, 2009) Cardiovascular Risk in Young Finns Study	Finland, Primarily White	3-18 (11) %M 45.2	(Number of cases not reported) /3596	21 years (33)	FFQ (19)	Change in sugar-sweetened beverages	BMI >25 Measured by clinician/ professional	Male	Increase vs Stable low				1.07 (0.74, 1.57)		Age, BMI, Education, PA, Smoking
14706 Cardiovascular Risk in Young Finns Study								Female	Increase vs Stable low				1.9 (1.38, 2.61)		Age, BMI, Education, PA, Smoking
14609/14610/ 14611 (Tam *et al.*, 2006) Nepean study	Australia, Ethnicity unknown	(8) %M 51	(32) /268	5.4 years	Food diary	Carbohydrates from soft drinks and cordial	BMI Z-score status Measured by clinician/ professional			g/day		Gainers: (n: 32) 29g/d Maintained acceptable BMI z-score: (n: 195) 20g/d Overweight maintainers: (n: 41) 30g/d Losers: (n: 13) 6.5g/d		0.005	
17600/17601/ 17602 Nepean study			(32) /268			Carbohydrates from fruit juice and fruit drinks	BMI Z-score status Measured by clinician/ professional			g/day		Gainers: (n: 32) 8.6g/d Maintained acceptable BMI z-score: (n: 195) 14g/d Overweight maintainers: (n: 41) 14g/d Losers: (n: 13) 13g/d		0.734	

Table 5.37 Overweight and obesity and sweetened beverages: RCT data

Result ID/ Author	Subgroup detail	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	Difference between groups in Δ from baseline	Outcome/Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
14933 (James *et al.*, 2004)		Control minus reduced carbonated beverages	Control: 279/319 Intervention: 295/325				7.7 (CI 2.2, 13.1)	Mean percentage >91 centile	Measured by clinician/ professional	1 year	Increase in both	Unclear
14934		Control	279/319	19.4 (SD 8.4)	26.9 (SD 12.3)	7.5 (SD 8)		Mean percentage >91 centile	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	20.3 (SD 6.3)	20.1 (SD 6.7)	-0.2 (SD 6.3)					Increase	
15918	Overweight Boys	Control	279/319	18.8	22.2			Overweight defined by International obesity task force	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	20.1	18.3						Increase	
15920	Overweight Girls	Control	279/319	28.0	29.6			Overweight defined by International obesity task force	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	27.6	23.1						Increase	
15921	Obese Boys	Control	279/319	1.7	1.7			Obese defined by International obesity task force	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	4.1	3.5						Increase	
15922	Obese Girls	Control	279/319	7.3	6.3			Obese defined by International obesity task force	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	5.7	4.7						Increase	
15923	Overweight Boys	Control	279/319	19.6	25.6			Overweight defined by 1990 British centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	19.2	19.9						Increase	
16018	Overweight Girls	Control	279/319	20.1	28.3			Overweight defined by 1990 British centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	19.2	20.9						Increase	
16019	Obese Boys	Control	279/319	7.0	9.0			Obese defined by 1990 British centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	6.9	7.1						Increase	
16020	Obese Girls	Control	279/319	7.5	9.0			Obese defined by 1990 British centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	6.6	6.5						Increase	
16021	Overweight Boys	Control	279/319	20.3	25.0			Overweight defined by British waist circumference centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	21.5	22.6						Increase	
16022	Overweight Girls	Control	279/319	24.4	36.9			Overweight defined by British waist circumference centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	22.1	29.6						Increase	
16023	Obese Boys	Control	279/319	9.9	10.4			Obese defined by British waist	Measured by	1 year	Increase	Unclear

Result ID/ Author	Subgroup detail	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	Difference between groups in Δ from baseline	Outcome/Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		Reduced carbonated beverages	295/325	9.5	8.4			circumference centile charts	clinician/ professional		Increase	
16025	Obese Girls	Control	279/319	10.7	19.0			Obese defined by British waist circumference centile charts	Measured by clinician/ professional	1 year	Increase	Unclear
		Reduced carbonated beverages	295/325	9.9	11.5						Increase	

Body fatness and body fat distribution

Studies included here provided information concerning the association between dietary carbohydrate and a range of different measures that reflect total body stores of adipose tissue and relative distribution around the body. The methods used to assess these outcomes include dual-energy X-ray absorptiometry (DEXA), the use of skinfold thicknesses (either singular or at multiple sites), bioimpedance, near infrared light interactance, and the body circumference at waist or hip level as well as the ratio of the latter two measures.

Body fatness, fat distribution and total carbohydrate intake or high carbohydrate diets

Summary of cohort results in adults

Data concerning total carbohydrate intake and measures of fat distribution were extracted from two publications, presenting results from the CARDIA study and the Danish Diet, Cancer and Health study in adults (Ludwig *et al.*, 1999;Halkjaer *et al.*, 2006) (see Table 5.38). There was no significant association between carbohydrate intake and waist-to-hip ratio in the CARDIA Study (Ludwig *et al.*, 1999). Similarly the results taken from the Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2006) indicate no association between total baseline carbohydrate consumption (grams per MJ/day) and change in waist circumference over 5 years of follow-up. However, in women, greater increases in waist circumference were associated with particular sources of carbohydrate; carbohydrates from refined-grain products and potatoes and carbohydrates from foods with ‘simple’ or added sugars were associated with significantly greater increases in waist circumference over time. The relationships did not achieve statistical significance when carbohydrate intake from wholegrain foods or total carbohydrates were considered, nor for analyses of the male subgroup.

The Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2006) assessed waist circumference change as the outcome, whereas the CARDIA study (Ludwig *et al.*, 1999) calculated waist-to-hip ratio. This was measured by clinicians or professionals in the CARDIA study and the Danish Diet, Cancer and Health Study.

One further US cohort study of male health professionals (HPFS), provided data concerning the impact of habitual consumption of high or low carbohydrate diets and change in waist circumference, but instead focused on the macronutrient source used to replace carbohydrate (Table 5.41) (Koh-Banerjee *et al.*, 2003). In this study, through modelling, the authors explored the impact on body fat distribution associated with diets in which the isoenergetic replacement of carbohydrate had been achieved with: trans fats, saturated fats, polyunsaturated fats or monounsaturated fats. Each 2% increment in energy intake from trans fats that was isoenergetically substituted for carbohydrates was significantly associated with a 0.77-cm waist gain over 9 years ($p < 0.001$). A similar, but smaller change was apparent for each unit increase in saturated fat that replaced carbohydrate. No such association was apparent when polyunsaturated or monounsaturated fats were explored as the replacement for carbohydrate.

These cohort studies suggest that there is no association between total carbohydrate and waist circumference change over time. However, since these studies found differences in the direction of the relationship depending on the source of the carbohydrate and the nature of its replacement as an energy source, further exploration of the relationship between sources of dietary carbohydrate and waist circumference change is warranted using clinical trials rather than observational approaches.

Exposure definition and assessment

All studies employed FFQs to collect dietary data on habitual carbohydrate intake.

Adjustment for appropriate confounders

All studies included a range of important covariates in the analyses.

Summary of cohort results in children

Data concerning total carbohydrate intake and body fatness or fat distribution were extracted from five publications, presenting results from three cohort studies: the Amsterdam Growth and Health Study, The Northern Ireland Young Hearts Project and the Adelaide Longitudinal Study of Growth and Nutrition (Twisk *et al.*, 1998;Van Lenthe *et al.*, 1998;Koppes *et al.*, 2009;Boreham *et al.*, 1999;Magarey *et al.*, 2001) (see Table 5.39).

Of the three publications that stemmed from the Amsterdam Growth and Health Study, two had a follow-up of 14 years, and the other of 23 years. There was no apparent association between carbohydrate intake assessed at age 12-15 years and sum of four skinfolds after 14 years of follow-up (Twisk *et al.*, 1998). However when only subscapular skinfolds were used, and analyses were conducted separately for males and females, a positive relationship – that is an increasing skinfold thickness with increasing consumption of total carbohydrate - was evident for girls (p=0.01) (Van Lenthe *et al.*, 1998). No significant associations were reported at the 23 year follow-up (Koppes *et al.*, 2009).

The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) found no significant relationship between skinfold thicknesses and carbohydrate intake in either boys or girls. In contrast, the Adelaide Longitudinal Study of Growth and Nutrition (Magarey *et al.*, 2001) reported a statistically significant negative relationship between total carbohydrate intake and triceps skinfold measurement in both males and females, which implies that as total carbohydrate intake increases, skinfold measurements decrease.

Duration of follow-up varied across publications, ranging from 4 to 23 years. The Amsterdam Growth and Health Study (Twisk *et al.*, 1998) experienced a higher percentage loss to follow-up than the other studies, at 50%.

Data from one further cohort study were extracted, assessing adiposity as a fat mass index (Johnson *et al.*, 2008) (see Table 5.40). In the ALSPAC cohort (Johnson *et al.*, 2008) adiposity was quantified with DEXA. Differences in reported carbohydrate intake between incident cases of excess adiposity and non-cases were found to be non-significant.

Collectively, these cohort studies of children and adolescents which have employed skinfold thickness measures or DEXA to assess body fatness do not provide clear evidence of an association between total dietary carbohydrate consumption and adiposity at follow-up.

Exposure definition and assessment

Two of the studies gathered dietary intake information via food diaries (Magarey *et al.*, 2001;Johnson *et al.*, 2008) the other two cohort studies employed the dietary history approach. Carbohydrate intake was expressed as per cent energy in all studies, with the exception of one of the publications from the Amsterdam Growth and Health Study (Twisk *et al.*, 1998) in which both grams per day and per cent energy were both reported.

Adjustment for appropriate confounders

Adjustments in the analyses from the 14-year follow-up of the Amsterdam Growth and Health Study were unclear (Twisk *et al.*, 1998;Van Lenthe *et al.*, 1998). The other publications appeared to be more fully adjusted, including covariates such as gender, parental BMI, and energy intake.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

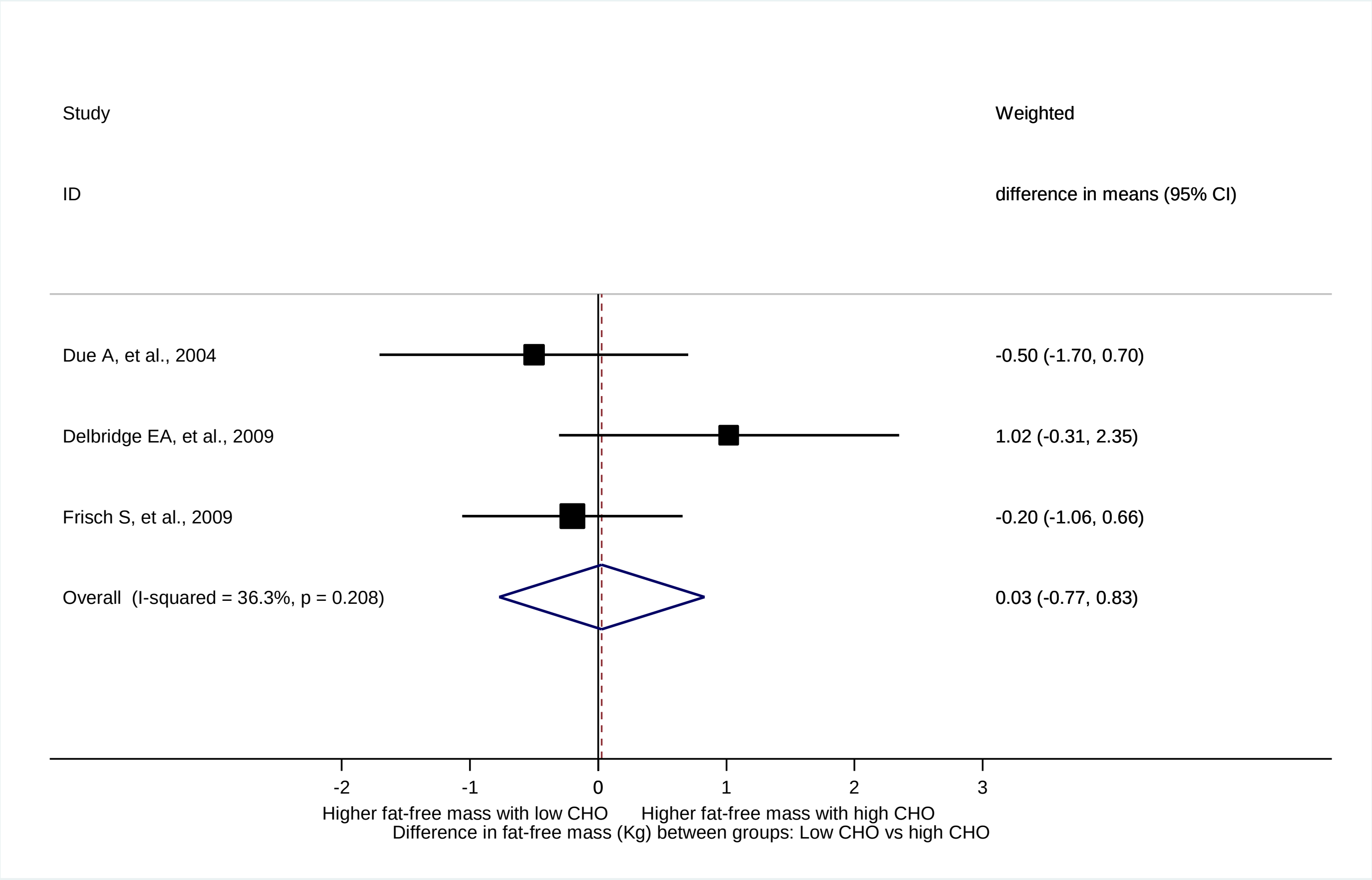
Fat free mass

Five trials which compared higher and lower carbohydrate diets on fat free mass provided data (Table 5.42). One study was not included in the meta-analysis because the carbohydrate percentage of energy difference between the diets was estimated to be less than 5% (Dale *et al.*, 2009).

Dale and colleagues randomly assigned 200 women who had previously lost 5% or more of their initial body weight to groups that were advised to follow a high carbohydrate or a relatively high monounsaturated fat diet (Dale *et al.*, 2009). These groups were also divided into those receiving either an intensive or less intensive form of support. After 2 years of follow-up, data were available from 174 (87%) participants. Change in fat free mass did not vary significantly between diet groups (both decreased). However, achieved differences in percentage energy from carbohydrate between groups were much less than intended (43 vs. 47% energy) and since this was less than our 5% cut-off, this study was not included in the meta-analysis.

Three studies which assessed the effect of diets that differed by more than 5% energy from carbohydrate between groups provided data and were included in the meta-analysis (Delbridge *et al.*, 2009;Due *et al.*, 2004;Frisch *et al.*, 2009). All included adults as participants. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.42. The first follow up reported at the end of the intervention was used. This was 12 months for all three of the studies. Fat free mass was measured in these studies by DEXA and bioimpedance. The overall pooled estimate indicated that fat-free mass was 0.03kg (95% CI, -0.77 to 0.83) higher with consumption of a low carbohydrate diet compared with a high carbohydrate diet, however this difference was not statistically different from zero (p=0.95). Heterogeneity denoted by I^2 was 36% (95% CI, 0 to 80%). There were insufficient studies to carry out a funnel plot. Statistically, there was no evidence of a difference in fat-free mass with higher compared to lower carbohydrate diets.

Figure 5.6 Forest plot for higher carbohydrate diets and fat free mass (kg)



Total body fat

Children and adolescents

Two trials using obese adolescents provided data on total body fat changes in response to high and low carbohydrate diets (Ebbeling *et al.*, 2003;Demol *et al.*, 2009). Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.43. These trials were not pooled with the data from adults in the meta-analysis. The study reported by Demol *et al.* compared the effects of a high carbohydrate low fat diet, with lower carbohydrate diets that varied in the proportion of energy derived from fat or protein on body weight and the metabolic profile of obese adolescents (Demol *et al.*, 2009). Both low carbohydrate diets aimed to deliver up to 20% of energy from carbohydrate (60g/d), which was compared with 50-60% of energy from carbohydrate in the high carbohydrate diet. The participants were free-living and were instructed to follow a pre-set rotating menu, which aimed to deliver 1200-1500 kcal per day. After one year, all groups had lost body fat, which was estimated using bioimpedance. However the changes in fat mass did not vary by diet group. All diets were equally effective in reducing fatness.

Ebbeling 2003 (Ebbeling *et al.*, 2003) compared the effects of a conventional reduced fat (25-30% energy), high carbohydrate (55-60% energy) diet with a moderate fat (30-35% energy), lower carbohydrate (45-50% energy, low/moderate GI) diet in 16 obese adolescents (13-21 years) over 12 months. Fourteen subjects completed the full trial. There was a decrease in fat mass (assessed by DEXA) at 12 months in the lower carbohydrate group (-2.6 SE 1.5), but an increase in the high carbohydrate group (+1.6 SE 0.9), and the difference between groups was statistically significant, p=0.01.

Adults

Nine studies of adults provided data on the effects of higher compared to lower carbohydrate diets on changes in total fat mass (in kg) (Clifton *et al.*, 2008;Dale *et al.*, 2009;Delbridge *et al.*, 2009;Due *et al.*, 2004;Ebbeling *et al.*, 2007;Frisch *et al.*, 2009;Gardner *et al.*, 2007;Layman *et al.*, 2009;McManus *et al.*, 2001).

Three studies were not included in the meta-analysis because the achieved carbohydrate percentage of energy difference between the diets was estimated to be less than 5%.
Clifton *et al.* (Clifton *et al.*, 2008) did not observe any differential effect of the high/low protein diets tested on body fat stores at 1 year post randomisation.

In the trial conducted by McManus *et al.* (McManus *et al.*, 2001) 101 overweight men and women were allocated to a lower carbohydrate, moderate-fat diet (35% of energy) or a higher carbohydrate, low-fat diet (20% of energy). After 18 months, 31/50 subjects in the moderate-fat group, and 30/51 in the low fat group were available for measurements. Percent body fat decrease assessed using a near-infrared light interactance device was similar in both diet groups at 18 months. However, at 18 months, the difference in percentage energy from carbohydrate was less than 5% between groups.

Dale and colleagues randomly assigned 200 women who had previously lost 5% or more of their initial body weight to groups that were advised to follow a high carbohydrate or a relatively high monounsaturated fat diet (Dale *et al.*, 2009). These groups were also divided into those receiving either an intensive or less intensive form of support. After 2 years of follow-up, data were available from 174 (87%) participants. Change in fat mass did not vary significantly between diet groups (both decreased). However, achieved differences in percentage energy from carbohydrate between groups were much less than intended (43 vs. 47% energy) and since this was less than our 5% cut-off, this study was not included in the meta-analysis.

For inclusion in the meta-analysis there was requirement for a difference of energy from carbohydrate between trial groups of 5% or more. Actual consumption was used rather than intended diet unless otherwise stated – see trial characteristics table.

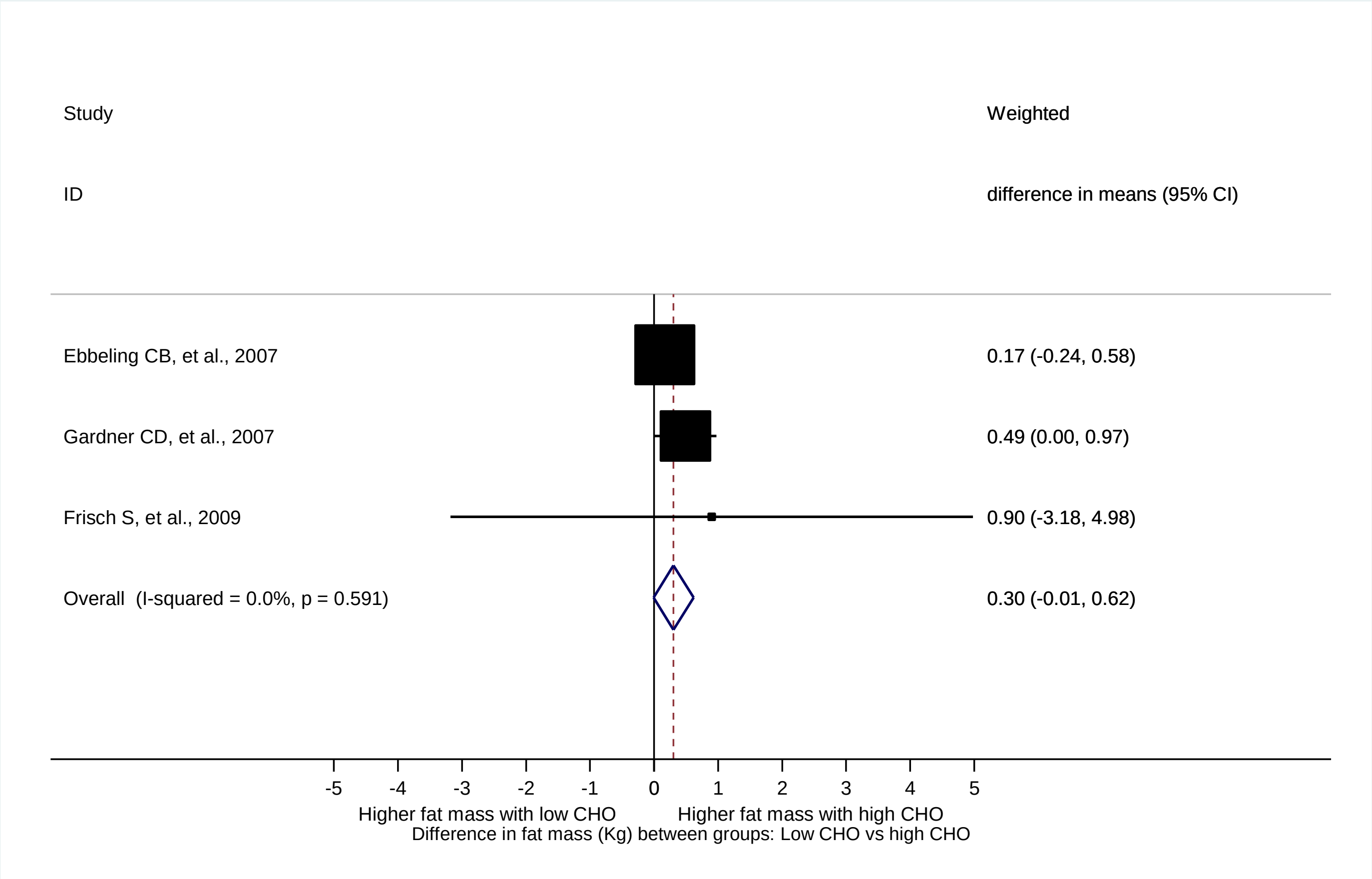
Six studies of adults were included in the meta-analysis comparing different carbohydrate intakes and changes in weight of fat mass (in kg) (Delbridge *et al.*, 2009;Due *et al.*, 2004;Ebbeling *et al.*, 2007;Frisch *et al.*, 2009;Gardner *et al.*, 2007;Layman *et al.*, 2009). Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.43. A variety of methods were used to estimate body fat content, including DEXA, bioimpedance and a near-infrared light interactance device. One study reported results from four groups (Dansinger *et al.*, 2005) and for this study the group with the lowest carbohydrate intake was compared with the group with highest carbohydrate intake.

The first follow up reported at the end of the intervention and over 12 months was used. This varied from 12 months to 2 years. The meta-analysis was split into 2 plots, the first looking at carbohydrate and fat swap and the second including studies with carbohydrate and protein or protein and fat swap.

Higher carbohydrate, low fat diets compared with lower carbohydrate, higher fat diets

The pooled estimate indicated that fat mass was 0.30kg higher (95% CI, -0.01 to 0.62) with consumption of a higher carbohydrate, lower fat diet. This was not significantly different from zero (p=0.06). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 80%). Statistically there was no evidence that diets higher in carbohydrate and lower in fat resulted in different fat mass.

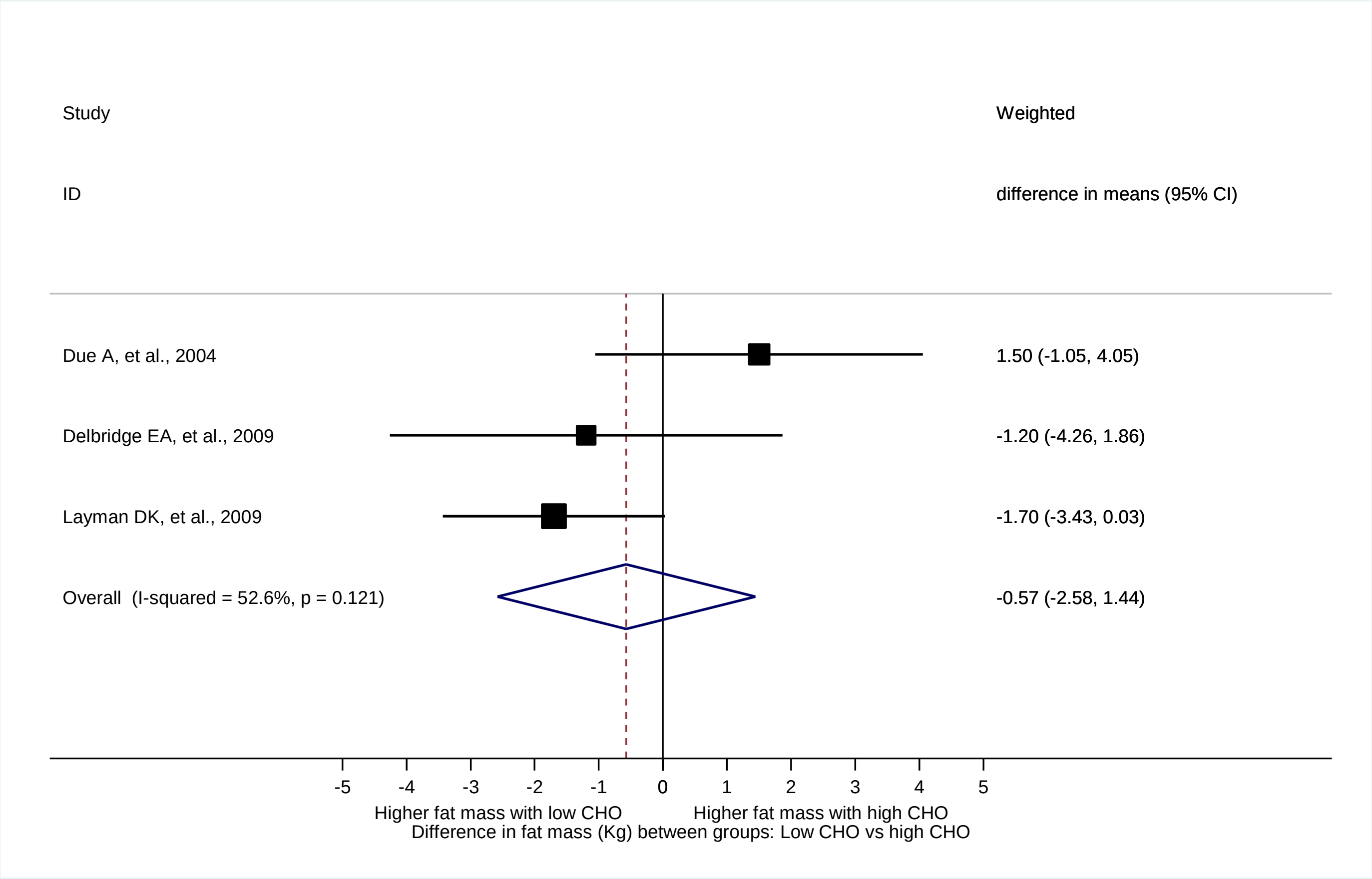
Figure 5.7 Forest plot for higher carbohydrate, lower fat diets and higher carbohydrate, higher fat diets and total body fat (kg)



Carbohydrate and protein or fat and protein swap

The pooled estimate indicated that fat mass was 0.57kg lower (95% CI, -1.44 to 2.58) with consumption of a high carbohydrate, lower protein diet. This was not significantly different from zero (p=0.58). Overall heterogeneity denoted by I^2 was 53% (95% CI 0 to 86%). A funnel plot was not carried out due to the small number of studies. Statistically, there was no evidence that a diet higher in carbohydrate and lower in fat and protein or protein only was associated with differences in fat mass.

Figure 5.8 Forest plot for carbohydrate and protein swap or fat and protein swap diets and total body fat (kg)



Waist circumference

Nine trials provided data on the relationship between waist circumference and diets high or low in carbohydrate (Bhargava, 2006;Dale *et al.*, 2009;Dansinger *et al.*, 2005;Delbridge *et al.*, 2009;Due *et al.*, 2004;Frisch *et al.*, 2009;Keogh *et al.*, 2007;McManus *et al.*, 2001;Sacks *et al.*, 2009). Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.44.

Three studies were excluded from the meta-analysis since the difference in percentage of carbohydrate energy was less than 5% between intervention groups (McManus *et al.*, 2001;Dale *et al.*, 2009;Bhargava, 2006). These three studies found inconsistent directions of effect.

In the trial conducted by McManus *et al.* (McManus *et al.*, 2001) 101 overweight men and women were allocated to a lower carbohydrate, moderate-fat diet (35% of energy) or a higher carbohydrate, low-fat diet (20% of energy). After 18 months, 31/50 subjects in the moderate-fat group, and 30/51 in the low fat group were available for measurements. In the lower carbohydrate, moderate-fat group, waist circumference decreased by 6.9 cm, compared to an increase in the low-fat group of 2.6cm ($p<0.001$). However, at 18 months, the difference in percentage energy from carbohydrate was less than 5% between groups.

Dale and colleagues randomly assigned 200 women who had previously lost 5% or more of their initial body weight to groups that were advised to follow a high carbohydrate or a relatively high monounsaturated fat diet (Dale *et al.*, 2009). These groups were also divided into those receiving either an intensive or less intensive form of support. After 2 years of follow-up, data were available from 174 (87%) participants. Waist circumference changes did not vary significantly between diet groups. However, achieved differences in percentage energy from carbohydrate between groups were much less than intended (43 vs. 47% energy) and since this was less than our 5% cut-off, this study was not included in the meta-analysis.

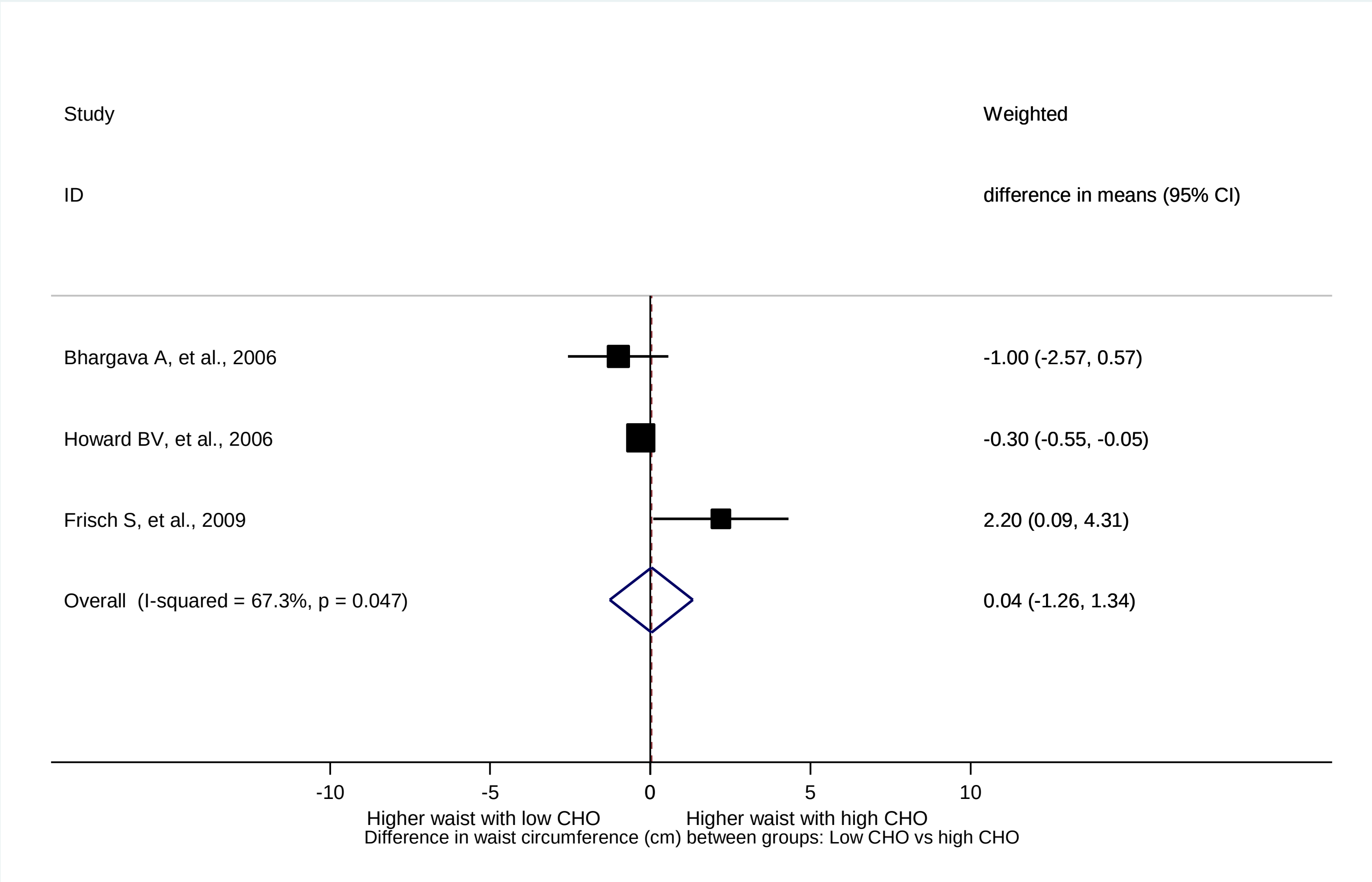
Data from the Women's Health Trial: Feasibility Study in Minority Populations (Bhargava, 2006) were also not included in the meta-analysis as the percentage energy derived from carbohydrate was not reported, although it was clear that the control diet was likely to be considerably lower in carbohydrate than the low fat, high fruit and vegetable and grain diet advocated to the intervention group. After one year of follow up, the women in the intervention group experienced a small decrease in waist circumference (-0.02cm), whereas women in the control group remained stable (difference between groups, $p<0.05$). This is despite the fact that the participants in this trial were not markedly overweight and the diet was not energy restricted.

For inclusion in meta-analysis there was a difference of energy from carbohydrate between trial groups of 5% or more. Actual consumption was used rather than intended diet unless otherwise stated – see trial characteristics table.

Seven studies were included in the meta-analyses comparing different carbohydrate intakes and changes in waist circumference (cm). One study reported results from four groups (Dansinger *et al.*, 2005). For this study the arm with the lowest carbohydrate intake was compared with the arm with highest carbohydrate intake (Atkins vs. Ornish diets). The remaining studies were stratified into two groups, one where fat and carbohydrate content of the diet was different between groups and one where either protein only or fat and protein and carbohydrate content were different. All studies included adults as participants. Levels of carbohydrate, fat and protein are reported in the trial characteristics table. The first follow up reported at the end of the intervention and over 12 months was used. This varied from 12 months to 7 years.

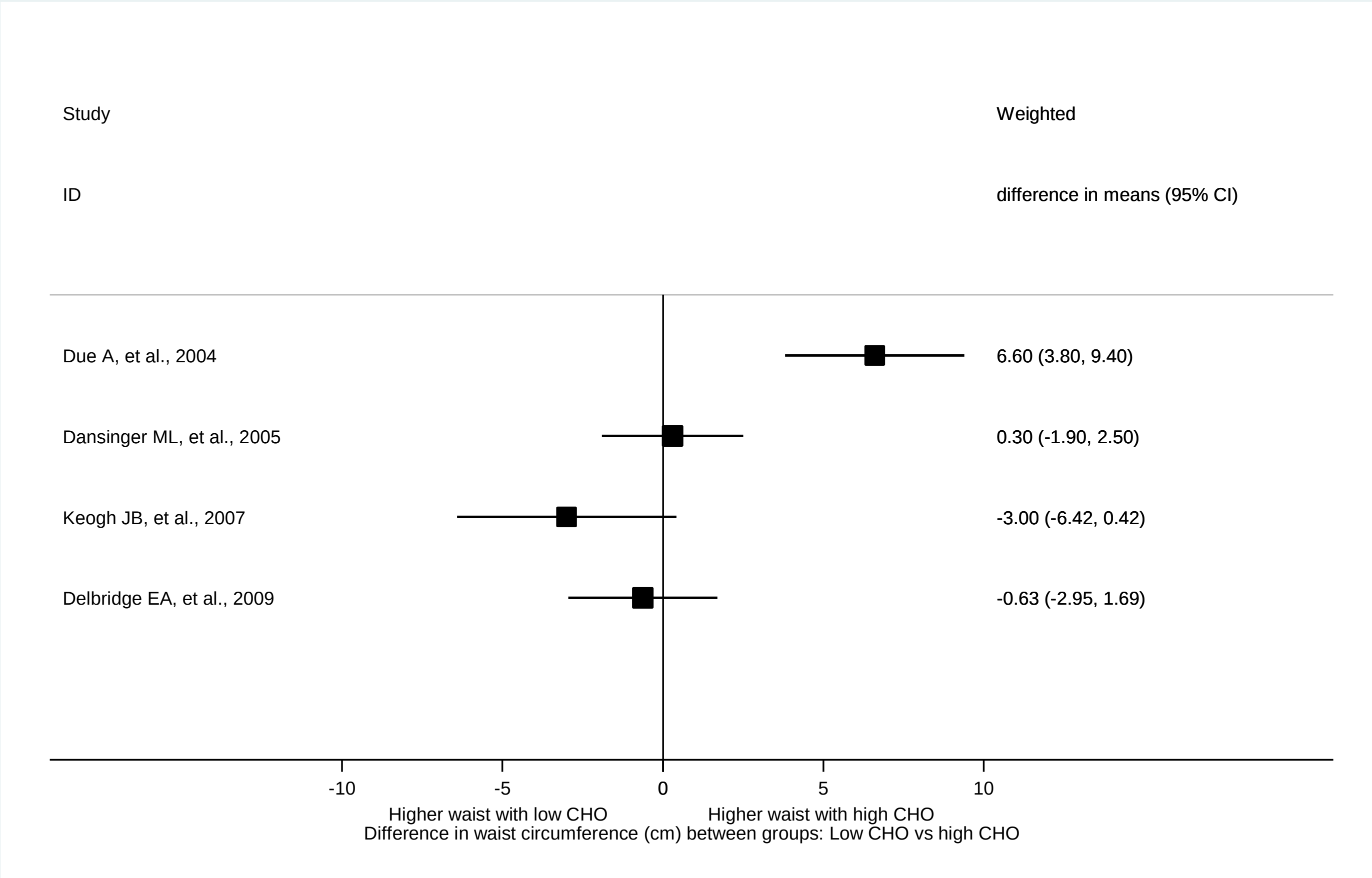
The pooled estimate for studies reporting differences in fat and carbohydrate indicated that waist circumference was 0.04cm (95% CI, -1.26 to 1.34) higher with consumption of a high carbohydrate, low fat diet but this was not significantly different from zero (p=0.96). Heterogeneity indicated by I^2 was 67% (95% CI 0 to 91%). A funnel plot was not carried out due to the small number of studies. Statistically, there was no evidence that a diet higher in carbohydrate and lower in fat was associated with changes in waist circumference.

Figure 5.9 Forest plot for carbohydrate and fat swap diets and waist circumference (cm)



Four studies explored the effects of high and low carbohydrate diets where carbohydrate was replaced with protein or a combination of fat and protein and these were pooled in a meta-analysis. No pooled estimate was reported for these studies as heterogeneity was high at 86%. Three studies reported no difference between high and low carbohydrate diets on waist circumference (Dansinger *et al.*, 2005;Delbridge *et al.*, 2009;Keogh *et al.*, 2007). However the study by Due et al. (Due *et al.*, 2004) which compared medium and low protein diets (12 vs. 25% energy) observed a significantly greater reduction in waist circumference in the high protein diet group.

Figure 5.10 Forest plot for high and low carbohydrate diets in which carbohydrate was replaced with protein or a combination of fat and protein on waist circumference (cm)



Hip circumference

Two trials provided data on the relationship between hip circumference and diets high or low in dietary carbohydrate (Bhargava, 2006;Delbridge *et al.*, 2009). Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.45. Bhargrava *et al.* (Bhargava, 2006) reported that the change from baseline to 12 months differed between control and low fat, high fibre groups in the Women's Health Trial Feasibility Study in Minority Populations, with a greater decrease in hip circumference in the intervention group. This was a US-based multi-centre trial of postmenopausal women, with an average initial BMI of 28. The intervention group received detailed group-based advice aimed at reducing total fat to 20% of energy and increasing consumption of fruit, vegetables and whole grains. Total fat intakes are not reported in this paper, but saturated fat intakes decreased by 10g/d in the intervention group and 5g/d in the control (no diet change) group. Dietary fibre intakes decreased in the control group, but were unchanged in the intervention group.

The study reported by Delbridge *et al.* (Delbridge *et al.*, 2009) explored the effects on weight maintenance of high protein or high carbohydrate diets after an acute weight loss phase of 3 months. Both diets were isoenergetic and individually prescribed. Subjects were initially overweight or obese, with no other health problems, and after the initial weight loss of 16.5 kg, maintained most of this weight loss at the 12 month weigh-in (14.5 ±1.2 kg). Hip circumferences in both groups increased slightly over the maintenance phase, but not to a greater extent in either group (p=0.53). The different approaches used and variation in outcome do not provide strong evidence of a difference in effect of high compared with low carbohydrate diets.

Waist to hip ratio

Five studies reported data on changes in waist to hip ratio after consuming high or low carbohydrate diets. Two of these studies could not be included in a meta-analysis since the difference in percent carbohydrate energy was less than 5%, and due to lack of information about total carbohydrate content of the intervention diets respectively (McManus *et al.*, 2001;Bhargava, 2006).

In the trial conducted by McManus *et al.* (McManus *et al.*, 2001) 101 overweight men and women were allocated to a lower carbohydrate, moderate-fat diet (35% of energy) or a higher carbohydrate, low-fat diet (20% of energy). After 18 months, 24/50 subjects in the moderate-fat group, and 10/51 in the low fat group provided measurements. In the lower carbohydrate, moderate-fat group, there was no change in waist: hip ratio, compared to a small decrease in the low-fat group of -0.02.

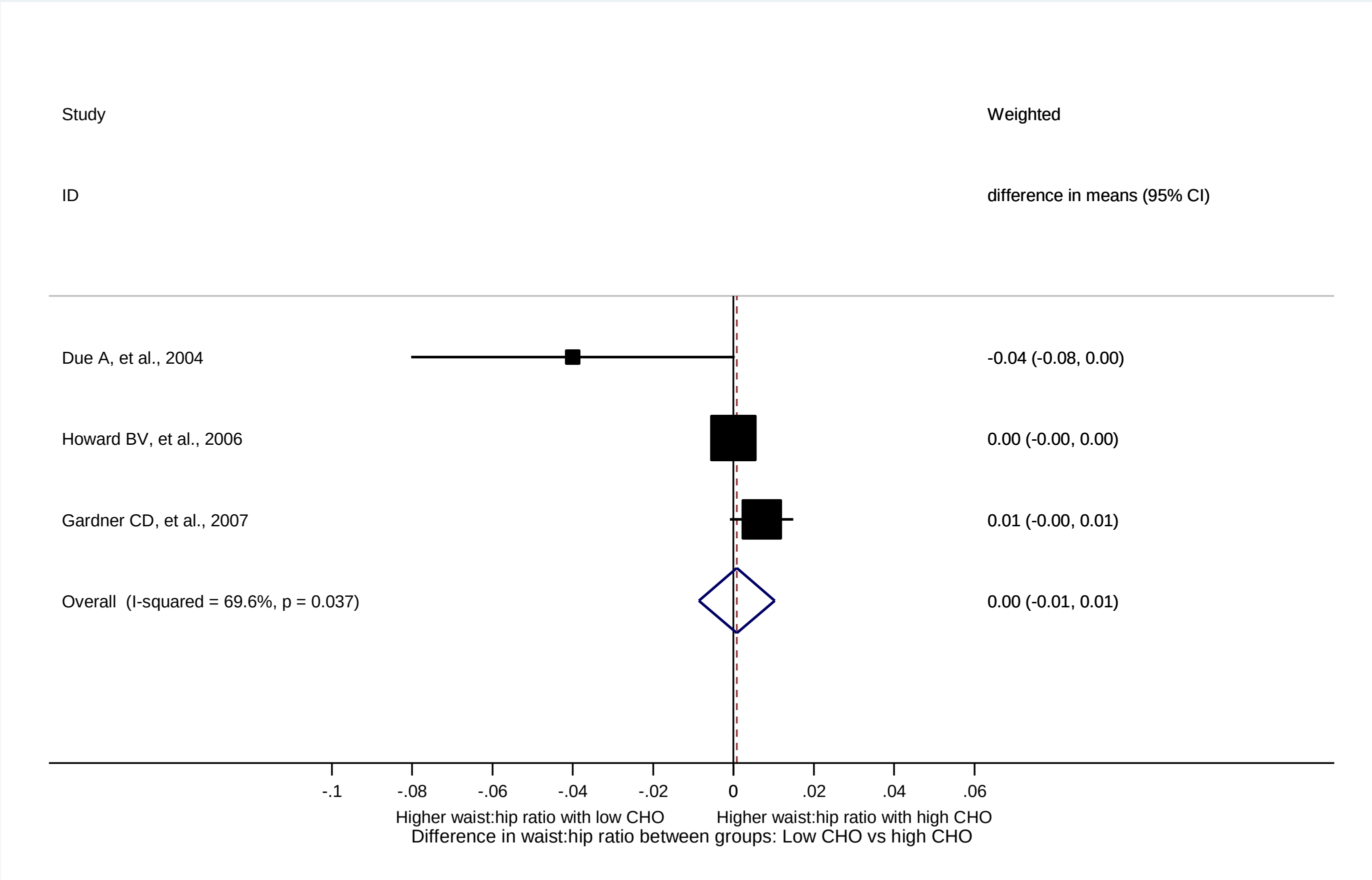
Data from the Women's Health Trial: Feasibility Study in Minority Populations (Bhargava, 2006) were also not included in the meta-analysis as the percentage energy derived from carbohydrate was not reported, although it was clear that the control diet was likely to be considerably lower in carbohydrate than the low fat, high fruit and vegetable and grain diet advocated to the intervention group. After one year of follow up, there was no difference in waist:hip ratio between the two diet groups.

For inclusion in meta-analysis there a difference of energy from carbohydrate between trial groups of 5% or more was taken as meaningful. Actual consumption was used rather than intended diet unless otherwise stated – see trial characteristics table.

Three studies were included in the meta-analyses comparing high carbohydrate with lower carbohydrate diets and changes in waist to hip ratio (Delbridge *et al.*, 2009;Due *et al.*, 2004;Gardner *et al.*, 2007). All studies included adults as participants. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.46. The first follow up reported at the end of the intervention was used. This was 12 months for two of the studies and 90 months for one study (Howard *et al.*, 2006).

The overall pooled estimate indicated that the waist:hip ratio was not differentially affected by the carbohydrate content of the intervention diets (difference of zero) (95% CI, -0.01 to 0.1) (p=0.87). Heterogeneity denoted by I^2 was 50% (95% CI, 0 to 82). Statistically, there was no evidence of a difference in waist:hip ratio with differences in dietary carbohydrate.

Figure 5.11 Forest plot for high carbohydrate diets and waist to hip ratio



Other measures of fat distribution – central and peripheral fat

Two trials provided data on the effects of high carbohydrate diets on intra and extra-abdominal fat deposition. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.47. Clifton *et al.* (Clifton *et al.*, 2008) provided data on change in peripheral and central body fat, citing DEXA as the method used. It is likely that fat distribution measures were achieved by combining this with some other measure of body dimensions, however this was not reported in the paper. In another paper from the same group, abdominal fat was defined as midriff fat plus pelvic fat and this was achieved by measuring body fat using DEXA at specific body locations (Clifton *et al.*, 2004). Due *et al.* (Due *et al.*, 2004) derived measures of intra-abdominal fat using DEXA and anthropometry (combining measures of waist circumference, sagittal diameter and trunk fat percentage).

Clifton *et al.* (Clifton *et al.*, 2008) did not observe any differential effect of the high/low protein diets tested on centrally or peripherally located body fat stores at 1 year post randomisation. However, Due *et al.* (Due *et al.*, 2004) reported a greater decrease in intra-abdominal adipose tissue in participants allocated to a high protein/lower carbohydrate diet compared with a moderate protein/higher carbohydrate diet at 1 year post randomisation (and at 6 months - data not in table). This was despite the fact that total fat mass was not differentially affected by the diets at 1 year. Additionally, the difference between dietary groups was still evident after statistical adjustment for body weight loss ($p<0.05$).

Table 5.38 Body fat distribution and total carbohydrates: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Means outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
13672 (Ludwig *et al.*, 1999) The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, Not diabetic	18-30 %M 45.9	5115	10 years	FFQ (700)	Total carbohydrate (% energy)	Waist to hip ratio Measured by clinician/ professional	Race - White	Q5 vs Q1 (51.9) vs (33.5)		0.801 vs. 0.805			0.21	Age, Alcohol, Centre, Education, Energy intake physical activity, Gender, Smoking, Baseline W:H, Vitamin use
13673 The CARDIA Study								Race - Black	Q5 vs Q1 (51.9) vs (33.5)		0.806 vs. 0.815			0.11	As above
13162 (Halkjaer *et al.*, 2006) Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M 47	56506	5 years (21)	FFQ (192)	Carbohydrate density (g/unit energy. Total CHO)	Waist circumference change Measured by clinician/ professional	Women		1 MJ/day		0.07 (-0.05, 0.19)	ns		Age, Alcohol, Baseline EI, Waist, BMI, PA, Smoking
13176 Danish Diet, Cancer and Health Study								Men		1 MJ/day		0.04 (-0.05, 0.12)	ns		As above
13173 Danish Diet, Cancer and Health Study						Carbohydrate density (g/unit energy. CHO from whole-grain foods)		Women		1 MJ/day		0.15 (-0.06, 0.36)	0.16		As above
13177 Danish Diet, Cancer and Health Study								Men		1 MJ/day		0.08 (-0.06, 0.22)	0.27		As above
13174 Danish Diet, Cancer and Health Study						Carbohydrate density (g/unit energy. CHO from refined-grain products and potatoes)		Women		1 MJ/day		0.48 (0.18, 0.78)	0.002		As above

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Means outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
13178 Danish Diet, Cancer and Health Study								Men		1 MJ/day		0.06 (-0.12, 0.25)	0.49		As above
13175 Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M 47	56506	5 years (21)	FFQ (192)	Carbohydrate density (g/unit energy. CHO from foods with simple or added sugars)	Waist circumference change Measured by clinician/ professional	Women		1 MJ/day		0.39 (0.18, 0.6)	<0.001		As above
13179 Danish Diet, Cancer and Health Study								Men		1 MJ/day		0.09 (-0.06, 0.23)	0.24		As above

Table 5.39 Skinfold thickness and total carbohydrates: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14164 (Boreham *et al.*, 1999) The Northern Ireland Young Hearts Project	Northern Ireland, Primarily White	12-15 %M 49.3	509	4 years (1.7)	Dietary history	Carbohydrate, total (% energy)	Sum of skinfolds (subscapular, triceps, biceps, suprailiac)	Males	1 % Energy	Beta coefficient not reported		NS	Social Class, Sexual maturity
14212 The Northern Ireland Young Hearts Project								Females	1 % Energy	Beta coefficient not reported		NS	Social Class, Sexual maturity
13364 (Koppes *et al.*, 2009) Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 48	698	23 years (50)	Dietary history	Carbohydrate, total (% energy)	Sum of skinfolds (subscapular, triceps, biceps, suprailiac)	Males	1 % Energy	-0.004	0.49		Age, Smoking, Education
13371 Amsterdam Growth and Health Study								Females	1 % Energy	-0.003	0.61		Age, Smoking, Education

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
14613 (Magarey *et al.*, 2001) Adelaide Longitudinal Study of Growth & Nutrition	Australia	2-15 %M 58	500	13 years	Food diary	Carbohydrate, total (% energy)	Triceps skinfold		1 % Energy	-0.003 (0.001)	0.02		Energy intake, gender, parental BMI, BMI at previous age
14614 Adelaide Longitudinal Study of Growth & Nutrition									1 % Energy	-0.003(0.001)	0.006		Energy intake, gender, parental BMI, BMI at previous age
13309 (Twisk *et al.*, 1998) Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 46	233	14 years (22)	Dietary history	Carbohydrate, total (grams/day)	Sum of skinfolds (subscapular, triceps, biceps, suprailiac)		1 g/day	-0.02 (-0.06, 0.01)	0.24		Time, Gender
13308 Amsterdam Growth and Health Study						Carbohydrate, total (% energy)			1 % Energy	0 (-0.03, 0.03)	0.96		Time, Gender
13284 (Van Lenthe *et al.*, 1998) Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 46	233	14 years (22)	Dietary history	Carbohydrate, total (% energy)	Subscapular skinfold	Males	1 % Energy	-0.01 (-0.05, 0.05)	0.56		Baseline subscapular skinfold
13286 Amsterdam Growth and Health Study								Females	1 % Energy	0.09 (0.02, 0.16)	0.01		Baseline subscapular skinfold

Table 5.40 Adiposity and total carbohydrate: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Exposure Units	Mean exposure (SD)	p	Adjustments

13374 (Johnson *et al.*, 2008) ALSPAC	UK, Age 5-10y	(5) %M 54	(97) /682	4 years (25)	Food diary	Carbohydrate, total (% energy)	Adiposity (Fat Mass Index) >80th centile DEXA	1 % Energy	Cases: (n: 97) 53 Non cases: (n: 424) 54	NS	Anthropometrics at baseline; energy intake; fat intake; fibre intake; drinks; TV parental BMI; parental SES

Table 5.41 Body fat distribution and dietary pattern analysis: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13457 (Koh-Banerjee *et al.*, 2003) HPFS	USA, Primarily White, Cancer free, No heart disease, Not diabetic	40-75 %M 100	51529	9 years (35)	FFQ (131)	CHO replaced by **trans fat** (Isoenergetic replacement of carbohydrates with trans fats)	Waist circumference change Self-reported	2 %	0.77 (0.21)	<0.001	Age, Alcohol, BMI, Waist, Smoking, Dietary changes, energy intake, fat intake, physical activity, Protein
13458 HPFS						CHO replaced by **saturated fat** (Isoenergetic replacement of carbohydrates with saturated fats)	Waist circumference change Self-reported	2 %	0.27 (0.07)	<0.001	As above
13460 HPFS						CHO replaced by **polyunsaturated fat** (Isoenergetic replacement of carbohydrates with polyunsaturated fats)	Waist circumference change Self-reported	2 %	0.02 (0.08)	0.82	As above
13459 HPFS						CHO replaced by **monounsaturated fat** (Isoenergetic replacement of carbohydrates with monounsaturated fats)	Waist circumference change Self-reported	2 %	-0.02 (0.05)	0.72	As above

Table 5.42 Fat-free mass and high carbohydrate diets: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15966 (Dale *et al.*, 2009)	High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.4 (CI -0.3, 1.1)	Fat free mass	Biompedance (kg)	2 years	Decrease in both	Unclear
17364	High carbohydrate diet	89/100	48.6 (SD 7.4)	47.7 (SD 7.4)			0.31		Fat free mass	Biompedance (kg)	2 years	Decrease	Unclear
	High MUFA diet	85/100	48.7 (SD 6.4)	48.2 (SD 6.9)								Decrease	
17390	High carbohydrate diet	89/100	48.6 (SD 7.4)	48.0 (SD 7.3)			0.31		Fat free mass	Biompedance (kg)	1 year	Decrease	Unclear
	High MUFA diet	85/100	48.7 (SD 6.4)	48.6 (SD 7.1)								Decrease	
*15321 (Delbridge *et al.*, 2009)	Low fat, high carbohydrate weight maintenance diet	70/70			0.85 (SE 0.56)				Fat free mass	Biompedance (kg)	1 year	Increase	Unclear
	Low fat, high protein weight maintenance diet	68/71			-0.17 (SE 0.38)		0.149					Increase	
*16030 (Due *et al.*, 2004)	High protein	18/23			-0.4 (CI -1.2, 0.4)				Fat free mass	DEXA (kg)	1 year	Decrease	Unclear
	Moderate protein	23/18			-0.9 (CI -1.8, 0)							Decrease	
*15155 (Frisch *et al.*, 2009)	High carbohydrate diet	100/100			-1.3 (SD 3.6)	0.05			Fat free mass	Bioimpedance (kg)	1 year	Decrease	Unclear
	Moderate carbohydrate diet	100/100			-1.5 (SD 2.5)	0.05	0.794					Decrease	

*This result was used in the meta-analysis for high carbohydrate diets and fat-free mass (Figure 5.6)

Table 5.43 Total body fat and high carbohydrate diets: RCT data

Result ID/ Author	Subgroup detail	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
Adolescent studies														
15398 (Demol *et al.*, 2009)		High carbohydrate, low fat	20/20	39.3 (SD 1.8)	37.6 (SD 2.2)					Total body fat	Bioimpedance (%)	1 year	Decrease	Bias
		Low carbohydrate, high fat	17/17	42.0 (SD 2.0)	42.3 (SD 2.3)			NS					Decrease	
		Low carbohydrate, high protein	18/18	43.2 (SD 1.9)	38.9 (SD 2.2)			NS					Decrease	
15020 (Ebbeling *et al.*, 2003)		Low fat diet	8/8			1.6 (SE 0.9)				Fat mass development	DEXA (kg)	1 year	No change	Bias
		Low GL diet	8/8			-2.6 (SE 1.5)		0.01					Decrease	
Adult studies														
17431 (Clifton *et al.*, 2008)		High carbohydrate diet	38/38			3.5 (SD 3.8)				Fat mass	DEXA (kg)	1.25 years	Decrease	Unclear
		High protein diet	40/41			3.5 (SD 3.8)							Decrease	
15979 (Dale *et al.*, 2009)		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.5 (CI -0.9, 2)	Fat mass	Biompedance (kg)	2 years	Decrease in both	Unclear
17365		High carbohydrate diet	89/100	37.0 (SD 9.6)	35.4 (SD 9.9)					Fat mass	Biompedance (kg)	2 years	Decrease	Unclear
		High MUFA diet	85/100	36.9 (SD 8.1)	35.8 (SD 9.3)								Decrease	
17391		High carbohydrate diet	89/100	37.0 (SD 9.6)	35.5 (SD 10.5)					Fat mass	Biompedance (kg)	1 year	Decrease	Unclear
		High MUFA diet	85/100	36.9 (SD 8.1)	36.0 (SD 9.9)								Decrease	
**15320 (Delbridge *et al.*, 2009)		Low fat, high carbohydrate weight maintenance diet	70/70			1.8 (SE 1)				Fat mass	Bioimpedance (kg)	1 year	Increase	Unclear
		Low fat, high	68/71			3 (SE 1.2)		0.43					Increase	

Result ID/ Author	Subgroup detail	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		protein weight maintenance diet												
**16029 (Due *et al.*, 2004)		High protein	23/23			-4.6 (CI -6.6, -2.7)				Fat mass	DEXA (kg)	1 year	Decrease	Unclear
		Moderate protein	18/18			-3.1 (CI -4.7, -1.4)							Decrease	
*15446 (Ebbeling *et al.*, 2007)		Low fat diet	37/37			-1.1 (SE 0.3)				percentage body fat	DEXA (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-1.5 (SE 0.4)		0.5					Decrease	
15447	Insulin (30m post 75gdose) <57.5µUI/ ml	Low fat diet	37/37			-1.4 (SE 0.6)				percentage body fat	DEXA (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-0.9 (SE 0.5)		0.56					Decrease	
15448	Insulin (30m post 75gdose) >57.5µUI/ ml	Low fat diet	37/37			-0.9 (SE 0.5)				percentage body fat	DEXA (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-2.6 (SE 0.6)		0.3					Decrease	
*15154 (Frisch *et al.*, 2009)		High carbohydrate diet	100/100			-3.3 (SD 4.4)	0.05			Total body fat	Bioimpedance (kg)	1 year	Decrease	Unclear
		Moderate carbohydrate diet	100/100			-4.2 (SD 4.9)	0.05	0.132					Decrease	
*15106 (Gardner *et al.*, 2007)		Atkins: low carbohydrate	77/77			-2.9 (SD 4.8)		NS		Percentage body fat	DEXA (%)	1 year	Decrease	No bias
		Ornish: high carbohydrate	76/76			-1.5 (SD 4)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			-1.3 (SD 3.4)							Decrease	
**14956 (Layman *et al.*, 2009)		High carbohydrate, low protein diet	51/66			4.9 (SE 0.54)	NS			Fat mass development	DEXA (kg)	1 year		Unclear
		Low carbohydrate, high protein diet	52/64			6.6 (SE 0.7)	<0.05							
14894		Low fat diet	13/51			-2.0 (SD 6)	NS			Body fat change	Near-infrared light	1 year	Decrease	Unclear

Result ID/ Author	Subgroup detail	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(McManus *et al.*, 2001)		Moderate fat diet	27/50			-5.0 (SD 5)	<0.001			(%)	interactance device		Decrease	
14895		Low fat diet	10/51			-3.0 (SD 4.0)	NS			Body fat change (%)	Near-infrared light interactance device	18 months	Increase	Unclear
		Moderate fat diet	24/50			-3.0 (SD 5.0)	<0.01						Decrease	
14897		Low fat diet	13/51			-3.5 (SD 6.4)	NS			Total body fat change	Near-infrared light interactance device (kg)	1 year	Decrease	Unclear
		Moderate fat diet	27/50			-5.9 (SD 5.8)	<0.001						Decrease	
14898		Low fat diet	10/51			-3.0 (SD 6.3)	NS			Total body fat change	Near-infrared light interactance device (kg)	18 months	Increase	Unclear
		Moderate fat diet	24/50			-4.2 (SD 5.1)	<0.001						Decrease	

*This result was used in the meta-analysis for carbohydrate/fat swap and total body fat (Figure 5.7)

**This results was used in the meta-analysis for carbohydrate/protein and carbohydrate/fat and protein swap and total body fat (Figure 5.8)

Table 5.44 Waist circumference and high carbohydrate diets: RCT

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
*16860 (Bhargava, 2006)	Control	379/allocated not reported	0.86 (SD 0.11)	0.86 (SD 0.11)	0	NS			Waist circumference	Measured by clinician/ professional (m)	1 year	Decrease	Unclear
	Low fat	615/allocated not reported	0.87 (SD 0.11)	0.85 (SD 0.11)	-0.02	0.05	0.05					Decrease	
15976 (Dale *et al.*, 2009)	High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.3 (CI -1.5, 2.1)	Waist circumference	Measured by clinician/ professional (cm)	2 years	Decrease in both	Unclear
17363	High carbohydrate diet	89/100	94 (SD 13.1)	90.3 (SD 12.5)					Waist circumference	Measured by clinician/ professional (cm)	2 years	Decrease	Unclear
	High MUFA diet	85/100	95.5 (SD 11.8)	91.4 (SD 11.7)								Decrease	

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
17385	High carbohydrate diet	89/100	94 (SD 13.1)	91.3 (SD 13.1)					Waist circumference	Measured by clinician/ professional (cm)	1 year	Decrease	Unclear
	High MUFA diet	85/100	95.5 (SD 11.8)	93.0 (SD 12.3)								Decrease	
**15694 (Dansinger *et al.*, 2005)	Atkins	40/40			-2.5 (SD 4.5)	0.01			Waist circumference	Measured by clinician/ professional (cm)	1 year	Decrease	No bias
	Ornish	40/40			-2.2 (SD 5.5)	0.05						Decrease	
	Weight watchers	40/40			-3.3 (SD 5.4)	0.01						Decrease	
	Zone	40/40			-2.9 (SD 5.3)	0.01						Decrease	
**15317 (Delbridge *et al.*, 2009)	Low fat, high carbohydrate weight maintenance diet	70/70			0.05 (SE 0.94)				Waist circumference	Measured by clinician/ professional (cm)	1 year	Increase	Unclear
	Low fat, high protein weight maintenance diet	68/71			0.68 (SE 0.72)		0.599					Increase	
**16038 (Due *et al.*, 2004)	High protein	23/23			-8.4 (CI -10.5, -6.3)				Waist circumference	Measured by clinician/ professional (cm)	1 year	Decrease	Unclear
	Moderate protein	18/18			-1.8 (CI -5.5, -1.8)							Decrease	
*15159 (Frisch *et al.*, 2009)	High carbohydrate diet	100/100			-4.7 (SD 8.9)	0.05			Waist circumference	Measured by clinician/ professional (cm)	1 year	Decrease	Unclear
	Moderate carbohydrate diet	100/100			-6.9 (SD 6.1)	0.05	0.037					Decrease	
(Howard *et al.*, 2006) *16242	Control	9517/29294	89.0 (SD 13.7)	90.4 (SD 14.2)	1.9 (SD 8.8)				Waist circumference	Measured by clinician/ professional (cm)	90 months	No change	No bias
	Low fat diet	6154/19541	89.0 (SD 13.9)	90.1 (SD 14.1)	1.6 (SD 8.6)		0.04					Decrease	
**15598 (Keogh *et al.*, 2007)	High carbohydrate diet	completers not reported/12			-7 (SE 0.7)	0.01			Waist circumference	Measured by clinician/ professional (cm)	1 year	Decrease	Unclear
	Low carbohydrate diet	completers not reported/13			-4 (SE 1.6)	0.01						Decrease	
14900 (McManus *et al.*, 2001)	Low fat diet	13/51			-1.6 (SD 9.2)	NS			Waist circumference change	Measured by clinician/ professional (cm)	1 year	Decrease	Unclear
	Moderate fat diet	27/50			-7.3 (SD 6.3)	<0.001						Decrease	
14901	Low fat diet	30/51			2.6 (SD 10.5)	NS			Waist	Measured by	18 months	Increase	Unclear

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
	Moderate fat diet	31/50			-6.9 (SD 9.1)	<0.001	<0.001		circumference change	clinician/ professional (cm)		Decrease	
16013 (Sacks *et al.*, 2009)	High-fat, average-protein	ITT: /204					NS		Waist circumference change	Measured by clinician/ professional (cm)	1 years	Decrease	No bias
	High-fat, high-protein	ITT: /201					NS					Decrease	
	Low-fat, average-protein	ITT: /204					NS					Decrease	
	Low-fat, high-protein	ITT: /202					NS					Decrease	
16016	High-fat, average-protein	ITT: /204					NS		Waist circumference change	Measured by clinician/ professional (cm)	18 months	Decrease	No bias
	High-fat, high-protein	ITT: /201					NS					Decrease	
	Low-fat, average-protein	ITT: /204					NS					Decrease	
	Low-fat, high-protein	ITT: /202					NS					Decrease	
16017	High-fat, average-protein	ITT: /204					NS		Waist circumference change	Measured by clinician/ professional (cm)	2 years	Decrease	No bias
	High-fat, high-protein	ITT: /201					NS					Decrease	
	Low-fat, average-protein	ITT: /204					NS					Decrease	
	Low-fat, high-protein	ITT: /202					NS					Decrease	
15365 (Tinker *et al.*, 2008)	Control	24800/29294	28.9 (SD 13.4)	87.9 (SD 13.3)					Waist circumference	Not reported (cm)	1 year	No change	Unclear
	Low fat diet	16864/19541	88.3 (SD 13.6)	86.3 (SD 13.4)			0.001					Decrease	
15366	Control	8092/29294	28.9 (SD 13.4)	89.6 (SD 13.8)					Waist circumference	Not reported (cm)	6 years	No change	Unclear
	Low fat diet	5237/19541	88.3 (SD 13.6)	89.1 (SD 14)								Decrease	

*This result was used in the meta-analysis for carbohydrate/fat swap and waist circumference (Figure 5.9)

**This results was used in the meta-analysis for carbohydrate/protein and carbohydrate/fat and protein swap and waist circumference (Figure 5.10)

Table 5.45 Hip circumference and high carbohydrate diets: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
16854 (Bhargava, 2006)	Control	379/allocated not reported	1.08 (SD 0.09)	1.08 (SD 0.1)	0	NS		Hip circumference	Measured by clinician/ professional (m)	1 year	Decrease	Unclear
	Low fat	615/allocated not reported	1.09 (SD 0.09)	1.07 (SD 0.1)	-0.02	0.05	0.05				Decrease	
15318 (Delbridge *et al.*, 2009)	Low fat, high carbohydrate weight maintenance diet	70/70			0.43 (SE 0.72)			Hip circumference	Measured by clinician/ professional (cm)	1 year	Increase	Unclear
	Low fat, high protein weight maintenance diet	68/71			1.02 (SE 0.63)		0.534				Increase	

Table 5.46 Waist to Hip Ratio and high carbohydrate diets: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
*16866 (Bhargava, 2006)	Control	379/allocated not reported	0.79 (SD 0.07)	0.79 (SD 0.07)		NS		Waist to hip ratio	Measured by clinician/ professional	1 year	Decrease	Unclear
	Low fat	615/allocated not reported	0.8 (SD 0.08)	0.79 (SD 0.07)		NS	NS				Decrease	
15319 (Delbridge *et al.*, 2009)	Low fat, high carbohydrate weight maintenance diet	70/70			0 (SE 0)			Waist to hip ratio	Measured by clinician/ professional	1 year	Increase	Unclear
	Low fat, high protein weight maintenance diet	68/71			0 (SE 0)		0.482				Increase	
*16039 (Due *et al.*, 2004)	High protein	18/23			0 (CI -0.03, 0.04)			Waist to hip ratio	Measured by clinician/ professional	1 year	Decrease	Unclear
	Moderate protein	23/18			-0.04 (CI -0.02, -0.06)						Decrease	
	Zone: moderate carbohydrate	79/79			-1.3 (SD 3.4)						Decrease	

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
*15107 (Gardner *et al.*, 2007)	Atkins: low carbohydrate	77/77			-0.019 (SD 0.026)		NS	Waist to hip ratio	Measured by clinician/ professional	1 year	Decrease	No bias
	Ornish: high carbohydrate	76/76			-0.012 (SD 0.024)		NS				Decrease	
	Zone: moderate carbohydrate	79/79			-0.013 (SD 0.023)						Decrease	
(Howard *et al.*, 2006) *16243	Control	9487/29294	0.82 (SD 0.1)	0.83 (SD 0.1)	0.02 (SD 0.1)			Waist to hip ratio	Measured by clinician/ professional	90 months	No change	No bias
	Low fat diet	6123/19541	0.82 (SD 0.1)	0.83 (SD 0.1)	0.02 (SD 0.1)		0.85				Decrease	
14903 (McManus *et al.*, 2001)	Low fat diet	13/51			-0.03 (SD 0.08)	NS		Waist to hip ratio change	Measured by clinician/ professional	1 year	Decrease	Unclear
	Moderate fat diet	27/50			0 (SD 0.05)	NS					Decrease	
14904	Low fat diet	10/51			0 (SD 0.06)	NS		Waist to hip ratio change	Measured by clinician/ professional	18 months	Increase	Unclear
	Moderate fat diet	24/50			-0.02 (SD 0.05)	<0.05					Decrease	

*This result was used in the meta-analysis for high carbohydrate diets and waist to hip ratio (Figure 5.11)

Table 5.47 Other measures of fat distribution and high carbohydrate diets: RCT data

Result ID/ Author	Intervention group	Completers / Allocated	Baseline	Within group Δ from baseline	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
17432 (Clifton *et al.*, 2008)	High carbohydrate diet	38/38		2.0 (SD 1.8)	NS	Central fat	DEXA (g) detail of method not provided	1.25 years	Decrease	Unclear
	High protein diet	40/41		1.8 (SD 1.9)					Decrease	
17433	High carbohydrate diet	38/38		1.5 (SD 2.0)	NS	Peripheral fat	DEXA (kg) detail of method not provided	1.25 years	Decrease	Unclear
	High protein diet	40/41		1.4 (SD 2.1)					Decrease	
16041 (Due *et al.*, 2004)	High protein	23/23	120.3 (CI 111, 129.7)	-22 (CI -15, -29)	0.03	Intra-abdominal adipose tissue	DEXA (cm^2) estimated from sagittal diameter, waist circumference, trunk fat %	1 year	Decrease	Unclear
	Moderate protein	18/18	126.3 (CI 115.4, 137.2)	-10.5 (CI -0.1, -20.8)					Decrease	

Total body fat and carbohydrate supplements

No cohort data reported results on carbohydrate supplements and total body fat.

Summary of RCT data

One randomised double blind trial explored the effects of a 50g carbohydrate supplement (42% glucose, 58% maltodextrins) compared with a control (do nothing) intervention on body fat mass and fat free mass using bioimpedance measures over 16 months (Pasman *et al.*, 1997b). An initial 2 month weight loss phase with a very low energy diet was employed, and then the trajectory of weight gain was compared in the 2 groups over a further 14 months in the carbohydrate and control groups. A further group was also followed which consumed a combination supplement of carbohydrate, chromium-picolinate, caffeine and soluble fibre. These data were not extracted. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.48. No difference between dietary groups was observed for either of these outcomes.

nb. while the authors claim the study was double-blind, it is unclear how this was achieved without a placebo product.

Table 5.48 Total body fat and carbohydrate supplements: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Follow-up	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15504 (Pasman *et al.*, 1997b)	Carbohydrate	11/11	decrease	<0.05	NS	Fat free mass	Bioimpedance	14 months	Increase	Unclear
	Control	9/9	decrease	<0.05					Increase	
15507	Carbohydrate	11/11		NS	NS	Fat mass	Bioimpedance	14 months	Increase	Unclear
	Control	9/9		NS					Increase	

Body fatness and fat distribution and dietary fibre

Summary of cohort results in adults

Data were extracted from two publications, presenting results from two US cohorts: the CARDIA study and the HPFS (Ludwig *et al.*, 1999;Koh-Banerjee *et al.*, 2003) (see Table 5.49). Change in waist circumference was the outcome assessed in the HPFS; whilst the CARDIA study measured waist-to-hip ratio. Clinical or professional measurements were used in the CARDIA study, but not the HPFS. The evidence from these is generally supportive of a negative dietary fibre – body fat distribution relationship: the CARDIA study (Ludwig *et al.*, 1999) reported a significantly lower adjusted mean waist-to-hip ratio in the highest quintile fibre consumers compared with the lowest quintile; and in the HPFS (Koh-Banerjee *et al.*, 2003) each additional 12g of fibre was associated with a small but statistically significant decrease in waist circumference over the 9 years of follow-up (beta coefficient -0.63, $p<0.01$).

Exposure definition and assessment

Both studies employed FFQs to collect dietary data. One of the studies recruited only males (Koh-Banerjee *et al.*, 2003). The CARDIA study (Ludwig *et al.*, 1999) expressed fibre intake as density (per unit of energy), whereas the HPFS (Koh-Banerjee *et al.*, 2003) reported grams per day.

Adjustment for appropriate confounders

Both studies incorporated several important covariates in their analyses, including age, smoking, physical activity and dietary variables.

Summary of cohort results in children

Data were extracted from three publications, presenting analyses from the following three cohort studies: the DONALD Study, the ALSPAC study and the National Heart, Lung, and Blood Institute Growth and Health Study (Cheng *et al.*, 2009;Johnson *et al.*, 2008;Albertson *et al.*, 2009). None of the results support a strong relationship between dietary fibre and adiposity in children. The outcomes of the studies were also assessed quite differently: by skinfold measurements (Cheng *et al.*, 2009), DEXA (Johnson *et al.*, 2008) or bioempedence (Albertson *et al.*, 2009) (Table 5.50).

Exposure definition and assessment

Food diaries were the dietary collection method used in all studies; however the exposure was defined or expressed differently in each. The DONALD study (Cheng *et al.*, 2009) determined grams of fibre per day using the LEBTAB database, the ALSPAC study (Johnson *et al.*, 2008) determined fibre density based on Englyst fibre values, and the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) calculated the proportion of cereal consumed that was fibre. It is unclear in the latter publication if this refers to breakfast cereals only or to total cereals.

Adjustment for appropriate confounders

Both the DONALD study (Cheng *et al.*, 2009) and the ALSPAC study (Johnson *et al.*, 2008) included several important covariates in the analyses. The National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) included main effects only in the final model.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Two studies provided data on the effects of dietary fibre supplementation on body fat content and distribution in children and adults respectively (Abrams *et al.*, 2007;Pasman *et al.*, 1997a).

Abrams *et al.* (Abrams *et al.*, 2007) conducted an exploratory study of the effects of a prebiotic supplement containing oligofructose and inulin and habitual calcium intake on changes in body composition of pre-pubertal children. This randomised, double-blind trial included 97 children initially aged 9-13 years who were allocated to consume either an 8g/d prebiotic supplement or an equivalent 8g/d maltodextrin control supplement for one year. The prebiotic supplement group exhibited a significantly smaller increase in total fat mass than the control group ($p=0.02$) after one year, but the changes in body fat percentage were similar in both groups. The control group tended towards an exaggerated increase in adiposity, whereas the prebiotic group grew along recommended lines.

One study of adults provided data on body fat distribution and fibre (Pasman *et al.*, 1997a) (see Table 5.51). This study explored the effects of partially hydrolysed guar gum supplements on body fat % and waist to hip ratio in weight-reduced subjects over 16 months. Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.51. The consumption of 20 g of water soluble fibre (guar gum) per day for 14 months, following a 2 month very low calorie diet weight loss phase did not markedly alter percentage body fat change or change in waist to hip ratio compared with the control group, although the low compliance group tended to have higher values at 16 months. nb. this is a rather curious design – the paper does not clarify whether the decision to split into high and low compliance groups was made *a priori.*

Table 5.49 Body fat distribution and dietary fibre: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	P trend	Adjustments
13670 (Ludwig *et al.*, 1999) The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, Not diabetic	18-30 %M 45.9	5115	10 years	FFQ (700)	Fibre density (g/unit energy. AOAC method)	Waist to hip ratio Measured by clinician/ professional	Race - White	Q5 vs Q1 (12.3) vs (5.2)	g/4184kJ /day	0.801 vs. 0.813			0.004	Age, Alcohol, Centre, Education, Energy Intake, physical activity, Gender, Smoking, Vitamin intake, W:H
13671 The CARDIA Study								Race - Black	Q5 vs Q1 (12.3) vs (5.2)	g/4184kJ /day	0.799 vs. 0.809			0.05	As above
13456 (Koh-Banerjee *et al.*, 2003) HPFS	USA, Primarily White, Cancer free, No heart disease, Not diabetic	40-75 %M 100	51529	9 years (35)	FFQ (131)	Dietary Fibre, g/d (AOAC method)	Waist circumference change Self-reported			12 g/day		-0.63 (0.1)	<0.001		Age, Alcohol, BMI, Waist, Alcohol, physical activity, Smoking, Dietary changes, energy intake

Table 5.50 Adiposity and total body fat and dietary fibre and sources of fibre: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Mean exposure (SD)	Beta coefficient (SE)/(CI)	p	Adjustments
13983 (Albertson *et al.*, 2009) National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	7 years	Food diary	Fibre from cereals g/d (Percent of cereal consumed that was fibre. Cereal was defined as 'ready-to-eat cereal and cooked cereal', but it is unclear if this refers only to breakfast cereals or total cereals.)	Percentage body fat Bioimpedance	1 %		0.01 (0.07)	0.087	
14002 (Cheng *et al.*, 2009) The DONALD Study	Germany	(9) %M 46	376	4 years	Food diary	Dietary Fibre, g/d (The dietary fibre content in the LEBTAB database was determined by different enzymatic methods as defined in the respective food table.)	Body fat change (%) Skinfolds	1 g/day		0.016 (0.137)	0.9	Age, Breastfed, EI, GI, Parental overweight, Gender
14305 (Johnson *et al.*, 2008) ALSPAC	UK, Age 5-10y	(5) %M 54	(97) /682	4 years (25)	Food diary	Fibre density (Englyst fibre)	Adiposity (Fat Mass Index) >80th centile DEXA	g/MJ	Cases: (n: 97) 1.4 Non cases: (n: 424) 1.5			Anthropometrics at baseline; EI; fat intake; fibre intake; drinks; TV parental BMI; parental SES

Table 5.51 Body fat distribution and dietary fibre: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	p-value Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
Children											
(Abrams *et al.*, 2007)	Prebiotic supplement	48/50	24.7 (SE 0.9)	23.3 (SE 0.4)		0.14	Body fat %	Anthropometry and dual-energy X-ray absorptiometry (details not provided in paper)	12 months	Increase	unclear
	Control – maltodextrin supplement	49/50	24.5 (SE 0.9)	24.2 (SE 0.4)						Increase	
	Prebiotic supplement	48/50	10.7 (SE 0.6)	11.24 (SE 0.25)		0.02	Total fat mass	Anthropometry and dual-energy X-ray absorptiometry (details not provided in paper)	12 months	Increase	unclear
	Control – maltodextrin supplement	49/50	10.4 (SE 0.6)	12.07 (SE 0.25)						Increase	
Adults											
15537 (Pasman *et al.*, 1997a)	Control	11/14	36.4 (SD 6.0)	38.9 (SD 5.1)	<0.05		Body fat % (maintenance after weight loss)	Deuterium dilution technique (%)	14 months	Increase	Unclear
	Guar gum - High compliance	10/10	37.3 (SD 3.7)	42.1 (SD 3.3)	<0.05	NA				Increase	
	Guar gum - Low compliance	10/10	38.7 (SD 5.8)	44.6 (SD 4.5)	<0.05	<0.05				Increase	
15540	Control	11/14	0.81 (SD 0.05)	0.83 (SD 0.04)	<0.05		Waist to hip ratio (maintenance after weight loss)	Measured by clinician/ professional	14 months	Increase	Unclear
	Guar gum - High compliance	10/10	0.82 (SD 0.04)	0.82 (SD 0.04)	<0.05	NS				Increase	
	Guar gum - Low compliance	10/10	0.81 (SD 0.06)	0.83 (SD 0.05)	<0.05	NS				Increase	

Body fatness and fat distribution and foods high in added sugars

Summary of cohort results in adults

Data were extracted from two publications, presenting results from two Danish cohort studies: the Danish Diet, Cancer and Health Study and the MONICA study (Halkjaer *et al.*, 2009;Halkjaer *et al.*, 2004). Direct measurements of waist circumference were used in MONICA, but the Danish Diet, Cancer and Health Study used a combination of both direct and self-reported measurements. The data indicated no apparent trend between sugar consumption and waist circumference change in adults (see Table 5.52).

Exposure definition and assessment

Both studies collected dietary data via FFQ. The Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009) included jam, sugar, honey and fruit syrups in the exposure called 'jam, syrups, sugar'; whereas MONICA (Halkjaer *et al.*, 2004) considered a wider range of sweet snack foods, such as chocolate and ice cream.

Adjustment for appropriate confounders

The analyses of both studies included several important covariates in the analyses.

Summary of cohort results in children

Two publications presented results concerning body fatness in children and foods rich in added sugars (candy, baked goods and sugar content of breakfast cereals) (Phillips *et al.*, 2004;Albertson *et al.*, 2009). The Massachusetts Institute of Technology Growth and Development Study (Phillips *et al.*, 2004) and the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) measured body fatness using bioimpedence. The cohorts studied were both female-only. Data are not supportive of a relationship between sweet food consumption and body fatness measures in children (see Table 5.53).

Exposure definition and assessment

The studies assessed intake by FFQ (Phillips *et al.*, 2004) or food diary (Albertson *et al.*, 2009). The MIT Growth and Development Study (Phillips *et al.*, 2004) categorised sugar-sweetened foods into 'sweets' or 'baked goods'. The National Heart, Lung and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) considered only sugar content from breakfast cereals.

Adjustment for appropriate confounders

The MIT Growth and Development Study (Phillips *et al.*, 2004) adjusted for parental overweight and protein intake. The National Heart, Lung and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) did not report whether or not any covariates were included in the regression model.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs concerning specific foods rich in added sugars and body fat distribution were identified in this review.

Table 5.52 Body fat distribution (waist circumference) and foods rich in added sugars: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13132 (Halkjaer *et al.*, 2009) Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M47	56506	5 years (21)	FFQ (192)	Table sugar (jam, sugar, honey, fruit syrups)	Waist circumference change Both self reported and direct measures	Women	60 Kcal/day	0.05 (-0.03, 0.13)	NS	Age, Alcohol, Baseline energy intake, Waist, BMI, physical activity, Smoking
13157 Danish Diet, Cancer and Health Study								Men	60 Kcal/day	-0.0004 (-0.06, 0.06)	NS	As above
13844 (Halkjaer *et al.*, 2004) MONICA I	Denmark, Primarily White	30-60 (45) %M 48.9	2436	6 years	FFQ (26)	Sweet snack foods (Cakes, biscuits, sweets, chocolate, jam, honey, soft drinks and ice cream)	Waist circumference change Measured by clinician/ professional	Men	per quintile increase	0.01 (-0.22, 0.25)	NS	Age, Alcohol, BMI, Waist, cakes and chocolate, Dietary changes, Education, Hip circumference, physical activity, Smoking
13849 MONICA I								Women	per quintile increase	-0.05 (-0.35, 0.24)	NS	As above

Table 5.53 Total body fat and foods rich in added sugars: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Contrast (mean)	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13985 (Albertson *et al.*, 2009) National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	7 years	Food diary	Sugar from breakfast cereals (Percent of cereal consumed that was sugar)	Percentage body fat Bioimpedance		1 %	0.01 (0.02)	0.75	None reported
14188 (Phillips *et al.*, 2004) Massachusetts Institute of Technology Growth and Development Study	USA, Primarily White, BMI <30, Generally healthy	8-12 %M 0	196	6.9 years (28)	FFQ (116)	Candy (chocolate and non chocolate types)	Percentage body fat Bioimpedance	1. <2 2. 2 -3.4 3. ≥3.5	% Energy	1. Referent 2. -0.051 3. 0.066	1. N/A 2. 0.52 3. 0.96 p trend 0.35	Parental Overweight, Protein
14190 Massachusetts Institute of Technology Growth and Development Study						Baked goods (buns, pastries, cookies, cakes, pies and brownies)	Percentage body fat Bioimpedance	1. <2.5 2. 2.5- 3.9 3. ≥4	% Energy	1. Referent 2. -0.103 3. -0.221	1. N/A 2. 0.57 3. 0.23 p trend 0.23	Parental Overweight, Protein

Body fatness and fat distribution and carbohydrate rich food groups

Summary of cohort results in adults

Data were extracted from two publications, presenting results from two cohort studies (Halkjaer *et al.*, 2004;Halkjaer *et al.*, 2009), concerning waist circumference change in relation to several carbohydrate-rich food groups, including rice and pasta, breads and potatoes (see Table 5.54). Professional measurements of waist circumference were used in MONICA (Halkjaer *et al.*, 2004), but the Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009) used a combination of both direct and self-reported measurements. In the Danish Diet, Cancer and Health Study women, energy intake from potatoes was positively associated with the 5-year change in waist circumference in women, but not in men. In the MONICA study, none of the associations between high carbohydrate food groups and changes in waist circumference achieved statistical significance.

Generally, these two Danish studies do not provide evidence of an association between high carbohydrate foods and waist circumference change over time.

Exposure definition and assessment

Both studies collected dietary data via FFQ. MONICA (Halkjaer *et al.*, 2004) collected consumption data on a number of carbohydrate-rich food categories, including rice and pasta, wholewheat bread, white bread and potatoes. The only carbohydrate-rich food group investigated by the Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009) was potatoes.

Adjustment for appropriate confounders

The analyses of both studies included several important covariates in the analyses.

Summary of cohort results in children

Data were extracted from two publications, presenting results from two cohort studies in female children (Albertson *et al.*, 2009;Phillips *et al.*, 2004). Percentage body fat was measured using bioempedence in both studies. Analyses from the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) indicate a negative relationship between cereal consumption frequency and percentage body fat – that is a decrease in percentage body fat with increasing cereal consumption - which achieved statistical significance. Results from the MIT Growth and Development Study (Phillips *et al.*, 2004), on the other hand, were found to be non-significant, when comparing quartiles of savoury starch-based snacks.

Exposure definition and assessment

Dietary data were collected by food diary (Albertson *et al.*, 2009) or FFQ (Phillips *et al.*, 2004). Each publication focused on a different carbohydrate-rich food group: the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) on cereals, and the MIT Growth and Development Study (Phillips *et al.*, 2004) on savoury starch-based snacks, such as potato and corn crisps. It is unclear in the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) publication if cereal consumption refers to breakfast cereals only or to total cereals.

Adjustment for appropriate confounders

The MIT Growth and Development Study (Phillips *et al.*, 2004) adjusted for parental overweight and protein intake. The National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) included main effects only in the final model.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs concerning specific carbohydrate rich foods and body fat distribution were identified in this review.

Table 5.54 Body fat distribution and high carbohydrate foods: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Subgroup detail	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13841 (Halkjaer *et al.*, 2004) MONICA I	Denmark, Primarily White	30-60 (45) %M 48.9	2436	6 years	FFQ (26)	Total Rice and pasta	Waist circumference change Measured by clinician/ professional	Men	per quintile increase	-0.1 (-0.34, 0.15)	NS	Age, Alcohol, BMI, Waist, Dietary changes, Education, Hip, physical activity, Rice, Smoking
13846 MONICA I								Women	per quintile increase	-0.03 (-0.32, 0.27)	NS	As above
13842 MONICA I						Wholewheat bread (Wholegrain rye bread, wholegrain wheat bread, oatmeal)		Men	per quintile increase	-0.07 (-0.3, 0.17)	NS	Age, Alcohol, BMI, Waist, Dietary changes, Education, Hip, physical activity, Smoking, Wholemeal bread
13847 MONICA I								Women	per quintile increase	-0.2 (-0.49, 0.09)	NS	As above
13843 MONICA I						White bread (Refined wheat bread, refined rye bread)		Men	per quintile increase	-0.24 (-0.5, 0.01)	NS	Age, Alcohol, BMI, Waist, Dietary changes, Education, Hip, physical activity, Refined bread, Smoking
13848 MONICA I								Women	per quintile increase	0.42 (0.11, 0.73)	<0.05	As above
13810 MONICA I						Potatoes		Men	per quintile increase	-0.51 (-1.04, 0.02)	NS	Age, Alcohol, BMI, Waist, Dietary changes, Education, Hip, physical activity, Potatoes, Smoking
13845 MONICA I								Women	per quintile increase	0.16 (-0.44, 0.76)	NS	As above
13122 (Halkjaer *et al.*, 2009) Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M 47	56506	5 years (21)	FFQ (192)	Potatoes	Waist circumference change Both self reported and direct measures	Women	60 Kcal/day	0.1 (0.006, 0.19)	<0.05	Age, Alcohol, Baseline EI, Waist, BMI, PA, Smoking
13154 Danish Diet, Cancer and Health Study								Men	60 Kcal/day	-0.01 (-0.07, 0.05)	NS	As above

Table 5.55 Body fat distribution and high carbohydrate foods: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assess-ment	Exposure	Outcome/ Assessment Details	Contrast (mean)	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13396 (Albertson *et al.*, 2009) National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M0	2379	7 years	Food diary	Cereal consumption frequency (Percentage of days consuming cereal. Cereal was defined as 'ready-to-eat cereal and cooked cereal', but it is unclear if this refers only to breakfast cereals or total cereals.)	Percentage body fat Bioimpedance		1 %	-0.04 (0.01)	0.01	None reported
14189 (Phillips *et al.*, 2004) Massachusetts Institute of Technology Growth and Development Study	USA, Primarily White, BMI <30, Generally healthy	8-12 %M0	196	6.9 years (28)	FFQ (116)	Starchy snack foods - Potato and corn crisps	Percentage body fat Bioimpedance	1. <1 2. 1 to 1.4 3. 1.5 to 2.4 4. ≥2.5	1 % Energy	1. Referent 2. -0.054 3. -0.101 4. -0.161	1. N/A 2. 0.86 3. 0.66 4. 0.56 p trend 0.63	Parental Overweight, Protein

Body fatness and fat distribution and whole grains

Summary of cohort results in adults

Data were extracted from one publication, presenting results from one cohort study, the Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009). The beta-coefficients reported do not support a relationship between intake of wholegrain foods and 5 year change in waist circumference in either men or women. Dietary intake was assessed using a FFQ, and a combination of self-report and direct measures of waist circumference were taken. Analyses included important covariates such as age, BMI, physical activity and smoking behaviour (see Table 5.56).

Summary of cohort results in children

Data from one cohort study, the DONALD Study, examining wholegrain food intake and change in body fatness were extracted (see Table 5.57) (Cheng *et al.*, 2009). Consumption was collected from food diaries, and body fatness assessed using skinfold measures. The analysis took into account several important confounders, including age, energy intake and gender. The beta coefficient was not statistically significant.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Data from one intervention, the Women's Health Initiative Dietary Modification Trial are tabulated in Table 5.58, using two publications with different follow-up points (Howard *et al.*, 2006;Tinker *et al.*, 2008). Details concerning the design, participants, duration and nature of the intervention are included in Table 5.4.

The Women's Health Initiative Randomized Controlled Dietary Modification Trial was designed to test the hypothesis that a low fat, high fruit and vegetable (F&V), high grain diet would reduce the risk of cardiovascular disease in middle-aged and older women. The goal of the dietary intervention was to decrease total fat to 20% of energy intake, to increase F&V portions to 5 or more per day and to increase servings of grains to a minimum of 6 per day. Changes in outcome may therefore not be attributed solely to the increase in whole grain intake. There were no weight loss or energy restriction goals for either group.

Dietary change was implemented through a behavioural modification program that ran intensively throughout the first year of the trial and then less intensively thereafter. At the year 1 assessment, wholegrain consumption in the intervention group had increased by one third of a serving, whilst the comparison group remained unchanged (Tinker *et al.*, 2008). At 1 year, the intervention group had a lower waist circumference than the control group ($p<0.01$) and a small difference was still observed at the 90 month follow-up ($p<0.04$).

Data on waist to hip ratio were provided at 90 months follow-up. At this point, no differences between the dietary groups were observed. These data provide some evidence that a lower fat, higher fruit, vegetable and whole grain diet may minimise some of the gain in waist circumference typically observed in postmenopausal women.

Table 5.56 Body fat distribution and whole grains: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13131 (Halkjaer *et al.*, 2009) Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M 47	56506	5 years (21)	FFQ (192)	Total wholegrain foods: (Whole-grain bread, rye bread, whole-grain flour, oatmeal, corncobs, muesli, whole-grain crispbread)	Waist circumference change Both self reported and direct measures	Women	60 Kcal/day	0.03 (-0.01, 0.07)	NS	Age, Alcohol, Baseline EI, Waist, BMI, PA, Smoking
13156								Men	60 Kcal/day	0.01 (-0.01, 0.04)	NS	As above

Table 5.57 Total body fat and whole grains: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14003 (Cheng *et al.*, 2009) The DONALD Study	Germany	(9) %M 46	376	4 years	Food diary	Total wholegrain foods (Amaranth, whole buckwheat, bulgar, spelt, spelt flakes, barley flakes, whole barley, unripe spelt, whole and rolled oats, oat flakes, oat flour, millet, millet flakes, whole kamut wheat, corn bran, unpolished rice, popcorn, puffed rice, puffed wheat, puffed spelt, whole rice flour, rice flakes, wholemeal rye, rye flour, whole triticale, whole wheat, wheat germ, wheat bran, whole-wheat flakes, and whole-wheat flour)	Body fat change (%) Skinfolds	1 g/day	0.089 (0.128)	0.5	Age, Breastfed, EI, GI, Parental overweight, Gender

Table 5.58 Body fat distribution and whole grains: RCT data

Result ID/ Author	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
16242 (Howard *et al.*, 2006)	Control	9517/29294	89.0 (SD 13.7)	90.4 (SD 14.2)	1.9 (SD 8.8)		Waist circumference	Measured by clinician/ professional (cm)	90 months	No change	No bias
	Low fat diet	6154/19541	89.0 (SD 13.9)	90.1 (SD 14.1)	1.6 (SD 8.6)	0.04				Decrease	
16243	Control	9487/29294	0.82 (SD 0.1)	0.83 (SD 0.1)	0.02 (SD 0.1)		Waist to hip ratio	Measured by clinician/ professional	90 months	No change	No bias
	Low fat diet	6123/19541	0.82 (SD 0.1)	0.83 (SD 0.1)	0.02 (SD 0.1)	0.85				Decrease	
15365 (Tinker *et al.*, 2008)	Control	24800/29294	28.9 (SD 13.4)*	87.9 (SD 13.3)			Waist circumference	Not reported (cm)	1 year	No change	unclear
	Low fat diet	16864/19541	88.3 (SD 13.6)	86.3 (SD 13.4)		0.001				Decrease	
15366	Control	8092/29294	28.9 (SD 13.4)*	89.6 (SD 13.8)			Waist circumference	Not reported (cm)	6 years	No change	unclear
	Low fat diet	5237/19541	88.3 (SD 13.6)	89.1 (SD 14)		NS				Decrease	

*this value is reported in the paper – clearly a mistake

Body fat distribution and refined grains

Summary of cohort results in adults

Data were extracted from one cohort study, the Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009) (see Table 5.59). The beta-coefficients reported do not support a relationship between intake of refined grain foods and change in waist circumference in either men or women. Dietary intake was assessed using a FFQ, and a combination of self-report and direct measures of waist circumference were taken. Analyses included important covariates such as age, BMI, physical activity and smoking behaviour.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No RCTs concerning refined grains and body fat distribution were identified in this review.

Table 5.59 Body fat distribution and refined grains: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) / Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group detail	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
13126 (Halkjaer *et al.*, 2009) Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M 47	56506	5 years (21)	FFQ (192)	Refined grain foods, total (white bread, wheat flour, rice, potato flour, cornflour/starch, pasta, breadcrumbs, crispbread (wheat/refined))	Waist circumference change Both self reported and direct measures	Women	60 Kcal/day	0.07 (-0.01, 0.14)	NS	Age, Alcohol, Baseline energy intake, Waist, BMI, physical activity, Smoking
13155 Danish Diet, Cancer and Health Study								Men	60 Kcal/day	0.03 (-0.02, 0.08)	NS	As above

Body fatness and fat distribution and glycaemic index or load

Summary of cohort results in adults

Data were extracted from two publications, presenting results from two cohorts (Hare-Bruun *et al.*, 2006;Du *et al.*, 2009). Anthropometry was directly assessed in the MONICA study, but a combination of direct measures and self-reported data was used in the EPIC study (see Table 5.60).

The MONICA study (Hare-Bruun *et al.*, 2006) investigated both dietary glycaemic index and load in relation to hip circumference and also waist circumference. The EPIC study (Du *et al.*, 2009) focused only on glycaemic index. Statistical significance was not reported separately for each subgroup for the quintile comparisons in the MONICA study (Hare-Bruun *et al.*, 2006), however none of the p values for men and women combined indicated statistical significance. Nor was statistical significance achieved by any of the beta coefficients reported in this study. The results of the EPIC study, on the other hand, showed a small but significant positive direction of change with increasing dietary glycaemic index.

Exposure definition and assessment

The glycaemic index (GI) is a relative measure of the plasma glucose response induced by a specific food, as compared to the response induced by the same amount of carbohydrate from a reference source, such as white bread or pure glucose (Liu *et al.*, 2000). The glycaemic load (GL) is the product of a specific food's GI and its carbohydrate content (Liu *et al.*, 2000), therefore taking into account both the quality and quantity of carbohydrate consumed. This may be interpreted as a measure of diet-induced insulin demand (Stevens *et al.*, 2002).

Intake was assessed using a dietary history in the MONICA study (Hare-Bruun *et al.*, 2006) and FFQ in EPIC (Du *et al.*, 2009). The studies used different reference sources to calculate GI values: white bread in the MONICA study (Hare-Bruun *et al.*, 2006) and glucose was used for the EPIC study (Du *et al.*, 2009).

Dietary GI and GL were calculated by summing the products of the GI for each food multiplied by its carbohydrate content per serving multiplied by the average number of servings of that food per day (to give dietary GL), then dividing by the average daily carbohydrate intake to give dietary GI:

Dietary GI = $\{\sum[(\text{servings of food per day}) \times (\text{CHO content}) \times \text{GI})]\}/\text{total CHO}$ (Meyer *et al.*, 2000).

The glycaemic index (and thus also GL) is determined not only by the nature of the carbohydrate component of a food or diet, but also by the types and amounts of protein, fat and dietary fibre, as well food processing and storage (Venn and Green, 2007). Unless tightly controlled in an experimental situation, in most cases high and low GI/GL diets differ in many ways other than the carbohydrate fraction, including dietary fibre content, energy density and sensory quality.

Adjustment for appropriate confounders
Both studies included important covariates in their adjustments.

Summary of cohort results in children

Data were extracted from one publication, presenting results from the DONALD Study (Cheng *et al.*, 2009). The study included both dietary glycaemic index and load as an exposure, with the outcome of body fat change, measured using skinfold thicknesses. None of the results were indicative of an association. Analyses were adjusted for several confounders, including age, energy intake, parental overweight, gender, whether breastfed, and fibre intake.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

Three trials explored the effects of manipulation of dietary GI or GL on body fat distribution (Carels *et al.*, 2005;Ebbeling *et al.*, 2007;Das *et al.*, 2007). Details concerning the design, participants, duration and nature of the interventions are included in Table 5.4 and results are included in Table 5.62.

All three studies were included in a meta-analysis. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from 12 months to 18 months.

The study by Carels *et al.* (Carels *et al.*, 2005) was a comparison between the LEARN weight loss program (low energy and fat diet with enhanced physical activity) with and without low GI education. The difference in average dietary glycaemic index and load between the two groups was small, and total carbohydrate and energy intakes were similar (GL in higher vs. lower group respectively 70.9 g/1000kcal and 67.0 g/1000kcal). The study by Ebbeling *et al.* (Ebbeling *et al.*, 2007) compared an *ad libitum* low GL diet (35g/1000 kcal) with an *ad libitum*, higher GL, low fat diet (70g/1000 kcal) in obese young adults. Reported energy and fibre intakes were similar between groups. The CALERIE study (Das *et al.*, 2007) compared high GI/GL (GL 116g/1000kcal) and low GI/GL (45g/1000kcal) diets that were equally energy restricted and similar in dietary fibre content.

The overall pooled estimate indicated that fat mass was 0.56kg (95% CI, -.63 to 1.75) higher with consumption of a higher GI or GL diet but this was not significantly different from zero (p=0.36).

Heterogeneity denoted by I^2 was 25% (95% CI, 0 to 92). Statistically, there was no evidence of a difference in fat mass with differences in dietary glycaemic index or glycaemic load.

Figure 5.12 Forest plot for high glycaemic index or glycaemic load diets and body fat mass (kg)

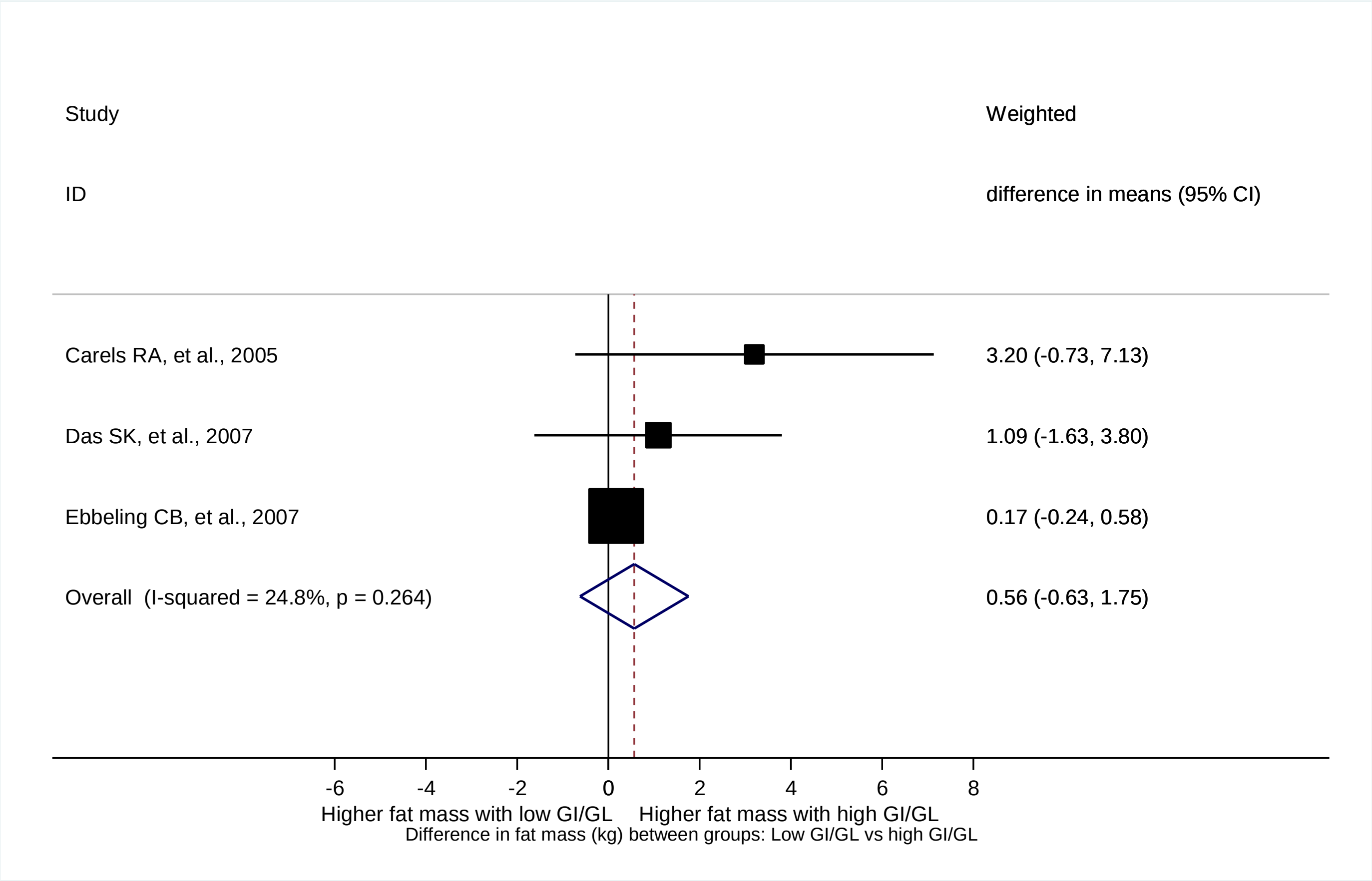


Table 5.60 Body fat distribution and glycaemic index and load: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/Assessment Details	Sub-group detail	Contrast (mean)	Expo sure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	P trend	Adjustments
13827 (Hare-Bruun *et al.*, 2006) MONICA	Denmark, Primarily White, Not diabetic	30-60 (45) %M 48.9	552	6 years	Dietary history	Glycaemic load	Waist circumference change Measured by clinician/ professional	Men	Q5 vs Q1		2.8 (5) vs. 1.3 (5.4)			
13828 MONICA							Hip circumference change Measured by clinician/ professional	Men	Q5 vs Q1		1.1 (2.7) vs. 0.8 (3.7)			
13831 MONICA							Waist circumference change Measured by clinician/ professional	Women	Q5 vs Q1		3.5 (5.1) vs.4.6 (6.2)			
13832 MONICA							Hip circumference change Measured by clinician/ professional	Women	Q5 vs Q1		2.7 (4) vs. 2.2 (5)			
13819 MONICA						Glycaemic index	Waist circumference change Measured by clinician/ professional	Men	Q5 vs Q1		1.9 (5) vs. 1.5 (5.9)			
13820 MONICA							Hip circumference change Measured by clinician/ professional	Men	Q5 vs Q1		1.2 (3.8) vs. 0.7 (3.3)			
13823 MONICA							Waist circumference change Measured by clinician/ professional	Women	Q5 vs Q1		2.4 (6.7) vs.2.4 (7.4)			
13824 MONICA							Hip circumference change Measured by clinician/ professional	Women	Q5 vs Q1		0.6 (4.6) vs.0.7 (5.6)			
13835 MONICA							Waist circumference change Measured by clinician/ professional	Men		1 Unit		0.02 (-0.14, 0.17)	NS	Age, Waist, Weight, Education, fat intake, Protein, energy intake, Fibre, Hip, physical activity, Smoking

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/Assessment Details	Sub-group detail	Contrast (mean)	Expo sure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	P trend	Adjustments
13836 MONICA							Hip circumference change Measured by clinician/ professional	Men		1 Unit		-0.003 (-0.1, 0.1)	NS	Age, Waist, Weight, Education, FAT, Protein, EI, Fibre, Hip, PA, Smoking
13827 (Hare-Bruun *et al.*, 2006) MONICA	Denmark, Primarily White, Not diabetic	30-60 (45) %M 48.9	552	6 years	Dietary history	Glycaemic index	Waist circumference change Measured by clinician/ professional	Women		1 Unit		0.16 (-0.05, 0.22)	0.07	Age, Waist, Weight, Education, FAT, Protein, EI, Fibre, Hip, PA, Smoking
13840 MONICA							Hip circumference change Measured by clinician/ professional	Women		1 Unit		0.08 (-0.05, 0.22)	NS	Age, Waist, Weight, Education, FAT, Protein, EI, Fibre, Hip, PA, Smoking
13658 (Du *et al.*, 2009) EPIC Denmark, Germany, Italy, The Netherlands and the United Kingdom	Europe, Cancer free, No heart disease, Not diabetic	20-78 (53) %M 40	146543	6.5 years (30)	FFQ	Glycaemic index	Waist circumference change Both self reported and direct measures			10 Units /day		0.26 (0.2, 0.33)		Age, Alcohol, Baseline weight, CHO, Smoking, Education, FAT, Fibre, Height, Meno Stat, PA, Protein, Gender

Table 5.61 Total body fat and glycaemic index and load: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14000 (Cheng *et al.*, 2009) The DONALD Study	Germany	(9) %M 46	376	4 years	Food diary	Glycaemic index	Body fat change (%) By skinfold thickness		1 Unit/day	-0.028 (0.113)	0.8	Age, Breastfed, EI, Fibre, Parental overweight, Gender
14009 The DONALD Study								BMI <25	1 Unit/day	0.018 (0.111)	0.9	As above
14010 The DONALD Study								BMI >25	1 Unit/day	0.088 (0.359)	0.8	As above

14001 The DONALD Study	Glycaemic load	Body fat change (%) By skinfold thickness	1 g/day	-0.009 (0.111)	0.9	As above

Table 5.62 Body fat percentage and glycaemic index and load: RCT data

Result ID/ Author	Subgroup detail	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
*14910 (Carels *et al.*, 2005)		Weight loss program	~19/~26	45.8 (SD 3.9)	41.9 (SD 5.5)		NS	percentage body fat	Bioimpedance (%)	1 year	Decrease	unclear
		Weight loss program + low GI education	~19/~26	42.5 (SD 4.0)	38.7 (SD 6.8)						Decrease	
*15225 (Das *et al.*, 2007)		Energy restricted high GL diet	15/17	35 (SD 7.1)		-14.8% (SD 8.8%)		percentage body fat	Air displacement plethysmography (BODPOD) (%)	1 year	Decrease	No bias
		Energy restricted low GL diet	14/17	35.2 (SD 8.7)		-17.9% (SD 12.5%)	NS				Decrease	
*15446 (Ebbeling *et al.*, 2007)		Low fat diet	37/37			-1.1 (SE 0.3)		percentage body fat	DEXA (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-1.5 (SE 0.4)	0.5				Decrease	
15447	Insulin (30m post 75gdose) <57.5µUI/ml	Low fat diet	37/37			-1.4 (SE 0.6)		percentage body fat	DEXA (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-0.9 (SE 0.5)	0.56				Decrease	
15448	Insulin (30m post 75gdose) >57.5µUI/ml	Low fat diet	37/37			-0.9 (SE 0.5)		percentage body fat	DEXA (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-2.6 (SE 0.6)	0.3				Decrease	

*This result was used in the meta-analysis for glycaemic index or glycaemic load diets and body fat percentage (Figure 5.12)

Body fatness and fat distribution and sweetened beverages

[Fruit juice data were only extracted from cohort studies which were already captured in the review. In accord with our original protocol, a specific search for studies reporting fruit juice consumption has not been undertaken.]

Summary of cohort results in adults

Data were extracted from two publications, presenting results from the Danish Diet, Cancer and Health Study and the Framingham Heart Study (Halkjaer *et al.*, 2009;Dhingra *et al.*, 2007) (table5.63). The studies used different outcome measures: waist circumference change was measured in the Danish Diet, Cancer and Health Study, and incident abdominal obesity in the Framingham Heart Study. The latter used professionally taken measurements, whereas in the Danish Diet, Cancer and Health Study relied on a combination of professionally measured and self-reported waist circumference data.

In the Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009) neither energy intake from sugar-sweetened soft drinks or from juices was significantly associated with 5-year difference in waist circumference in women. A decrease in waist circumference with increased consumption was suggested in men. Neither result was statistically significant, however. The authors commented that overall, soft drink consumption was low in this cohort.

The Framingham Heart Study (Dhingra *et al.*, 2007) reported a relative risk, as opposed to beta coefficients. The authors reported a 30% increased risk of incident abdominal obesity defined as a waist circumference in excess of 102cm in consumers of one or more sweetened (sugars and non-calorically sweetened combined) beverages per day compared to non consumers.

These 2 cohort studies of adults provide contradictory results with regard to body fat distribution and consumption of sweetened beverages.

Exposure definition and assessment

The Danish Diet, Cancer and Health Study (Halkjaer *et al.*, 2009) reported data separately on full-calorie soft drinks or fruit and vegetable juices; whilst the Framingham Heart Study (Dhingra *et al.*, 2007) considered sugar- and non-calorically sweetened beverages, without considering fruit juices (see note above).

Adjustment for appropriate confounders

Both studies included important covariates in their adjustments, such as age, energy intake, gender (where appropriate), physical activity and smoking behaviour.

Summary of cohort results in children

Data were extracted from five publications, presenting results from the following five cohort studies: the DONALD Study, the Pennsylvania Study of Health and Development of Young Girls, the MIT growth and development study, the Paediatric Bone Mineral Accrual Study and ALSPAC (Libuda *et al.*, 2008;Fiorito *et al.*, 2009;Johnson *et al.*, 2007;Mundt *et al.*, 2006;Phillips *et al.*, 2004) (see Table 5.64). Two cohorts did not recruit males (Phillips *et al.*, 2004;Fiorito *et al.*, 2009). Percentage body fat was the outcome measured in three of the studies (Libuda *et al.*, 2008;Fiorito *et al.*, 2009;Phillips *et al.*, 2004); total body fat in one study (Johnson *et al.*, 2007), and development of fat mass in another (Mundt *et al.*, 2006). The Pennsylvania Study of Health and Development of Young Girls (Fiorito *et al.*, 2009) additionally assessed waist circumference. The methods used for measuring body fat included skinfolds (Libuda *et al.*, 2008), DEXA (Fiorito *et al.*, 2009;Johnson *et al.*, 2007;Mundt *et al.*, 2006) and bioimpedence (Phillips *et al.*, 2004).

Four of the five cohort studies did not find statistically significant associations. The exception to this was the Pennsylvania Study of Health and Development of Young Girls (Fiorito *et al.*, 2009) which found that sweetened beverage intake at age 5 was a significant predictor of adiposity (DEXA measures) at ages 5 through 15 years after controlling for differences in energy intake at age 5, Parental education, family income, maternal BMI and sweetened beverage intake at the time of outcome assessment. This study also reported a statistically significant relationship between sugar-sweetened beverage consumption at age five and difference in waist circumference at ages 5-15 years (see Table 5.24). Girls drinking two or more servings per day at age 5 years had higher waist circumference at all subsequent ages compared with lower consumers ($p<0.05$).

Three studies provided data on fruit juice consumption and measures of adiposity (Libuda *et al.*, 2008;Fiorito *et al.*, 2009;Johnson *et al.*, 2007). None found any association.

The cohort studies presented here do not provide consistent evidence of a change in body fat amount or distribution with sweetened beverage consumption assessed in childhood or adolescence.

Exposure definition and assessment

Dietary data were collected by diet recall (3 days) (Fiorito *et al.*, 2009), food diaries (Libuda *et al.*, 2008;Johnson *et al.*, 2007) and FFQ (Phillips *et al.*, 2004) or a combination of FFQ and questionnaire (Mundt *et al.*, 2006). Three studies distinguished between sweetened drinks and 100% fruit juice (Libuda *et al.*, 2008;Fiorito *et al.*, 2009;Johnson *et al.*, 2007); two studies considered only sugar-sweetened beverages (Mundt *et al.*, 2006;Phillips *et al.*, 2004). One study also included non-calorically sweetened drinks, although the authors reported that at age 5 y, non-calorically sweetened beverages formed a small proportion of beverage intake and thus all sweetened beverages were grouped together (Fiorito *et al.*, 2009).

Adjustment for appropriate confounders

Each study included a different set of covariates in their analyses. The MIT Growth and Development Study (Phillips *et al.*, 2004) appeared to be less fully adjusted than the other studies.

Please interpret observational data with caution: With observational studies there is substantial potential for biases.

Summary of RCT data

No trials provided data concerning the effects of sweetened beverages on body fat distribution.

Table 5.63 Body fat distribution and sweetened beverages: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/Assessment Details	Sub-group detail	Contrast (mean)	Exposure Units	RR (CI)	Beta coefficient (SE)/(CI)	p	Adjustments
14261 (Dhingra *et al.*, 2007) The Framingham Heart Study	USA, No heart disease, Without metabolic syndrome	(53) %M43	(1302) /8997	4 years	Questionnaire (general)	Mixed sugar and non-calorically sweetened beverages	Incident Abdominal Obesity - Waist>102cm Measured by clinician/ professional		≥1 vs 0	12 oz serving/ day	1.3 (1.09, 1.56)			Age, Waist, Smoking, Ssaturated fat intake, energy intake, Fibre, GI, Magnesium Intake, Physical activity, Gender, trans-fat intake
13153 (Halkjaer *et al.*, 2009) Danish Diet, Cancer and Health Study	Denmark, Primarily White, Cancer free	50-64 (56) %M47	56506	5 years (21)	FFQ (192)	Full-calorie sugar sweetened soft drinks	Waist circumference change Both self reported and direct measures	Women		60 Kcal/day		0.01 (-0.24, 0.27)	NS	Age, Alcohol, Baseline energy intake, Waist, BMI, physical activity, Smoking
13160 Danish Diet, Cancer and Health Study								Men		60 Kcal/day		-0.02 (-0.12, 0.08)	NS	As above
17603 Danish Diet, Cancer and Health Study						Fruit and vegetable juices	Waist circumference change Both self reported and direct measures	Women		60 Kcal/day		-0.15 (-0.38, 0.09)	NS	As above
17604 Danish Diet, Cancer and Health Study								Men		60 Kcal/day		0.11 (-0.09, 0.31)	NS	As above

Table 5.64 Body fatness, fat distribution and sweetened beverages: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group details	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	Adjustments
13377 (Fiorito *et al.*, 2009) Pennsylvania Study of Health and Development of Young girls	USA, Primarily White, Age 5-18y	(5) %M 0	170	10 years (15)	Dietary recall	All sugar-sweetened and non-calorically sweetened beverages including fruit flavour drinks (<100% fruit juice), sports drinks and sodas. Authors report that non-calorically sweetened were small proportion	Percentage body fat at age 9 DEXA			1 serving/ day		0.3	<0.01	Energy intake, Income, Maternal BMI, Parental education, sugar-sweetened drinks intake at measurement age
14198							Percentage body fat at age 11 DEXA			1 serving/ day		0.2	<0.05	As above
14199							Percentage body fat at age 13 DEXA			1 serving/ day		0.21	<0.05	As above
14200							Percentage body fat at age 15 DEXA			1 serving/ day		0.17	<0.05	As above
17585						100% fruit juice	Percentage body fat at age 9 DEXA			1 serving/ day		0.02	NS	As above
17586							Percentage body fat at age 11 DEXA			1 serving/ day		0.03	NS	As above
17587							Percentage body fat at age 13 DEXA			1 serving/ day		0.00	NS	As above

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group details	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	Adjustments
17588							Percentage body fat at age 15 DEXA			1 serving/ day		-0.02	NS	As above
14202						All sugar-sweetened and non-calorically sweetened beverages including fruit flavour drinks (<100% fruit juice), sports drinks and sodas	Waist circumference at age 9 (cm) Measured by clinician/ professional		1.<1 2. ≥ 1 to 2 3.>2	Servings/ day	1. 65.5 (SD 7.7) 2. 66.4 (SD 8.3) 3. 72.5 (SD 9.9)		Main effect of beverage frequency group p<0.05	As above
14207							Waist circumference at age 11 (cm)		1.<1 2. ≥ 1 to 2 3.>2	Servings/ day	1. 71.5 (SD 9.7) 2. 74.1 (SD 11.6) 3. 78.1 (SD 10.6)			As above
14208							Waist circumference at age 13 (cm)		1.<1 2.≥ 1 to 2 3.>2	Servings/ day	1. 77 (SD 9.8) 2. 79 (SD 11.1) 3. 83.7 (SD 11.8)			As above
14209							Waist circumference at age 15 (cm)		1.<1 2.≥ 1 to 2 3.>2	Servings/ day	1. 77.8 (SD 9.3) 2. 79.1 (SD 10.6) 3. 81.6 (SD 11.9)			As above
13375 (Johnson *et al.*, 2007) ALSPAC	UK	(5) %M not reported	692	4 years (25)	Food diary	Full-calorie sugar sweetened beverages (fruit squash, cordial, fizzy drinks with added sugar)	Total body fat DEXA			1 serving/ day		-0.15 (-0.54, 0.24)	0.45	BMI, Education, Energy density, fat intake, Fibre, Height, TV, Parental overweight, SES/Class, Gender
17599 ALSPAC						100% fruit juices and concentrates	Total body fat DEXA			1 serving/ day		-0.11 -0.61, -0.38)	0.66	As above
13608 (Libuda *et al.*, 2008) The DONALD Study	Germany, Age 11-18y	9-18 (12) %M 51	1170	5 years	Food diary	Energy from regular soft drinks at baseline (carbonated and	Percentage body fat Skinfolds	Male		1 MJ/day		-0.033	0.92	Age, energy intake, Birth-weight, Maternal BMI, Maternal Education,

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases) /Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group details	Contrast (mean)	Exposure Units	Mean outcome (SD)	Beta coefficient (SE)/(CI)	p	Adjustments
						non-carbonated sugar sweetened drinks such as lemonades, iced tea and both diluted and sugar sweetened fruit juice drinks)								Assessment period, Year at birth, Years of adolescence
13609 The DONALD Study								Female		1 MJ/day		0.006	0.987	As above
17592 The DONALD Study						Energy from 100% fruit juice at baseline	Percentage body fat Skinfolds	Male		1 MJ/day		-0.058	0.874	As above
17596 The DONALD Study						Energy from 100% fruit juice at baseline	Percentage body fat Skinfolds	Female		1 MJ/day		-0.265	0.426	As above
14176 (Mundt *et al.*, 2006) Pediatric Bone Mineral Accrual Study	Australia, Primarily White	8-19 %M 50.4	208	5 years	FFQ / Question-naire	Full-calorie sugar sweetened beverages	Fat mass development DEXA	Female		1 Oz/day		-0.0188 (0.0118)	NS	Age, fat free mass, physical activity, energy intake
14182 Pediatric Bone Mineral Accrual Study								Male		1 Oz/day		-0.0141 (0.00846)	NS	As above
14187 (Phillips *et al.*, 2004) Massachusetts Institute of Technology Growth and Development Study	USA, Primarily White, BMI <30, Generally healthy	8-12 %M 0	196	6.9 years (28)	FFQ (116)	Energy from soda (only sugar-sweetened)	Percentage body fat Bioimpedance		1. <0.74 2. 0.74 to 1.4 3. 1.5 to 3.1 4. ≥3.2	% Energy		1. Referent 2. 0.15 3. 0.41 4. 0.31	1. N/A 2. 0.57 3. 0.18 4. 0.35 P trend 0.23	Parental Overweight, %Energy from protein

References

Abete I, Parra D, Martinez JA (2008) Energy-restricted diets based on a distinct food selection affecting the glycemic index induce different weight loss and oxidative response. *Clin Nutr* **27** (4): 545-551

Abrams SA, Griffin IJ, Hawthorne KM, Ellis KJ (2007) Effect of prebiotic supplementation and calcium intake on body mass index. *J Pediatr* **151** (3): 293-298

Albertson AM, Thompson D, Franko DL, Holschuh NM, Bauserman R, Barton BA (2009) Prospective associations among cereal intake in childhood and adiposity, lipid levels, and physical activity during late adolescence. *J Am Diet Assoc* **109** (10): 1775-1780

Barton BA, Eldridge AL, Thompson D, Affenito SG, Striegel-Moore RH, Franko DL, Albertson AM, Crockett SJ (2005) The relationship of breakfast and cereal consumption to nutrient intake and body mass index: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Am Diet Assoc* **105** (9): 1383-1389

Bates, B., Lennox, A, and Swan, G. National Diet and Nutrition Survey: Headline results from Year 1 of the rolling programme. [Online]. 2009. 21-2-2011.

Bazzano LA, Song Y, Bubes V, Good CK, Manson JE, Liu S (2005) Dietary intake of whole and refined grain breakfast cereals and weight gain in men. *Obes Res* **13** (11): 1952-1960

Bellisle F, Drewnowski A (2007) Intense sweeteners, energy intake and the control of body weight. *Eur J Clin Nutr* **61** (6): 691-700, doi:1602649 [pii];10.1038/sj.ejcn.1602649 [doi]

Bhargava A (2006) Fiber intakes and anthropometric measures are predictors of circulating hormone, triglyceride, and cholesterol concentrations in the women's health trial. *J Nutr* **136** (8): 2249-2254

Boreham C, Twisk J, van MW, Savage M, Strain J, Cran G (1999) Relationships between the development of biological risk factors for coronary heart disease and lifestyle parameters during adolescence: The Northern Ireland Young Hearts Project. *Public Health* **113** (1): 7-12

Bornet FR, Jardy-Gennetier AE, Jacquet N, Stowell J (2007) Glycaemic response to foods: impact on satiety and long-term weight regulation. *Appetite* **49** (3): 535-553, doi:S0195-6663(07)00280-2 [pii];10.1016/j.appet.2007.04.006 [doi]

Burr ML, Fehily AM, Gilbert JF, Rogers S, Holliday RM, Sweetnam PM, Elwood PC, Deadman NM (1989) Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: diet and reinfarction trial (DART). *Lancet* **2** (8666): 757-761, doi:S0140-6736(89)90828-3 [pii]

Butland, B., Jeeb, S. A., Kopelman, K., McPherson, S., and Thomas, J. Tackling Obesity: Future Choices - Project report (2nd ed) [Online]. 2007. 21-2-2011.

Carels RA, Darby LA, Douglass OM, Cacciapaglia HM, Rydin S (2005) Education on the glycemic index of foods fails to improve treatment outcomes in a behavioral weight loss program. *Eating Behaviors* **6** (2): 145-150

Cheng G, Karaolis-Danckert N, Libuda L, Bolzenius K, Remer T, Buyken AE (2009) Relation of dietary glycemic index, glycemic load, and fiber and whole-grain intakes during puberty to the concurrent development of percent body fat and body mass index. *Am J Epidemiol* **169** (6): 667-677

Clifton PM, Keogh JB, Noakes M (2008) Long-term effects of a high-protein weight-loss diet. *Am J Clin Nutr* **87** (1): 23-29

Clifton PM, Noakes M, Keogh JB (2004) Very Low-Fat (12%) and High Monounsaturated Fat (35%) Diets Do Not Differentially Affect Abdominal Fat Loss in Overweight, Nondiabetic Women. *J Nutr* **134** (7): 1741-1745

Colditz GA, Willett WC, Stampfer MJ, London SJ, Segal MR, Speizer FE (1990) Patterns of weight change and their relation to diet in a cohort of healthy women. *Am J Clin Nutr* **51** (6): 1100-1105

Cole TJ, Bellizzi MC, Flegal KM, Dietz WH (2000) Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* **320** (7244): 1240-1243

Committee on Medical Aspects of Food Policy (1989) *Dietary Sugars and Human Disease*. London, HMSO

Committee on Medical Aspects of Food Policy (1994) *Nutritional aspects of cardiovascular disease*. HMSO: London

Dale KS, McAuley KA, Taylor RW, Williams SM, Farmer VL, Hansen P, Vorgers SM, Chisholm AW, Mann JI (2009) Determining optimal approaches for weight maintenance: a randomized controlled trial. *Can Med Assoc J* **180** (10): E39-E46

Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ (2005) Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA* **293** (1): 43-53

Das SK, Gilhooly CH, Golden JK, Pittas AG, Fuss PJ, Cheatham RA, Tyler S, Tsay M, McCrory MA, Lichtenstein AH, Dallal GE, Dutta C, Bhapkar MV, Delany JP, Saltzman E, Roberts SB (2007) Long-term effects of 2 energy-restricted diets differing in glycemic load on dietary adherence, body composition, and metabolism in CALERIE: a 1-y randomized controlled trial. *Am J Clin Nutr* **85** (4): 1023-1030

Delbridge EA, Prendergast LA, Pritchard JE, Proietto J (2009) One-year weight maintenance after significant weight loss in healthy overweight and obese subjects: does diet composition matter? *Am J Clin Nutr* **90** (5): 1203-1214

Demol S, Yackobovitch-Gavan M, Shalitin S, Nagelberg N, Gillon-Keren M, Phillip M (2009) Low-carbohydrate (low & high-fat) versus high-carbohydrate low-fat diets in the treatment of obesity in adolescents. *Acta Paediatr* **98** (2): 346-351

Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, D'Agostino RB, Gaziano JM, Vasan RS (2007) Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community.[erratum appears in Circulation. 2007 Dec 4;116(23):e557]. *Circulation* **116** (5): 480-488

Dreon DM, Frey-Hewitt B, Ellsworth N, Williams PT, Terry RB, Wood PD (1988) Dietary fat:carbohydrate ratio and obesity in middle-aged men. *Am J Clin Nutr* **47** (6): 995-1000

Du H, van der AD, van Bakel MM, Slimani N, Forouhi NG, Wareham NJ, Halkjaer J, Tjonneland A, Jakobsen MU, Overvad K, Schulze MB, Buijsse B, Boeing H, Palli D, Masala G, Sorensen TI, Saris WH, Feskens EJ (2009) Dietary glycaemic index, glycaemic load and subsequent changes of weight and waist circumference in European men and women. *Int J Obes* **33** (11): 1280-1288

Due A, Toubro S, Skov AR, Astrup A (2004) Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised 1-year trial. *Int J Obes* **28** (10): 1283-1290

Dyson PA (2008) A review of low and reduced carbohydrate diets and weight loss in type 2 diabetes. *J Hum Nutr Diet* **21** (6): 530-538, doi:JHN896 [pii];10.1111/j.1365-277X.2008.00896.x [doi]

Ebbeling CB, Leidig MM, Feldman HA, Lovesky MM, Ludwig DS (2007) Effects of a low-glycemic load vs low-fat diet in obese young adults: a randomized trial.[erratum appears in JAMA. 2007 Aug 8;298(6):627]. *JAMA* **297** (19): 2092-2102

Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS (2003) A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med* **157** (8): 773-779

Ebbeling CB, Leidig MM, Sinclair KB, Seger-Shippee LG, Feldman HA, Ludwig DS (2005) Effects of an ad libitum low-glycemic load diet on cardiovascular disease risk factors in obese young adults. *Am J Clin Nutr* **81** (5): 976-982

Fiorito LM, Marini M, Francis LA, Smiciklas-Wright H, Birch LL (2009) Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. *Am J Clin Nutr* **90** (4): 935-942

Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S (2003) A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* **348** (21): 2082-2090

Frisch S, Zittermann A, Berthold HK, Gotting C, Kuhn J, Kleesiek K, Stehle P, Kortke H (2009) A randomized controlled trial on the efficacy of carbohydrate-reduced or fat-reduced diets in patients attending a telemedically guided weight loss program. *Cardiovasc Diabetol* **8** 36

Gardner CD, Kiazand A, Alhassan S, Kim S, Stafford RS, Balise RR, Kraemer HC, King AC (2007) Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial.[see comment][erratum appears in JAMA. 2007 Jul 11;298(2):178]. *JAMA* **297** (9): 969-977

Gibson S (2008) Sugar-sweetened soft drinks and obesity: a systematic review of the evidence from observational studies and interventions. *Nutr Res Rev* **21** (2): 134-147, doi:S0954422408110976 [pii];10.1017/S0954422408110976 [doi]

Greenberg I, Stampfer MJ, Schwarzfuchs D, Shai I, DIRECT Group (2009) Adherence and success in long-term weight loss diets: the dietary intervention randomized controlled trial (DIRECT). *J Am Coll Nutr* **28** (2): 159-168

Haffner SM, Stern MP, Mitchell BD, Hazuda HP (1991) Predictors of obesity in Mexican Americans. *Am J Clin Nutr* **53** (6:Suppl): Suppl-1576S

Halkjaer J, Sorensen TI, Tjonneland A, Togo P, Holst C, Heitmann BL (2004) Food and drinking patterns as predictors of 6-year BMI-adjusted changes in waist circumference. *Br J Nutr* **92** (4): 735-748

Halkjaer J, Tjonneland A, Overvad K, Sorensen TI (2009) Dietary predictors of 5-year changes in waist circumference. *J Am Diet Assoc* **109** (8): 1356-1366

Halkjaer J, Tjonneland A, Thomsen BL, Overvad K, Sorensen TI (2006) Intake of macronutrients as predictors of 5-y changes in waist circumference. *Am J Clin Nutr* **84** (4): 789-797

Hare-Bruun H, Flint A, Heitmann BL (2006) Glycemic index and glycemic load in relation to changes in body weight, body fat distribution, and body composition in adult Danes. *Am J Clin Nutr* **84** (4): 871-879

Hays NP, Bathalon GP, Roubenoff R, McCrory MA, Roberts SB (2006) Eating behavior and weight change in healthy postmenopausal women: Results of a 4-year longitudinal study. *Journals of Gerontology Series A-Biological Sciences and Medical Sciences* **61** (6): 608-615

Howard BV, Manson JE, Stefanick ML, Beresford SA, Frank G, Jones B, Rodabough RJ, Snetselaar L, Thomson C, Tinker L, Vitolins M, Prentice R (2006) Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial. *JAMA* **295** (1): 39-49

Iqbal SI, Helge JW, Heitmann BL (2006) Do energy density and dietary fiber influence subsequent 5-year weight changes in adult men and women? *Obesity* **14** (1): 106-114

James J, Thomas P, Cavan D, Kerr D (2004) Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial[erratum appears in BMJ. 2004 May 22;328(7450):1236]. *British Medical Journal* **328** (7450): 1237

James J, Thomas P, Kerr D (2007) Preventing childhood obesity: two year follow-up results from the Christchurch obesity prevention programme in schools (CHOPPS). *BMJ* **335** (7623): 762, doi:bmj.39342.571806.55 [pii];10.1136/bmj.39342.571806.55 [doi]

Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA (2007) Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition* **23** (7-8): 557-563

Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA (2008) A prospective analysis of dietary energy density at age 5 and 7 years and fatness at 9 years among UK children. *Int J Obes* **32** (4): 586-593

Kanders BS, Lavin PT, Kowalchuk MB, Greenberg I, Blackburn GL (1988) An evaluation of the effect of aspartame on weight loss. *Appetite* **11 Suppl 1** 73-84

Keen H, Thomas BJ, Jarrett RJ, Fuller JH (1979) Nutrient intake, adiposity, and diabetes. *Br Med J* **1** (6164): 655-658

Keogh JB, Brinkworth GD, Clifton PM (2007) Effects of weight loss on a low-carbohydrate diet on flow-mediated dilatation, adhesion molecules and adiponectin. *Br J Nutr* **98** (4): 852-859

Koh-Banerjee P, Chu NF, Spiegelman D, Rosner B, Colditz G, Willett W, Rimm E (2003) Prospective study of the association of changes in dietary intake, physical activity, alcohol consumption, and smoking with 9-y gain in waist circumference among 16 587 US men. *Am J Clin Nutr* **78** (4): 719-727

Koppes LL, Boon N, Nooyens AC, van MW, Saris WH (2009) Macronutrient distribution over a period of 23 years in relation to energy intake and body fatness. *Br J Nutr* **101** (1): 108-115

Kromhout D (1983) Energy and macronutrient intake in lean and obese middle-aged men (the Zutphen study). *Am J Clin Nutr* **37** (2): 295-299

Kvaavik E, Andersen LF, Klepp KI (2005) The stability of soft drinks intake from adolescence to adult age and the association between long-term consumption of soft drinks and lifestyle factors and body weight. *Public Health Nutr* **8** (2): 149-157

Layman DK, Evans EM, Erickson D, Seyler J, Weber J, Bagshaw D, Griel A, Psota T, Kris-Etherton P (2009) A moderate-protein diet produces sustained weight loss and long-term changes in body composition and blood lipids in obese adults. *J Nutr* **139** (3): 514-521

Lewington S, Whitlock G, Clarke R, Sherliker P, Emberson J, Halsey J, Qizilbash N, Peto R, Collins R (2007) Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet* **370** (9602): 1829-1839, doi:S0140-6736(07)61778-4 [pii];10.1016/S0140-6736(07)61778-4 [doi]

Libuda L, Alexy U, Sichert-Hellert W, Stehle P, Karaolis-Danckert N, Buyken AE, Kersting M (2008) Pattern of beverage consumption and long-term association with body-weight status in German adolescents--results from the DONALD study. *Br J Nutr* **99** (6): 1370-1379

Lissau I, Breum L, Sorensen TI (1993) Maternal attitude to sweet eating habits and risk of overweight in offspring: a ten-year prospective population study. *Int J Obes* **17** (3): 125-129

Lissner L, Levitsky DA, Strupp BJ, Kalkwarf HJ, Roe DA (1987) Dietary fat and the regulation of energy intake in human subjects. *Am J Clin Nutr* **46** (6): 886-892

Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, Hennekens CH, Manson JE (2000) A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *Am J Clin Nutr* **71** (6): 1455-1461

Lock S, Ford MA, Bagley R, Green LF (1980) The effect on plasma lipids of the isoenergetic replacement of table sucrose by dried glucose syrup (maize-syrup solids) in the normal diet of adult men over a period of 1 year. *Br J Nutr* **43** (2): 251-256, doi:S0007114580000335 [pii]

Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van HL, Slattery ML, Jacobs DR, Jr. (1999) Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. *JAMA* **282** (16): 1539-1546

Macdonald I (1967) Interrelationship between the influences of dietary carbohydrates and fats on fasting serum lipids. *Am J Clin Nutr* **20** (4): 345-351

Magarey AM, Daniels LA, Boulton TJ, Cockington RA (2001) Does fat intake predict adiposity in healthy children and adolescents aged 2--15 y? A longitudinal analysis. *Eur J Clin Nutr* **55** (6): 471-481

Malik VS, Schulze MB, Hu FB (2006) Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* **84** (2): 274-288

Mann JI, Hendricks DA, Truswell AS, Manning E (1970) Effects on serum-lipids in normal men of reducing dietary sucrose or starch for five months. *Lancet* **1** (7652): 870-872

Mann JI, Truswell AS (1972) Effects of isocaloric exchange of dietary sucrose and starch on fasting serum lipids, postprandial insulin secretion and alimentary lipaemia in human subjects. *Br J Nutr* **27** (2): 395-405, doi:S0007114572000467 [pii]

Mann JI, Truswell AS, Manning EB, Randles J (1974) Effects of omitting dietary sucrose and isoenergetic substitution of starch in primary type IV hyperlipoproteinaemia. *Proc Nutr Soc* **33** (1): 2A-3A

McManus K, Antinoro L, Sacks F (2001) A randomized controlled trial of a moderate-fat, low-energy diet compared with a low fat, low-energy diet for weight loss in overweight adults. *Int J Obes* **25** (10): 1503-1511

Meyer KA, Kushi LH, Jacobs DR, Jr., Slavin J, Sellers TA, Folsom AR (2000) Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. *Am J Clin Nutr* **71** (4): 921-930

Mundt CA, Baxter-Jones AD, Whiting SJ, Bailey DA, Faulkner RA, Mirwald RL (2006) Relationships of activity and sugar drink intake on fat mass development in youths. *Medicine & Science in Sports & Exercise* **38** (7): 1245-1254

NHS Information Centre. Health Surveys for England, 2009. [Online]. 2010. 11-4-2011.

Nissinen K, Mikkila V, Mannisto S, Lahti-Koski M, Rasanen L, Viikari J, Raitakari O (2009) Sweets and sugar-sweetened soft drink intake in childhood in relation to adult BMI and overweight. The Cardiovascular Risk in Young Finns Study. *Public Health Nutr* **12** (11): 2018-2026

Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L (2008) Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med* **168** (14): 1487-1492

Parker DR, Gonzalez S, Derby CA, Gans KM, Lasater TM, Carleton RA (1997) Dietary factors in relation to weight change among men and women from two southeastern New England communities. *Int J Obes* **21** (2): 103-109

Pasman WJ, Westerterp-Plantenga MS, Muls E, Vansant G, van RJ, Saris WH (1997a) The effectiveness of long-term fibre supplementation on weight maintenance in weight-reduced women. *Int J Obes* **21** (7): 548-555

Pasman WJ, Westerterp-Plantenga MS, Saris WH (1997b) The effectiveness of long-term supplementation of carbohydrate, chromium, fibre and caffeine on weight maintenance. *Int J Obes* **21** (12): 1143-1151

Phelan S, Wyatt H, Nassery S, DiBello J, Fava JL, Hill JO, Wing RR (2007) Three-year weight change in successful weight losers who lost weight on a low-carbohydrate diet. *Obesity* **15** (10): 2470-2477

Phillips SM, Bandini LG, Naumova EN, Cyr H, Colclough S, Dietz WH, Must A (2004) Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. *Obes Res* **12** (3): 461-472

Poppitt SD, Swann D, Black AE, Prentice AM (1998) Assessment of selective under-reporting of food intake by both obese and non-obese women in a metabolic facility. *Int J Obes* **22** (4): 303-311

Porikos KP, Booth G, Van Itallie TB (1977) Effect of covert nutritive dilution on the spontaneous food intake of obese individuals: a pilot study. *Am J Clin Nutr* **30** (10): 1638-1644

Porikos KP, Hesser MF, Van Itallie TB (1982) Caloric regulation in normal-weight men maintained on a palatable diet of conventional foods. *Physiol Behav* **29** (2): 293-300

Reiser S, Hallfrisch J, Michaelis OE, Lazar FL, Martin RE, Prather ES (1979a) Isocaloric exchange of dietary starch and sucrose in humans. I. Effects on levels of fasting blood lipids. *Am J Clin Nutr* **32** (8): 1659-1669

Reiser S, Handler HB, Gardner LB, Hallfrisch JG, Michaelis OE, Prather ES (1979b) Isocaloric exchange of dietary starch and sucrose in humans. II. Effect on fasting blood insulin, glucose, and glucagon and on insulin and glucose response to a sucrose load. *Am J Clin Nutr* **32** (11): 2206-2216

Richardson JF (1972) The sugar intake of businessmen and its inverse relationship with relative weight. *Br J Nutr* **27** (3): 449-460

Rifkind BM, Lawson DH, Gale M (1966) Effect of short-term sucrose restriction on serum-lipid levels. *Lancet* **2** (7478): 1379-1381

Rissanen AM, Heliovaara M, Knekt P, Reunanen A, Aromaa A (1991) Determinants of weight gain and overweight in adult Finns. *Eur J Clin Nutr* **45** (9): 419-430

Romieu I, Willett WC, Stampfer MJ, Colditz GA, Sampson L, Rosner B, Hennekens CH, Speizer FE (1988) Energy intake and other determinants of relative weight. *Am J Clin Nutr* **47** (3): 406-412

Rosenthal MB, Barnard RJ, Rose DP, Inkeles S, Hall J, Pritikin N (1985) Effects of a high-complex-carbohydrate, low-fat, low-cholesterol diet on levels of serum lipids and estradiol. *Am J Med* **78** (1): 23-27, doi:0002-9343(85)90456-5 [pii]

Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, McManus K, Champagne CM, Bishop LM, Laranjo N, Leboff MS, Rood JC, de JL, Greenway FL, Loria CM, Obarzanek E, Williamson DA (2009) Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *N Engl J Med* **360** (9): 859-873

Sammel MD, Grisso JA, Freeman EW, Hollander L, Liu L, Liu S, Nelson DB, Battistini M (2003) Weight gain among women in the late reproductive years. *Family Practice* **20** (4): 401-409

Scottish Intercollegiate Guidelines Network. Risk estimation and the prevention of cardiovascular disease:national clinical guideline 97. 2007. Edinburgh:Scottish Intercollegiate Guidelines Network.

Shah M, Baxter JE, McGovern PG, Garg A (1996) Nutrient and food intake in obese women on a low-fat or low-calorie diet. *Am J Health Promot* **10** (3): 179-182

Sheppard L, Kristal AR, Kushi LH (1991) Weight loss in women participating in a randomized trial of low-fat diets. *Am J Clin Nutr* **54** (5): 821-828

Sichieri R, Moura AS, Genelhu V, Hu F, Willett WC (2007a) An 18-mo randomized trial of a low-glycemic-index diet and weight change in Brazilian women. *Am J Clin Nutr* **86** (3): 707-713, doi:86/3/707 [pii]

Sichieri R, Moura AS, Genelhu V, Hu F, Willett WC (2007b) An 18-mo randomized trial of a low-glycemic-index diet and weight change in Brazilian women. *Am J Clin Nutr* **86** (3): 707-713

Stevens J, Ahn K, Juhaeri, Houston D, Steffan L, Couper D (2002) Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults: The ARIC study. *Diabetes Care* **25** (10): 1715-1721

Striegel-Moore RH, Thompson D, Affenito SG, Franko DL, Obarzanek E, Barton BA, Schreiber GB, Daniels SR, Schmidt M, Crawford PB (2006) Correlates of beverage intake in adolescent girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr* **148** (2): 183-187

Swinburn BA, Metcalf PA, Ley SJ (2001) Long-term (5-year) effects of a reduced-fat diet intervention in individuals with glucose intolerance. *Diabetes Care* **24** (4): 619-624

Tam CS, Garnett SP, Cowell CT, Campbell K, Cabrera G, Baur LA (2006) Soft drink consumption and excess weight gain in Australian school students: results from the Nepean study. *Int J Obes* **30** (7): 1091-1093

Thornton JR, Emmett PM, Heaton KW (1983) Diet and gall stones: effects of refined and unrefined carbohydrate diets on bile cholesterol saturation and bile acid metabolism. *Gut* **24** (1): 2-6

Tinker LF, Bonds DE, Margolis KL, Manson JE, Howard BV, Larson J, Perri MG, Beresford SA, Robinson JG, Rodriguez B, Safford MM, Wenger NK, Stevens VJ, Parker LM, Women's Health Initiative (2008) Low-fat dietary pattern and risk of treated diabetes mellitus in postmenopausal women: the Women's Health Initiative randomized controlled dietary modification trial. *Arch Intern Med* **168** (14): 1500-1511

Twisk JW, Kemper HC, van MW, Post GB, Van Lenthe FJ (1998) Body fatness: longitudinal relationship of body mass index and the sum of skinfolds with other risk factors for coronary heart disease. *Int J Obes* **22** (9): 915-922

Van Dam RM, Seidell JC (2007) Carbohydrate intake and obesity. *Eur J Clin Nutr* **61 Suppl 1** S75-S99, doi:1602939 [pii];10.1038/sj.ejcn.1602939 [doi]

Van Lenthe FJ, van MW, Kemper HC, Post GB (1998) Behavioral variables and development of a central pattern of body fat from adolescence into adulthood in normal-weight whites: the Amsterdam Growth and Health Study. *Am J Clin Nutr* **67** (5): 846-852

Vega-Lopez S, Mayol-Kreiser SN (2009) Use of the glycemic index for weight loss and glycemic control: a review of recent evidence. *Curr Diab Rep* **9** (5): 379-388

Venn BJ, Green TJ (2007) Glycemic index and glycemic load: measurement issues and their effect on diet-disease relationships. *Eur J Clin Nutr* **61 Suppl 1** S122-S131

Werner D, Emmett PM, Heaton KW (1984) Effects of dietary sucrose on factors influencing cholesterol gall stone formation. *Gut* **25** (3): 269-274

WHO expert consultation (2004) Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* **363** (9403): 157-163, doi:S0140-6736(03)15268-3 [pii];10.1016/S0140-6736(03)15268-3 [doi]

Willett WC, Howe GR, Kushi LH (1997) Adjustment for total energy intake in epidemiologic studies. *Am J Clin Nutr* **65** (4 Suppl): 1220S-1228S

World Health Organisation (1995) *Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854.* Geneva: World Health Organization.:

World Health Organization (2007) *The challenge of obesity in the WHO European region and the strategies for response.* Geneva: The World Health Organization.: