Chapter 2: Markers of Cardiovascular Disease

Incident hypertension and blood pressure

Contents

Background

Hypertension or ‘high blood pressure’ is a condition in which the systemic arterial blood pressure is elevated. It is suggested that hypertension affects up to one quarter of the population worldwide (Kearney *et al.*, 2005). Hypertension is a major risk factor for stroke and myocardial infarction. It is also a common cause of kidney disease. Hypertension, therefore, contributes significantly to morbidity and mortality rates (Whelton, 1994).

Blood pressure readings relate both to when the heart is contracting (systole) and when it is relaxing (diastole), thereby giving rise to the SBP and the DBP. Typically a sustained systolic blood pressure (SBP) greater than 140mmHg and a sustained diastolic blood pressure (DBP) greater than 90mmHg are termed hypertension. Guidance from the National Institute for Health and Clinical excellence recommends that hypertension is diagnosed when after 2 visits the systolic or diastolic or both are above 140/90mmHg. The threshold for offering drug treatment is 160/100mmHg or a SBP of more than 160mmHg. There are, however, a number of different cut-offs relating to the degree of hypertension, and also to the thresholds for the treatment of hypertension in different populations, for example the treatment threshold is 140/90mmHg if the estimated 10-year cardiovascular risk is more than 20% or there is existing cardiovascular disease or target organ damage. The aim of treatment is to reduce the blood pressure to 140/90mmHg or below.

Although the diagnostic thresholds for hypertension imply a cut-off of CVD risk, this is not likely to be the case (Collins *et al.*, 1990;Lewington *et al.*, 2002;MacMahon *et al.*, 1990). Analyses of many observational studies suggest that the lowering of systolic and diastolic readings are associated with significant reductions in rates of stroke and coronary heart disease. In one study, a reduction in DBP of 10mmHg was associated with reductions in death from stroke and heart disease by one half – and that this effect was consistent across a range of blood pressures (Lewington *et al.*, 2002).

There are many possible causes of hypertension. Commonly, however, no cause is identified, and this is termed essential hypertension. Essential hypertension accounts for over 90% of cases of hypertension. The remaining 10% of cases are termed secondary hypertension and usually are due to diseases affecting the heart, blood vessels, kidneys or endocrine system. Usually there are no symptoms associated with hypertension. It is therefore important to screen for and treat the condition.

There are many treatments recommended for the management of hypertension including lifestyle changes, pharmacological therapies and surgical options (National Institute of Clinical Excellence, 2004;British Cardiac Society *et al.*, 2005;Wright and Musini, 2009). The prevention of hypertension involves lifestyle changes such as the maintenance of a healthy weight, stopping smoking, reducing alcohol consumption, and dietary changes including a low salt diet, rich in fruit and vegetables (British Cardiac Society *et al.*, 2005).

The average effect size noted in dietary intervention trials is generally relatively small. However, these small effects, can translate into important reductions in the incidence of hypertension and community burden of cardiovascular disease (Klag *et al.*, 1990). A reduction in the SBP of 20mmHg or DBP of 10mmHg reduces the risk of death from stroke and ischaemic heart disease by approximately one half (Lewington *et al.*, 2002). Specifically, a reduction in DBP of 5, 7.5 and 10mmHg is associated with a reduction in stroke of 34%, 46% and 56% and coronary heart disease of 21%, 29% and 37% respectively (MacMahon *et al.*, 1990).

Hypertension commonly occurs in conjunction with other cardiovascular risk factors such as diabetes mellitus, dyslipidaemia and obesity (Kannel, 2000). Whilst there is greater understanding of the pathophysiology behind the rarer secondary hypertension, less is known about the link between essential hypertension and cardiovascular sequelea. That is, secondary hypertension may be due to an abnormality of a hormonal regulatory system (such as Cushing's disease) or a structural abnormality of the blood vessels (such as coarctation of the aorta). For essential hypertension, it is believed that abnormalities resulting in excess blood volume, or increased total peripheral resistance, or over-activation of the sympathetic nervous system contribute to its pathophysiology.

Notably, just as there are many environmental causes of hypertension, including physical activity, nutrition, social deprivation, alcohol consumption, there is a significant genetic element to the aetiology of hypertension

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;Committee on Medical Aspects of Food Policy, 1991). Studies were initially scanned by title and abstract for relevance. Those deemed non-relevant were omitted and those of relevance were passed through the inclusion/ exclusion criteria applied to the current review. The papers noted below would not have been eligible for inclusion in this review for the reasons listed.

Papers from COMA reports that did not meet inclusion criteria

The paper published before 1990 noted in the table below would not have been eligible for inclusion in this review for the reasons listed.

Table 2.1 Study identified from previous COMA reports*: excluded

Authors, Year	Intervention description	Intervention duration/ follow up	Exclusion code that would be applied in this review	Exclusion detail
(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.

*(Committee on Medical Aspects of Food Policy, 1989;Committee on Medical Aspects of Food Policy, 1994;Committee on Medical Aspects of Food Policy, 1991)

Papers from COMA reports that met inclusion criteria

The following paper published before 1990 would have been eligible for inclusion in this review.

Table 2.2 Previous cohort study in COMA reports*: included study

Authors/ Reference	Population characteristics	Recruitment of participants	Dietary assessment method	Length of follow-up (years)	Initial cohort size	Losses to follow-up (%)
(Morris *et al.,* 1977)	Middle-aged men Mean age: 30-67 %Male: 100 Country: UK Ethnicity: Not stated	Community cohort	Diet was assessed via 7-day weighed dietary surveys administered twice. No details concerning validation of the dietary assessment method were reported.	20 years	337	10

*(Committee on Medical Aspects of Food Policy, 1989;Committee on Medical Aspects of Food Policy, 1994;Committee on Medical Aspects of Food Policy, 1991)

One small cohort study published in 1977 provided evidence of intakes of total carbohydrate, sugar and dietary fibre (from fruit, vegetables, potatoes, pulses, whole grain and cereal foods) and blood pressure (Morris *et al.*, 1977). No association was reported between blood pressure and the nutrients of interest.

Summary of the evidence base

Cohort Studies

An overview of each cohort study that provides data for this chapter may be seen in Table 2.3.

In total, 12 papers provided data on 9 cohort studies. Of these, 8 studies followed cohorts of adults (Schroeder *et al.*, 2007;Ludwig *et al.*, 1999;Steffen *et al.*, 2005;Stamler *et al.*, 2002;Dhingra *et al.*, 2007;Ascherio *et al.*, 1992;Flint *et al.*, 2009;Ascherio *et al.*, 1996;Forman *et al.*, 2009;Wang *et al.*, 2007), and one study followed participants aged 12-15 years at baseline (Boreham *et al.*, 1999).

These cohort studies were mainly conducted in the USA (6 studies). One cohort study was conducted in Japan (Kanda *et al.*, 1999), and the study of adolescents was conducted in Northern Ireland (Boreham *et al.*, 1999). Most included both male and female participants, but 2 cohorts studied only females (Ascherio *et al.*, 1996;Forman *et al.*, 2009;Wang *et al.*, 2007) and 2 included men only (Ascherio *et al.*, 1992;Flint *et al.*, 2009;Stamler *et al.*, 2002).

The cohort study of Middle Aged Runners is unusual in that it tracked the health status of chronic exercisers over a 10 year period (Schroeder *et al.*, 2007). On average, at baseline these cohort participants ran approximately 61 km/week. This decreased to 44 km/week after 10 years.

Dietary assessment was mostly achieved through comprehensive food frequency questionnaires (FFQ), but the Middle-Aged Runners study employed food diaries (Schroeder *et al.*, 2007) and the Northern Ireland Young Hearts Cohort used dietary recall (Boreham *et al.*, 1999).

Length of follow-up ranged from a minimum of 4 years to a maximum of 18 years in the Health Professionals Follow-up Study (Flint *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 middle-aged runners had just 91 participants at baseline (Schroeder *et al.*, 2007), and the largest was the Nurse's Health Study with in excess of 121,000 participants (Ascherio *et al.*, 1996;Forman *et al.*, 2009).

Observational data should be interpreted 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

Sixty two publications from 59 randomised controlled trials provided information on the relationship between blood pressure and aspects of dietary carbohydrate.

Details concerning the design, participants, duration and nature of the interventions are included in Table 2.4. Seven studies employed a cross-over design (Swain *et al.*, 1990;Black *et al.*, 2006;Lehtimaki *et al.*, 2005;Landin *et al.*, 1992;Appel *et al.*, 2005;Andersson *et al.*, 2007;Kleemola *et al.*, 1999), but the majority used a parallel group design.

None of the trials used children or adolescents as subjects, all were studies of adults. Most studies included both male and female participants. Eight trials studied male participants only (Sciarrone *et al.*, 1993;Landin *et al.*, 1992;Black *et al.*, 2006;Philippou *et al.*, 2009;Bell *et al.*, 1990;Lovejoy *et al.*, 2003;Davy *et al.*, 2002;Wood *et al.*, 2007), and 12 studies recruited females only (Clifton *et al.*, 2004;Meckling and Sherfey, 2007;Bellisle *et al.*, 2007;Dale *et al.*, 2009;Birketvedt *et al.*, 2000;Pasman *et al.*, 1997;Surwit *et al.*, 1997;Gardner *et al.*, 2007;Howard *et al.*, 2006b;Tinker *et al.*, 2008;Jensen *et al.*, 2008;Leidy *et al.*, 2007;Brehm *et al.*, 2003;Brehm *et al.*, 2005;O'Brien *et al.*, 2005).

Of the 59 trials that reported data on blood pressure, a minority used participants with a BMI less than 25kg/m^2. Four studies included participants with either a mean BMI less than 25kg/m^2, or specifically recruited individuals who were not overweight or obese (Landin *et al.*, 1992;Kleemola *et al.*, 1999;Lehtimaki *et al.*, 2005;Swain *et al.*, 1990). Additionally, the Women's Health Initiative Trial (Tinker *et al.*, 2008;Howard *et al.*, 2006a) included a large proportion of participants who were less than BMI 25kg/m^2. The evidence base is therefore mainly reliant on studies that have explored the impact of dietary carbohydrate on individuals with pre-existing excess adiposity. The aim of many of the trials included here was to determine the effect of dietary interventions (involving carbohydrate) on body weight or fatness and there was frequently an explicit energy restriction component to the interventions.

Neter *et al*. suggested that for every 1kg weight loss, SBP and DBP would decrease by 1mmHg (Neter *et al.*, 2003). A more recent systematic review of evidence linking long-term (2+ years) weight and lifestyle changes to blood pressure changes for those with a body mass index (BMI) of ≤35kg/m^2 reported that their findings were similar for SBP, but less predictable for DBP, possibly due to the relative importance of initial DBP level in influencing degree of change (Aucott *et al.*, 2009).

Trials were conducted in the USA (25), Denmark (3), Australia (7), Spain (2), New Zealand (2), the UK (3), Germany (1), Finland (3), France (2), Italy (1), Norway (1), Sweden (2), Switzerland (1), the Netherlands (2), and 1 study was a European multi-centre trial. The evidence base therefore comprises studies that are fairly well spread across Europe, the Antipodes and North America.

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

The average number of participants across all trials, except the very large Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006a;Tinker *et al.*, 2008), was 87. Forty seven trials were rather small, with less than 100 participants in total. Nine trials other than the Women's Health Initiative Trial recruited more than 150 participants (Sacks *et al.*, 2009;Gardner *et al.*, 2007;Kleemola *et al.*, 1999;de Luis *et al.*, 2008;Frisch *et al.*, 2009;Dale *et al.*, 2009;Appel *et al.*, 2005;Ley *et al.*, 2004;Dansinger *et al.*, 2005).

Using data derived from a study designed to compare the reproducibility of blood pressure measurements achieved using 24-ambulatory monitoring, clinic measurements or an oscillometric wrist instrument used within the home environment, Uen *et al.* (Uen *et al.*, 2009) estimated the number of patients needed to detect a SBP and DBP difference of 5mmHg with a two-sided α risk of 5% and a statistical power of 80%. Reproducibility of blood pressure measurements over a one week period was highest using the wrist instrument, followed by 24-hour ambulatory monitoring.

Reproducibility was similar between 24-hour ambulatory monitoring and clinic assessments. Accordingly, the numbers needed to detect a 5mmHg difference between two groups of a trial were estimated to be 65 and 19 for systolic and diastolic measures for 24-hour ambulatory assessments and 64 and 23 respectively for clinic measures. On the basis of these estimates, on an individual basis, a large number of studies included in this review are likely to have insufficient power to detect a difference of 5mmHg between dietary groups or groups. Therefore, where meta-analysis was not possible, these individual studies should be interpreted cautiously.

In this review, most studies assessed blood pressure within a clinic setting. However, two trials employed 24-hour ambulatory monitoring (Davy *et al.*, 2002;Lee *et al.*, 2009). For inclusion in the meta-analysis however, where data from both methods were provided, we opted to include the clinic measurements to maintain consistency between studies. We did not discriminate between seated and supine blood pressure measurements within meta-analyses however.

It is generally observed that interventions that influence blood pressure usually have a more marked effect in hypertensive persons (Appel *et al.*, 1997). We have only included studies here on individuals with normal or modestly raised blood pressure (see protocol) and so the majority of studies have recruited subjects with blood pressure within the normal range. However, a small number of studies have specifically recruited participants with pre-hypertension or slightly elevated blood pressure (Appel *et al.*, 2005;Davy *et al.*, 2002;Maki *et al.*, 2007a;Philippou *et al.*, 2009;Wood *et al.*, 2007).

Risk of bias

A summary of the risk of bias assessment is provided in Table 2.5. 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. The majority 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. Only one was judged to be 'biased' with regard to allocation concealment (Brehm *et al.*, 2003). Blinding of participants and researchers to the various dietary approaches was more difficult to achieve, as might be anticipated with dietary intervention trials. However, sixteen trials were judged to have 'no bias' in respect of participants' awareness of the dietary intervention, and 21 trials were judged to have 'no bias' in respect of researcher awareness (these generally overlapped). There was some evidence of incomplete outcome reporting in 19 publications.

Table 2.3 Characteristics of cohort studies (studies with grey shading are on children and adolescents)

Cohort Name	Authors/ Reference	Population characteristics	Recruitment of participants	Dietary assessment method	Length of follow-up (years)	Criteria for defining hypertension	Initial cohort size	Losses to follow-up (%)
The CARDIA Study	(Ludwig *et al.*, 1999)	Young Black and White Adults Mean age: 18-30 %Male: 45.9 Country: USA Ethnicity: Multi-ethnic	Community cohort (4 sites: Alabama, Illinois, Minnesota, California)	Diet was assessed from a 700-item FFQ for intake over the previous month and it was reported to be validated.	10	Measured by research staff	5115	Not reported
	(Steffen *et al.*, 2005)				15	Incident hypertension was defined as SBP ≥ 130mmHg, DBP ≥ 85mmHg or use of antihypertensive medication.	5115	Not reported
Chicago Western Electric Study	(Stamler *et al.*, 2002)	Employed middle-aged men Mean age: 47.5 (40-55) %Male: 100 Country: USA Ethnicity: Not reported	Community cohort	Diet was assessed by standardised interviews which were cross-checked using a 195-item FFQ to assess usual intake of foods and beverages during the preceding 28 days. This was administered twice. No detail was reported concerning validation of the dietary assessment methods.	9	Seated blood pressure measurements made by study physicians. Data expressed as a continuous variable.	2107	Not reported
The Framingham Heart Study	(Dhingra *et al.*, 2007)	Mean age: 53 %Male: 43 Country: USA Ethnicity: not stated	Community cohort	Diet was assessed using a general questionnaire administered three times and it was reported to be validated.	4	Seated blood pressure measurements made by study physicians. Incident hypertension was defined as BP ≥135/85mmHg or by treatment for hypertension.	8997	Not reported
Health Professionals' Follow-Up Study	(Ascherio *et al.*, 1992)	Male health professionals Mean age: 40-75 %Male:100 Country: USA Ethnicity: Primarily white	Occupational cohort	Diet was assessed from a 131-item FFQ administered at baseline and then 4 years subsequently. This method was reported to be validated. The FFQ referred to the diet over the previous year.	4	Blood pressure was self reported as a categorical variable. Validity of self-reports in a sub-sample confirmed by medical record review.	51529	9.7
	(Flint *et al.*, 2009)				18	Self-reported physician diagnosis of high blood pressure.	51529	Not reported
Japanese Blood Pressure Study	(Kanda *et al.*, 1999)	Normotensive elderly subjects Mean age: 60-69 %Male: 37 Country: Japan Ethnicity: Japanese	Community cohort	Diet was assessed using a general questionnaire administered four times. No details concerning validation of the dietary assessment method were reported.	4	Seated blood pressure was measured by study physicians on an annual basis. Hypertension was defined as SBP ≥ 140mmHg and/or DBP ≥ 90mmHg and/or diagnosed as hypertensive before or during follow-up.	948	Not reported
Middle-aged Runners Study	(Schroeder *et al.*, 2007)	Chronically endurance-trained runners Mean age: 51 %Male: 62 Country: USA Ethnicity: Not stated	Community cohort	Diet was assessed using 3-day food diary records administered once.	10	Measured by research staff and reported as a continuous variable	91	Not reported

Cohort Name	Authors/ Reference	Population characteristics	Recruitment of participants	Dietary assessment method	Length of follow-up (years)	Criteria for defining hypertension	Initial cohort size	Losses to follow-up (%)
The Northern Ireland Young Hearts Project	(Boreham *et al.*, 1999)	Representative sample of adolescents from Northern Ireland. Mean age: 12-15 %Male: 49.5 Country: Northern Ireland Ethnicity: Primarily White	Population sampled cohort	Diet was assessed by dietary recall over the previous month. It was administered twice and reported to be validated.	4	Measured by research staff and reported as a continuous variable	509	1.7
Nurses' Health Study	(Ascherio *et al.*, 1996)	Female Health Professionals Mean age: 30-55 %Male: 0 Country: USA Ethnicity: Primarily white	Occupational cohort	Diet was assessed from a semi-quantitative 126-item FFQ which was reported to be validated.	4	Incident hypertension was defined by meeting one of two criteria: previously being diagnosed with high BP (excluding during pregnancy) or being newly diagnosed with high BP.	121700	0.9
Nurses' Health Study/ Nurses' Health Study II/ Health Professionals' Follow-Up Study	(Forman *et al.*, 2009)	Male and female health professionals Mean age: 25-75 %Male: not stated Country: USA Ethnicity: Primarily white	Occupational cohort	Diet was assessed from a FFQ for intakes over the previous year and it was reported to be validated.	14 (NHS II) 18 (HPFS) 20 (NHS)	Incident hypertension was defined by meeting one of two criteria: previously being diagnosed with high BP (excluding during pregnancy) or being newly diagnosed with high BP.	88,540 (NHS) 97,315 (NHS II) 37,375 (HPFS)	Not reported
The Women's Health Study	(Wang *et al.*, 2007)	US female health professionals free of CVD, cancer and hypertension at baseline. Mean age: 54 %Male: 0 Country: USA Ethnicity: Primarily White	Occupational cohort	Diet was assessed once using a validated 131-item FFQ.	10	Incident hypertension was defined by meeting ≥ 1 of 4 criteria: self-reports of new physician diagnosis, self-reports of new antihypertensive treatment, self-reports of SBP ≥ 140mmHg or self reports of DBP ≥ 90mmHg.	39876	Not reported

Table 2.4 Trial characteristics

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	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 maintenance	Free living diet plan	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 support
(Andersson *et al.*, 2007) Uppsala Wholegrain Trial	≥ 1 CHD risk factor Age 30-70y BMI 26-35	Sweden 27% Male Age: 35 - 70(59) BMI: (28)	Crossover (washout 6 weeks)	6 weeks	Supplement	34	1. Wholegrain products 2. Refined grain products	1. Usual diet + whole grain foods (Bread, bread, muesli & pasta) Minimum 50% wholegrain in provided foods = 112g wholegrain/day 2. Usual diet + refined grain foods (Bread, muesli & pasta)	1. g/d: C 143 P 28 F 8 Energy: 3180kJ/d Fibre g/d:18 2. g/d: C 145 P 23 F 14 Energy: 3340kJ/d Fibre g/d:6	Yes	Government /Research institute funding Swedish Diabetes Association
(Appel *et al.*, 2005) OMNI-Heart	Age >30y Generally healthy No CVD, T2DM No medications which influence outcomes Not hyperlipidaemic/ hypercholesterolaemic Prehypertension/ stage 1 HTN Weight < 160kg	USA 55% Male Age: (54) BMI: (30)	Crossover (washout 3 weeks)	6 weeks	All food provided	191	1. High carbohydrate 2. High protein 3. High PUFA	1. High CHO diet provided 2. High protein diet provided 3. High unsaturated fat diet provided.	1. %E: C 58 P 15 F 27 2. %E: C 48 P 25 F 27 3. %E: C 48 P 15 F 37	Intended diet	Government/ NIH
(Bell *et al.*, 1990)	Age 24-59y Body weight >130% of ideal Cholesterol between the 50th and 90th centile Free of chronic disease No medications which influence outcomes	USA 100% Male Age: mean not reported BMI: mean not reported	Parallel Group	6 weeks	Substitution	60	1. Placebo 2. Pectin enriched cereal 3. Psyllium enriched cereal	1. Step 1 diet with 57g of cornflakes consumed each morning. 2. Step 1 diet with 57g of cornflakes containing oat bran, sugar-beet fibre, white wheat bran and high-methoxyl pectin consumed each morning. 50% total soluble fibre in cereal was from pectin. 3. Step 1 diet with 57g of		Yes	General Mills Inc.

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
								cornflakes containing oat bran, sugar-beet fibre, white wheat bran and psyllium consumed each morning. 50% total soluble fibre in cereal was from psyllium.			
(Bellisle *et al.*, 2007)	Age >18y BMI >25 Free of chronic disease No medication Women	France 0% Male Age: 20 - 72 BMI:25 - 40	Parallel Group	12 weeks	Free living diet plan	96	1. Low GI 2. Control	1. Weight watchers program with a focus on low GI foods. 2. Weight watchers program		Yes	Weight Watchers International Inc
(Birketvedt *et al.*, 2000)	Age 18-70y BMI >27.5 Generally healthy	Norway 0% Male Age: (40) BMI: (28)	Parallel Group	24 weeks	Supplement	53	1. Energy restricted diet + mixed fibre tablets 2. Energy restricted diet + placebo tablets	In both groups: 24 tablets/d for 8 weeks then 15 tablets/d up to 24 weeks + 1200kcal, 15g fibre weight reducing diet 1. Supplement tablets contained grain/citrus fibre. 6g fibre, 15% soluble/85% insoluble. 2. Placebo tablets content not reported		Yes	Not reported
(Black *et al.*, 2006)	BMI <35 No CHD, T2DM or HTN Not hyperlipidaemic/ hypercholesterolaemic	UK 100% Male Age: (33) BMI: (27)	Crossover (washout 4 weeks)	6 weeks	All food provided	14	1. High sucrose diet 2. Low sucrose diet	1. 25% energy provided as sucrose (solid food & beverages). 55% CHO, 10-15% PRO, 30-35% FAT, 18g/d fibre 2. 10% energy provided as sucrose (solid food & beverages). 55% CHO, 10-15% PRO, 30-35% FAT, 18g/d fibre	1. %E: C 55 P 11 F 33 Energy 2484 kcal/d Fibre g/d:17 2. %E: C 55 P 12 F 33 Energy 3176 kcal/d Fibre g/d:18	Yes	The Sugar Bureau and Suikerstichting. Government funding
(Brehm *et al.*, 2003) American LC study 1	Age >18y BMI 30-35 Familial CVD/CHD Generally healthy No HTN or T2DM Weight stable	USA 0% Male Age: (44) BMI: (34)	Parallel Group	6 months	Free living diet plan	53	1. Low carbohydrate 2. Moderate fat	1. Ad libitum food intake. Max CHO intake 20g/d. CHO increased to 40-60g/d if ketosis was induced after 2 weeks. 2. American Heart Association Step 1 diet + restrict to 1200kcal/d. Intended intake: 55% CHO, 15% PRO, 30% FAT	1. %E: C 30 P 23 F 46 Energy 1302 kcal/d Fibre g/d:8.4 2. %E: C 53 P 18 F 29 Energy 1247 kcal/d Fibre g/d:12.35	Yes	American Heart Association, research institute funding and NIH

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(Brehm *et al.*, 2005) American LC study 2	<10% Δ body weight in previous 6m Age >18y BMI 30-35 Free of chronic disease	USA 0% Male Age: 44 BMI: (34)	Parallel Group	4 months	Free living diet plan	50	1. Low carbohydrate 2. Moderate fat	1. Ad libitum food intake. Max CHO intake 20g/d. CHO increased to 40-60g/d if ketosis was induced after 2 weeks. 2. American Heart Association Step 1 diet + restrict to 1200kcal/d. Intended intake: 55% CHO, 15% PRO, 30% FAT	1. %E: C 15 P 28 F 57 Energy 1288 kcal/d 2. %E: C 53 P 18 F 29 Energy 1339 kcal/d	Yes	American Heart Association, research institute funding and NIH
(Cairella *et al.*, 1995)	BMI >30 No CHD Sedentary occupation	Italy 27% Male Age: (36) BMI:31 - 47(37)	Parallel Group	60 days	Supplement	30	1. Balanced diet + fibre tablets 2. Balanced diet + placebo tablets	1. Fibre tablets (vegetable, citrus, cereal fibre, 6g/d) + balanced diet following 2 week VLCD 2. Placebo tablets, plus balanced diet following 2 week VLCD	1. Fibre g/d:6	Yes	Not reported
(Claessens *et al.*, 2009)	BMI >27 No HTN Normal glucose tolerance Normal lipid profile Weight loss >5% during run-in Weight stable	The Netherlands 28% Male Age: 30 - 60(45) BMI: (33)	Parallel Group	12 weeks	Supplement	60	1. High carbohydrate supplement 2. High protein supplement - casein 3. High protein supplement - whey	1. 50g/d consumed as a flavoured drink 2. 50g/d consumed as a flavoured drink 3. 50g/d consumed as a flavoured drink		Yes	Kerry Bio-Science, Almere, The Netherlands
(Clifton *et al.*, 2004)	BMI >27 No medications which influence outcomes No T2DM	Australia 0% Male Age: (47) BMI: (35)	Parallel Group	12 weeks	Free living diet plan	70	1. Very low fat 2. High MUFA	1. Diet was closely prescribed and key foods were provided 2. Diet was closely prescribed and key foods were provided	1. %E: C 65.4 P 21.7 F 11.6 Energy: 6004kJ/d Fibre g/d:31.2 2. %E: C 43.7 P 21.3 F 35.3 Energy: 5972kJ/d Fibre g/d:32	Yes	Meadow Lea Foods, Australia
(Dale *et al.*, 2009)	BMI >27.5	New Zealand 0% Male Age: (45) BMI: (32)	Factorial	2 years	Free living diet plan	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	Yes	Health Research Council of New Zealand

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
									g/d: C 183 P 77 F 46 Energy: 6192kJ/d Fibre g/d:23		
(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	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
(Davy *et al.*, 2002) American Cereal Study	50-75 year old men BMI 25-35 DBP 85-99mm and/ or SBP 130-159mmHg Fibre <30g/d No CHD, T2DM No medical conditions which influence outcomes Non smokers Normal glucose tolerance Not extremely athletic/active	USA 100% Male Age: (59) BMI: (29)	Parallel Group	12 weeks	Substitution	36	1. Wheat group 2. Oat group	1. 60g wheat cereal and 81g Frosted Mini-Wheats (14g/d of dietary fibre) 2. 60g oatmeal and 76g oat bran ready-to-eat cold cereal (14g/day of fibre, 5.5g/d beta glucan).	1. g/d: C 112 P 14 F 3 Energy: 2008kJ/d Fibre g/d:14 2. g/d: C 95 P 21 F 8 Energy: 2146kJ/d Fibre g/d:14	Yes	Quaker Oats and NIH
(de Luis *et al.*, 2008) Spanish Hypocaloric Diet Study	BMI >30 No CHD, T2DM or HTN	Spain 24.5% Male Age: (46) BMI: (34)	Parallel Group	2 months	Free living diet plan	204	1. Low fat 2. Low carbohydrate	1. Intended diet: 1500 kcal/d. 52% CHO, 20% PRO, 27% FAT 2. Intended diet: 1507kcal/d. 38% CHO, 26% PRO, 36% FAT	1. %E: C 52 P 20 F 27 Energy 1500 kcal/d 2. %E: C 38 P 26 F 36 Energy 1507 kcal/d	No, intended diet only	Not reported

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(de Luis *et al.*, 2009a) Spanish Hypocaloric Diet Study	BMI >30 No CHD or T2DM No medications which influence outcomes Not hyperlipidaemic/ hypercholesterolaemic	Spain 22% Male Age: (46) BMI: (35)	Parallel Group	2 months	Free living diet plan	131	1. Low fat 2. Low carbohydrate	1. Intended diet: 1500 kcal/d. 52% CHO, 20% PRO, 27% FAT 2. Intended diet: 1507kcal/d. 38% CHO, 26% PRO, 36% FAT	1. %E: C 53 P 20 F 27 Energy 1500 kcal/d 2. %E: C 38 P 26 F 36 Energy 1507 kcal/d	No, intended diet only	Not reported
(de Luis *et al.*, 2009b) Spanish Hypocaloric Diet Study	BMI >30 No CHD, T2DM or HTN	Spain 28% Male Age: (46) BMI: (35)	Parallel Group	3 months	Free living diet plan	118	1. Low carbohydrate 2. Low fat	1. Intended diet: 1507kcal/d. 38% CHO, 26% PRO, 36% FAT 2. Intended diet: 1500 kcal/d. 52% CHO, 20% PRO, 27% FAT	1. %E: C 30.8 Energy 1548 kcal/d 2. %E: F 25.3 Energy 1613 kcal/d	Yes	Not reported
(Delbridge *et al.*, 2009)	Age 18-70y BMI >27 Generally healthy	Australia 50% Male Age: 44 BMI: 39	Parallel Group	12 months Weight maintenance plan following 3 month weight loss	Free living diet plan	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	No, intended diet only	Meat & Livestock Australia
(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 month follow up. Monthly group workshops through-out 18 mo	Free living diet plan	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 National Centre for Research Resources
(Ebbeling *et al.*, 2005)	Age 18-35y BMI >27.5 Healthy	USA 12% Male Age: 28	Parallel Group	6 months strict, 6-12 mo less strict	Free living diet plan	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	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	Yes	National Institute of Diabetes & Digestive & Kidney Diseases,

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
		BMI: obese						of 250-500kcal/d. GL 77 g/1000 kcal	18.7 F 23.4 Energy 1409 kcal/d Fibre g/d:17.8		Charles H. Hood Foundation and National Centre for Research Resources
(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	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	No, intended diet only	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	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 & the 'Institute for Applied Telemedicine'
(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	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

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(Golay *et al.*, 2000)	Able to participate in physical activity BMI >30 Highly motivated to lose weight	Switzerland 24.1% Male Age: (44) BMI: (39)	Parallel Group	6 weeks	All food provided	54	1. Dissociated low energy diet 2. Balanced low energy diet	1. 1100 kcal/day. 47% carbohydrates and 25% lipids. Participants were not allowed to consume lipids and carbohydrates simultaneously. 2. 1100 kcal/day. 42% carbohydrates and 31% lipids. Participants were allowed to consume all macronutrients simultaneous	1. %E: C 47 P 27 F 25 g/d: C 123 P 71 F 29 Energy: 4600kJ/d 2. %E: C 42 P 27 F 31 g/d: C 114 P72 F 38 Energy: 4600kJ/d	No, intended diet only	Not reported
(He *et al.*, 2004) American Fibre Study	BMI <35 Generally healthy No CHD, T2DM or HTN No medications which influence outcomes Not hyperlipidaemic/ hypercholesterolaemic	USA 40% Male Age: (48) BMI: (29)	Parallel Group	12 weeks	Substitution	110	1. Oat bran and oatmeal 2. Refined wheat and corn	1. 60g oat bran in a muffin and 84g of oatmeal squares cereal daily. Soluble fibre 8.1g/d, beta glucan 7.3g/d, insoluble fibre 7.7g/d 2. 93g refined wheat in a muffin and 42g of corn flakes cereal daily. Soluble fibre 0.9g/d, beta glucan 0g/d, insoluble fibre 1.5g/d	1. g/d: C 113.3 P 24 F 13.7 Energy 652 kcal/d Fibre g/d:15.9 2. g/d: C 108.4 P 10.8 F 11 Energy 567 kcal/d Fibre g/d:2.7	No, intended diet only	NIH & Research institute funding
(Howard *et al.*, 2006b) 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	6 years	Free living diet plan	48835	1. Low fat 2. Control	1. Advice: reduce fat intake to 20%, increase fruit, vegetables and wholegrains 2. Received information relating to health and healthy diets	1. %E: C 53.9 P 17.7 F 28.8 Energy 1432 kcal/d Fibre g/d:19.6 2. %E: C 45.9 P 17.1 F 37 Energy 1546 kcal/d Fibre g/d:14.4	Yes	National Heart, Lung, and Blood Institute
(Jensen *et al.*, 2008)	Age 20-40y BMI 25-30 Generally healthy Moderate alcohol intake No medical conditions which influence outcomes No medication, HTN, smokers Not extremely athletic/active	Denmark 0% Male Age: 20 - 40 BMI: (28)	Parallel Group	10 weeks	Substitution	55	1. Low GI diet 2. High GI diet	1. Received low GI test foods in place of their usual CHO rich foods (wholegrain wheat bread, wholegrain rye bread, mashed potato, pasta, long grain rice) 2. Received high GI test foods in place of their usual CHO rich foods (wheat bread, rye bread, mashed potato, pasta, round grain rice)	1. %E: C 81.2 P 12.8 F 5.9 Energy: 4860kJ/d Fibre g/d:29.3 2. %E: C 81.7 P 12.6 F 5.7 Energy: 4886kJ/d Fibre g/d:32.2 Aim for 55-60% energy from carbohydrate, 20-	Nutrients of provided foods	Danone Vitapole. Food donated by Masterfoods a.s., Denmark, Euryza GmbH, Germany, and by Cerealia R&D, Schulstad Brød A/S, Denmark

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
									30% energy from fat		
(Keogh *et al.*, 2007)	Age 20-65y BMI 27-40 Moderate alcohol intake No HTN or T2DM No medications which influence outcomes	Australia 32% Male Age: (49) BMI: (33)	Parallel Group	12 weeks Active weight loss phase 1-12 wk, monthly dietitian meeting until wk 52	Free living diet plan	44	1. Low carbohydrate diet 2. High carbohydrate diet	1. Energy restricted, low CHO diet, low in saturated fat. 2. Energy restricted, high CHO diet, low in saturated fat.	1. %E: C 33 P 40 F 27 Fibre g/d:26 2. %E: C 60 P 20 F 20 Fibre g/d:40	No, intended diet only	Research institute funding
(Keogh *et al.*, 2008)	≥ 1 metabolic syndrome risk factor Abdominal obesity No CHD or T2DM	Australia % Male: men and women Age: 24 - 64(50) BMI:27 - 44(34)	Parallel Group	8 weeks	Free living diet plan	117	1. Low carbohydrate, high SFA 2. High carbohydrate, low SFA	1. 30% energy restriction. Some key foods were provided top aid compliance. Intended diet: 4%CHO, 35%PRO, 61%FAT 2. 30% energy restriction. Some key foods were provided top aid compliance. Intended diet: 46%CHO, 24%PRO, 30%FAT	1. %E: C 5 P 35 F 59 g/d: C 20 P 133 F 103 Energy: 6608kJ/d Fibre g/d:13 2. %E: C 47 P 24 F 28 g/d: C 172 P 87 F 47 Energy: 6590kJ/d Fibre g/d:32	Yes	Research institute funding
(Kleemola *et al.*, 1999)	BMI >20 Not breakfast cereal eater Moderate alcohol intake No medications which influence outcomes Non diabetic Not very low saturated fat intake	Finland 45% Male Age: 29 - 71 BMI:>20	Crossover (washout 6 weeks)	6 weeks	Substitution	224	1. Group 1- Cereal diet first 2. Group 2- Control diet first 3. Group 1- Control diet second 4. Group 2- Cereal diet second	Cereal diet: 60 g/d for women and 80 g/d for men, either Cornflakes or Rice Krispies. Control diet: follow usual habits	1. %E: C 55.3 P 16.3 F 28.5 Energy 2094 kcal/d Fibre g/d:22.3 2. %E: C 49 P 16.3 F 34.6 Energy 2063 kcal/d Fibre g/d:21.3 3. %E: C 50.5 P 16.6 F 32.9 Energy 2004 kcal/d	Yes	Not reported

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
									Fibre g/d:22.3 4. %E: C 55.4 P15.7 F 28.8 Fibre g/d: 21.3 Energy 1963 kcal/d		
(Landin *et al.*, 1992)	Generally healthy Middle-aged adults Not extremely athletic/active Not obese WHR of 0.91	Sweden 100% Male Age: (52) BMI: (25)	Crossover (washout 2 weeks)	6 weeks	Supplement	25	1. Guar gum 2. Placebo	1. Ten grams granulated guar given in a glass of water, 3 times a day before meals. 2. Granulated gelling starch given in a glass of water, 3 times a day before meals.	1. g/d: C 445 P 14 F 92 Energy 2875 kcal/d 2. g/d: C 445 P 14 F 92 Energy 2875 kcal/d	Yes	Research institute funding: Nordisk Insulin fond and the Swedish Nutrition Foundation & Goteborg Medical Society.
(Lee *et al.*, 2009)	Age 20-70y BMI 25-35 Fasting plasma glucose <5.6mmol/l Free of chronic disease Moderate alcohol intake No change in medications which influence outcomes within previous 3m No untreated HTN Non smokers Weight stable	Australia 25% Male Age: mean not reported BMI: mean not reported	Parallel Group	16 weeks	Substitution	88	1. Control bread 2. Lupin flour bread	1. Replaced 15-20% TE with white bread 2. Replaced 15-20% TE with lupin kernal flour-enriched bread (high protein, high fibre)	1. g/d: C 45.2 P 9.6 F 4.6 Fibre g/d:2.7 2. g/d: C 24.9 P 15.8 F 3.6 Fibre g/d:9.5	Yes	Government funding

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(Lehtimaki *et al.*, 2005)	Age 18-65y Healthy Not recently involved in any trial Stratified by apolipoprotein E genotype	Finland 42% Male Age: (44) BMI: (26)	Crossover (washout 0 days)	3 months	Supplement	130	1. Encapsulated microcrystalline chitosan 2. Starch capsules	1. 1.2 g chitosan twice daily (total 2.4g/d). 2. 1.2 g starch twice daily.		Yes	Research institute & University funding and the Finnish Cultural Foundation
(Leidy *et al.*, 2007) American Protein Study	Age >18y BMI >25 Non smokers Normal blood profiles Normal glucose tolerance Stable activity level Weight stable Women	USA 0% Male Age: 28 - 80 BMI:26 - 37	Parallel Group	12 weeks	Free living diet plan	54	1. High protein, energy restricted 2. Moderate protein, energy restricted	1. 750 kcal/d energy-deficit diet, 30% PRO 2. 750 kcal/d energy-deficit diet, 18% PRO	1. %E: C 45 P 30 F 25 Energy: 1560 kcal/d 2. %E: C 57 P 18 F 25 Energy: 1440 kcal/d	No, intended diet only	University funding & the National Pork Board
(Ley *et al.*, 2004) New Zealand Diabetic Workforce Study	Age >40y Impaired glucose tolerance	New Zealand 74% Male Age: >40 (53) BMI: 29	Parallel Group	12 months	Free living diet plan	176	1. Control 2. Low fat	1. No intervention 2. Education for dietary fat reduction	1. %E: C 45.6 P 16.6 F 33.8 Energy: 9500kJ/d Fibre g/d:19.95 2. %E: C 54.5 P 18.6 F 25.9 Energy: 7900kJ/d Fibre g/d:21.33	Yes	National Heart Foundation of New Zealand, Research funding and the Lotteries Medical Board
(Lovejoy *et al.*, 2003) Ole Study	Age 18-70y BMI 25-35 Generally healthy Non smokers Not extremely athletic/active Weight stable	USA 100% Male Age: (37) BMI: (31)	Parallel Group	9 months	All food provided	45	1. Control 2. Fat reduced 3. Fat substituted	1. 33%FAT 2. 25%FAT. Diet designed to be 11% lower energy than control diet 3. 1/3 of dietary fat replaced by olestra (25% metabolizable fat). This group will not be included in the review.	1. %E: C 52 P 15 F 33 2. %E: C 58 P 17 F 25	No, intended diet only	Government funding & Procter & Gamble Co.
(Maki *et al.*, 2007a)	Age >40y DBP 85-109mmHg Fibre <20g/d Mid upper arm circumference <42cm No CHD or T2DM SBP 130-179mmHg	USA 55% Male Age: >40 BMI: (32)	Parallel Group	12 weeks	Substitution	97	1. Oat beta-glucan cereal 2. Wheat cereal	1. 90g/d oat bran cereal + 60g/d oatmeal + 20g/d powdered oat beta-glucan. 7.7g/d beta glucan 2. 90g/d wheat cereal + 65g/d low fibre hot cereal oatmeal + 12g/d maltodextrin powder	1. g/d: C 124.3 P 20.3 F 8.9 Energy: 658 kcal/d Fibre g/d:17.3 2. g/d: C 139.5 P 10 F 2.1 Energy: 641 kcal/d	Yes	Quaker Oats Company

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
	Waist circumference >96.5 (m) >88.9 (f)								Fibre g/d:1.9		
(Maki *et al.*, 2007b)	<4.5kg Δ weight in previous 2m Age 18-65y Generally healthy No untreated HTN Non smokers No T2DM Waist >87cm(F) or >90cm(M)	USA 32.6% Male Age: (50) BMI: (32)	Parallel Group	36 weeks	Free living diet plan	86	1. Ad libitum low GL diet 2. Low fat, energy restricted	1. Dietary advice ad libitum reduced-glycaemic-load (GI average = 48, GL = 8173 carb*GI) 2.Reduce fat intake, decrease portion sizes, target energy deficit 500-800 kcal/d (GI average = 51, GL= 12118 carb*GI)	1. g/d: C 69 P 97 F 80 Energy: 1365 kcal/d Fibre g/d:11 2. g/d: C 168 P 75 F 62 Energy: 1525 kcal/d Fibre g/d:12	Yes	Kraft Foods
(Marett and Slavin, 2004)	Age 18-55y Generally healthy No BMI or BP criteria	USA 52% Male Age: (29) BMI: mean not reported	Parallel Group	6 months	Supplement	54	1. Placebo 2. Larch arabinogalactan 3. Tamarack arabinogalactan	1. Rice starch 8.4g/d added to food or drinks 2. 8.4g/d Larch arabinogalactan (non viscous soluble fibre) added to food or drinks 3. 8.4g/d Tamarack arabinogalactan (non viscous soluble fibre) added to food or drinks		Yes	The Sota-Tec Fund
(Meckling *et al.*, 2004)	BMI >25 Generally healthy Highly motivated to lose weight No medications which influence outcomes	Canada 29% Male Age: 24 - 61 BMI: (32)	Parallel Group	10 weeks	Free living diet plan	40	1. Low fat 2. Low carbohydrate	1. Energy restriction was matched to the low CHO group 2. CHO 50-70 g/d plus concomitant energy restriction	1. %E: C 61.9 P 19.5 F 17.8 Energy: 6077kJ/d Fibre g/d:20.3 2. %E: C 15.4 P 26.2 F 55.5 Energy: 6421kJ/d Fibre g/d:8.9	Yes	Research institute funding
(Meckling and Sherfey, 2007)	BMI 25-30 No chronic illness No CHD/ T2DM No medication Pre-menopausal	Canada 0% Male Age: (43) BMI: (30)	Parallel Group	12 weeks	Free living diet plan	60	1. Hypocaloric control diet 2. Hypocaloric control diet + exercise 3. Hypocaloric protein rich diet 4. Hypocaloric protein rich diet + exercise	1. Hypoenergetic (-500kcal/day). Target PRO:CHO ratio 1:3 (WHO standards) 2. Hypoenergetic (-500kcal/day). Target PRO:CHO ratio 1:3 (WHO standards). Supervised circuit training exercise 3d/week 3. Hypoenergetic (-500kcal/day). Target PRO:CHO ratio 1:1 (Fat	1. %E: C 49.5 P 16 F 33.8 g/d: C 171 P 56 F 53 Energy: 5822kJ/d 2. %E: C 50.2 P 18.4 F 29.4 g/d: C 160 P 59 F 42 Energy: 5271kJ/d	Yes	Not reported

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
								intake >30%).	3. %E: C 36.6 P 24.3 F 38.6 g/d: C 127 P 84 F 60 Energy: 5787kJ/d		
(Noakes *et al.*, 2006)	≥ 1 CHD risk factor BMI >28	Australia 17% Male Age: (48) BMI: (33)	Parallel Group	12 weeks	Free living diet plan	83	1. Very low carbohydrate 2. Very low fat 3. High unsaturated fat	All groups were isocaloric with 30% energy restriction during weeks 1-8, weight maintenance weeks 9-12. 36% of key foods provided to aid compliance	1. %E: C 12.4 P 30.5 F 54.3 Energy: 7706kJ/d 2. %E: C 66 P 20.3 F 12.5 Energy: 7000kJ/d 3. %E: C 48.7 P 21.4 F 28 Energy: 7659kJ/d	Yes	The National Heart Foundation of Australia
(O'Brien *et al.*, 2005) American LC study	Age >18y BMI 30-35 No CHD, T2DM or HTN No weight Δ >10% in past 6m	USA 0% Male Age: (44) BMI: (34)	Parallel Group	3 months	Free living diet plan	42	1. Moderate fat 2. Low carbohydrate	1. American Heart Association Step 1 diet + restrict to 1200kcal/d. Intended intake: 55% CHO, 15% PRO, 30% FAT 2. Ad libitum food intake. Max CHO intake 20g/d. CHO increased to 40-60g/d if ketosis was induced after 2 weeks.		Yes	University funding, NIH & American Heart Association Grant-in-Aid
(Olendzki *et al.*, 2009)	Age 18-70y BMI >25	USA 16% Male Age: (48) BMI: (31)	Parallel Group	3 months	Free living diet plan	31	1. Hypo-energetic high fibre 2. Hypo-energetic low saturated fat 3. Hypo=energetic high fibre and low saturated fat	In all conditions, energy restriction goal plus: 1. Increase fibre to 30g/day 2. saturated fat < 7% 3. low saturated fat <7% and high fibre > 30g	1. %E: C 51.4 P F 27.6 Energy: 1511 kcal/d Fibre g/d:24.6 2. %E: C 49.9 P F 27.5 Energy: 1523 kcal/d Fibre g/d:17.4 3. %E: C 52.1 P F 26.2 Energy: 1511 kcal/d Fibre g/d:23.7	Yes	Not reported

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(Pasman *et al.*, 1997)	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	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)
(Pereira *et al.*, 2004)	Age 18-35y BMI >25 Generally healthy No medications which influence outcomes No recent weight loss program Non smokers Not extremely athletic/active Weight stable	USA 23.7% Male Age: (31) BMI: mean not reported	Parallel Group	Mean interval from baseline to follow-up = 65d in low GL group and 69d in low fat	All food provided	39	1.Hypoenergetic low GL diet 2.Hypoenergetic low fat diet	1. Energy restricted low glycaemic load diet (60% of predicted requirements). GI 50, GL 82 2. Energy restricted low fat diet (60% of predicted requirements). 18%FAT. GI 82, GL 205. NCEP Step 1 diet	1. %E: C 43 P 27 F 30 Energy: 1500 kcal/d Fibre g/d:32 2. %E: C 65 P 17 F 18 Energy: 1500 kcal/d Fibre g/d:20	Yes	National Institute of Diabetes, NIH, Digestive and Kidney Diseases, Charles H. Hood Foundation and General Mills
(Philippou *et al.*, 2009)	≥1 cardiac risk factor (BMI 27-35 kg/m2, waist ≥94 cm, total cholesterol to high-density lipoprotein ratio ≥5.0, raised BP up to a maximum of 140/90mmHg) No medication	UK 100% Male Age: 35 - 65 BMI: mean not reported	Parallel Group	6 months	Substitution	56	1. High GI 2. Low GI	Those with BMI>25 also received weight management advice 1. High GI, carbohydrate foods (e.g. white/wholemeal bread, cornflakes, weetabix, potatoes, couscous, risotto rice, melon, pineapple, rice cakes) 2. Low GI, carbohydrate foods (e.g. seeded bread, wholemeal pita, muesli, porridge, sweet potatoes, pasta, noodles, basmati slow-cook rice, beans, lentils, apples, dried fruit, nuts)	Both groups decreased EI (greater in low GI group), but no macronutrient differences between groups	Yes	British Heart Foundation
(Phillips *et al.*, 2008)	Age 18-50y BMI 29-39 Generally healthy No CHD, T2DM or HTN Non smokers Not hyperlipidaemic/	USA 25% Male Age: mean not reported	Parallel Group	6 weeks	All food provided	28	1. Low carbohydrate diet 2. Low fat diet	1. Isocaloric groups. Low carbohydrate Atkins-style diet (20g/d CHO). 750kcal/d energy deficit weeks 1-4 weeks. 2. AHA low fat diet (30% total energy from fat). 750kcal/d	1. g/d: C 20 2.%E: F 30	No, all food provided	NIH & the Medical College of Wisconsin Cardiovascular

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
	hyper-cholesterolaemic	BMI: mean not reported						energy deficit weeks 1-4.			Centre
(Poppitt *et al.*, 2002)	≥3 metabolic syndrome risk factors Age >38y No intention to begin a weight loss program Not on weight loss diet Overweight/ Obese	Europe 31% Male Age: (46) BMI: (32)	Parallel Group	6 months	Free living diet plan	46	1. Low-fat, high-simple carbohydrate diet 2. Low-fat high-complex carbohydrate diet 3. Control diet	1. 60-70% of the diet was provided. 17.6% energy from simple CHO, 35.5% energy from complex CHO 2. 60-70% of the diet was provided. 28.9% energy from simple CHO, 28.5% energy from complex CHO 3. 60-70% of the diet was provided. 20.6% energy from simple CHO, 28.6% energy from complex CHO	1. %E: F 26 Energy: 7316kJ/d 2. %E: F 19.6 Energy: 9790kJ/d 3. %E: F 31.2 Energy: 8281kJ/d	Yes	EU-FAIR program and European Sugar Industries
(Raben *et al.*, 2002) Danish Sweetened Beverage Study	Age 20-50y BMI 25-30 Generally healthy Not on weight loss diet	Denmark 15% Male Age: mean not reported BMI: 28	Parallel Group	10 weeks	Supplement	42	1. Sucrose 2. Sweetener	1. Sucrose-containing food and drinks provided ~2g/kg/day (~23% total energy). 80% of sucrose within drinks and 20% within food. 2. Food and drinks provided matched sucrose intervention but contained artificial sweeteners	From supplements: 1. g/d: C 176 P 9 F 9 Energy: 3349kJ/d 2. g/d: C 31 P 9 F 9 Energy: 963kJ/d	Yes	Government funding & Danisco Sugar.
(Rigaud *et al.*, 1990)	Age 16-60y BMI >25 No medications which influence outcomes No T2DM Weight stable	France 21% Male Age: (37) BMI: (29)	Parallel Group	6 months	Free living diet plan	52	1. Hypocaloric diet + fibre tablets 2. Hypocaloric diet + placebo tablets	1. Hypoenergetic (25-30% below run-in period diet) diet with a dietary fibre tablets (beet, barley, citrus fibre, 90% insoluble) providing 7g/day. 2. Hypoenergetic (25-30% below run-in period diet) diet with placebo tablets containing 1g fibre/d.		Yes	Not reported

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(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	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
(Saltzman *et al.*, 2001) American Oat Study	BMI 25-35 Generally healthy Moderate alcohol intake No HTN No medications which influence outcomes Non smokers Not extremely athletic/active Weight stable	USA 49% Male Age: (44.7) BMI: (26.3)	Parallel Group	6 weeks	All food provided	43	1. Control 2. Oats	1. Hypocaloric (minus 4.2 MJ/d). Same macronutrient composition as intervention but with 45g/1000 kcal of wheat products instead of oats. 2. Hypocaloric (minus 4.2 MJ/d). Same macronutrient composition as control but with 45g/1000 kcal of rolled oats.	1. g/d: C 234 P 82 F 69 Energy: 7833kJ/d Fibre g/d:12.5 2. g/d: C 229 P 79 F 67 Energy: 7645kJ/d Fibre g/d:16.3	Yes	Quaker Oats Company, NIH and Government funding
(Schwab *et al.*, 2006)	Abnormal glucose metabolism Age 30-65y BMI <35 No CHD No insulin treatment Not taking lipid lowering drugs Plasma glucose <8 mmol/l TC <7.5 mmol/l TG <4 mmol/l	Finland 43.9% Male Age: (53) BMI: (29)	Parallel Group	12 weeks	Supplement	70	1. Pectin 2. Polydextrose 3. Placebo	1. Sugar-beet pectin, drinks. 400ml/day, containing 16g pectin, of which 76% soluble fibre 2. Polydestrose, drinks. 400ml/day, containing 40g/d polydextrose 3. Placebo drinks 400ml/d	1. %E: C 51.3 P 17.8 F 28.4 Energy: 7768kJ/d 2. %E: C 51.3 P 17.8 F 26.4 Energy: 7978kJ/d 3. %E: C 53.2 P 18.8 F 26.3 Energy: 7978kJ/d	Yes	Danisco Ltd
(Sciarrone *et al.*, 1993)	Age 30-59y No chronic illness Normal BP only >120% ideal body weight Omnivorous	Australia 100% Male Age: (41)	Parallel Group	6 weeks	Free living diet plan	21	1. Omnivorous diet 2. Lacto-ovovegetarian	1. Omnivorous diet 25% total energy complex carbohydrates, 20% sugar + fibre intake <8g/1000kcal 2. Lacto-ovovegetarian diet 35%	1. g/d: C 314 P 100 F 114 Energy: 2658 kcal/d Fibre g/d:24	Yes	Research institute funding; the National Heart Foundation; the Clive &

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
		BMI: (26)					diet	total energy complex carbohydrates, 20% sugar + fibre intake of approx 20g/1000kcal	2. g/d: C 339 P 78 F 86 Energy: 2437 kcal/d Fibre g/d:41		Vera Ramaciotti Foundation and Sanitarium Health Foods
(Smith *et al.*, 2008)	<5kg Δ weight in previous 3m Age 22-66y BMI <30 Free of chronic disease Generally healthy Mild to moderate lipidaemias No medications which influence outcomes Non smokers	USA 29% Male Age: mean not reported BMI: mean not reported	Parallel Group	6 weeks	Supplement	90	1. Beta glucan, low molecular weight 2. Beta glucan, high molecular weight	1. Low molecular weight barley B-glucan. 6g B-glucan per day was given as a dietary supplement powder, consumed as a beverage with morning and evening meals. 2. High molecular weight barley B-glucan. 6g B-glucan per day was given as a dietary supplement		Yes	NIH
(Surwit *et al.*, 1997)	Generally healthy No medications which influence outcomes Non smokers Sedentary only	UK 0% Male Age: 41 BMI: 36	Parallel Group	6 weeks	All food provided	52	1. High sucrose diet 2. Low sucrose diet	1. Hypoenergetic diet: low fat high sucrose diet (43% TE from sucrose) 2. Hypoenergetic diet: low fat, low sucrose diet (4% TE from sucrose)	1. %E: C 73.3 P 18.7 F 10.8 Energy: 4552.2kJ/d Fibre g/d:10.4 2. %E: C 70.9 P 19.3 F 10.6 Energy: 4840.9kJ/d Fibre g/d:14.9	Yes	NIH and The Sugar Association, Inc and the Kellogg Company, Inc
(Swain *et al.*, 1990)	No HTN Not hyperlipidaemic/ hyper-cholesterolaemic Not obese(<120% desirable body weight) Not taking lipid lowering drugs	USA 20% Male Age: 23 - 49(30) BMI: mean not reported	Crossover (washout 2 weeks)	6 weeks	Supplement	24	1. Oat bran supplement 2. Low fibre wheat supplement	1. Participants were asked to eat muffins or entrees containing a total of 100g oat bran/d. 2. Participants were asked to eat muffins or entrees containing a total of 100g low fibre wheat/d.	1.%E: Fat 35, fibre 39g/d, 2429 kcal 2. .%E: Fat 30, fibre 18g/d, 2315 kcal	Yes	National, Heart, lung and Blood Institute & NIH
(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	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	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	Yes	NIH

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
									Fibre g/d:18.5		
(Vasilaras *et al.*, 2001) CARMEN	Age 20-55y BMI 26-35 Generally healthy Moderate alcohol intake No medications which influence outcomes No weight loss >5kg in past 6m Not extremely athletic/active Not on weight loss diet	Denmark 45.8% Male Age: (42) BMI: (31)	Parallel Group	6 months	All food provided	30	1. Low-fat, high-simple carbohydrate diet 2. Low-fat high-complex carbohydrate diet 3. Control diet	1. Consumption of provided food was ad libitum and participants were allowed additional food outside those provided. 2. Consumption of provided food was ad libitum and participants were allowed additional food outside those provided. 3. Consumption of provided food was ad libitum and participants were allowed additional food outside those provided. Control diet corresponds to average national intake.	1. %E: C 51.6 P 15.3 F 25.7 Energy: 10.8kJ/d 2. %E: C 49.3 P 18.8 F 26.4 Energy: 10.5kJ/d 3. %E: C 47.7 P 17.2 F 31.3 Energy: 9.6kJ/d	Yes	EU-FAIR program, research institute funding & the European Sugar Industries
(Wolever and Mehling, 2002) American GI & carbohydrate study	≥1 diabetes risk factor Age 30-65y BMI <40 Impaired glucose tolerance Not hyperlipidaemic/ hypercholesterolaemic	USA 20% Male Age: (57) BMI: (30)	Parallel Group	4 months	Free living diet plan	37	1. High carbohydrate, high GI 2. High carbohydrate, low GI 3. Low carbohydrate, high MUFA	1. Ad libitum diet. 55%CHO, 30%FAT. At least one serving of a high GI food with each meal. Provided foods included breakfast cereal, breads, polished rice, crackers and instant potato 2. Ad libitum diet. 55%CHO, 30%FAT. At least one serving of a low GI food with each meal. 3. Ad libitum diet. 45%CHO, 40%FAT (20%MUFA).	1. %E: C 52.8 P 17.4 F 27.9 Energy: 1712 kcal/d Fibre g/d:22.7 2. %E: C 54.8 P 19.4 F 24.7 Energy: 1693kcal/d Fibre g/d:36.2 3. %E: C 47.4 P 16.4 F 35.4 Energy: 1877 kcal/d Fibre g/d:23.7	Yes	Canadian Diabetes Association and the International Olive Oil Council

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial design (washout duration)	Length of intervention	Intervention style	Total n	Intervention groups	Intervention description	Diet/ Supplement nutritional characteristics	Actual diet consumed reported?	Funding source
(Wood *et al.*, 2007) American Soluble Fibre Study	<2.5kg Δ weight in previous 6m Age 20-69y BMI 25-35 DBP <90mmHg No CHD or T2DM Not taking lipid lowering drugs SBP <160mmHg	USA 100% Male Age: 20 - 69(39) BMI:25 - 35(30)	Parallel Group	12 weeks	Free living diet plan	30	1. Low carbohydrate diet + konjac-mannan 2. Low carbohydrate diet + maltodextrin	1. Ad libitum diet: 13% CHO, 27% PRO, 60% FAT. Supplement: Konjac-mannan 3g/d 2. Ad libitum diet: 13% CHO, 27% PRO, 60% FAT. Supplement: Maltodextrin 3g/d	1. %E: C 12.5 P 28.4 F 60.7 Energy: 6866kJ/d Fibre g/d:12.7 2. %E: C 13.3 P 27.1 F 59.6 Energy: 7017kJ/d Fibre g/d:9.6		University funding & Nutraquest

Table 2.5 Risk of bias

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
(Andersson *et al.*, 2007)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Appel *et al.*, 2005)	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Bell *et al.*, 1990)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Bellisle *et al.*, 2007)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Birketvedt *et al.*, 2000)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Black *et al.*, 2006)	No Bias	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Brehm *et al.*, 2003)	No Bias	Bias	Bias	Unclear	Unclear	No Bias	No Bias
(Brehm *et al.*, 2005)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Cairella *et al.*, 1995)	No Bias	Unclear	No Bias	No Bias	Unclear	Bias	Bias
(Claessens *et al.*, 2009)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Clifton *et al.*, 2004)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(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
(Davy *et al.*, 2002)	Unclear	Unclear	Bias	No Bias	No Bias	No Bias	No Bias
(de Luis *et al.*, 2008)	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(de Luis *et al.*, 2009a)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(de Luis *et al.*, 2009b)	No Bias	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Delbridge *et al.*, 2009)	No Bias	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Ebbeling *et al.*, 2007)	No Bias	No Bias	Bias	No Bias	No Bias	No Bias	No Bias
(Ebbeling *et al.*, 2005)	Unclear	Unclear	Unclear	Unclear	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
(Golay *et al.*, 2000)	Unclear	Unclear	Unclear	Bias	Bias	No Bias	No Bias
(He *et al.*, 2004)	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Howard *et al.*, 2006b)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias

Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Keogh *et al.*, 2007)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Keogh *et al.*, 2008)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Kleemola *et al.*, 1999)	Unclear	Unclear	Bias	Bias	Unclear	No Bias	No Bias
(Landin *et al.*, 1992)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Lee *et al.*, 2009)	No Bias	No Bias	Bias	Bias	Bias	No Bias	No Bias
(Lehtimaki *et al.*, 2005)	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Leidy *et al.*, 2007)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Ley *et al.*, 2004)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Lovejoy *et al.*, 2003)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Maki *et al.*, 2007a)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Maki *et al.*, 2007b)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Marett and Slavin, 2004)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Meckling *et al.*, 2004)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Meckling and Sherfey, 2007)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Noakes *et al.*, 2006)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(O'Brien *et al.*, 2005)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Olendzki *et al.*, 2009)	No Bias	Unclear	Bias	Bias	Bias	Unclear	Unclear
(Pasman *et al.*, 1997)	Unclear	Unclear	Bias	Unclear	Bias	Bias	Bias
(Pereira *et al.*, 2004)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Philippou *et al.*, 2009)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Phillips *et al.*, 2008)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Poppitt *et al.*, 2002)	Unclear	Unclear	Bias	Bias	Unclear	No Bias	No Bias
(Raben *et al.*, 2002)	No Bias	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Rigaud *et al.*, 1990)	Unclear	Unclear	No Bias	No Bias	Bias	Unclear	Unclear
(Sacks *et al.*, 2009)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Saltzman *et al.*, 2001)	No Bias	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Schwab *et al.*, 2006)	Unclear	Unclear	No Bias	No Bias	Unclear	No Bias	No Bias
(Sciarrone *et al.*, 1993)	Unclear	Unclear	Bias	No Bias	No Bias	No Bias	No Bias

Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Smith *et al.*, 2008)	No Bias	Unclear	No Bias	No Bias	No Bias	Bias	Bias
(Surwit *et al.*, 1997)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Swain *et al.*, 1990)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Tinker *et al.*, 2008)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias
(Vasilaras *et al.*, 2001)	No Bias	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias
(Wolever and Mehling, 2002)	No Bias	Unclear	Bias	Unclear	Unclear	Bias	Bias
(Wood *et al.*, 2007)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias

Results – Incident Hypertension

This section includes studies that have reported the influence of dietary carbohydrates on incident hypertension only. No randomised controlled trials provided data on the number of new cases of hypertension that were diagnosed within the time frame of the trial, therefore this evidence base is reliant on results of cohort studies only.

Incident hypertension and fructose

Summary of cohort results

Data were extracted from one publication, reporting results from three cohort studies: the Nurses' Health Study (NHS), the Nurses' Health Study II and the Health Professionals' Follow-up Study (Forman *et al.*, 2009). The studies estimated fructose intake using FFQs and present results by quintile of per cent total energy. All three cohorts reported risk estimates close to 1, implying that there is no association between fructose intake and risk of incident hypertension. Appropriate confounders were adjusted for including age, BMI and smoking status.

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

No RCTs reported outcomes concerning fructose and incident hypertension.

Table 2.6 Incident hypertension and fructose: 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	Contrast (mean)	RR (CI)	Adjustments
(Forman *et al.*, 2009) 13913 HPFS	USA, Primarily White, Cancer free, No CHD, No hypertension, No T2DM	40-75 %M 100	(11192) /51529	18 years	FFQ (131)	Fructose intake (% of total energy)	Incident hypertension Self-reported	Q5 vs. Q1	0.99 (0.93, 1.05)	Age, alcohol, BMI, caffeine, family history of hypertension, folate, physical activity, smoking, vitamin C
13354 NHS		30-55 %M 0	(31107) /121700	20 years	FFQ (126)	Fructose intake (% of total energy)	Incident hypertension Self-reported	Q5 vs. Q1	1.02 (0.99, 1.06)	As above
13912 NHS II	USA, Primarily White, Cancer free, No CHD, No T2DM	24-44 %M 0	(15863) /116671	14 years	FFQ (133)	Fructose intake (% of total energy)	Incident hypertension Self-reported	Q5 vs. Q1	1.03 (0.98, 1.08)	As above

Incident hypertension and dietary fibre

Summary of cohort results

Two studies: the Nurses' Health Study (Ascherio *et al.*, 1996) and the Health Professionals' Follow-up Study (Ascherio *et al.*, 1992) presented data on dietary fibre (calculated using the Association of Official Analytical Chemist (AOAC) method) and incident hypertension. Both studies relied on self-reports of blood pressure and incident hypertension and dietary fibre was assessed by FFQs. Findings from the Nurses' Health Study (Ascherio *et al.*, 1996) did not suggest an association with dietary fibre, while the Health Professionals' Follow-up Study (HPFS) (Ascherio *et al.*, 1992) provided evidence of an increased risk of hypertension in participants with the lowest intakes of dietary fibre, 11.9g/d vs. >24g/d (Relative Risk (RR) 1.57 95% Confidence Intervals (Cl) 1.2 to 2.05). Risk estimates tended to be elevated in all cohort subgroups, but were statistically significant only in participants younger than age 50, with BMI less than 28kg/m^2 and in very low or high alcohol consumers. Both studies included appropriate adjustments such as age, alcohol and BMI. There was an insufficient number of studies to conduct a meta-analysis.

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

No RCTs reported outcomes concerning dietary fibre and incident hypertension.

Table 2.7 Incident hypertension 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 Assessment	Exposure	Outcome/ Assessment Details	Sub-group Detail	Contrast (mean)	Exposure Units	RR (CI)	p	p trend	Adjustments
(Ascherio *et al.*, 1996) 13806 NHS	USA, Primarily White, Cancer free, No CHD, No T2DM	30-55 %M 0	(2526) /121700	4 years (0.9)	FFQ (126)	Dietary Fibre, g/d (AOAC method)	Incident hypertension Self-reported		>25 vs. <10	g/day	1.01 (0.8, 1.29)		0.75	Age, alcohol, BMI
(Ascherio *et al.*, 1992) 13484 HPFS	USA, Primarily White, Cancer free, No CHD, Normal BP only, No T2DM	40-75 %M 100	(1248) /51529	4 years (9.7)	FFQ (131)	Dietary Fibre, g/d (AOAC method)	Incident hypertension Self-reported		<11.9 vs. >24	g/day	1.57 (1.2, 2.05)		0.0001	Age, alcohol, BMI
13857 HPFS			Not reported					Age <50	<11.9 vs. >24	g/day	2.04	<0.001		Age, alcohol, BMI
13859 HPFS			Not reported					Age >50	<11.9 vs. >24	g/day	1.42			Age, alcohol, BMI
13860 HPFS			Not reported					BMI <23	<11.9 vs. >24	g/day	1.68			Age, alcohol
13861 HPFS			Not reported					BMI 23-28	<11.9 vs. >24	g/day	1.77	<0.001		Age, alcohol
13862 HPFS			Not reported					BMI >28	<11.9 vs. >24	g/day	1.22			Age, alcohol
13863 HPFS			Not reported					Alcohol <0.1 g/d	<11.9 vs. >24	g/day	2.0	<0.01		Age, BMI
13864 HPFS			Not reported					Alcohol 0.1-19 g/d	<11.9 vs. >24	g/day	1.29			Age, BMI
13865 HPFS			Not reported					Alcohol >19 g/d	<11.9 vs. >24	g/day	1.98	<0.01		Age, BMI

Incident hypertension and cereal foods

Summary of cohort results

Data were extracted from two publications reporting results from two cohort studies, one including Japanese participants (Kanda *et al.*, 1999) and one US participants (Flint *et al.*, 2009). Rice intake was assessed through a questionnaire in the Japanese Blood Pressure Study (Kanda *et al.*, 1999) and a 131 item FFQ was used in the Health Professionals' Follow-up Study to assess total bran and germ intake (Flint *et al.*, 2009). Both studies described a negative association with increasing cereal food intake, suggesting a lower risk of incident hypertension with increasing consumption of cereal foods (Kanda *et al.*, 1999) (Flint *et al.*, 2009); only the US study (Flint *et al.*, 2009), however, found a trend with total bran to be statistically significant (p trend=0.002). The Health Professionals' Follow-up Study (Flint *et al.*, 2009) used appropriate adjustments but no confounders were adjusted for in the Japanese Blood Pressure Study (Kanda *et al.*, 1999). There was an insufficient number of studies to conduct a meta-analysis.

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

No RCTs reported outcomes concerning cereal foods and incident hypertension.

Table 2.8 Incident hypertension and cereal 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)	p trend	Adjustments
(Kanda *et al.*, 1999) 14159 Japanese Blood Pressure Study	Japan	60-69 %M 37	(54) /948	4 years	Questionnaire (general)	Rice, total boiled	Incident hypertension Clinic BP	>4 vs. <4	Bowls /day	0.5 (0.05, 5.09)		No adjustments
(Flint *et al.*, 2009) 13358 HPFS	USA, Primarily White, Cancer free, No CHD, No hypertension	40-75 %M 100	(9227) /51529	18 years	FFQ (131)	Bran, Total	Incident hypertension Self-reported	(12) vs. (0.3)	g/day	0.85 (0.78, 0.92)	0.002	Age, alcohol, blood total cholesterol, energy intake, family history of CVD, family history of hypertension, Fruit intake, height, marital status, occupation, physical activity, salt intake, smoking, supplements, vegetable intake
13359 HPFS						Germ, total	Incident hypertension Self-reported	(2.4) vs. (0.1)	g/day	0.96 (0.88, 1.04)	0.11	As above

Incident hypertension and legumes

Summary of cohort results

Data were extracted from one publication reporting results from one cohort study: the CARDIA study (Steffen *et al.*, 2005). In this study, black and white men and women aged 18-30 years underwent a baseline examination and dietary assessment and were then followed up over a period of 15 years. Legume consumption was measured using a 700 item FFQ. This study does not provide evidence of an association between intake of legumes and risk of incident hypertension. The CARDIA study (Steffen *et al.*, 2005) adjusted for appropriate confounders such as age, alcohol, gender and smoking status.

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

No RCTs reported outcomes concerning legumes and incident hypertension.

Table 2.9 Incident hypertension and legumes: 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	RR (CI)	p trend	Adjustments
(Steffen *et al.*, 2005) 13718 The CARDIA Study	USA, No hypertension, No T2DM	18-30 %M 45.9	(997) /5115	15 years	FFQ (700)	Legumes (excluding soy)	Incident hypertension Physician diagnosed/ medication use	0.2 vs. 0.1	times /day	0.88 (0.75, 1.03)	0.11	Age, alcohol, centre, education, energy intake, ethnicity, physical activity, gender, smoking, vitamin intake

Incident hypertension and wholegrains

Summary of cohort results

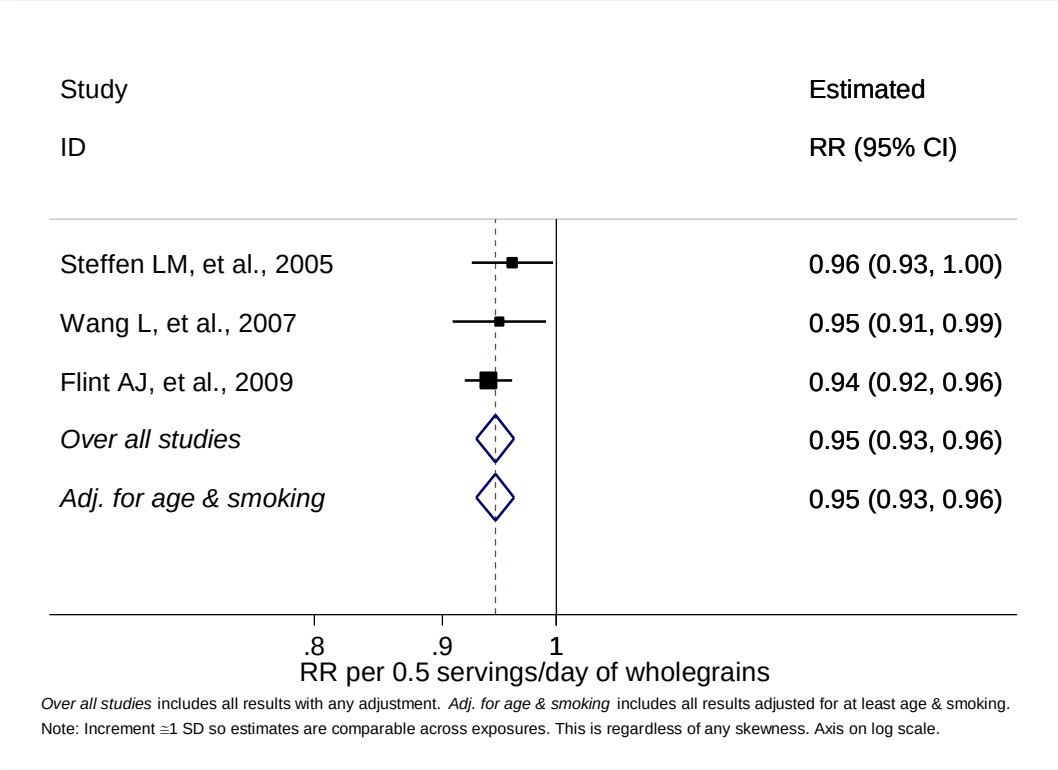
Data were extracted from three publications reporting results from three cohort studies: the HPFS (Flint *et al.*, 2009), the Women's Health Study (Wang *et al.*, 2007) and the CARDIA study ((Flint *et al.*, 2009;Wang *et al.*, 2007;Steffen *et al.*, 2005).

In these cohort studies, incident hypertension was defined by meeting ≥ 1 of 4 criteria in one study (the CARDIA study): self-reports of new physician diagnosis, self-reports of new antihypertensive treatment, self-reports of SBP ≥140mmHg or self reports of DBP ≥ 90mmHg (Wang *et al.*, 2007). The remaining studies either relied on self-reports of incident hypertension (Flint *et al.*, 2009) or defined hypertension as SBP ≥130mmHg, DBP ≥85mmHg or the use of hypertensive medications (Steffen *et al.*, 2005).

So that one study could be included in the meta-analysis, we assumed that the mean of the lowest category was half the upper limit of that category, and that the mean of the highest category was 1.5 times the lower limit of that category (Steffen *et al.*, 2005). So that another study could be included, we assumed that a standard serving of wholegrains is approximately 28g (Flint *et al.*, 2009).

The pooled estimate of relative risk from the cohort studies was 0.95 (95% CI: 0.93 to 0.96) per half serving per day of wholegrains ($p<0.001$).

Figure 2.1 Forest plot for wholegrains and incident hypertension



There was no excess heterogeneity between the cohort studies (I^2=0 %, 95% CI: 0% to 80%, Q=1.0, df=2, p=0.6). There were insufficient studies to explore sources of heterogeneity through subgroup analysis or meta-regression. There were insufficient studies to explore small-study effects, such as publication bias, through funnel plots or hypothesis tests. No one study had a dominant influence on the pooled estimate from the random effects analysis.

These studies collectively provide evidence of a reduction in risk of high blood pressure with increasing consumption of wholegrains.

Exposure definition and assessment

The American Association of Cereal Chemists International and the American Food and Drug Administration (FDA) defines whole grains as “intact, ground, cracked or flaked fruit of the grain whose principal components, the starchy endosperm, germ and bran, are present in the same relative proportions as they exist in the intact grain” (American Association of Cereal Chemists International, 1999;United States FDA, 2006). This approach therefore includes all foods with more than 51% whole-grain content.

Wholegrains were measured using 131- item or 700-item FFQs, in all studies. The studies differed in their definition of whole grain foods: the CARDIA study (Wang *et al.*, 2007) and The Women's Health Study (Steffen *et al.*, 2005) used non-FDA definitions which were based on the approach used by Jacobs *et al.* (Jacobs, Jr. *et al.*, 1998). This approach considered cereals to be wholegrain if the product contained >25% wholegrain or bran by weight. The Health Professionals Follow-up Study (Flint *et al.*, 2009) estimated wholegrains using the FDA definition.

While it is recommended that Americans eat at least three portions (around 85g) of whole grains per day, the UK does not currently have any specific recommendations other than the recommendation "to choose whole-grain varieties whenever you can" (USDA, 2010;Food Standards Agency, 2010).

All three studies included in the meta-analysis were conducted in the USA where the dominant grain type may differ from that in the UK and the rest of Europe.

Adjustment for appropriate confounders

The CARDIA study (Steffen *et al.*, 2005) and the Health Professionals Follow-up Study (Flint *et al.*, 2009) adjusted for variables such as age and energy intake but did not adjust for BMI. In the Health Professionals Follow-Up Study, further adjustment for the components of whole grains (bran, germ, total fibre etc.) did not influence the risk estimates materially. However, the inclusion of cereal fibre in the model did attenuate the association with risk of hypertension (RR of 0.94 (95% CI: 0.84, 1.05) for the highest quintile of whole grains (p for trend of 0.23). The Women's Health Study (Wang *et al.*, 2007) was the most fully adjusted.

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

No RCTs reported outcomes concerning wholegrain and incident hypertension.

Table 2.10 Incident hypertension and wholegrains: 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	Contrast (mean)	Exposure Units	RR (CI)	p trend	Adjustments
(Flint *et al.*, 2009) *13357 HPFS	USA, Primarily White, Cancer free, No CHD, No HTN	40-75 %M 100	(9227) /51529	18 years	FFQ (131)	Wholegrains (FDA definition)	Incident hypertension Self-reported		(46) vs. (3.3)	g/day	0.81 (0.75, 0.87)	<0.0001	Age, alcohol, blood total cholesterol, energy intake, family history of CVD, family history of HTN, fruit intake, height, marital status, occupation, physical activity, salt intake, smoking, supplements, vegetable intake
(Steffen *et al.*, 2005) *13716 The CARDIA Study	USA, No HTN, No T2DM	18-30 %M 45.9	(997) /5115	15 years	FFQ (700)	Wholegrains (non-FDA definition, Jacobs et al. 1998)	Incident hypertension Physician diagnosed/ medication use		>1.9 vs.<0.4	times /day	0.83 (0.67, 1.03)	0.03	Age, alcohol, centre, education, energy intake, ethnicity, physical activity, gender, smoking, vitamin intake
(Wang *et al.*, 2007) 13447 The Women's Health Study	USA, Primarily White, No CHD	(54) %M 0	(8722) /39876	10 years	FFQ (131)	Wholegrain proportion (whole grains consumed as a percent of total grains) (non-FDA definition, Jacobs et al. 1998)	Incident hypertension Self-reported		58-100 (69) vs. 0-18 (10)	% total grains	0.9 (0.84, 0.98)	0.002	Age, alcohol, BMI, dairy, energy intake, ethnicity, family history of MI, fruit intake, DM, hypercholesterolaemia, meat intake, menopause status, physical activity, smoking, supplements, group allocation, postmenopausal HRT, vegetable intake
*13434 The Women's Health Study						Total wholegrain foods			0.82-0.97 (0.89) vs. 0-0.49 (0.21)	servings/day	0.89 (0.82, 0.97)	0.007	As above
13448 The Women's Health Study			(3770) /39876					BMI <25	>4 (5) vs. <0.5 (0.28)	servings/day	0.72 (0.57, 0.9)		Age, dairy, energy intake, ethnicity, family history of MI, fruit intake, DM, hypercholesterolaemia, meat intake, menopause status, supplements, group allocation, postmenopausal HRT, vegetable intake
13450 The Women's Health Study			(4770) /39876					BMI >25	>4 (5) vs. <0.5 (0.28)	servings/day	0.81 (0.67, 0.98)		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	RR (CI)	p trend	Adjustments
13452 The Women's Health Study			(3441) /39876					No vigorous PA	>4 (5) vs. <0.5 (0.28)	servings/day	0.72 (0.56, 0.93)		As above
13453 The Women's Health Study			(5277) /39876					Some vigorous PA	>4 (5) vs. <0.5 (0.28)	servings/day	0.79 (0.66, 0.95)		As above
13454 The Women's Health Study			(7552) /39876					Never or former smoker	>4 (5) vs. <0.5 (0.28)	servings/day	0.8 (0.68, 0.93)		As above
13455 The Women's Health Study			(1160) /39876					Smokers	>4 (5) vs. <0.5 (0.28)	servings/day	0.52 (0.3, 0.9)		As above
13480 The Women's Health Study			(3985) /39876					Alcohol Non-drinkers	>4 (5) vs. <0.5 (0.28)	servings/day	0.71 (0.58, 0.88)		As above
13481 The Women's Health Study			(4732) /39876					Alcohol Drinkers	>4 (5) vs. <0.5 (0.28)	servings/day	0.83 (0.68, 1.02)		As above

*This result was used in the meta-analysis of wholegrains and incident hypertension

Incident hypertension and refined grains

Summary of cohort results

Two cohort studies provided data on refined grains intake in relation to incident hypertension (Wang *et al.*, 2007;Steffen *et al.*, 2005). In both The Women's Health Study (Wang *et al.*, 2007) and The CARDIA Study (Steffen *et al.*, 2005) intake of refined grains was measured using FFQs. No association was observed between refined grain consumption and risk of incident hypertension in these studies. Adjustments were made for variables such as age, alcohol, smoking status and physical activity. There was an insufficient number of studies to conduct a meta-analysis.

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

No RCTs reported outcomes concerning refined grains and incident hypertension.

Table 2.11 Incident hypertension and refined grains: 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)	p trend	Adjustments
(Wang *et al.*, 2007) 13437 The Women's Health Study	USA, Primarily White, No CHD	(54) %M 0	(8722) /39876	10 years	FFQ (131)	Refined grains	Incident hypertension Self-reported	3.12-24.5 (4.06) vs. 0-1.057 (0.76)	servings/day	0.97 (0.89, 1.06)	0.8	Age, alcohol, BMI, dairy, energy intake, ethnicity, family history of MI, fruit intake, DM, hypercholesterolaemia, meat intake, menopause status, physical activity, smoking, supplements, group allocation, postmenopausal HRT, vegetable intake
(Steffen *et al.*, 2005) 13717 The CARDIA Study	USA, No HTN, No T2DM	18-30 %M 45.9	(997) /5115	15 years	FFQ (700)	Refined grains	Incident hypertension Physician diagnosed/medication use	>4.3 vs. <1.8	times /day	0.87 (0.68, 1.12)	0.7	Age, alcohol, centre, education, energy intake, ethnicity, physical activity, gender, smoking, vitamin intake

Incident hypertension and sweetened beverages

Summary of cohort results

Data were extracted from one publication, reporting results from one cohort study (Dhingra *et al.*, 2007). In this study (The Framingham Heart Study) (Dhingra *et al.*, 2007), diet was assessed using a general questionnaire and blood pressure was measured by a physician. Findings from this study provide some evidence of an elevation in risk of high blood pressure in those that consumed at least one 12oz soft drink can per day compared to those who do not. Whilst the age, and gender-adjusted model was statistically significant, the fully adjusted model was not (p=0.10). It should be noted that the 'soft drink' exposure reported here included both sugar-sweetened and artificially sweetened carbonated beverages.

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

No RCTs reported outcomes concerning sweetened beverages and incident hypertension.

Table 2.12 Incident hypertension 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	RR (CI)	Adjustments
(Dhingra *et al.*, 2007) 14263 The Framingham Heart Study	USA, No CHD, no metabolic syndrome	(53) %M 43	(1004) /8997	4 years	Questionnaire (general)	Total sugar or artificially sweetened beverages (number of 12oz soft drink cans per day)	Incident hypertension Clinic BP	≥1 vs. 0	servings/day	1.18 (0.96, 1.44)	Age, smoking, SFA, energy intake, dietary fibre, magnesium Intake, physical activity, gender, trans-fatty acid intake

Results - Blood pressure

In the following section, data from studies that reported blood pressure as a continuous variable are provided.

Blood pressure and total carbohydrate and high carbohydrate diets

Summary of cohort results

Four publications from four cohort studies (Boreham *et al.*, 1999;Ludwig *et al.*, 1999;Schroeder *et al.*, 2007;Stamler *et al.*, 2002) reported the association between total carbohydrate as per cent energy on SBP and DBP. All but one of the studies involved adults; the exception was The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) which studied males and females aged 12-15 years at baseline. Two out of four studies measured blood pressure in clinics, whereas the Chicago Electric Western study (Stamler *et al.*, 2002) used measurements by a physician. The Middle-aged Runners Study (Schroeder *et al.*, 2007) did not report blood pressure assessment details.

Findings from the Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) were reported separately for males and females. In females no association was observed between dietary carbohydrate intake and blood pressure assessments. However, in males, there was some evidence of a positive association between blood pressure and energy derived from carbohydrate, with increasing intakes being reflected in increasing SBP (p=0.010). However, it should be noted that the duration of follow-up in this study was relatively short (4 years) and the authors caution that results should be interpreted cautiously.

The CARDIA study of young multi-ethnic adults, (Ludwig *et al.*, 1999) did not find evidence of statistically significant trends in either white or black adults in relation to total carbohydrate intake and SBP or DBP. Similarly, results from the Chicago Electric Western Study (Stamler *et al.*, 2002) described negative beta correlations, indicating an inverse relationship between total carbohydrate and SBP and DBP; however these were not found to be statistically significant.

One final study, The Middle-aged Runners Study (Schroeder *et al.*, 2007) which estimated total carbohydrate using a food diary, reported no effect on regression direction (beta-coefficients not provided in the paper).

The Chicago Western Electric study (Stamler *et al.*, 2002) reported an annual change in blood pressure (assessed by physician), whereas the Northern Ireland Young Hearts Study and the CARDIA study blood pressure provided measurements at the end of the period of follow-up only. The Middle-aged Runners Study (Schroeder *et al.*, 2007) did not provide assessment details.

Collectively, these studies provide inconsistent results concerning the association between blood pressure and dietary carbohydrate intake.

Unfortunately, the three studies of adults provided insufficient information (generally a lack of measures of variance around the risk estimates or an insufficient number of exposure categories or quantiles to derive a dose response estimate) to permit a meta-analysis.

Exposure definition and assessment

Diet was assessed using a dietary history, a 700-item FFQ, a food diary and both a dietary history and 195-item FFQ in the Northern Ireland Young Hearts Project (Boreham *et al.*, 1999), the CARDIA study (Ludwig *et al.*, 1999), the Middle-aged Runners Study (Schroeder *et al.*, 2007) and the Chicago Electric Western study (Stamler *et al.*, 2002) respectively. Total carbohydrate intake was recorded as either % energy (Boreham *et al.*, 1999;Ludwig *et al.*, 1999;Stamler *et al.*, 2002) or grams per day (Schroeder *et al.*, 2007).

Adjustment for appropriate confounders

The Chicago Electric Western Study (Stamler *et al.*, 2002) included appropriate covariates such as age, education and body weight in their analyses. The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999), the Middle-aged Runners Study (Schroeder *et al.*, 2007) and the CARDIA study (Ludwig *et al.*, 1999) did not adjust for BMI or body weight and should therefore be interpreted with caution.

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

Summary of RCT data

This section contains details of randomised controlled trials that assessed the effect of high carbohydrate compared to low carbohydrate diets on blood pressure as a marker of CVD. It should be noted that some of these trials were designed to assess other aspects of diet such as the nature of the carbohydrate (high or low GI, or whole grain content). However, if the difference in percentage carbohydrate from energy was greater than 5% between diet groups and all other inclusion criteria were met, the study was deemed to be eligible for inclusion in the meta-analysis. Details of trial characteristics are contained in the trial characteristics table (Table 2.4).

There were 32 publications identified which reported data on blood pressure and high carbohydrate diets. Most studies presented results for both SBP and DBP (or change in these), with just one study reporting only SBP (Dale *et al.*, 2009). Most studies used routine measures recorded in a clinic, four of which reported measurements as being recorded whilst seated (Ebbeling *et al.*, 2005;Ley *et al.*, 2004;de Luis *et al.*, 2009b;Maki *et al.*, 2007b). Two studies reported supine blood pressure (Leidy *et al.*, 2007;Phillips *et al.*, 2008). However, a large proportion of studies (6 trials) did not provide this detail (Golay *et al.*, 2000;Delbridge *et al.*, 2009;Tinker *et al.*, 2008;Dale *et al.*, 2009;de Luis *et al.*, 2008;Dale *et al.*, 2009;O'Brien *et al.*, 2005). Length of follow-up varied greatly between 6 weeks (Golay *et al.*, 2000) and 6 years (Tinker *et al.*, 2008), introducing some potential for heterogeneity in the study results. Whilst the longer follow-up is most relevant to cardiovascular disease risk, the shorter follow-up is most relevant to assessing the impact of the intervention.

Twenty five studies were included in the meta-analyses comparing different carbohydrate intakes and changes in blood pressure. One study reported results from four groups (Dansinger *et al.*, 2005) and two studies reported results from three groups (Gardner *et al.*, 2007;Sacks *et al.*, 2009). For these studies the group with the lowest carbohydrate intake was compared with the group with highest carbohydrate intake. One study was excluded that had differences in carbohydrate of less than 5% between groups (Dale *et al.*, 2009;Sacks *et al.*, 2009). One paper (Tinker *et al.*, 2008) was excluded because it duplicated results from another paper (Howard *et al.*, 2006b). In this case we selected the result closest to the end of the intervention, which is still ongoing in this trial, so the longest recorded follow-up was selected. Results from O'Brien *et al.* (O'Brien *et al.*, 2005) were also not included in the meta-analysis as these data are also provided in another publication from the same study (Brehm *et al.*, 2003). Two studies were excluded because they presented insufficient information to be included, e.g. no estimates or confidence intervals (Wolever and Mehling, 2002;Noakes *et al.*, 2006).

Studies were stratified according to whether fat or protein or both were adjusted as a result of changes in carbohydrate levels. Reported differences of more than 2% were taken as important. Seven studies reported differences in percentage fat with less than 2% difference in protein. Three studies reported changes in protein with less than 2% difference in fat and five studies reported differences in fat and protein of more than 2%. Studies where blood pressure was presented as percentage were converted to actual values in mmHg. The first follow up reported at the end of the intervention and over 6 weeks was used. For the studies included in the meta-analysis, this varied from 6 weeks to 6 years. The study reported by Golay *et al.* (Golay *et al.*, 2000) was unusual in that the aim was to evaluate the effect of `food combining' compared with a balanced macronutrient intake on metabolic parameters such as blood pressure. However, it was included in the meta-analysis as the carbohydrate differences between the groups met our inclusion criteria of >5% of energy.

Diastolic blood pressure (DBP)

Comparison of higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets

The pooled estimate for the 14 studies where carbohydrate was replaced with fat only, indicated that DBP was 0.02mmHg higher (95% CI, -0.81 to 0.86mmHg) in diets higher in carbohydrate and lower in fat diet compared with a low carbohydrate and high fat diet. This was not significantly different from zero (p=0.96). Heterogeneity denoted by I^2 was 37% (95% CI, 0 to 66%). A funnel plot indicated that there was some evidence of publication bias. Statistically, the pooled estimate indicates that diets higher in carbohydrate and lower in fat are not associated with a difference in DBP.

Figure 2.2 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and DBP

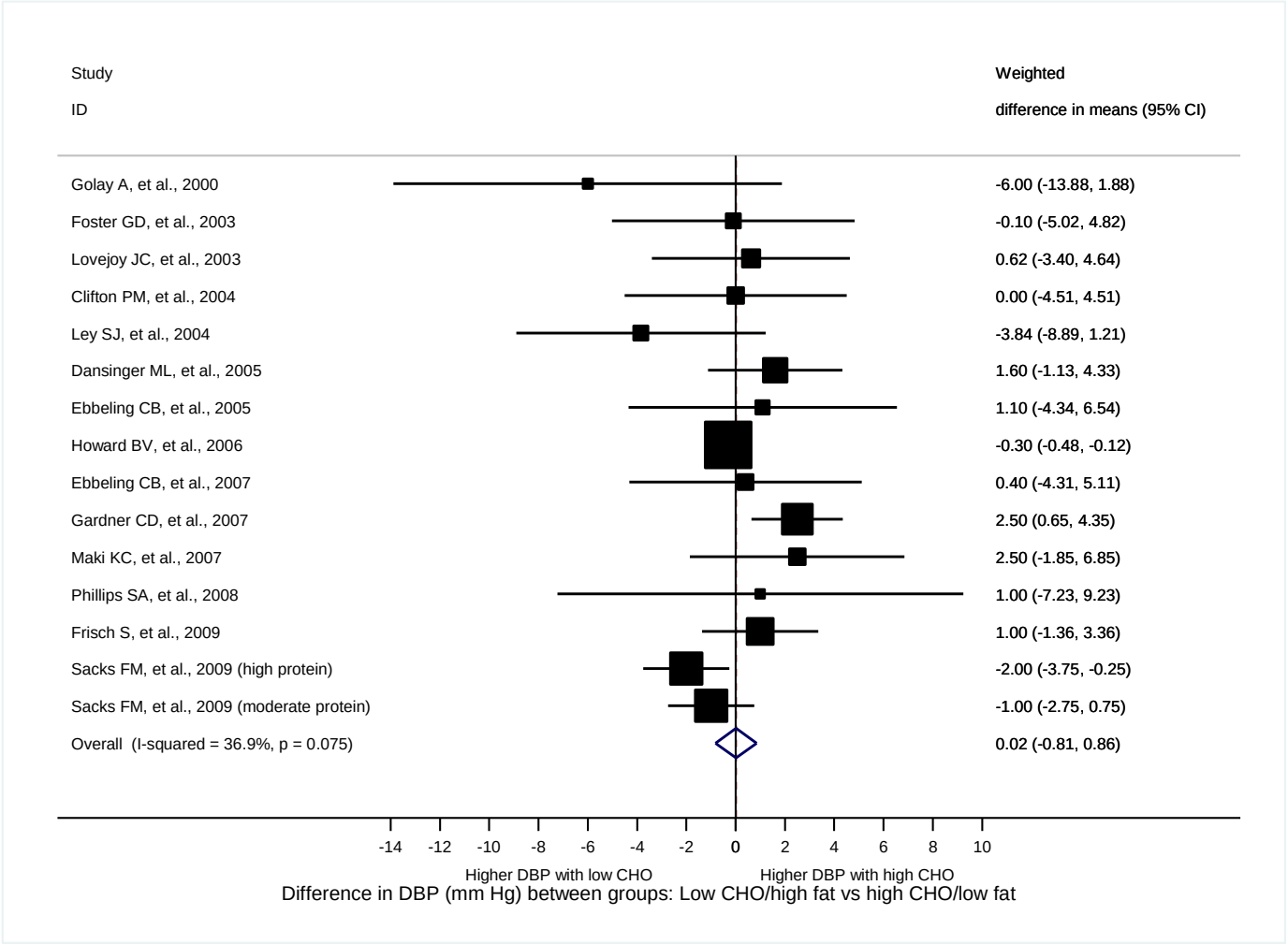
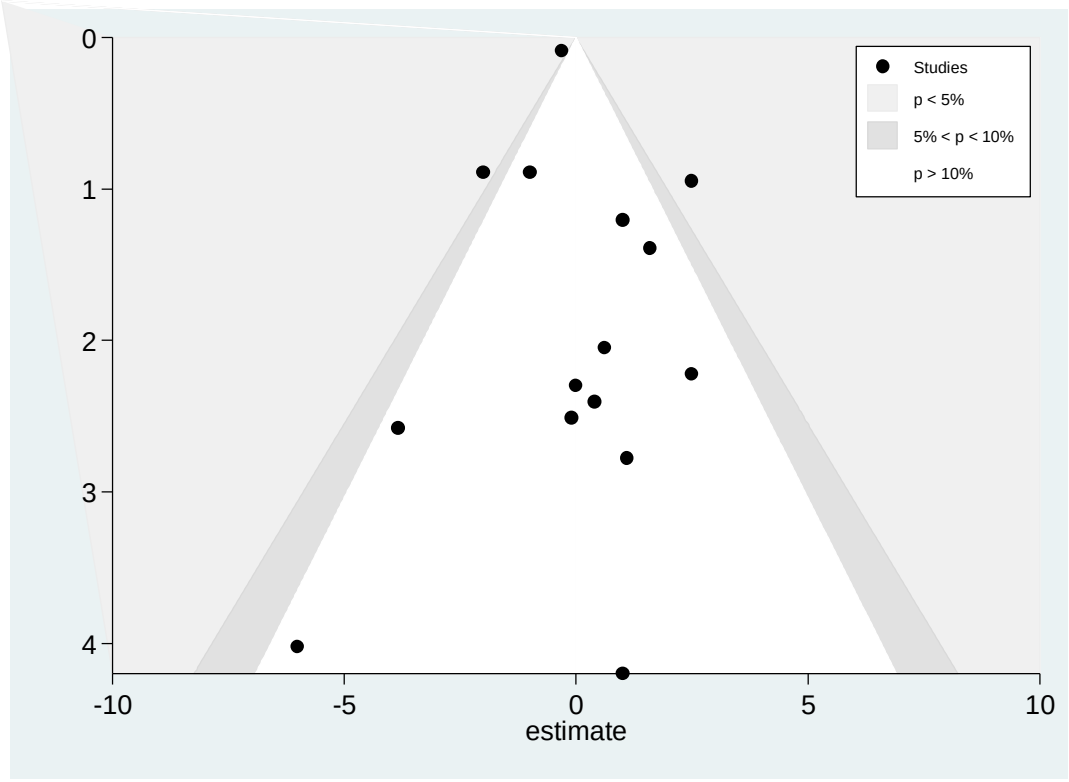


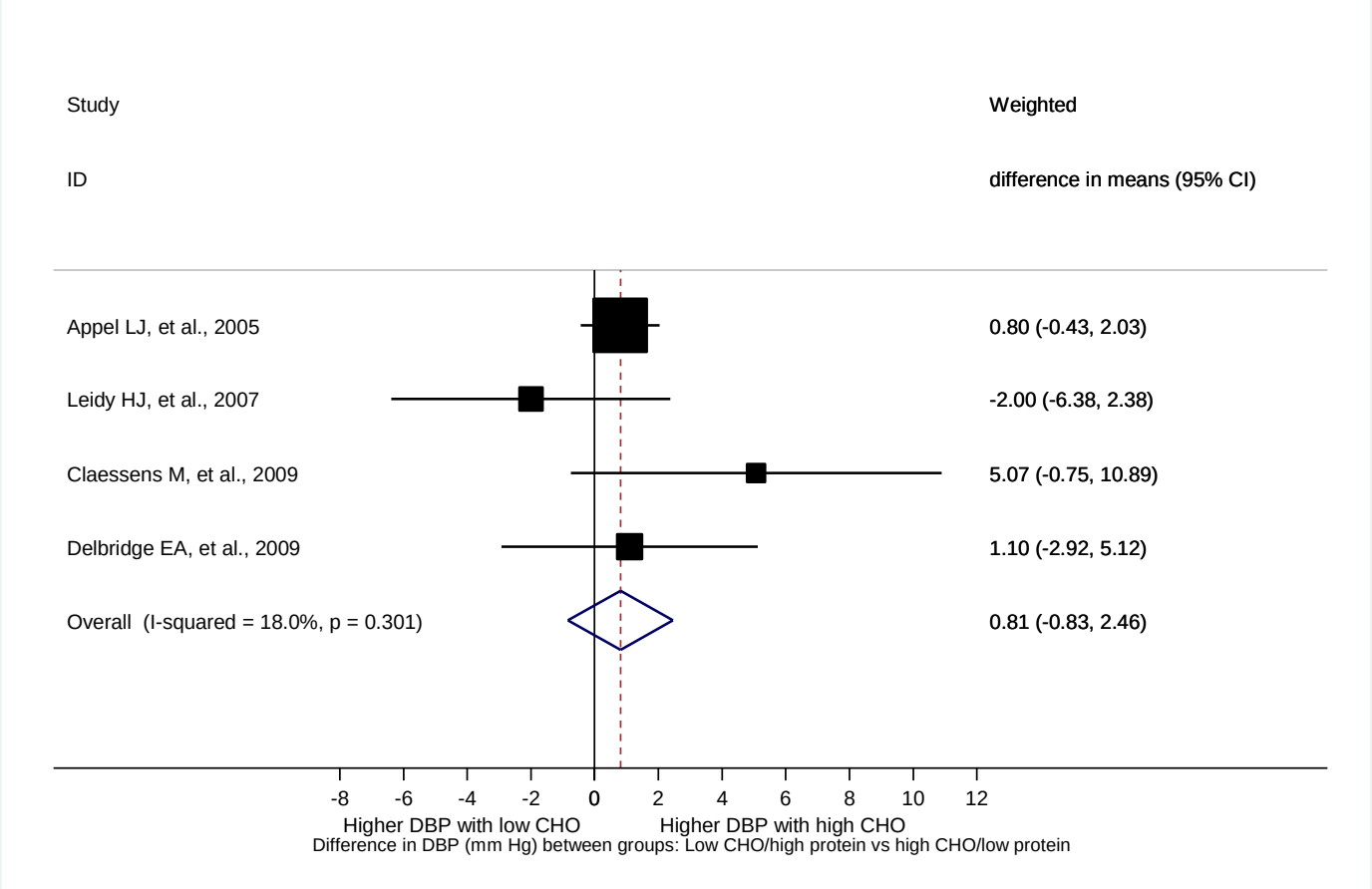
Figure 2.3 Contour-enhanced funnel plot for publications presenting DBP and higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets



Comparison of higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets

For the four studies with a change in protein and carbohydrate only, DBP was 0.81mmHg higher (95% CI, -0.83 to 2.46mmHg) with consumption of a higher carbohydrate lower protein diet compared with a lower carbohydrate, higher protein diet. This was not significantly different from zero (p=0.33). Overall heterogeneity denoted by I^2 was 18% (95% CI, 0 to 87%). There were insufficient studies to present a funnel plot. Statistically, the pooled estimate indicates that diets higher in carbohydrate and lower in protein are not associated with a difference in DBP.

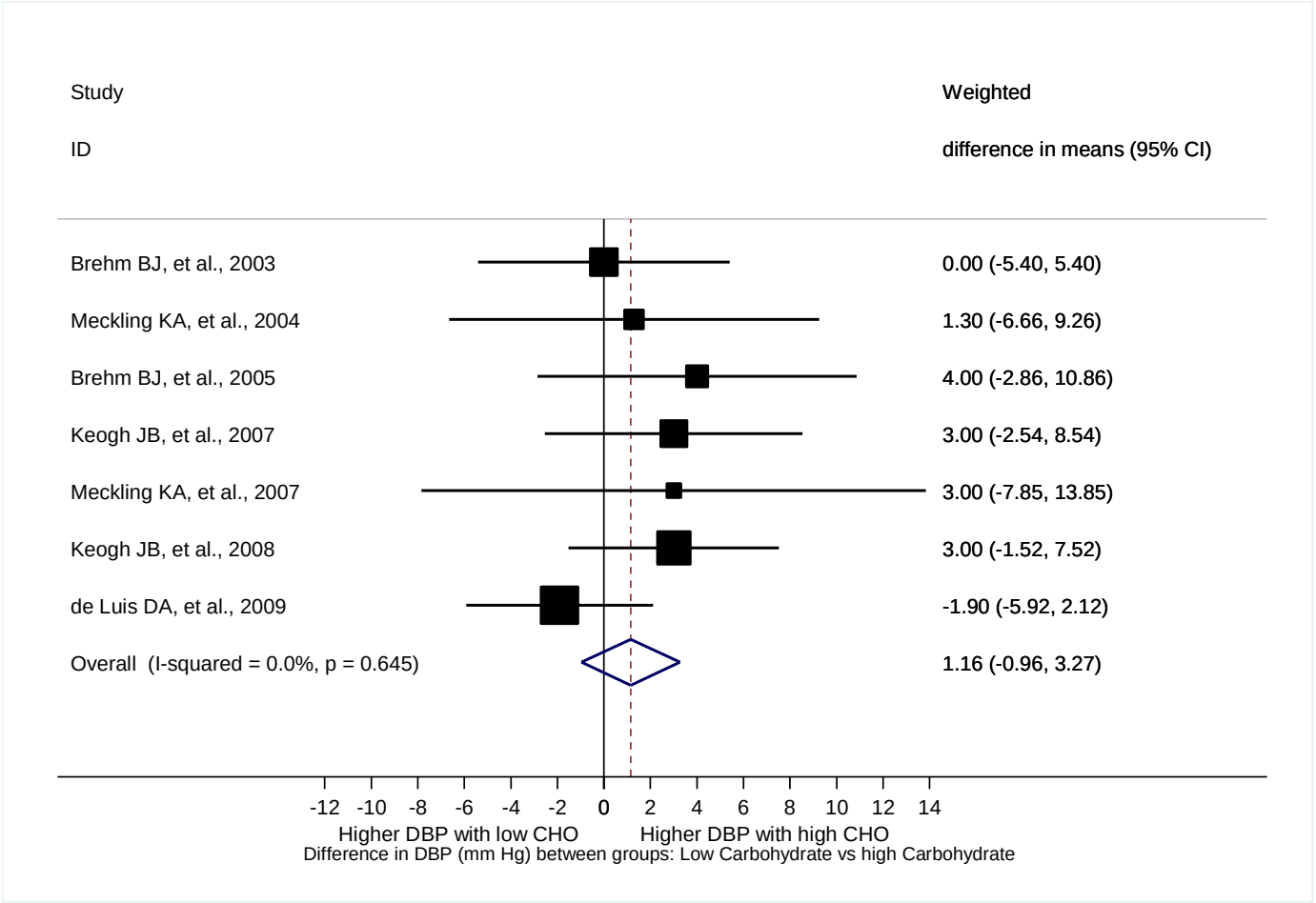
Figure 2.4 Forest plot for higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets and DBP



Comparison of higher carbohydrate, lower fat and protein diets and lower carbohydrate, higher fat and protein diets

For the seven studies with a change in carbohydrate, fat and protein, DBP was 1.16mmHg higher (95% CI, -0.96 to 3.27mmHg) with consumption of a higher carbohydrate lower fat and protein diet compared with a lower carbohydrate, higher fat and protein diet. This was not significantly different from zero (p=0.29). Overall heterogeneity denoted by I^2 was 0% (95% CI, 0 to 59%). There were insufficient studies to present a funnel plot. Statistically, the pooled estimate indicates that diets higher in carbohydrate and lower in fat and protein are not associated with a difference in DBP.

Figure 2.5 Forest plot for higher carbohydrate, lower fat and protein diets and lower carbohydrate, higher fat and protein diets and DBP



Systolic blood pressure (SBP)

Comparison of higher carbohydrate, lower fat diets with lower carbohydrate, higher fat diets

The pooled estimate for the 14 studies where carbohydrate was replaced with fat only, indicated that SBP was 0.71mmHg higher (95% CI, -0.71 to 2.14mmHg) in diets higher in carbohydrate and lower in fat diet compared with a low carbohydrate and high fat diet. This was not significantly different from zero (p=0.33). Heterogeneity denoted by I^2 was 59% (95% CI, 26 to 77%). A funnel plot indicated that there was some evidence of publication bias (see below). Statistically, the pooled estimate indicates that diets higher in carbohydrate and lower in fat are not associated with a difference in SBP.

Figure 2.6 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and SBP

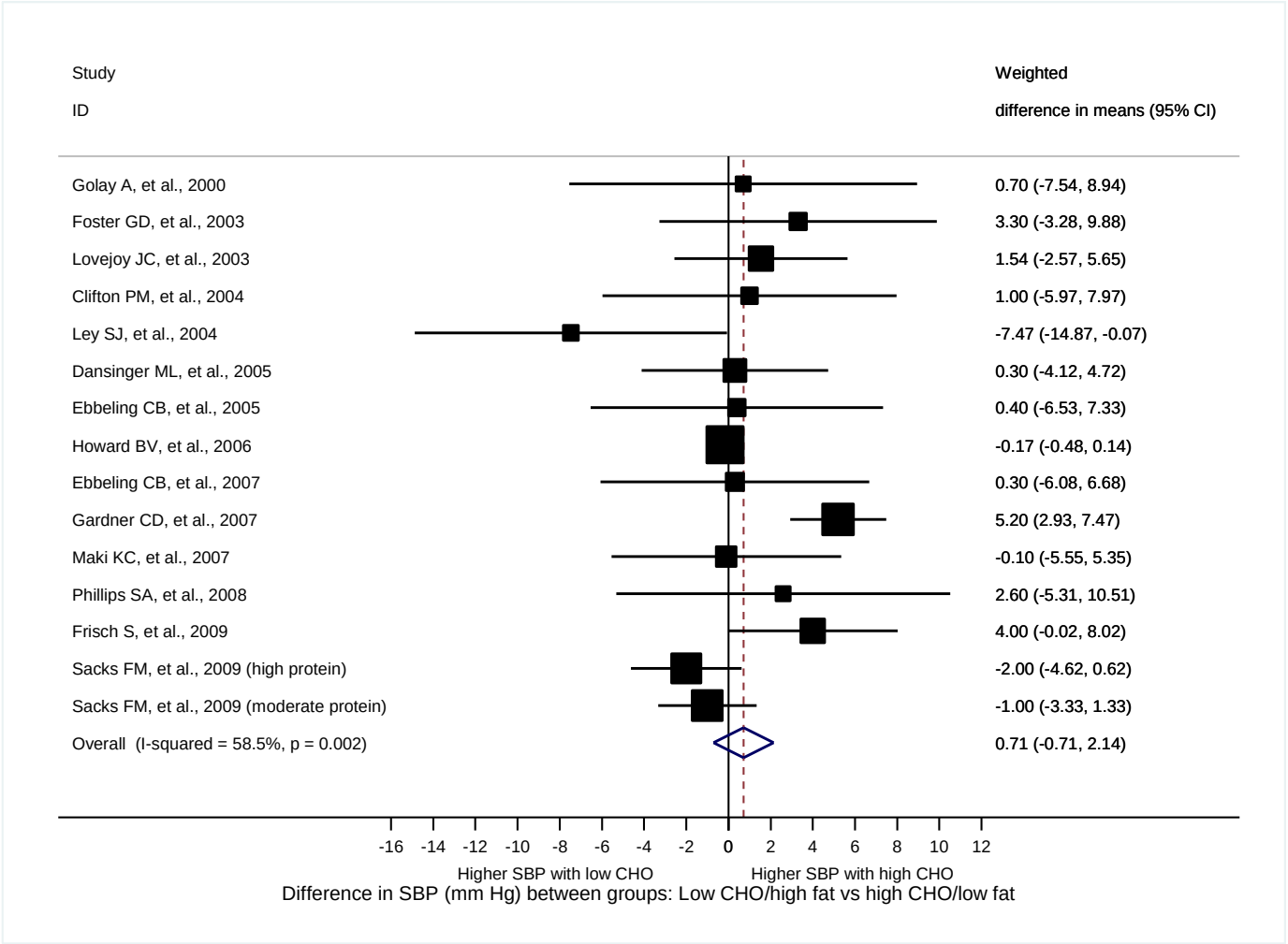
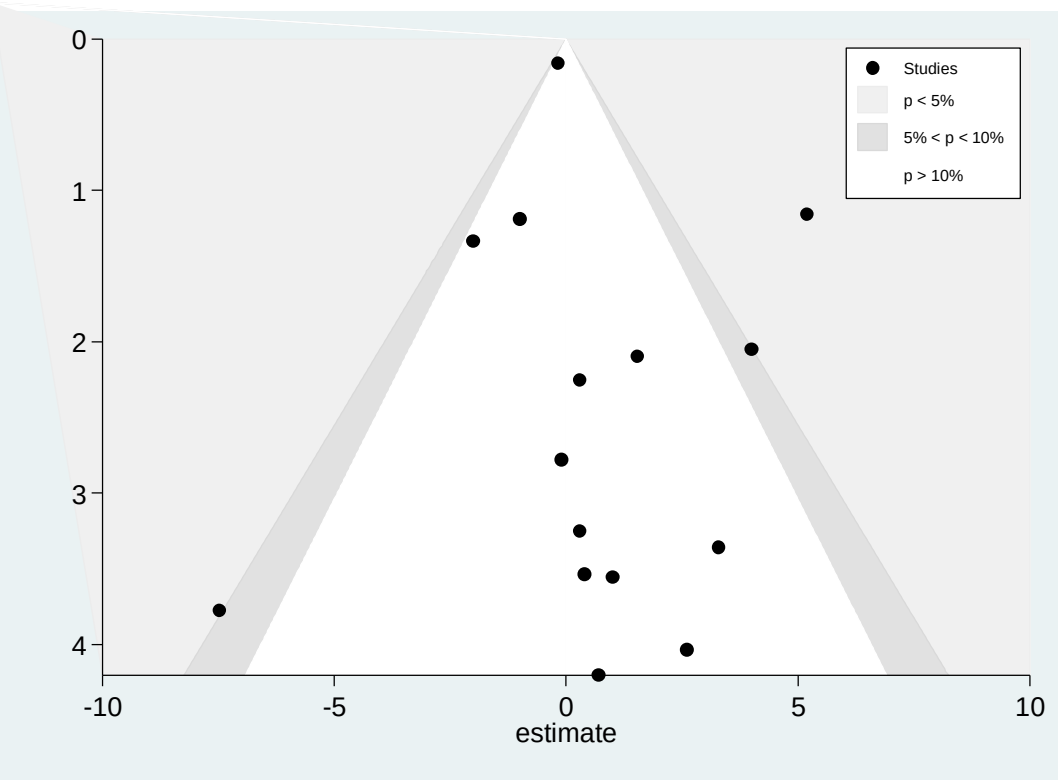


Figure 2.7 Contour-enhanced funnel plot for publications presenting SBP and higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets

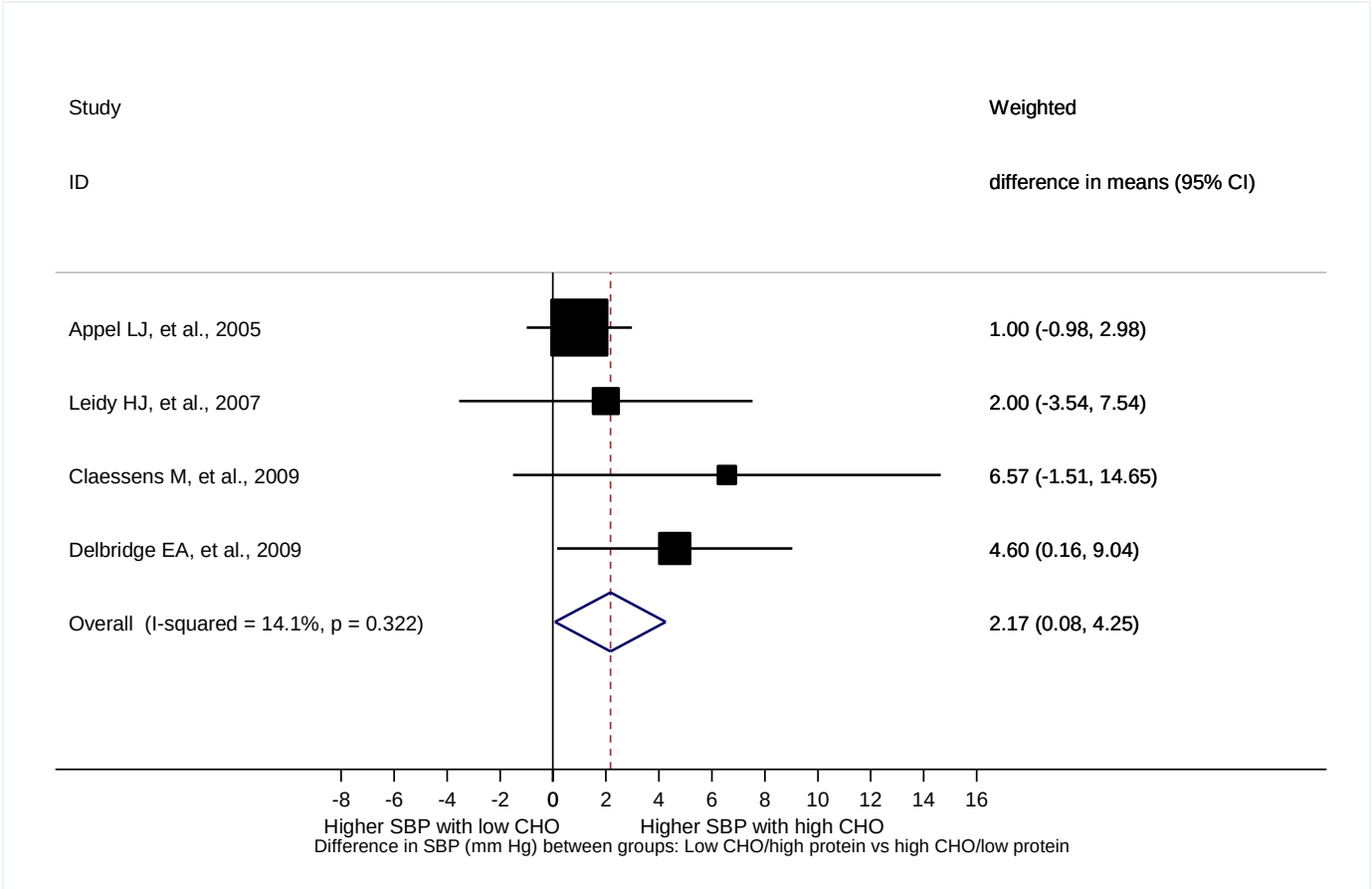


Comparison of higher carbohydrate, lower protein diets with lower carbohydrate, higher protein diets

For the four studies with a change in protein and carbohydrate, SBP was 2.17mmHg higher (95% CI, 0.08 to 4.25mmHg) with consumption of a higher carbohydrate lower protein diet compared with a lower carbohydrate, higher protein diet. This was significantly different from zero (p=0.04). Overall heterogeneity denoted by I^2 was 14% (95% CI, 0 to 87%). There were insufficient studies to present a funnel plot. Statistically, the pooled estimate indicates that diets higher in carbohydrate and lower in protein are associated with increased SBP.

The studies by Claessens *et al.* (Claessens *et al.*, 2009) and Delbridge *et al.* (Delbridge *et al.*, 2009) were weight maintenance studies, which tracked participants after completion of a more intensive phase of weight loss. In the former, the high carbohydrate group re-gained weight, but the low carbohydrate, high protein diet groups lost additional weight. In the study by Delbridge *et al.* there was no significant difference in degree of weight change between diet groups, however, in an analysis of study completers, mean weight regain was somewhat higher in the high carbohydrate group. In the energy-restricted trial reported by Leidy *et al.* (Leidy *et al.*, 2007), weight losses were similar between high carbohydrate and high protein groups, although lean body mass losses were lower in the high protein group. In the 6-week study reported by Appel *et al.* (Appel *et al.*, 2005) body weights remained unchanged in all intervention groups

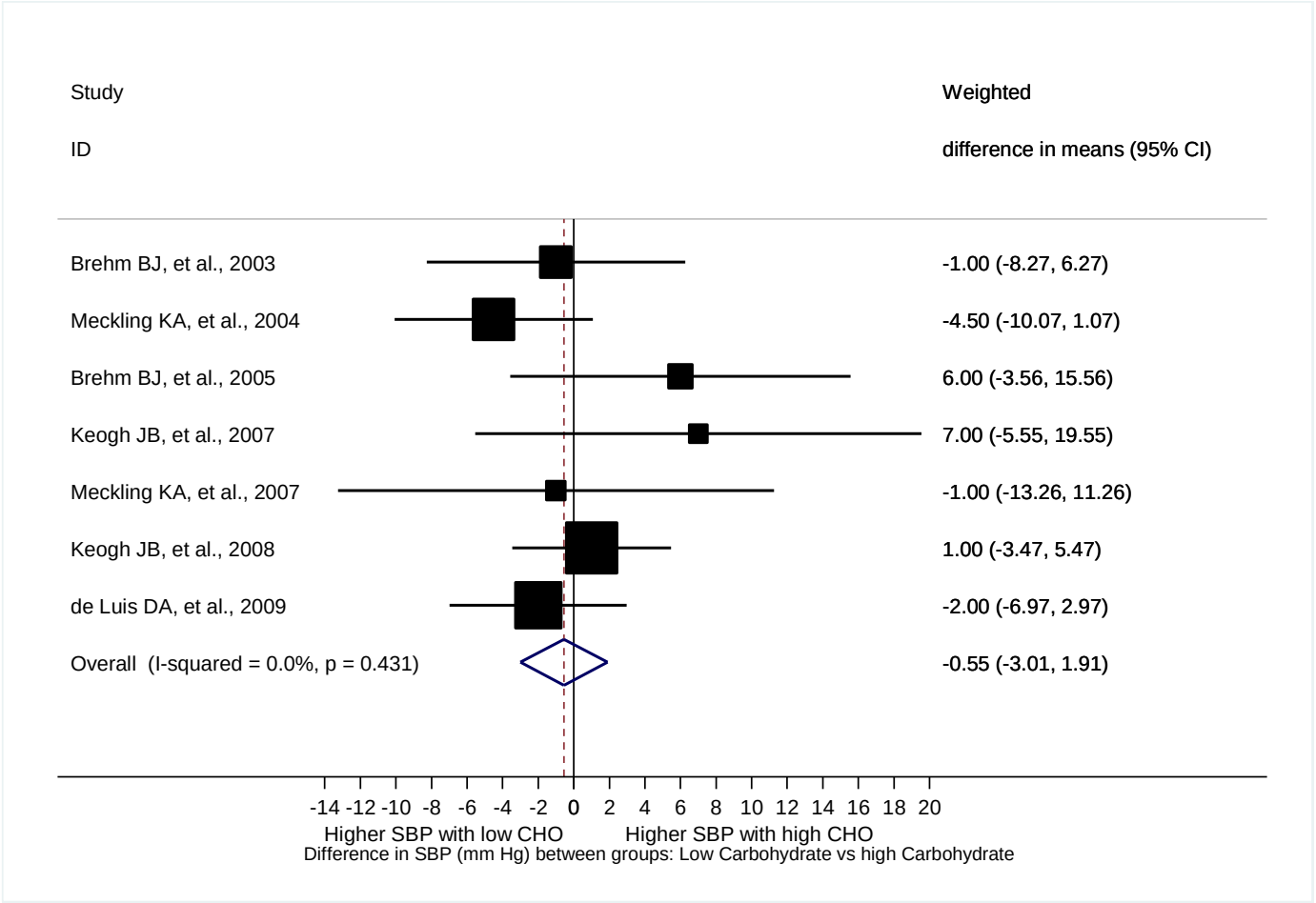
Figure 2.8 Forest plot for higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets and SBP



Comparison of higher carbohydrate, lower fat and protein diets with lower carbohydrate, higher fat and protein diets

For the 7 high versus low carbohydrate studies with a change in fat *and* protein, SBP was 0.55mmHg lower (95% CI, -1.91 to 3.01mmHg) with consumption of a higher carbohydrate lower fat and protein diet compared with a lower carbohydrate, higher fat and protein diet. This was not significantly different from zero (p=0.44). Overall heterogeneity denoted by I^2 was 0% (95% CI, 0 to 71%). There were insufficient studies to present a funnel plot. Statistically, the pooled estimate indicates that diets higher in carbohydrate and lower in fat and protein are not associated with differences in SBP.

Figure 2.9 Forest plot for higher carbohydrate, lower fat and protein diets with lower carbohydrate, higher fat and protein diets and SBP



Six studies did not contribute to any of the meta-analyses. The meta-analysis excluded (Tinker *et al.*, 2008) because it duplicated results from another study (Howard *et al.*, 2006b) whose results were closer to the end of the intervention, which is still ongoing in this trial, so the longest recorded follow-up was selected. This comparison also reported significantly lower SBP and DBP in the women following the high carbohydrate/low fat diet than the control diet, but this is likely to be attributable to the substantial decrease in weight recorded in the low fat diet group, rather than an inherent property of increasing carbohydrate intake. The three studies not presenting sufficient information (e.g. estimate and confidence interval) to be included in the meta-analysis (Wolever and Mehling, 2002;Noakes *et al.*, 2006;O'Brien *et al.*, 2005) all reported no significant difference between intervention groups, thereby introducing some potential for publication bias, where non-significant results are more likely to be excluded from meta-analyses. The study excluded because of less than 5% difference in carbohydrate between study groups did not report any significant differences in SBP or DBP between intervention groups (Dale *et al.*, 2009;Sacks *et al.*, 2009).

Table 2.13 Blood pressure and total carbohydrate: cohort studies in children and adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	Total n	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
(Boreham *et al.*, 1999) 14162 The Northern Ireland Young Hearts Project	Northern Ireland, Primarily White	12-15 %M 49.3	509	4 years (1.7)	Dietary history	Total Carbohydrate (% energy)	SBP Clinic BP	Male		1 % energy/ day		0.27 (0.11)	0.010		Social class, Sexual maturity
14163 The Northern Ireland Young Hearts Project							DBP Clinic BP	Male		1 % energy/ day		Beta coefficient not reported		NS	As above
14191 The Northern Ireland Young Hearts Project							SBP Clinic BP	Female		1 % energy/ day		Beta coefficient not reported		NS	As above
14211 The Northern Ireland Young Hearts Project							DBP Clinic BP	Female		1 % energy/ day		Beta coefficient not reported		NS	As above
(Ludwig *et al.*, 1999) 13684 The CARDIA Study	USA, Multi-ethnic, Generally healthy, No HTN, No T2DM	18-30 %M 45.9	5115	10 years	FFQ (700)	Total Carbohydrate (% energy)	SBP Clinic BP	Race - White	(51.9) vs. (33.5)	% Energy	106.5 vs. 107.8			0.13	Age, alcohol, centre, education, energy intake, physical activity, gender, smoking, baseline outcome variable, vitamin intake
13688 The CARDIA Study							DBP Clinic BP	Race - White	(51.9) vs. (33.5)	% Energy	70.3 vs. 69.7			0.79	
13685 The CARDIA Study							SBP Clinic BP	Race - Black	(51.9) vs. (33.5)	% Energy	111 vs.112.7			0.08	
13689 The CARDIA Study							DBP Clinic BP	Race - Black	(51.9) vs. (33.5)	% Energy	73.3 vs. 74.8			0.10	
(Schroeder *et al.*, 2007) 14180 Middle-aged Runners Study	USA, Active people only, No CHD, No hypertension	(51) %M 62	91	10 years	Food diary	Total Carbohydrate (grams/day)	SBP Not reported			1 g/day		No effect on regression direction			Age
14181 Middle-aged Runners Study							DBP Not reported			1 g/day		No effect on regression direction			Age

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	Total n	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
(Stamler *et al.*, 2002) 14129 Chicago Western Electric Study	USA	40-55 %M100	2107	9 years	Dietary history + FFQ (195)	Total Carbohydrate (% energy)	Annual SBP change Physician diagnosed/ medication use			1 %		-0.0189 (1.77)			Age, BP, education, height, assessment period, Weight
14131 Chicago Western Electric Study							Annual DBP change Physician diagnosed/ medication use			1 %		-0.0014 (0.23)			Age, BP, education, height, assessment period, weight

Table 2.14 Blood pressure 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
(Golay *et al.*, 2000) *14848		Higher carbohydrate, macronutrients not eaten simultaneously	26/26	130 (SE 4.1)	125 (SE 3.8)					SBP	Not reported (mm/Hg)	6 weeks	Decrease	unclear
		Lower carbohydrate, macronutrients eaten simultaneously	28/28	135 (SE 3.5)	124.3 (SE 1.8)		<0.01						Decrease	
**14849		Higher carbohydrate, macronutrients not eaten simultaneously	26/26	85.6 (SE 5)	75 (SE 3.3)					DBP		6 weeks	Decrease	unclear
		Lower carbohydrate,	28/28	85.8 (SE 2.1)	81 (SE 2.3)		<0.05						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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		macronutrients eaten simultaneously												
(Meckling *et al.*, 2004) *14870		Low carbohydrate	15/10	124.8 (SE 2.6)	114.6 (SE 1.8)		0.05	NS		SBP	Clinic BP (mm/Hg)	10 weeks	Decrease	No bias
		Low fat	16/10	121.3 (SE 1.5)	110.1 (SE 2.2)		0.05						Decrease	
**14871		Low carbohydrate	15/10	77.7 (SE 3.8)	71.6 (SE 3.2)		0.05	NS		DBP	Clinic BP (mm/Hg)	10 weeks	Decrease	No bias
		Low fat	16/10	77.9 (SE 3.1)	72.9 (SE 2.5)		0.05						Decrease	
(Lovejoy *et al.*, 2003) 14999		Control	13/15	117.24 (SE 2.33)		-1.71 (SE 1.81)				SBP	Clinic BP (mm/Hg)	3 months	Decrease	unclear
		Fat reduced	13/15	119.84 (SE 2.62)		-1.98 (SE 2.22)							Decrease	
15002		Control	13/15	76.67 (SE 2.18)		-4.21 (SE 1.51)				DBP	Clinic BP (mm/Hg)	3 months	Decrease	unclear
		Fat reduced	13/15	73.62 (SE 1.7)		-2.48 (SE 1.24)							Decrease	
15000		Control	13/15	117.24 (SE 2.33)		-1.76 (SE 1.54)				SBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
		Fat reduced	13/15	119.84 (SE 2.62)		-2.34 (SE 1.89)							Decrease	
15003		Control	13/15	76.67 (SE 2.18)		-5.26 (SE 1.93)				DBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
		Fat reduced	13/15	73.62 (SE 1.7)		-4.4 (SE 1.61)							Decrease	
*15001		Control	13/15	117.24 (SE 2.33)		-1.72 (SE 1.74)				SBP	Clinic BP (mm/Hg)	9 months	Decrease	unclear
		Fat reduced	13/15	119.84 (SE 2.62)		-0.18 (SE 1.17)							Decrease	
**15004		Control	13/15	76.67 (SE 2.18)		-0.54 (SE 1.43)				DBP	Clinic BP (mm/Hg)	9 months	Decrease	unclear
		Fat reduced	13/15	73.62 (SE 1.7)		0.08 (SE 1.47)							Decrease	
(Gardner		Atkins: low	77/77			-6.8 (SD		NS		SBP	Clinic BP	2 months	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 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
et al., 2007) *15126		carbohydrate				8)					(mm/Hg)			
		Ornish: high carbohydrate	76/76			-1.6 (SD 6.3)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			-3.2 (SD 8.2)							Decrease	
**15129		Atkins: low carbohydrate	77/77			-2.9 (SD 6.2)		NS		DBP	Clinic BP (mm/Hg)	2 months	Decrease	No bias
		Ornish: high carbohydrate	76/76			-0.4 (SD 5.5)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			-2.1 (SD 5.6)							Decrease	
15127		Atkins: low carbohydrate	77/77			-6.4 (SD 9.5)		NS		SBP	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		Ornish: high carbohydrate	76/76			-1.7 (SD 7)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			-3.6 (SD 8)							Decrease	
15130		Atkins: low carbohydrate	77/77			-3.3 (SD 6.9)		NS		DBP	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		Ornish: high carbohydrate	76/76			-1 (SD 5.6)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			-1.8 (SD 5.6)							Decrease	
15128		Atkins: low carbohydrate	77/77			-7.6 (SD 11)		0.05		SBP	Clinic BP (mm/Hg)	1 year	Decrease	No bias
		Ornish: high carbohydrate	76/76			-1.9 (SD 7.7)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			-3.3 (SD 8.1)							Decrease	
15131		Atkins: low carbohydrate	77/77			-4.4 (SD 8.4)		NS		DBP	Clinic BP (mm/Hg)	1 year	Decrease	No bias
		Ornish: high carbohydrate	76/76			-0.7 (SD 6)		NS					Decrease	
		Zone: moderate carbohydrate	79/79			2.1 (SD 5.8)							Decrease	
(Frisch *et*		High	100/100			-4 (SD 15)	0.05			SBP	Clinic BP	6 months	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
al., 2009) *15160		carbohydrate diet									(mm/Hg)			
		Moderate carbohydrate diet	100/100			-6 (SD 16)	0.05	0.102					Decrease	
15162		High carbohydrate diet	100/100			-3 (SD 9)	0.05			DBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
		Moderate carbohydrate diet	100/100			-3 (SD 8)	0.05	0.884					Decrease	
**15161		High carbohydrate diet	100/100			-1 (SD 15)	NS			SBP	Clinic BP (mm/Hg)	1 year	Decrease	unclear
		Moderate carbohydrate diet	100/100			-5 (SD 14)	0.05	0.007					Decrease	
15163		High carbohydrate diet	100/100			-2 (SD 8)	0.05			DBP	Clinic BP (mm/Hg)	1 year	Decrease	unclear
		Moderate carbohydrate diet	100/100			-3 (SD 9)	0.05	0.44					Decrease	
(Foster *et al.*, 2003) 15197		Conventional diet plan	30/30			-0.6 (SD 11.9)	NS			SBP change	Clinic BP (%)	3 months	Decrease	unclear
		Low carbohydrate diet	33/33			-2.6 (SD 11.2)	NS	0.59					Decrease	
15200		Conventional diet plan	30/30			-3.5 (SD 10.3)	<0.05			DBP change	Clinic BP (%)	3 months	Decrease	unclear
		Low carbohydrate diet	33/33			-3.0 (SD 13.4)	NS	0.84		DBP change			Decrease	
15198		Conventional diet plan	30/30			1.0 (SD 12.2)	NS			SBP change	Clinic BP (%)	6 months	Decrease	unclear
		Low	33/33			-2.3 (SD	NS	0.28					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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		carbohydrate diet				11.7)								
15201		Conventional diet plan	30/30			-2.9 (SD 14.2)	NS			DBP change	Clinic BP (%)	6 months	Decrease	unclear
		Low carbohydrate diet	33/33			-4.0 (SD 12.7)	<0.05	0.84					Decrease	
*15199		Conventional diet plan	30/30			1.7 (SD 11.8)	NS			SBP change	Clinic BP (%)	1 year	Decrease	unclear
		Low carbohydrate diet	33/33			-1.0 (SD 9.4)	NS	0.43					Decrease	
**15202		Conventional diet plan	30/30			-3.8 (SD 13.2)	NS			DBP change	Clinic BP (%)	1 year	Decrease	unclear
		Low carbohydrate diet	33/33			-3.7 (SD 12.4)	<0.05	0.84					Decrease	
(Delbridge *et al.*, 2009) *15326		Low fat, high carbohydrate weight maintenance diet	70/70			5.8 (SE 1.5)				SBP	Not reported (mm/Hg)	1 year	Increase	unclear
		Low fat, high protein weight maintenance diet	68/71			1.2 (SE 1.7)		0.042					Increase	
**15327		Low fat, high carbohydrate weight maintenance diet	70/70			3.4 (SE 1.5)				DBP	Not reported (mm/Hg)	1 year	Increase	unclear
		Low fat, high protein weight maintenance diet	68/71			2.3 (SE 1.4)		0.58					Increase	
(Tinker *et*		Control	25173/29294	127.4 (SD	125.4 (SD 16.8)					SBP	Not	1 year	No	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
al., 2008) 15368				17.1)							reported (mm/Hg)		change	
		Low fat diet	17126/19541	127.1 (SD 17.2)	124.4 (SD 17.1)			0.001					Decrease	
15370		Control	25169/29294	76 (SD 9)	74.7 (SD 9.1)					DBP	Not reported (mm/Hg)	1 year	No change	unclear
		Low fat diet	17125/19541	75.9 (SD 9.1)	73.9 (SD 9.2)			0.001					Decrease	
15369		Control	22532/29294	127.4 (SD 17.1)	124.6 (SD 16.3)					SBP	Not reported (mm/Hg)	6 years	No change	unclear
		Low fat diet	14543/19541	127.1 (SD 17.2)	124.5 (SD 16.5)								Decrease	
15371		Control	22532/29294	76 (SD 9)	71.9 (SD 9.2)					DBP	Not reported (mm/Hg)	6 years	No change	unclear
		Low fat diet	14540/19541	75.9 (SD 9.1)	71.7 (SD 9.2)								Decrease	
(Ebbeling *et al.*, 2007) *15455		Low fat diet	37/37			-4.8 (SE 2.3)				SBP change	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-5.1 (SE 2.3)		0.93					Decrease	
**15457		Low fat diet	37/37			-2.0 (SE 1.7)				DBP change	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-2.4 (SE 1.7)		0.88					Decrease	
15456		Low fat diet	37/37			1.1 (SE 2.3)				SBP change	Clinic BP (mm/Hg)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-3.2 (SE 2.3)		0.18					Decrease	
15458		Low fat diet	37/37			2.9 (SE 1.7)				DBP change	Clinic BP (mm/Hg)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			0 (SE 1.7)		0.22					Decrease	
(Ebbeling *et al.*, 2005) 15513		Low fat diet	12/17	105 (SE 4)		-0.5% (CI -5.3, 4.4)				SBP	Seated (mm/Hg)	6 months	Decrease	unclear
		Low GI diet	11/17	106 (SE 2)		-0.9% (CI -5.9, 4.2)							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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15515		Low fat diet	12/17	63 (SE 2)		0.3% (CI -4.8, 5.6)				DBP	Seated (mm/Hg)	6 months	Decrease	unclear
		Low GI diet	11/17	64 (SE 3)		-2% (CI -7.2, 3.4)							Decrease	
*15514		Low fat diet	12/17	105 (SE 4)		0.6% (CI -4.1, 5.5)				SBP	Seated (mm/Hg)	1 year	Decrease	unclear
		Low GI diet	11/17	106 (SE 2)		0.2% (CI -4.7, 5.3)							Decrease	
**15516		Low fat diet	12/17	63 (SE 2)		1.4% (CI -4.4, 7.6)				DBP	Seated (mm/Hg)	1 year	Decrease	unclear
		Low GI diet	11/17	64 (SE 3)		-0.3% (CI -6.2, 6)							Decrease	
(Sacks *et al.*, 2009) 15587		High-fat, average-protein	ITT: /204	120 (SD 13)	118 (SD 12)	-1.5%				SBP	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		High-fat, high-protein	ITT: /201	120 (SD 15)	119 (SD 12)	-1.7%							Decrease	
		Low-fat, average-protein	ITT: /204	118 (SD 13)	116 (SD 12)	-1.2%							Decrease	
		Low-fat, high-protein	ITT: /202	120 (SD 13)	117 (SD 12)	-2.6%							Decrease	
15589		High-fat, average-protein	ITT: /204	76 (SD 9)	74 (SD 9)	-2.3%				DBP	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		High-fat, high-protein	ITT: /201	76 (SD 10)	74 (SD 9)	-1.8%							Decrease	
		Low-fat, average-protein	ITT: /204	75 (SD 9)	74 (SD 9)	-1.4%							Decrease	
		Low-fat, high-protein	ITT: /202	75 (SD 9)	73 (SD 9)	-3.1%							Decrease	
15588		High-fat, average-protein	ITT: /204	120 (SD 13)	118 (SD 12)	-1.3%				SBP	Clinic BP (mm/Hg)	2 years	Decrease	No bias
		High-fat, high-protein	ITT: /201	120 (SD 15)	120 (SD 14)	-0.7%							Decrease	
		Low-fat, average-protein	ITT: /204	118 (SD 13)	117 (SD 12)	-0.8%							Decrease	
		Low-fat, high-protein	ITT: /202	120 (SD 13)	118 (SD 13)	-1.7%							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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15590		High-fat, average-protein	ITT: /204	76 (SD 9)	75 (SD 9)	-1.5%				DBP	Clinic BP (mm/Hg)	2 years	Decrease	No bias
		High-fat, high-protein	ITT: /201	76 (SD 10)	76 (SD 9)	-0.3%							Decrease	
		Low-fat, average-protein	ITT: /204	75 (SD 9)	74 (SD 9)	-0.8%							Decrease	
		Low-fat, high-protein	ITT: /202	75 (SD 9)	74 (SD 9)	-1.3%							Decrease	
(Keogh *et al.*, 2007) 15600		High carbohydrate diet	12/12	123 (SE 4)	115 (SE 4)		0.01			SBP	Clinic BP (mm/Hg)	6 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	122 (SE 4)	110 (SE 4)		0.05						Decrease	
15602		High carbohydrate diet	12/12	76 (SE 2)	72 (SE 1)		0.01			DBP	Clinic BP (mm/Hg)	6 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	74 (SE 2)	68 (SE 2)		0.05						Decrease	
*15601		High carbohydrate diet	12/12	123 (SE 4)	118 (SE 5)		0.01			SBP	(mm/Hg)	12 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	122 (SE 4)	111 (SE 4)		0.01						Decrease	
**15603		High carbohydrate diet	12/12	76 (SE 2)	70 (SE 2)		0.01			DBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	74 (SE 2)	67 (SE 2)		0.01						Decrease	
15607		High carbohydrate diet	completers not reported/12	128 (SE 3)	130 (SE 4)					SBP	Clinic BP (mm/Hg)	1 year	Decrease	unclear
		Low	completers	117 (SE 4)	115 (SE 5)								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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		carbohydrate diet	not reported/13											
15608		High carbohydrate diet	completers not reported/12	80 (SE 2)	74 (SE 3)		0.05			DBP	Clinic BP (mm/Hg)	1 year	Decrease	unclear
		Low carbohydrate diet	completers not reported/13	72 (SE 2)	68 (SE 3)		0.05						Decrease	
(Brehm *et al.*, 2003) 15719		Low carbohydrate	22/22	116 (SE 3.23)	112 (SE 2.36)			NS		SBP	Clinic BP (mm/Hg)	3 months	Decrease	unclear
		Moderate fat	20/20	115 (SE 2.47)	116 (SE 2.01)								Decrease	
15721		Low carbohydrate	22/22	79 (SE 2.69)	72 (SE 2.06)			NS		DBP	Clinic BP (mm/Hg)	3 months	Decrease	unclear
		Moderate fat	20/20	75 (SE 1.99)	75 (SE 1.79)								Decrease	
*15720		Low carbohydrate	22/22	116 (SE 3.23)	114 (SE 2.82)			NS		SBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
		Moderate fat	20/20	115 (SE 2.47)	113 (SE 2.41)								Decrease	
**15722		Low carbohydrate	22/22	79 (SE 2.69)	74 (SE 2.23)			NS		DBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
		Moderate fat	20/20	75 (SE 1.99)	74 (SE 1.62)								Decrease	
(Dansinger *et al.*, 2005) 15819		Atkins	40/40			-4.2 (SD 13)	0.05			SBP	Clinic BP (mm/Hg)	2 months	Decrease	No bias
		Ornish	40/40			-1.3 (SD 8.8)	NS						Decrease	
		Weight watchers	40/40			-4.8 (SD 13)	0.05						Decrease	
		Zone	40/40			-4.1 (SD 14)	NS						Decrease	
15822		Atkins	40/40			-4.2 (SD 8.3)	0.01			DBP	Clinic BP (mm/Hg)	2 months	Decrease	No bias
		Ornish	40/40			-2.5 (SD 7.1)	0.05						Decrease	
		Weight watchers	40/40			-3.1 (SD 7.4)	0.05						Decrease	
		Zone	40/40			-4.8 (SD 7.6)	0.01						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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15820		Atkins	40/40			-3.7 (SD 10)	0.05			SBP	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		Ornish	40/40			-0.6 (SD 8.7)	NS						Decrease	
		Weight watchers	40/40			-4.8 (SD 14)	0.05						Decrease	
		Zone	40/40			-3.9 (SD 14)	NS						Decrease	
15823		Atkins	40/40			-4 (SD 6.5)	0.01			DBP	Clinic BP (mm/Hg)	6 months	Decrease	No bias
		Ornish	40/40			-0.3 (SD 6.2)	NS						Decrease	
		Weight watchers	40/40			-1.8 (SD 6.9)	NS						Decrease	
		Zone	40/40			-4 (SD 9.1)	0.01						Decrease	
*15821		Atkins	40/40			0.2 (SD 12)	NS			SBP	Clinic BP (mm/Hg)	1 year	Decrease	No bias
		Ornish	40/40			0.5 (SD 7.7)	NS						Decrease	
		Weight watchers	40/40			-2.7 (SD 13)	NS						Decrease	
		Zone	40/40			1.4 (SD 15)	NS						Decrease	
**15824		Atkins	40/40			-1.4 (SD 7.5)	NS			DBP	Clinic BP (mm/Hg)	1 year	Decrease	No bias
		Ornish	40/40			0.2 (SD 4.6)	NS						Decrease	
		Weight watchers	40/40			-1.7 (SD 6.4)	NS						Decrease	
		Zone	40/40			-1.2 (SD 9.5)	NS						Decrease	
(Wolever and Mehling, 2002)		High carbohydrate, high GI	11/11					NS		SBP	Clinic BP	16 weeks	Decrease	unclear
		High	13/13					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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15940		carbohydrate, low GI												
		Low carbohydrate, high MUFA	11/11					NS					Increase	
15960		High carbohydrate, high GI	11/11					NS		DBP	Clinic BP (mm/Hg)	16 weeks	Decrease	unclear
		High carbohydrate, low GI	13/13					NS					Decrease	
		Low carbohydrate, high MUFA	11/11					NS					Increase	
(Ley *et al.*, 2004) 15965		Control	70/70			-1.99 (SE 2.97)				SBP	Clinic BP Seated (mm/Hg)	6 months	No change	unclear
		Low fat	66/66			-4.31 (SE 1.96)		0.05					Decrease	
15971		Control	70/70			-1.57 (SE 2.1)				DBP	Clinic BP Seated (mm/Hg)	6 months	No change	unclear
		Low fat	66/66			-1.31 (SE 1.54)		NS					Decrease	
*15967		Control	70/70			3.37 (SE 3.21)				SBP	Clinic BP Seated (mm/Hg)	1 year	No change	unclear
		Low fat	66/66			-4.1 (SE 1.99)		NS					Decrease	
**15972		Control	70/70			2.11 (SE 2.09)				DBP	Clinic BP Seated (mm/Hg)	1 year	No change	unclear
		Low fat	66/66			-1.73 (SE 1.51)		0.01					Decrease	
15968		Control	57/70			2.56 (SE 3.18)				SBP	Clinic BP Seated	2 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 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
											(mm/Hg)			
		Low fat	47/66			-2.99 (SE 2.19)		NS					Decrease	
15973		Control	57/70			-1.87 (SE 2)				DBP	Clinic BP Seated (mm/Hg)	2 years	No change	unclear
		Low fat	47/66			-6.1 (SE 1.49)		0.01					Decrease	
15969		Control	51/70			2.81 (SE 3.1)				SBP	Clinic BP Seated (mm/Hg)	3 years	No change	unclear
		Low fat	48/66			-3.63 (SE 1.6)		NS		SBP			Decrease	
15974		Control	51/70			-2.47 (SE 2.05)				DBP	Clinic BP Seated (mm/Hg)	3 years	No change	unclear
		Low fat	48/66			-5.78 (SE 1.27)		NS					Decrease	
15970		Control	52/70			1.31 (SE 3.38)				SBP	Clinic BP Seated (mm/Hg)	5 years	No change	unclear
		Low fat	51/66			-3.54 (SE 2.48)		NS					Decrease	
15975		Control	52/70			-4.2 (SE 1.92)				DBP	Clinic BP Seated (mm/Hg)	5 years	No change	unclear
		Low fat	51/66			-7.16 (SE 1.68)		0.05					Decrease	
(Dale *et al.*, 2009) 17392		High carbohydrate diet	89/100	124 (SD 15)	118 (SD 14)					SBP	(mm/Hg)	1 year	Decrease	unclear
		High MUFA diet	85/100	124 (SD 14)	119 (SD 12)								Decrease	
17396		High carbohydrate diet	89/100	79 (SD 9)	75 (SD 8)					DBP	(mm/Hg)	1 year	Decrease	unclear
		High MUFA diet	85/100	78 (SD 9)	75 (SD 7)								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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
17366		High carbohydrate diet	89/100	124 (SD 15)	120 (SD 14)					SBP	(mm/Hg)	2 years	Decrease	unclear
		High MUFA diet	85/100	124 (SD 14)	121 (SD 14)								Decrease	
15980		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.4 (CI -2.2, 3)	SBP	(mm/Hg)	2 years	Decrease in both	unclear
17367		High carbohydrate diet	89/100	79 (SD 9)	76 (SD 8)					DBP	(mm/Hg)	2 years	Decrease	unclear
		High MUFA diet	85/100	78 (SD 9)	76 (SD 8)								Decrease	
15981		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.4 (CI -1, 1.9)	DBP	(mm/Hg)	2 years	Decrease in both	unclear
(de Luis *et al.*, 2009a) 16689	Genetics - UCP3 Gene - 55CC poly-morphism	Low carbohydrate	54/67	139 (SD 16)	121 (SD 12)		<0.05			SBP	Clinic BP (mm/Hg)	2 months	Decrease	unclear
		Low fat	40/64	136 (SD 15)	118.2 (SD 41)		<0.05						Decrease	
16690	Genetics - UCP3 Gene - 55CT/TT poly-morphism	Low carbohydrate	13/67	142 (SD 12)	138 (SD 12)		NS			SBP	Clinic BP (mm/Hg)	2 months	Decrease	unclear
		Low fat	24/64	151 (SD 12)	138 (SD 12)		NS						Decrease	
16691	Genetics - UCP3 Gene - 55CC poly-morphism	Low carbohydrate	54/67	80.2 (SD 9.2)	77.4 (SD 14.8)		<0.05			DBP	Clinic BP (mm/Hg)	2 months	Decrease	unclear
		Low fat	40/64	82.1 (SD 8.9)	80.8 (SD 7.8)		NS						Decrease	
16692	Genetics - UCP3 Gene - 55CT/TT poly-morphism	Low carbohydrate	13/67	83.6 (SD 15.2)	82.9 (SD 10.4)		NS			DBP	Clinic BP (mm/Hg)	2 months	Decrease	unclear
		Low fat	24/64	87.6 (SD 17.1)	86.9 (SD 11.4)		NS						Decrease	
(de Luis *et al.*, 2009b) *16078		Low carbohydrate	66/52	142 (SD 16)	126 (SD 16)					SBP	Clinic BP Seated (mm/Hg)	3 months	Decrease	unclear
		Low fat	52/66	139 (SD 18)	124 (SD 13)								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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
**16079		Low carbohydrate	52/52	85.8 (SD 7)	81.1 (SD 7.4)					DBP	Clinic BP Seated (mm/Hg)	3 months	Decrease	unclear
		Low fat	66/66	80.7 (SD 6.8)	79.2 (SD 12.8)								Decrease	
(de Luis *et al.*, 2008) 16136	Genetics - wild-type Ala54/Ala54	Low carbohydrate	55/105	138.5 (SD 12.1)	122.5 (SD 12.7)					SBP	Not reported (mm/Hg)	2 months	Decrease	unclear
		Low fat	55/99	133.5 (SD 15)	110.8 (SD 14.5)								Decrease	
16141	Genetics - wild-type Ala54/Ala54	Low carbohydrate	55/105	87.1 (SD 9.2)	77.5 (SD 14.8)					DBP	Not reported (mm/Hg)	2 months	Decrease	unclear
		Low fat	55/99	81.3 (SD 8.9)	71.1 (SD 7.8)								Decrease	
16158	Genetics - mutant-type Ala54/Thr54 or Thr54/Thr54	Low carbohydrate	50/105	143.9 (SD 12)	137.8 (SD 12.5)					SBP	Not reported (mm/Hg)	2 months	Decrease	unclear
		Low fat	44/99	149 (SD 12)	135 (SD 12.5)								Decrease	
16159	Genetics - mutant-type Ala54/Thr54 or Thr54/Thr54	Low carbohydrate	50/105	83.6 (SD 17)	82.9 (SD 11.4)					DBP	Not reported (mm/Hg)	2 months	Decrease	unclear
		Low fat	44/99	77.6 (SD 17)	81.9 (SD 11.4)								Decrease	
(Howard *et al.*, 2006b) *16244		Control	approx 1699 participants included as a 5.8% sub-sample of 29294 in group	127.9 (SD 17.2)	125.7 (SD 16.8)	-2.1 (SD 16.4)				SBP	Clinic BP (mm/Hg)	3 years	No change	No bias
		Low fat	approx 1132 participants included as a 5.8% sub-sample of 19541 in group	127.5 (SD 17.2)	125.1 (SD 16.9)	-2.2 (SD 16.3)		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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
17611		Low fat minus control	Low fat: approx 1132 participants included as a 5.8% sub-sample of 19541 in group Control: approx 1699 participants included as a 5.8% sub-sample of 29294 in group						-0.17 (CI -0.49,0.15)	SBP	Clinic BP (mm/Hg)	3 years	No change in control group, decrease in low fat group	No bias
**16245		Control	approx 1699 participants included as a 5.8% sub-sample of 29294 in group	76.0 (SD 9.1)	73.6 (SD 9.3)	-2.3 (SD 9.4)				DBP	Clinic BP (mm/Hg)	3 years	No change	No bias
		Low fat	approx 1132 participants included as a 5.8% sub-sample of 19541 in group	75.9 (SD 9.1)	73.1 (SD 9.4)	-2.6 (SD 9.4)		<0.001					Decrease	
17610		Low fat minus control	Low fat: approx 1132 participants included as a 5.8% sub-sample of 19541 in group Control: approx 1699 participants included as a 5.8% sub-sample of 29294 in group						-0.31 (CI -0.5, -0.13)	DBP	Clinic BP (mm/Hg)	3 years	No change in control group, decrease in low fat group	No bias
(Brehm *et al.*, 2005) 16359		Low carbohydrate	20/25	119 (SE 3.5)	114 (SE 3.8)			NS		SBP	Clinic BP (mm/Hg)	2 months	Decrease	unclear
		Moderate fat	20/25	119 (SE 2.9)	116 (SE 2.8)								Decrease	
16361		Low carbohydrate	20/25	76 (SE 1.7)	73 (SE 2.4)			NS		DBP	Clinic BP (mm/Hg)	2 months	Decrease	unclear
		Moderate fat	20/25	77 (SE 1.7)	74 (SE 2)								Decrease	
*16360		Low carbohydrate	20/25	119 (SE 3.5)	110 (SE 3.4)			NS		SBP	Clinic BP (mm/Hg)	4 months	Decrease	unclear
		Moderate fat	20/25	119 (SE 2.9)	116 (SE 3.5)								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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
**16363		Low carbohydrate	20/25	76 (SE 1.7)	71 (SE 2.1)			NS		DBP	Clinic BP (mm/Hg)	4 months	Decrease	unclear
		Moderate fat	20/25	77 (SE 1.7)	75 (SE 2.8)								Decrease	
(Meckling and Sherfey, 2007) *16366		Hypocaloric control diet	8/15	127 (SD 14)	118 (SD 12)		<0.05			SBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet	10/15	128 (SD 19)	119 (SD 13)		<0.05						Decrease	
16367		Hypocaloric control diet + exercise	11/15	129 (SD 7)	122 (SD 10)		<0.05			SBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet + exercise	14/15	134 (SD 12)	127 (SD 17)		<0.05						Decrease	
**16369		Hypocaloric control diet	8/15	81 (SD 11)	75 (SD 14)		<0.05			DBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet	10/15	79 (SD 13)	72 (SD 7)		<0.05						Decrease	
16370		Hypocaloric control diet + exercise	11/15	82 (SD 8)	77 (SD 9)		NS			DBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet + exercise	14/15	82 (SD 7)	78 (SD 10)		<0.05						Decrease	
(Noakes *et al.*, 2006) 16598		High unsaturated fat	21/27					NS		SBP	Clinic BP	12 weeks	Decrease	unclear
		Very low carbohydrate	24/28					NS					Decrease	
		Very low fat	22/28					NS					Decrease	
16599		High unsaturated fat	21/27					NS		DBP	Clinic BP	12 weeks	Decrease	unclear
		Very low carbohydrate	24/28					NS					Decrease	
		Very low fat	22/28					NS					Decrease	
(Keogh *et al.*, 2008)		High carbohydrate,	47/50	136 (SD 12)	123 (SD 10)					SBP	Clinic BP (mm/Hg)	8 weeks	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
*16730		low SFA												
		Low carbohydrate, high SFA	51/57	133 (SD 14)	122 (SD 12)								Decrease	
**16731		High carbohydrate, low SFA	47/50	77 (SD 11)	70 (SD 9)		<0.05			DBP	Clinic BP (mm/Hg)	8 weeks	Decrease	unclear
		Low carbohydrate, high SFA	51/57	74 (SD 12)	67 (SD 13)								Decrease	
(Clifton *et al.*, 2004) 16757		High MUFA	31/35	132 (SD 17)	122 (SD 14)					SBP	Clinic BP (mm/Hg)	8 weeks	Decrease	unclear
		Very low fat	31/35	131 (SD 17)	122 (SD 13)								Decrease	
16760		High MUFA	31/35	76 (SD 10)	72 (SD 8)					DBP	Clinic BP (mm/Hg)	8 weeks	Decrease	unclear
		Very low fat	31/35	76 (SD 10)	71 (SD 7)								Decrease	
*16758		High MUFA	31/35	132 (SD 17)	121 (SD 14)		<0.01			SBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Very low fat	31/35	131 (SD 17)	122 (SD 14)		<0.01						Decrease	
**16761		High MUFA	31/35	76 (SD 10)	72 (SD 8)		<0.01			DBP	Clinic BP (mm/Hg)	12 weeks	Decrease	unclear
		Very low fat	31/35	76 (SD 10)	72 (SD 10)		<0.01						Decrease	
(Claessens *et al.*, 2009) *16814		High carbohydrate supplement	16/allocated not reported	125.88 (SE 3.78)	127.5 (SE 5.6)	1.63 (SE 4.67)	NS			SBP	Clinic BP (mm/Hg)	12 weeks	increase	unclear
		High protein supplement - casein	14/allocated not reported	122.57 (SE 5.02)	119.64 (SE 4.96)	-2.93 (SE 3.27)	NS						decrease	
		High protein supplement - whey	18/allocated not reported	124.33 (SE 1.77)	117.83 (SE 2.34)	-6.5 (SE 1.87)	<0.05						decrease	
**16815		High carbohydrate supplement	16/allocated not reported	81.56 (SE 1.9)	84.19 (SE 4.38)	2.63 (SE 3.52)	NS			DBP	Clinic BP (mm/Hg)	12 weeks	increase	unclear
		High protein supplement -	14/allocated not reported	78.43 (SE 2.53)	76.64 (SE 2.13)	-1.79 (SE 2.03)	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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		casein												
		High protein supplement - whey	18/allocated not reported	79.0 (SE 1.32)	76.06 (SE 1.72)	-2.94 (SE 1.37)	<0.05						decrease	
(Leidy *et al.*, 2007) 16843		High protein, energy restricted	21/27	109 (SE 3)	104 (SE 2)	-6 (SE 3)		NS		SBP	Supine (mm/Hg)	12 weeks	Decrease	unclear
		Moderate protein, energy restricted	25/27	114 (SE 2)	110 (SE 2)	-4 (SE 2)							Decrease	
16844		High protein, energy restricted	21/27	68 (SE 1)	65 (SE 1)	-3 (SE 1)		NS		DBP	Supine (mm/Hg)	12 weeks	Decrease	unclear
		Moderate protein, energy restricted	25/27	72 (SE 2)	64 (SE 1)	-6 (SE 1)							Decrease	
*16845		High protein, energy restricted	21/27	109 (SE 3)	104 (SE 2)	-5 (SE 2)		NS		SBP	Supine (mm/Hg)	12 weeks	Decrease	unclear
		Moderate protein, energy restricted	25/27	113 (SE 2)	110 (SE 2)	-3 (SE 2)							Decrease	
**16846		High protein, energy restricted	21/27	69 (SE 1)	66 (SE 1)	-4 (SE 1)		NS		DBP	Supine (mm/Hg)	12 weeks	Decrease	unclear
		Moderate protein, energy restricted	25/27	73 (SE 2)	65 (SE 2)	-6 (SE 2)							Decrease	
(O'Brien *et al.*, 2005) 16950		Low carbohydrate	22/22					0.22		SBP change	Not reported (mm/Hg)	3 months	Decrease	unclear
		Moderate fat	19/19										Decrease	
16951		Low carbohydrate	22/22					0.06		DBP change	Not reported (mm/Hg)	3 months	Decrease	unclear
		Moderate fat	19/19										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	Difference between groups in Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Maki *et al.*, 2007b) 17294		Ad libitum low GL diet	42/43	112.7 (SE 1.6)		-0.6 (SE 2)				SBP	Seated (mm/Hg)	12 weeks	Decrease	unclear
		Low fat, energy restricted	42/43	114.7 (SE 1.6)		-1.2 (SE 2.3)				SBP			Decrease	
17296		Ad libitum low GL diet	42/43	74.4 (SE 1.5)		-3.3 (SE 1.8)				DBP	Seated (mm/Hg)	12 weeks	Decrease	unclear
		Low fat, energy restricted	42/43	73.4 (SE 1.1)		-0.6 (SE 1.4)							Decrease	
*17295		Ad libitum low GL diet	42/43	112.7 (SE 1.6)		0.2 (SE 1.7)				SBP	Seated (mm/Hg)	36 weeks	Decrease	unclear
		Low fat, energy restricted	42/43	114.7 (SE 1.6)		0.1 (SE 2.2)							Decrease	
**17297		Ad libitum low GL diet	42/43	74.4 (SE 1.5)		-4.1 (SE 1.8)				DBP	Seated (mm/Hg)	36 weeks	Decrease	unclear
		Low fat, energy restricted	42/43	73.4 (SE 1.1)		-1.6 (SE 1.3)							Decrease	
(Phillips *et al.*, 2008) *17424		Low carbohydrate diet	10/~14	123.3 (SE 3.1)	112.6 (SE 2.7)		0.05	NS		SBP	Supine (mm/Hg)	6 weeks	Decrease	unclear
		Low fat diet	10/~14	124.1 (SE 4)	115.2 (SE 3)		0.05						Decrease	
**17425		Low carbohydrate diet	10/~14	70 (SE 3.5)	65.8 (SE 2.6)		0.05	NS		DBP	Supine (mm/Hg)	6 weeks	Decrease	unclear
		Low fat diet	10/~14	73.2 (SE 3.6)	66.8 (SE 3.3)		0.05						Decrease	
(Appel *et al.*, 2005) OMNI-Heart Study *16309	All	High carbohydrate	164	131.2 (SD 9.4)		-8.2 (-9.6 to -6.8)				SBP	Seated (mm/Hg)	6 weeks	No change	No bias
		High protein	164	131.2 (SD 9.4)		-9.5 (-10.9 to -8.2)							No change	
		Unsaturated fat	164	131.2 (SD 9.4)		-9.3 (-10.6 to -8.0)							No change	
16311	Pre-hypertensives	High carbohydrate	132	127.5 (SD 5.5)		-7.0 (-8.5 to -5.6)				SBP	Seated (mm/Hg)	6 weeks	No change	No bias
		High protein	132	127.5 (SD 5.5)		-8.0 (-9.3							No change	

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
						to -6.6)								
		Unsaturated fat	132	127.5 (SD 5.5)		-7.7 (-8.9 to -6.4)							No change	
**16312	All	High carbohydrate	164	77.0 (SD 8.2)		-4.1 (-5.0 to 3.3)				DBP	Seated (mm/Hg)	6 weeks	No change	No bias
		High protein	164	77.0 (SD 8.2)		-5.2 (-6.1 to -4.4)							No change	
		Unsaturated fat	164	77.0 (SD 8.2)		-4.8 (-5.6 to 4.0)							No change	
16314	Pre-hypertensives	High carbohydrate	132	75.3 (SD 7.4)		-3.6 (-4.5 to -2.7)				DBP	Seated (mm/Hg)	6 weeks	No change	No bias
		High protein	132	75.3 (SD 7.4)		-4.4 (-5.3 to -3.6)							No change	
		Unsaturated fat	132	75.3 (SD 7.4)		-3.9 (-4.7 to -3.2)							No change	

*This result was used in the meta-analysis for high carbohydrate diets and SBP

**This result was used in the meta-analysis for high carbohydrate diets and DBP

Blood pressure, dietary fibre and high fibre diets

Summary of cohort results

Data were extracted from three publications, recording results from three cohort studies (Ascherio *et al.*, 1992;Ascherio *et al.*, 1996;Ludwig *et al.*, 1999). One of these reported the effect of fibre density in grams per unit energy on SBP and DBP. Findings from this study (the CARDIA study) (Ludwig *et al.*, 1999) indicate that SBP and DBP were lower by 2-4% among white high-fibre density consumers than low-fibre density consumers. This association was not apparent in the black participants of the study, however. The Health Professionals Follow-up Study and the Nurse's Health Study (Ascherio *et al.*, 1992;Ascherio *et al.*, 1996) which both reported dietary fibre as grams per day consistently reported statistically significant results indicative of a decrease in SBP and DBP with each additional 1g per day of dietary fibre.

The Health Professionals Follow-up Study and the Nurse's Health Study (Ascherio *et al.*, 1992;Ascherio *et al.*, 1996) relied upon self-reports of blood pressure. Participants were asked to report their usual SBP and DBP (presumably measured by their physician) within seven categories. The CARDIA study (Ludwig *et al.*, 1999), however, measured blood pressure at clinic.

As the units of expression for dietary fibre intake differed between studies, a meta-analysis with 3 or more cohort studies was not possible. However, these three US studies provide evidence of consistent negative association between dietary fibre intake and blood pressure.

Exposure definition and assessment

All three studies measured dietary intake using FFQs, and the number of food items varied from 126 to 700.

Adjustment for appropriate confounders

The HPFS and NHS were adjusted for important covariates including age, alcohol and BMI. The CARDIA study adjusted for additional appropriate confounders such as education, gender and smoking.

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

Summary of RCT data

Data were extracted from three RCTs reporting information on blood pressure in relation to diets higher in dietary fibre from food sources, rather than from fibre isolates. Insufficient information was presented in one study for it to be included in meta-analysis (Sciarrone *et al.*, 1993) and so there were too few studies remaining for any meta-analysis to be conducted. The study by Sciarrone compared lacto-ovo-vegetarian and omnivorous diets with markedly different fibre contents (20g/1000 kcal vs. <8g/1000kcal) in overweight males. The authors reported that blood pressure was 'lower' with the vegetarian diet. Of the two remaining studies (Olendzki *et al.*, 2009;Andersson *et al.*, 2007), one presented results at 3 and 6 months (Olendzki *et al.*, 2009) and the other at 6 weeks (Andersson *et al.*, 2007). Andersson *et al.* (Andersson *et al.*, 2007) explored blood pressure differences in men and women consuming their usual diet with whole grain foods (bread, crisp bread, muesli & pasta - minimum 50% wholegrain in provided foods = 112g wholegrain/day) or with refined grain foods (bread, crisp bread, muesli & pasta). There was a marked difference in fibre content between the diets, and body weight increased in both groups possibly due to the test foods supplementing rather than substituting for usual foods. Olenzki *et al.* (Olendzki *et al.*, 2009) compared 3 hypoenergetic diets (high fibre, high fibre/low saturated fat and low fat). Body weight decreased in all 3 diet groups. Neither study reported evidence of an effect of high fibre diets on either SBP or DBP with a higher fibre diet.

Table 2.15 Blood pressure 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 Assessment	Exposure	Outcome/ Assessment Details	Sub-group Detail	Contrast (mean)	Exposure Units	Mean Outcome (SD)	Beta coefficient (SE)/(CI)	p	p trend	Adjustments
(Ascherio *et al.*, 1992) 13893 HPFS	USA, Primarily White, Cancer free, No CHD, Normal BP only, No T2DM	40-75 %M 100	51529	4 years (9.7)	FFQ (131)	Dietary Fibre, g/d (AOAC method)	SBP change (4 years) Self-reported			1 g/day		-0.027 (0.009)	<0.01		Age, alcohol, BMI
13910 HPFS							DBP change (4 years) Self-reported			1 g/day		-0.015 (0.006)	<0.05		Age, alcohol, BMI
(Ascherio *et al.*, 1996) 13923 NHS	USA, Primarily White, Cancer free, No CHD, No T2DM	30-55 %M 0	121700	4 years (0.9)	FFQ (126)	Dietary Fibre, g/d (AOAC method)	SBP Self-reported			1 g/day		-0.069 (0.009)	<0.0001		Age, alcohol, BMI
14039 NHS							DBP Self-reported			1 g/day		-0.056 (0.006)	<0.001		Age, alcohol, BMI
(Ludwig *et al.*, 1999) 13683 The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, No T2DM	18-30 %M 45.9	5115	10 years	FFQ (700)	Fibre density (g/unit energy. AOAC method)	SBP Clinic BP	Race - Black	(12.3) vs. (5.2)	g/4184kJ / day	111.5 vs.111.6			0.77	Age, alcohol, centre, education, energy intake, physical activity, gender, smoking, SBP, vitamin intake
13687 The CARDIA Study							DBP Clinic BP	Race - Black	(12.3) vs. (5.2)	g/4184kJ / day	73.3 vs.74			0.7	Age, alcohol, centre, DBP, education, energy intake, physical activity, gender, smoking, vitamin intake
13682 The CARDIA Study							SBP Clinic BP	Race - White	(12.3) vs. (5.2)	g/4184kJ / day	106.9 vs. 109.1			0.01	Age, alcohol, centre, education, energy intake, physical activity, gender, smoking, SBP, vitamin intake
13687 The CARDIA Study							DBP Clinic BP	Race - White	(12.3) vs. (5.2)	g/4184kJ / day	69.7 vs.72.4			<0.001	Age, alcohol, centre, DBP, education, energy intake, physical activity, gender, smoking, vitamin intake

Table 2.16 Blood pressure and high fibre diets: RCT data

Author/ Result ID	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
(Olendzki *et al.*, 2009) 14586	Hypoenergetic high fibre	12/12	74 (SE 2.3)		-4.17 (SE 2.3)			SBP	Not reported (mm/Hg)	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	70 (SE 2.6)		-1.67 (SE 2.7)						Decrease	
	Hypoenergetic low saturated fat	10/10	72 (SE 2.5)		-1.9 (SE 2.5)						Decrease	
14587	Hypoenergetic high fibre	12/12	74 (SE 2.3)		-2.08 (SE 2.3)			SBP	Not reported (mm/Hg)	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	70 (SE 2.6)		1.67 (SE 2.6)						Decrease	
	Hypoenergetic low saturated fat	10/10	72 (SE 2.5)		-3.5 (SE 2.5)						Decrease	
14588	Hypoenergetic high fibre	12/12	122 (SE 4.4)		-5.8 (SE 3.8)			DBP	Not reported (mm/Hg)	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	119 (SE 5.1)		-6.9 (SE 4.6)						Decrease	
	Hypoenergetic low saturated fat	10/10	125 (SE 4.8)		-0.9 (SE 4.2)						Decrease	
14589	Hypoenergetic high fibre	12/12	122 (SE 4.4)		-4.4 (SE 3.8)			DBP	Not reported (mm/Hg)	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	119 (SE 5.1)		1.4 (SE 4.2)						Decrease	
	Hypoenergetic low saturated fat	10/10	125 (SE 4.8)		0.8 (SE 4.4)						Decrease	
(Andersson *et al.*, 2007) 16305	Refined grain products	15/30	130 (SD 16)	130 (SD 15)		NS		SBP	Clinic BP Supine (mm/Hg)	6 weeks	Increase	unclear
	Wholegrain products	15/30	130 (SD 17)	129 (SD 15)		NS	0.35				Increase	
16306	Refined grain products	30/30	80 (SD 10)	81 (SD 9)		NS		DBP	Clinic BP Supine (mm/Hg)	6 weeks	Increase	unclear
	Wholegrain products	30/30	81 (SD 9)	81 (SD 8)		NS					Increase	
(Sciarrone *et al.*, 1993) 17463	Lacto-ovovegetarian diet	10/~10		lower				SBP	Daytime	6 weeks	Decrease	No bias
	Omnivorous diet	10/~10		higher							Decrease	

Blood pressure and food sources of dietary fibre

Summary of cohort results

One publication from one cohort study reported the association between food sources of fibre and blood pressure (Ascherio *et al.*, 1996). Findings from the Nurses' Health Study (Ascherio *et al.*, 1996) provide evidence of an inverse association between intake of fibre from cereals, fruit and vegetables and DBP, as well as an inverse association between intake of fibre from fruit and vegetables and SBP. That is, blood pressure tended to be lower with increasing intakes of the sources of fibre described. All achieved statistical significance. Results were not statistically significant, however, when investigating the relationship between intake of fibre from cereals and SBP (Ascherio *et al.*, 1996). The NHS (Ascherio *et al.*, 1996) adjusted for several suitable covariates including age, alcohol and BMI.

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

No RCTs reported outcomes concerning food sources of fibre and blood pressure as a continuous outcome.

Table 2.17 Blood pressure and food sources of fibre: 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
(Ascherio *et al.*, 1996) 13928 NHS	USA, Primarily White, Cancer free, No CHD, No T2DM	30-55 %M 0	121700	4 years (0.9)	FFQ (126)	Fibre within cereals g/d (AOAC method)	SBP Self-reported	1 g/day	-0.017 (0.034)	0.6	Age, alcohol, BMI, fibre from other sources, EI
13931 NHS							DBP Self-reported	1 g/day	-0.082 (0.023)	0.003	Age, alcohol, BMI, fibre from other sources, EI
13926 NHS						Fibre within fruit g/d (AOAC method)	SBP Self-reported	1 g/day	-0.133 (0.019)	<0.0001	Age, alcohol, BMI, fibre from other sources, EI
13929 NHS							DBP Self-reported	1 g/day	-0.079 (0.013)	<0.001	Age, alcohol, BMI, fibre from other sources, EI
13927 NHS						Fibre within vegetables g/d (AOAC method)	SBP Self-reported	1 g/day	-0.036 (0.017)	0.034	Age, alcohol, BMI, fibre from other sources, EI
13930 NHS							DBP Self-reported	1 g/day	-0.037 (0.012)	0.0013	Age, alcohol, BMI, fibre from other sources, EI

Blood pressure and fibre isolates, insoluble-type fibre

No cohort studies reported outcomes concerning insoluble fibre and continuous blood pressure.

Summary of RCT data

Data were extracted from 3 RCTs reporting results of interventions involving insoluble-type fibre which was administered in the form of fibre 'tablets' containing variable proportions of vegetable, citrus and cereal-derived fibre as outlined in the trial characteristics tables. Two studies reported results for SBP and DBP separately (Rigaud *et al.*, 1990;Birketvedt *et al.*, 2000) whilst one reported results for the two combined as mean arterial pressure (Cairella *et al.*, 1995). Trial duration ranged from 2 months (Cairella *et al.*, 1995) to 6 months (Rigaud *et al.*, 1990). The trial comparing mean arterial pressure (Cairella *et al.*, 1995) did not present sufficient information on the results to include in a meta-analysis, just stating that there was no significant effect. This left too few studies to be included in a meta-analysis. These remaining studies were both small, with one suggesting a significant reduction in both DBP and SBP in the fibre intervention after 24 weeks (Birketvedt *et al.*, 2000) and the other finding no evidence of any benefit after 6 months (Rigaud *et al.*, 1990).

These studies provide no consistent evidence of an effect of insoluble fibre isolates on blood pressure. It should be noted however, that all three studies were designed to assess the impact of dietary fibre tablets (using small doses of fibre) compared to placebo tablets within the context of an energy restriction weight loss regimen. Participants lost weight in all studies, and against a background of decreasing body weight, the impact of the small doses of fibre administered may have been difficult to detect.

Table 2.18 Blood pressure and fibre isolates, insoluble-type fibre: RCT data

Author/ Result ID	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
(Rigaud *et al.*, 1990) 16873	Fibre tablets	14/26		74.4 (SE 1.4)		NS	NS	DBP	Seated (mm/Hg)	6 months	Decrease	No bias
	Placebo tablets	9/26		75 (SE 1.8)		0.05					Decrease	
16872	Fibre tablets	14/26	126.5 (SE 2)		4.4	NS		SBP	Seated (mm/Hg)	6 months	Decrease	No bias
	Placebo tablets	9/26	126.7 (SE 2.5)		3.1	NS					Decrease	
(Cairella *et al.*, 1995) 15690	Balanced diet and fibre tablets, following weight loss with VLCD	completers not reported/15						Mean arterial pressure	Method not reported	60 days	Decrease	No bias
	Balanced diet and placebo tablets, following weight loss with VLCD	completers not reported/15					NS				Decrease	
(Birketvedt *et al.*, 2000) 14921	Energy restricted diet and mixed fibre tablets	28/28	81.1 (SE 2.3)	70.5 (SE 1.6)		<0.01		DBP	Not reported (mm/Hg)	24 weeks	Decrease	No bias
	Energy restricted diet and placebo tablets	25/25	82.9 (SE 2.2)	74.6 (SE 1.5)		<0.01					Decrease	
14916	Energy restricted diet and mixed fibre tablets	28/28	127.1 (SE 2.7)	120.5 (SE 2.6)		<0.01		SBP	Not reported (mm/Hg)	24 weeks	Decrease	No bias
	Energy restricted diet and placebo tablets	25/25	134 (SE 3.1)	126.8 (SE 2.1)		<0.01					Decrease	

Blood pressure and fibre isolates, psyllium

No cohort studies reported outcomes concerning psyllium and continuous blood pressure.

Summary of RCT data

In the study conducted by Bell *et al.* (Bell *et al.*, 1990), a Step 1 diet was employed during the first 6 weeks of the trial, after which participants were randomised to receive pectin-enriched cereal (10.76% soluble fibre), psyllium-enriched cereal (10.2% soluble fibre) or a placebo (cornflakes) whilst continuing with the Step 1 diet over a second 6-week period. Cereals were administered as 57g portions and were consumed as part of breakfast. Body weights remained unchanged in all diet groups, and there were decreases in blood pressure in all diet groups. The statistical significance of the differences between diet groups was not reported.

Table 2.19 Blood pressure and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Bell *et al.*, 1990) 17161	Pectin enriched cereal	20/20	122.1 (SE 2.5)	118.6 (SE 3.6)	-2.90%	SBP	Assessment details not reported (mm/Hg)	6 weeks	No change	No bias
	Placebo (cornflakes)	19/20	123.9 (SE 2.6)	118 (SE 2.7)	-4.80%				No change	
	Psyllium enriched cereal	19/20	123.5 (SE 2.9)	120.2 (SE 2.7)	-2.70%				No change	
17162	Pectin enriched cereal	20/20	80.5 (SE 1.6)	75.4 (SE 1.6)	-6.30%	DBP	Assessment details not reported (mm/Hg)	6 weeks	No change	No bias
	Placebo (cornflakes)	19/20	80.6 (SE 2.2)	77.1 (SE 2.2)	-4.30%				No change	
	Psyllium enriched cereal	19/20	81.9 (SE 1.9)	79.3 (SE 2.2)	-3.20%				No change	

Blood pressure and fibre isolates, gums and extracts

No cohort studies reported outcomes concerning gums and extracts and continuous blood pressure.

Summary of RCT data

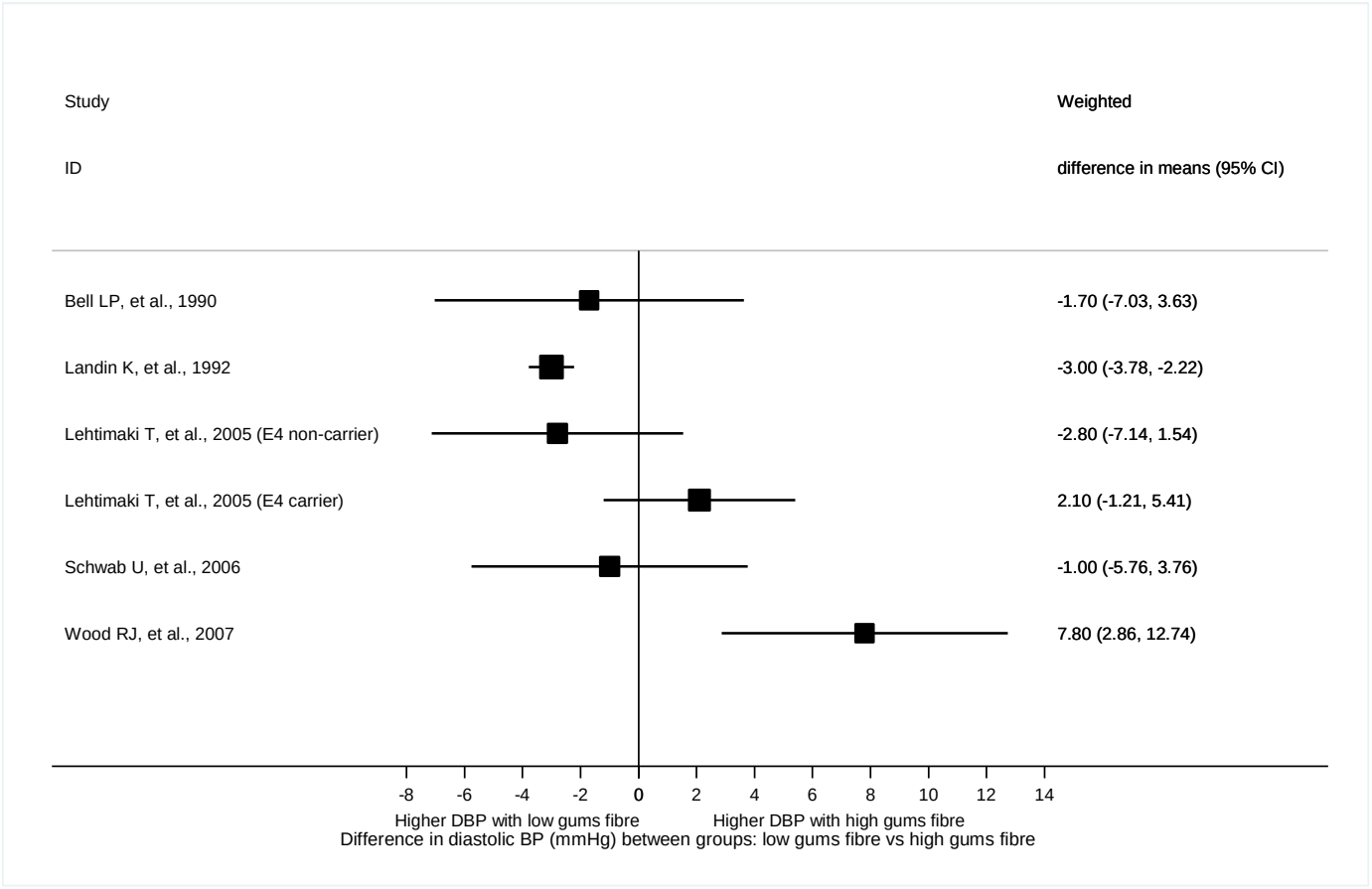
Seven RCTs exploring the effects of fibre isolates, gums and extracts on blood pressure, including pectin and guar gums, were identified. Two small studies reporting no evidence of any effect could not be included in a meta-analysis because they did not provide any estimates or confidence intervals (Pasman *et al.*, 1997;Marett and Slavin, 2004). These studies found no evidence of any effect on either SBP or DBP after 6 months (Marett and Slavin, 2004) and 14 months (Pasman *et al.*, 1997).

The remaining five studies providing dietary differences in gums and extracts including pectin (Bell *et al.*, 1990;Schwab *et al.*, 2006), guar gum (Landin *et al.*, 1992), chitosan (Lehtimaki *et al.*, 2005), and konjac-mannan (Wood *et al.*, 2007) were included in the meta-analysis. The control groups were largely starch-based. All studies included adults as participants and measured DBP and SBP in mmHg. Results for each are reported separately. The first follow up reported at the end of the intervention was used. This varied from 6 to 12 weeks.

Diastolic blood pressure (DBP)

The overall pooled estimate was not reported as heterogeneity denoted by I^2, quoted at 81% (95% CI, 59 to 91%), was more than the 75% threshold stipulated in our protocol. However, statistically there was no evidence of a difference in DBP with differences in dietary gums or extracts.

Figure 2.10 Forest plot for fibre isolates, gums and extracts and DBP



Systolic blood pressure (SBP)

The overall pooled estimate indicated that SBP was 0.82mmHg (95% CI, -3.8 to 5.2) higher with consumption of a diet higher in gums or extracts. This was not significantly different from zero (p=0.72). Heterogeneity denoted by I^2 was high at 66% (95% CI, 19 to 86%). Statistically, there was no evidence of a change in SBP with changes in gums or extracts. There were too few studies to present a funnel plot.

Figure 2.11 Forest plot for fibre isolates, gums and extracts and SBP

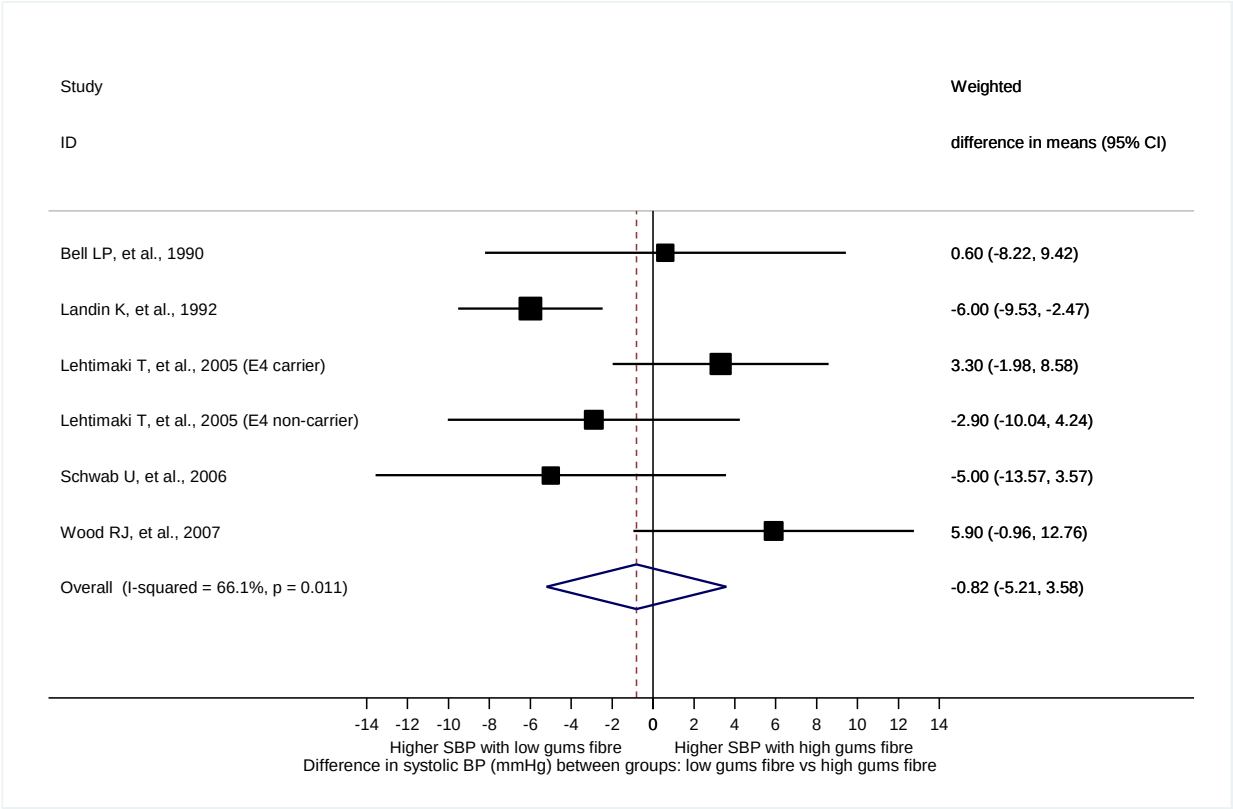


Table 2.20 Blood pressure and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Subgroup detail	Intervention group	Completers / Allocated	Base-line	Follow -up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value diff' between groups	Diff' between groups at follow-up	Diff' between groups in Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
(Pasman *et al.*, 1997) 15519		Control	11/14				NS					SBP	Clinic BP	14 months	Increase	unclear
		Guar gum - High compliance	10/10				NS	NS							Increase	
		Guar Gum - Low compliance	10/10				NS	NS							Increase	
15528		Control	11/14				NS					DBP	Clinic BP	14 months	Increase	unclear
		Guar gum - High compliance	10/10				NS	NS							Increase	
		Guar Gum - Low compliance	10/10				NS	NS							Increase	
(Schwab *et al.*, 2006) 16469		Sugar Beet Pectin	22/22	134 (SD 13)	131 (SD 13)			NS				SBP	Clinic BP (mm/Hg)	8 weeks	No change	No bias
		Placebo	22/22	135 (SD 14)	131 (SD 15)										Small decrease	
		Polydextrose	22/22	135 (SD 17)	140 (SD 16)			NS							Small decrease	
*16470		Sugar Beet Pectin	22/22	134 (SD 13)	129 (SD 15)		<0.05	NS				SBP	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Placebo	22/22	135 (SD 14)	134 (SD 14)		NS								Small decrease	
		Polydextrose	22/22	135 (SD 17)	135 (SD 18)		NS	NS							Small decrease	
16472		Placebo	22/22	86 (SD 9)	80 (SD 9)							DBP	Clinic BP (mm/Hg)	8 weeks	Small decrease	No bias
		Polydextrose	22/22	85 (SD 8)	84 (SD 8)			NS							Small decrease	
		Sugar Beet Pectin	22/22	86 (SD 9)	82 (SD 8)			NS							No change	

Author/ Result ID	Subgroup detail	Intervention group	Completers / Allocated	Base-line	Follow -up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value diff' between groups	Diff' between groups at follow-up	Diff' between groups in Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
**16473		Polydextrose	22/22	85 (SD 8)	84 (SD 9)		NS	NS				DBP	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Sugar Beet Pectin	22/22	86 (SD 9)	80 (SD 7)		<0.05	NS							No change	
		Placebo	22/22	86 (SD 9)	81 (SD 9)		<0.05								No change	
(Marett and Slavin, 2004) 16664		Larch arabino-galactan	18/18				NS					DBP	Clinic BP	6 months	No change	No bias
		Placebo	17/17				NS								No change	
		Tamarack arabino-galactan	19/19				NS								No change	
16665		Larch arabino-galactan	18/18				NS					SBP	Clinic BP	6 months	No change	No bias
		Placebo	17/17				NS								No change	
		Tamarack arabino-galactan	19/19				NS								No change	
(Landin *et al.*, 1992) *17121		Guar gum minus placebo	Crossover: 25/25						-6 (SD 9)		<0.01	SBP	Clinic BP (mm/Hg)	6 weeks	No change in both	No bias
**17122		Guar gum minus placebo	Crossover: 25/25						-3 (SD 2)		<0.001	DBP	Clinic BP (mm/Hg)	6 weeks	No change in both	
(Bell *et al.*, 1990) *17161		Pectin enriched cereal	20/20	122.1 (SE 2.5)	118.6 (SE 3.6)	-2.90%						SBP	Assess-ment details not reported (mm/Hg)	6 weeks	No change	No bias
		Placebo	19/20	123.9 (SE 2.6)	118 (SE 2.7)	-4.80%									No change	
		Psyllium enriched cereal	19/20	123.5 (SE 2.9)	120.2 (SE 2.7)	-2.70%									No change	
**17162		Pectin enriched	20/20	80.5	75.4	-6.30%						DBP	Assess-	6	No	No bias

Author/ Result ID	Subgroup detail	Intervention group	Completers / Allocated	Base-line	Follow -up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value diff' between groups	Diff' between groups at follow-up	Diff' between groups in Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
		cereal		(SE 1.6)	(SE 1.6)								ment	weeks	change	
		Placebo	19/20	80.6 (SE 2.2)	77.1 (SE 2.2)	-4.30%							details not reported (mm/Hg)		No change	
		Psyllium enriched cereal	19/20	81.9 (SE 1.9)	79.3 (SE 2.2)	-3.20%									No change	
(Wood *et al.*, 2007) 17242		Low carbohydrate diet + placebo	15/15	124.3 (SD 10)	117.7 (SD 8.3)		NS					SBP	Seated (mm/Hg)	6 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	124.4 (SD 10.6)	123.3 (SD 8.5)		NS	NS							Decrease	
*17243		Low carbohydrate diet + placebo	15/15	124.3 (SD 10)	113.7 (SD 9.4)	-10.5 (SD 8.7)	NS					SBP	Seated (mm/Hg)	12 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	124.4 (SD 10.6)	119.9 (SD 7.7)	-4.6 (SD 10.4)	NS	NS							Decrease	
17244		Low carbohydrate diet + placebo	15/15	85.2 (SD 9)	79.3 (SD 5.7)		NS					DBP	Seated (mm/Hg)	6 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	84 (SD 7.7)	84.9 (SD 7.3)		NS	0.05							Decrease	
**17245		Low carbohydrate diet + placebo	15/15	85.2 (SD 9)	77.7 (SD 4.6)	-7.5 (SD 6.9)	NS					DBP	Seated (mm/Hg)	12 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	84 (SD 7.7)	84.3 (SD 7.4)	0.3 (SD 6.9)	NS	0.05							Decrease	
(Lehtimaki *et al.*, 2005)	Apo E genotype E4 carrier	Encapsulated microcrystalline chitosan	86/96	134.4 (SD 16.2)	132.3 (SD 16.6)							SBP	Clinic BP (mm/Hg)	3 months	Not reported	No bias

Author/ Result ID	Subgroup detail	Intervention group	Completers / Allocated	Base-line	Follow -up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value diff' between groups	Diff' between groups at follow-up	Diff' between groups in Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assess-ment Bias
*17494		Starch capsules	85/96	134.4 (SD 16.2)	129 (SD 18.7)										Not reported	
*17495	Apo E genotype E4 non-carrier	Encapsulated microcrystalline chitosan	86/96	132.6 (SD 17.6)	122.2 (SD 29.6)							SBP	Clinic BP (mm/Hg)	3 months	Not reported	No bias
		Starch capsules	85/96	132.6 (SD 17.6)	125.1 (SD 16.3)										Not reported	
**17496	Apo E genotype E4 carrier	Encapsulated microcrystalline chitosan	86/96	87.8 (SD 11.5)	86.1 (SD 9.8)							DBP	Clinic BP (mm/Hg)	3 months	Not reported	No bias
		Starch capsules	85/96	87.8 (SD 11.5)	84 (SD 12.2)										Not reported	
**17497	Apo E genotype E4 non-carrier	Encapsulated microcrystalline chitosan	86/96	84.6 (SD 11.4)	77.7 (SD 18.5)							DBP	Clinic BP (mm/Hg)	3 months	Not reported	No bias
		Starch capsules	85/96	84.6 (SD 11.4)	80.5 (SD 8.9)										Not reported	

*This result was used in the meta-analysis for gums and extracts and SBP

**This result was used in the meta-analysis for gums and extracts and DBP

Blood pressure and fibre isolates, beta-glucan (oat and barley)

No cohort studies reported outcomes concerning beta-glucan (oat and barley) and continuous blood pressure.

Summary of RCT data

Data were extracted from six RCTs reporting on trials of beta-glucans (oat and barley) and blood pressure. Follow-up was between 6 and 12 weeks across all the studies. Most trials used standard clinic measures of blood pressure, but one study used a variety of positions, e.g. seated, supine and 24-hour ambulatory measures (Davy *et al.*, 2002). For comparability with the other studies, we used the seated measure from this study in the meta-analysis. Another study compared clinic, self-reported and seated measures (Swain *et al.*, 1990). All studies reported SBP and DBP, but one also reported mean arterial pressure (Davy *et al.*, 2002).

Beta-glucan is a viscous soluble polysaccharide that occurs in the endosperm cell walls of grains. It is composed of glucose molecules with mixed β-(1→4) and β-(1→3) bonds. Oats and barley are recognised as particularly rich sources. Considerable variation in the amount of beta-glucans in oats and oat products exists which is due to varietal and processing influences. Commercial rolled oats may contain in the region of 3-5% beta-glucan and oat bran between 6-10% (Wursch and Pi-Sunyer, 1997). The majority of the trials studied the effects of whole oats, oat bran-supplemented foods or oat-based breakfast cereals compared with similar wheat-based test foods. However, (Smith *et al.*, 2008) compared high and low molecular weight barley-derived beta-glucans. One small study compared wheat-based breakfast cereal with oat-based cereal (Saltzman *et al.*, 2001), two studies compared wheat with oats and/oat bran (He *et al.*, 2004;Davy *et al.*, 2002) and Swain *et al*. compared foods with either low fibre wheat or an oat bran supplement (100g/d) (Swain *et al.*, 1990). Maki *et al.* compared a high oat beta-glucan diet (from oatmeal, ready-to-eat cereal with oat bran and a powdered form of oat beta-glucan, which provided 7.7.g beta-glucan per day) with a control diet (wheat-based cereal, maltodextrin powder and a low fibre hot cereal, providing 0g beta-glucan per day).

Two studies could not be included in a meta-analysis. One study did not report sufficient information to be included in meta-analysis; Swain *et al.* (Swain *et al.*, 1990) did not provide a measure of variation such as standard errors or confidence intervals around the mean change in blood pressure. In contrast to the other studies which explored the effects of oat-derived beta-glucans, Smith *et al.* (Smith *et al.*, 2008) compared the effects of high and low molecular weight barley-derived beta glucans in 90 mildly hypercholesterolaemic non-obese individuals over 6 weeks. These beta glucan supplements (6g/day) were consumed stirred into beverages twice daily. The change in blood pressure from baseline to follow-up was not different between the two intervention groups.

In the study reported by Salzman *et al*. the oat diet resulted in greater decrease in mean SBP (oats -6mmHg, control -1mmHg, p=0.026), whereas DBP change did not differ between the two groups. An analysis of covariance of these data which explored the effects of the oat diet whilst holding constant the variation in initial blood pressure, extent of weight loss and initial BMI indicated that the oat diet reduced SBP, but not DBP independent of changes in these covariates (p<0.026).

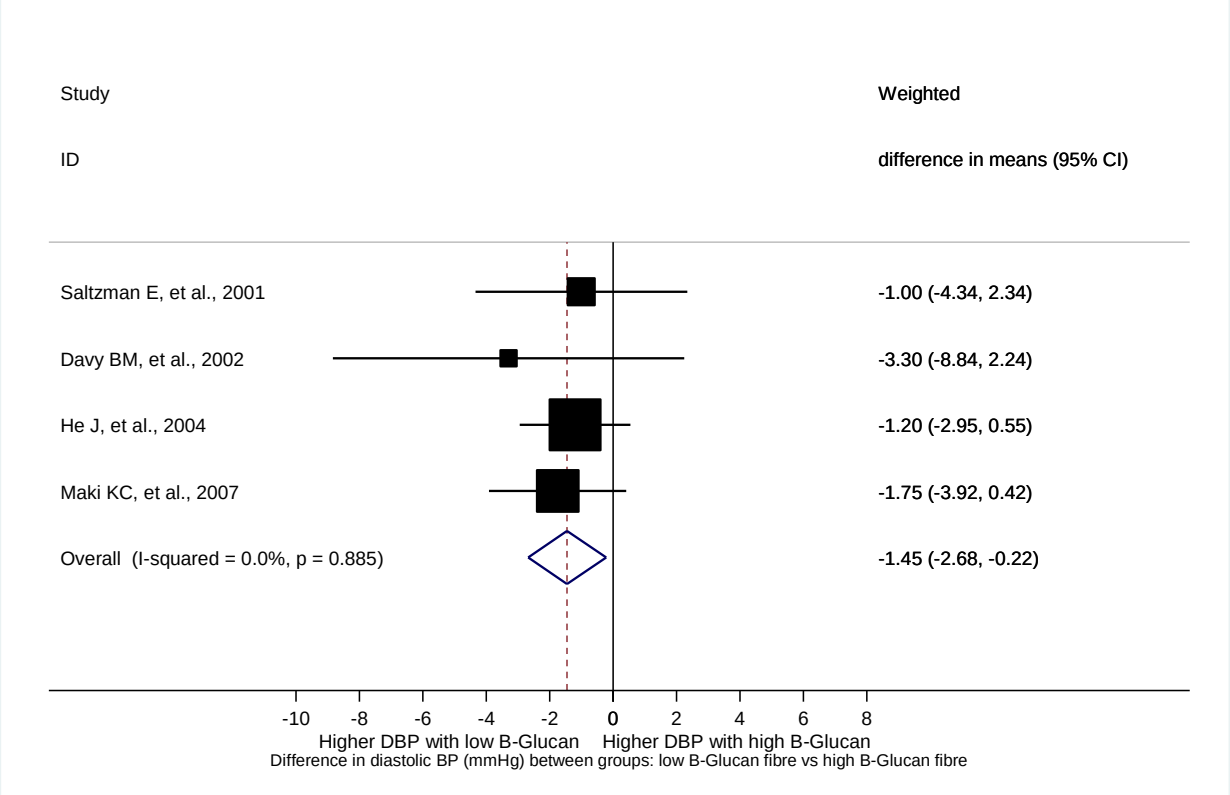
Four studies tested the effects of high and low oat beta-glucan diets on blood pressure and were similar enough to pool in a meta-analysis (Davy *et al.*, 2002;He *et al.*, 2004;Saltzman *et al.*, 2001;Maki *et al.*, 2007a). Studies varied in duration from 6 to 12 weeks and were all conducted on adults. Mean baseline participant BMI in each trial ranged from 26 to 32kg/m^2.

Diastolic blood pressure (DBP)

Comparison of diets high or low in beta-glucans from oats

The overall pooled estimate indicated that DBP was 1.45mmHg (95% CI, 0.22 to 2.68mmHg) lower with consumption of a high oat beta-glucan diet. This was significantly different from zero (p=0.02). Heterogeneity denoted by I^2 was 0% (95% CI, 0 to 29%). Statistically, diets high in oat beta-glucan diet were associated with lower DBP compared to low beta-glucan diets.

Figure 2.12 Forest plot for high versus low oat beta-glucan diets and DBP



Systolic blood pressure (SBP)

Comparison of diets high or low in beta-glucans from oats

The overall pooled estimate indicated that SBP was 2.86mmHg (95% CI, 0.85 to 4.87mmHg) lower with consumption of a diet high in oat beta-glucans. This was significantly different from zero ($p<0.01$). Heterogeneity denoted by I^2 was 0% (95% CI, 0 to 77%). High oat beta-glucan diets were associated with lower SBP compared with diets low in oat beta-glucans.

Figure 2.13 Forest plot for high versus low oat beta-glucan diets and SBP

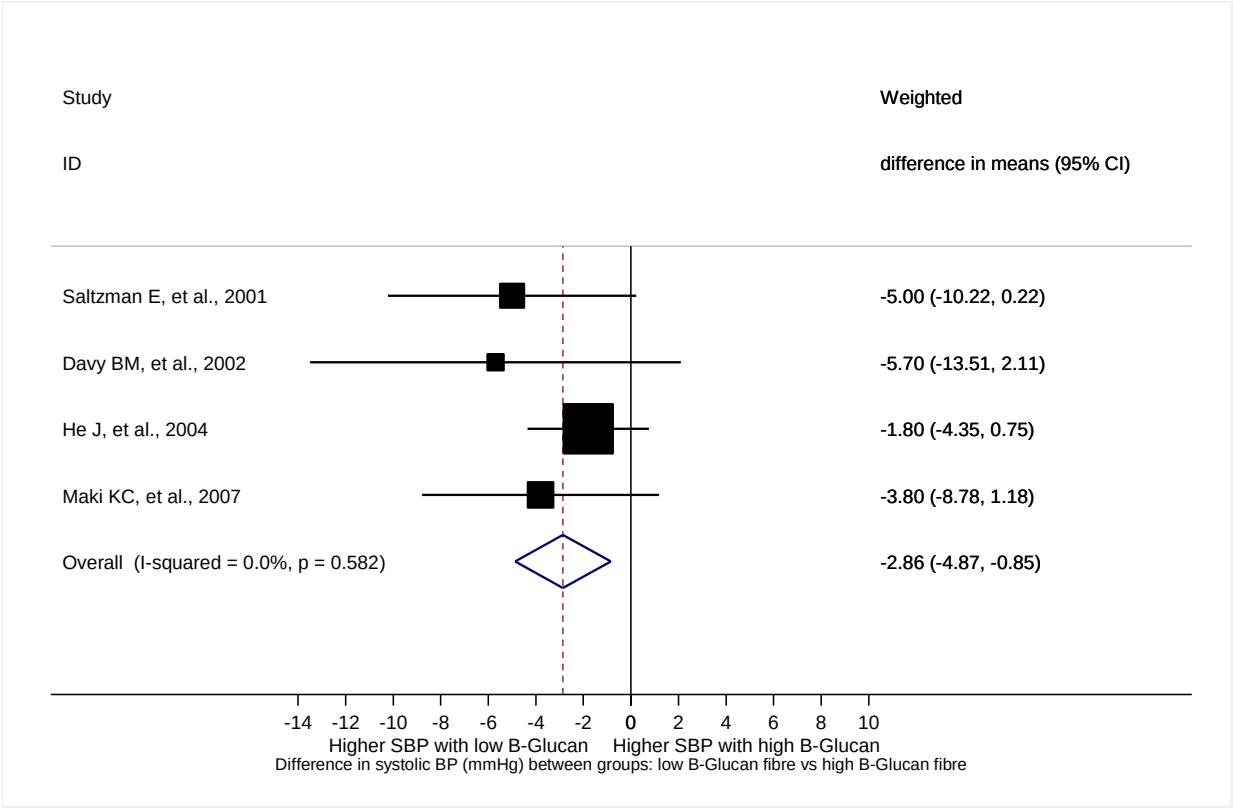


Table 2.21 Blood pressure and fibre isolates, beta-glucans (oat and barley): RCT data

Author/ Result ID	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	p-value difference between groups	Outcome/ Assess-ment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(He *et al.*, 2004) 14728		Oat bran and oatmeal minus refined wheat and corn	Oat: 50/54 Wheat and corn: 52/56						-2.2 (CI -5.3, 1)	0.18	SBP change	Clinic BP (mm/Hg)	6 weeks	No change in oat group, increase in wheat and corn group	No bias
14734		Oat bran and oatmeal	50/54			-3.5 (CI -5.8, -1.1)	0.005				SBP change	Clinic BP (mm/Hg)	6 weeks	No change	No bias
		Refined wheat and corn	52/56			-1.3 (CI -3.5, 0.9)	0.2							Increase	
14735		Oat bran and oatmeal minus refined wheat and corn	Oat: 50/54 Wheat and corn: 52/56						-1.8 (CI -4.3, 0.8)	0.17	SBP change	Clinic BP (mm/Hg)	12 weeks	No change in oat group, increase in wheat and corn group	No bias
14736		Oat bran and oatmeal	50/54			-3.4 (CI -5.4, -1.4)	0.001				SBP change	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Refined wheat and corn	52/56			-1.6 (CI -3.3, 0)	0.06							Increase	
14737		Oat bran and oatmeal minus refined wheat and corn	Oat: 50/54 Wheat and corn: 52/56						-0.8 (CI -3.1, 1.4)	0.47	DBP change	Clinic BP (mm/Hg)	6 weeks	No change in oat group, increase in wheat and corn group	No bias
14741		Oat bran and oatmeal	50/54			-2 (CI -3.5, -0.4)	0.02				DBP change	Clinic BP (mm/Hg)	6 weeks	No change	No bias
		Refined wheat and corn	52/56			-1.1 (CI -2.8, 0.6)	0.2							Increase	
14742		Oat bran and oatmeal	Oat: 50/54 Wheat and						-1.2 (CI -3, 0.5)	0.17	DBP change	Clinic BP (mm/Hg)	12 weeks	No change in oat	No bias

Author/ Result ID	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	p-value difference between groups	Outcome/ Assess-ment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		minus refined wheat and corn	corn: 52/56											group, increase in wheat and corn group	
14743		Oat bran and oatmeal	50/54			-2.2 (CI -3.3, -1)	0.001				DBP change	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Refined wheat and corn	52/56			-1.0 (CI -2.3, 0.4)	0.2							Increase	
(Maki *et al.*, 2007a) 15061		Oat beta-glucan cereal	26/26					NS			DBP	Clinic BP (mm/Hg)	8 weeks	No change	No bias
		Wheat cereal	34/34											No change	
15062		Oat beta-glucan cereal	26/26					NS			SBP	Clinic BP (mm/Hg)	8 weeks	No change	No bias
		Wheat cereal	34/34											No change	
15063		Oat beta-glucan cereal	26/26					NS			DBP	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Wheat cereal	34/34											No change	
15064		Oat beta-glucan cereal	26/26					NS			SBP	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Wheat cereal	34/34											No change	
15066	BMI > 31.5	Oat beta-glucan cereal	26/26			-2.1		0.018			DBP change	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Wheat cereal	34/34			1.9								No change	
15068	BMI > 31.5	Oat beta-glucan cereal	26/26			-5.6					SBP change	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Wheat cereal	34/34			2.7		0.008						No change	
15069	BMI < 31.5	Oat beta-glucan cereal	26/26					NS			DBP change	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Wheat cereal	34/34					NS						No change	
15070	BMI < 31.5	Oat beta-glucan cereal	26/26					NS			SBP change	Clinic BP (mm/Hg)	12 weeks	No change	No bias
		Wheat cereal	34/34					NS						No change	
(Davy *et al.*,		Oat group	18/18	138.2 (SE 2.4)	137 (SE 2)						SBP	Clinic BP Seated	8 weeks	Small increase	No bias

Author/ Result ID	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	p-value difference between groups	Outcome/ Assess-ment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
2002) 15337		Wheat group	18/18	142.3 (SE 2.4)	141.1 (SE 2.7)			NS				(mm/Hg)		Small increase	
15338		Oat group	18/18	138.2 (SE 2.4)	134.6 (SE 3.1)						SBP	Clinic BP Seated	12 weeks	Small increase	No bias
		Wheat group	18/18	142.3 (SE 2.4)	140.3 (SE 2.5)			NS				(mm/Hg)		Small increase	
15340		Oat group	18/18	88.5 (SE 1.6)	88.1 (SE 2.1)						DBP	Clinic BP Seated	8 weeks	Small increase	No bias
		Wheat group	18/18	90.4 (SE 1.5)	89.7 (SE 1.6)			NS				(mm/Hg)		Small increase	
15341		Oat group	18/18	88.5 (SE 1.6)	87.6 (SE 2)						DBP	Clinic BP Seated	12 weeks	Small increase	No bias
		Wheat group	18/18	90.4 (SE 1.5)	90.9 (SE 2)			NS				(mm/Hg)		Small increase	
15342		Oat group	18/18	132.6 (SE 2.3)	131.6 (SE 3.1)						SBP	Clinic BP Supine	12 weeks	Small increase	No bias
		Wheat group	18/18	139.2 (SE 2.4)	135.3 (SE 2.8)			NS				(mm/Hg)		Small increase	
15344		Oat group	18/18	83.6 (SE 1.4)	83.6 (SE 1.6)						DBP	Clinic BP Supine	12 weeks	Small increase	No bias
		Wheat group	18/18	85.1 (SE 1.8)	88.3 (SE 4)			NS				(mm/Hg)		Small increase	
15348		Oat group	18/18	133.3 (SE 1.5)	132.8 (SE 2)						DBP	24-hour Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	141.7 (SE 2.2)	143 (SE 2.4)			NS						Small increase	
15349		Oat group	18/18	84.1 (SE 1.6)	84.1 (SE 1.3)						DBP	24-hour Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	87.9 (SE 1.5)	88.3 (SE 1.7)			NS						Small increase	
15350		Oat group	18/18	137.5 (SE 1.6)	136.3 (SE 2)						SBP	Ambulatory BP Daytime (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	146.4 (SE 2.2)	147.4 (SE 2.7)			NS						Small	

Author/ Result ID	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	p-value difference between groups	Outcome/ Assess-ment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
														increase	
15351		Oat group	18/18	117.4 (SE 2.1)	116.6 (SE 4.7)						SBP	Ambulatory BP Night-time (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	126.6 (SE 2.5)	129.8 (SE 2.6)			NS						Small increase	
15352		Oat group	18/18	86.1 (SE 1.9)	87 (SE 1.4)						DBP	Ambulatory BP Daytime (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	91 (SE 1.9)	91.6 (SE 1.9)			NS						Small increase	
15353		Oat group	18/18	73 (SE 1.9)	75 (SE 2)		0.01				DBP	Ambulatory BP Night-time (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	74.4 (SE 1.7)	78.5 (SE 2.2)			NS						Small increase	
15355		Oat group	18/18	104.1 (SE 1.5)	103.2 (SE 1.6)						Mean arterial pressure	Ambulatory BP Daytime (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	110.1 (SE 1.6)	110.3 (SE 2.3)			NS						Small increase	
15356		Oat group	18/18	88.1 (SE 1.8)	90.2 (SE 2)		0.02				Mean arterial pressure	Ambulatory BP Night-time (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	90.3 (SE 2.9)	96.2 (SE 2.2)			NS						Small increase	
15361		Oat group	18/18	100.7 (SE 1.5)	100 (SE 1.4)						Mean arterial pressure	24-hour Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	106.1 (SE 1.6)	106.7 (SE 2) (SD			NS						Small increase	
15357		Oat group	18/18	13.6 (SE 0.6)	13.6 (SE 1)						SBP variability	Ambulatory BP	12 weeks	Small increase	No bias

Author/ Result ID	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	p-value difference between groups	Outcome/ Assess-ment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		Wheat group	18/18	12.9 (SE 0.9)	13.5 (SE 0.5)			NS				(mm/Hg)		Small increase	
15358		Oat group	18/18	10.2 (SE 0.4)	11.1 (SE 0.7)						DBP variability	Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
		Wheat group	18/18	10.9 (SE 0.5)	10.8 (SE 0.6)			NS						Small increase	
15359		Oat group	18/18	14.6 (SE 1.4)	14.7 (SE 3)						SBP nocturnal dip	Ambulatory BP (%)	12 weeks	Small increase	No bias
		Wheat group	18/18	13.7 (SE 1.3)	11.7 (SE 1.7)			NS						Small increase	
15360		Oat group	18/18	14.9 (SE 2.2)	13.9 (SE 1.7)						DBP nocturnal dip	Ambulatory BP (%)	12 weeks	Small increase	No bias
		Wheat group	18/18	18 (SE 1.4)	13.9 (SE 2.5)			NS						Small increase	
(Saltzman *et al.*, 2001) 16187		Control	21/21	118 (SD 15)		-1 (SD 10)					SBP	Clinic BP Seated (mm/Hg)	6 weeks	Decrease	unclear
		Oats	20/22	117 (SD 9)		-6 (SD 7)		<0.05						Decrease	
16188		Control	21/21	70 (SD 8)		-3 (SD 5)					DBP	Clinic BP Seated (mm/Hg)	6 weeks	Decrease	unclear
		Oats	20/22	72 (SD 6)		-4 (SD 6)		NS						Decrease	
(Smith *et al.*, 2008) 16562		High molecular weight beta glucan	45/45			1.4 (SE 1)	NS	0.37			DBP	Not reported (mm/Hg)	6 weeks	No change	No bias
		Low molecular weight beta glucan	45/45			0.2 (SE 0.8)	NS							Increase	
16564		High molecular weight beta	45/45			2.4 (SE 1.5)	NS	0.62			SBP	Not reported (mm/Hg)	6 weeks	No change	No bias

Author/ Result ID	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	p-value difference between groups	Outcome/ Assess-ment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		glucan													
		Low molecular weight beta glucan	45/45			1.5 (SE 1.2)	NS							Increase	
(Swain *et al.*, 1990) 17351		Low fibre wheat supplement	11/11	112	No change						SBP	Clinic BP (mm/Hg)	6 weeks	No change	No bias
		Oat bran supplement	9/9	112	No change									No change	
17352		Low fibre wheat supplement	11/11	68	No change						DBP	Clinic BP (mm/Hg)	6 weeks	No change	No bias
		Oat bran supplement	9/9	68	No change									No change	
17353		Low fibre wheat supplement	11/11	112	107		NS				SBP	Self reported (mm/Hg)	6 weeks	No change	No bias
		Oat bran supplement	9/9	112	110		NS							No change	
17354		Low fibre wheat supplement	11/11	68	65		NS				DBP	Seated (mm/Hg)	6 weeks	No change	No bias
		Oat bran supplement	9/9	68	67		NS							No change	No bias

Blood pressure and added sugars and sugar reduction trials

Summary of cohort results

Data were extracted from one publication, reporting results from the NHS (Ascherio *et al.*, 1996). Consumption of sweets, which included added sugars and sweet snack foods such as cookies, pies and jam, was assessed using a 126-item FFQ and blood pressure was self-reported. The analyses from this study (Ascherio *et al.*, 1996) did not indicate an association between intake of sweets and blood pressure. Adjustments were made for suitable covariates.

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

Summary of RCT data

Five studies providing dietary differences in sugars were identified and data extracted. One small study with 6 month follow-up did not present sufficient information to be included in any meta-analyses (Vasilaras *et al.*, 2001). This study found no evidence for any effect of sugar reduction, but numbers were very small and weight changed differently between study groups, so any effect on blood pressure would not necessarily be attributable to the sugars per se, but weight change generally. One further small study (Poppitt *et al.*, 2002) could not be included in the meta-analysis because insufficient information was provided on the intervention effects and a measure of uncertainty in that estimate such as standard error or confidence interval (data presented in figures only). This study found that consumption of a high carbohydrate, low fat diet, whether high in 'complex' or simple carbohydrate reduced SBP ($p<0.01$). At 6 months, DBP was lowered by the high simple carbohydrate diet compared with the control diet (but 'complex' carbohydrate and control diets did not differ). However, some of these dietary effects may be attributable to differences in extent of weight change in each diet group. Some potential for bias was also identified in this study in that neither patients nor researchers were blind to the intervention group.

The three remaining studies providing dietary differences in sugars were included in the meta-analysis. All studies included adults as participants and measured DBP and SBP in mmHg. Results for each are reported separately. The first follow up reported at the end of the intervention was used. This varied from 6 to 10 weeks.

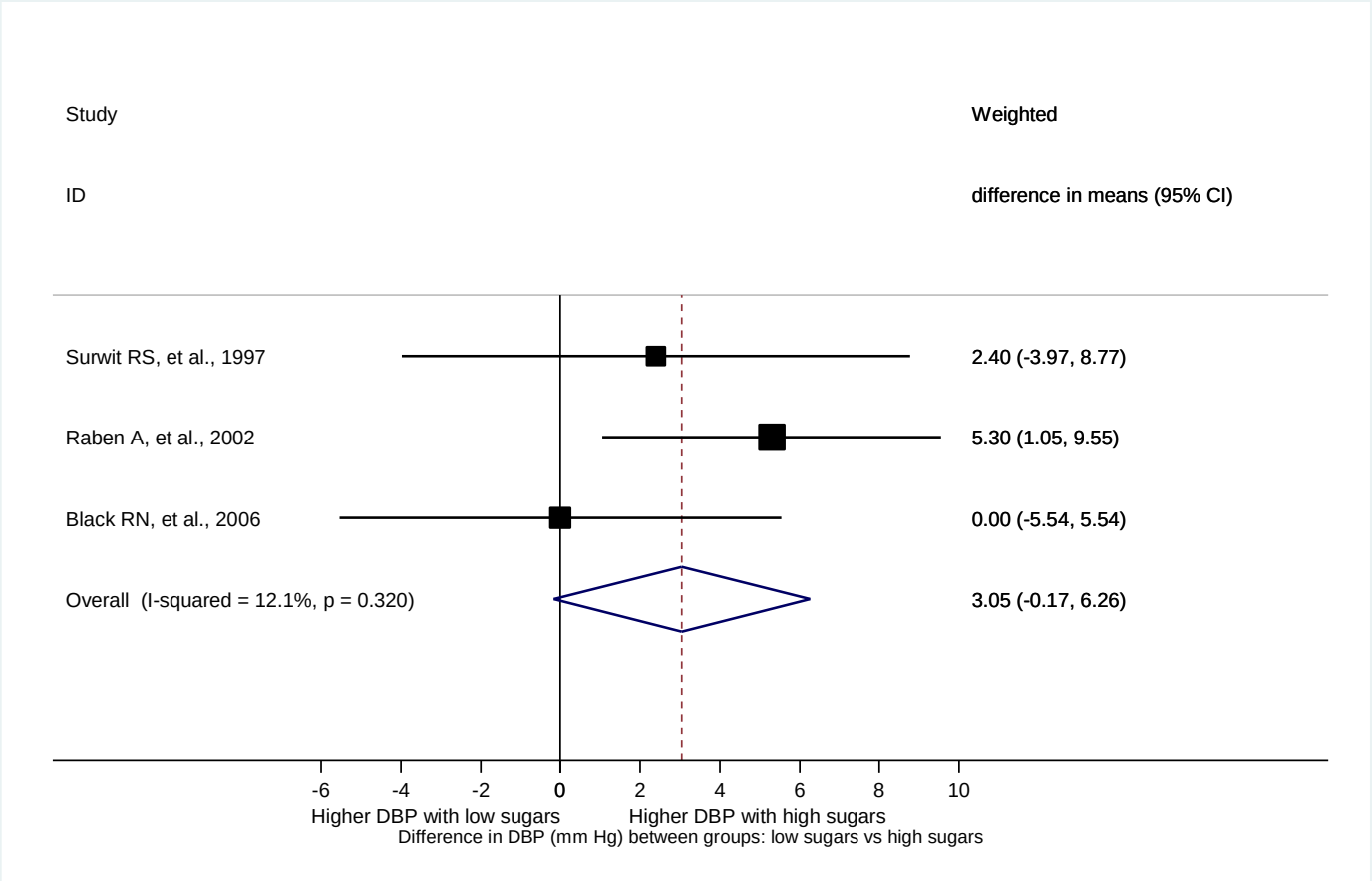
Raben *et al.* compared (Raben *et al.*, 2002) blood pressure after 10 weeks dietary supplementation with sucrose-sweetened foods and drinks (152g sucrose per day) compared with similar food and drink sweetened with artificial sweeteners (0g sucrose per day). Body weight increased in the sucrose-supplemented group, but not in the control group and this may have impacted on blood pressure changes. Black *et al.* (Black *et al.*, 2006) tested a smaller sucrose difference between groups (10 vs. 25% energy from sucrose) but maintained a similar

macronutrient composition and dietary fibre intake between groups. After 6 weeks body weights were unchanged in both groups. The study by Surwit *et al.* (Surwit *et al.*, 1997) compared two low fat (11% energy) hypoenergetic diets: a low sucrose diet (4% energy from sucrose) with a high sucrose diet (43% energy from sucrose). Total carbohydrate was high in both groups (71% energy), and dietary fibre was somewhat higher in the low sucrose group. After 6 weeks body weights decreased in both groups.

Diastolic blood pressure (DBP)

The overall pooled estimate indicated that DBP was 3.1mmHg (95% CI, -0.2 to 6.3mmHg) higher with higher consumption of sugars. This was not significantly different from zero (p=0.06). Heterogeneity denoted by I^2 was 12% (95% CI, 0 to 91%). The pooled estimate of the blood pressure difference between high and low sugars diets suggests higher DBP with higher sugars diets. However, the pooled estimate was of borderline statistical significance and conflicts with the results of studies that could not be included in the meta-analysis. It should therefore be interpreted cautiously.

Figure 2.14 Forest plot for sugar reduction trials and DBP



Systolic blood pressure (SBP)

The overall pooled estimate indicated that SBP was 1.4mmHg (95% CI, -5.4 to 8.3mmHg) higher with higher consumption of sugars. This was not significantly different from zero (p=0.69). Heterogeneity denoted by I^2 was 65% (95% CI, 0 to 90%). Statistically, there was no evidence of a difference in SBP with differences in consumption of sugars.

Figure 2.15 Forest plot for sugar reduction trials and SBP

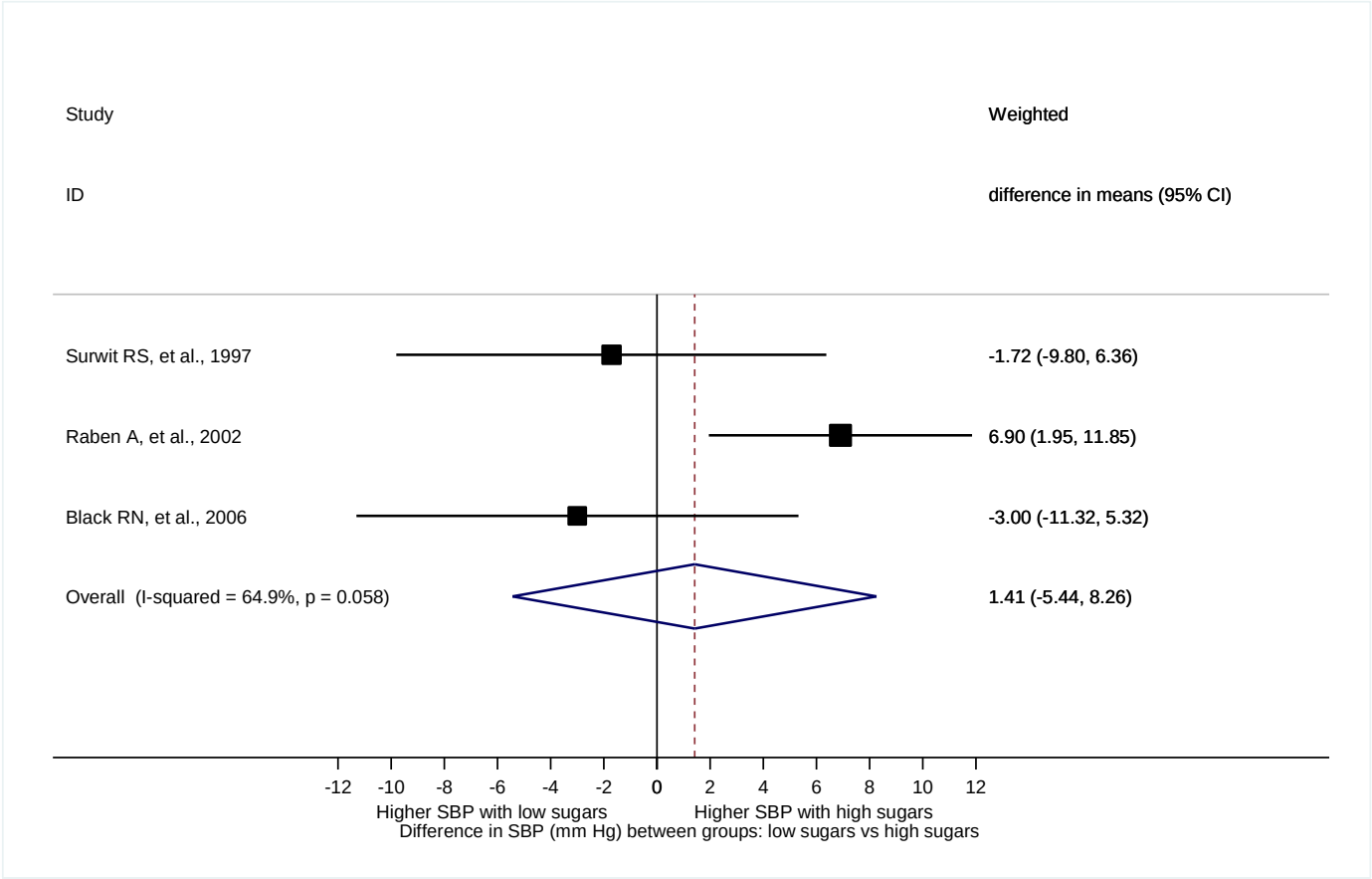


Table 2.22 Blood pressure and added sugars: cohort study in adults

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
(Ascherio *et al.*, 1996) 13933 NHS	USA, Primarily White, Cancer free, No CHD, No T2DM	30-55 %M 0	121700	4 years (0.9)	FFQ (126)	Added sugars, Sweet snack foods (chocolate, candies, cookies, brownies, donuts, cakes, pies, sweet rolls, jam)	SBP Self-reported	1 g/day	0.059 (0.041)	0.2	Age, alcohol, BMI, cakes and chocolate, energy intake
13935 NHS							DBP Self-reported	1 g/day	0.03 (0.028)	0.3	Age, alcohol, BMI, cakes and chocolate, energy intake

Table 2.23 Blood pressure and sugar reduction trials: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	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
(Vasilaras *et al.*, 2001) 15038	Control diet	7/7			NS		DBP	Clinic BP (mm/Hg)	6 months	Increase	unclear
	Low-fat high-complex carbohydrate diet	9/9			NS					Decrease	
	Low-fat, high-simple carbohydrate diet	8/8			NS					No change	
15040	Control diet	7/7			NS		SBP	Clinic BP (mm/Hg)	6 months	Increase	unclear
	Low-fat high-complex carbohydrate diet	9/9			NS					Decrease	
	Low-fat, high-simple carbohydrate diet	8/8			NS					No change	
(Surwit *et al.*, 1997) *15048	High sucrose diet	20/28	139.5 (SD 16.02)	127.95 (SD 14.59)			SBP	Clinic BP (mm/Hg)	6 weeks	Decrease	unclear
	Low sucrose diet	22/24	131.82 (SD 13.52)	129.67 (SD 11.28)		NS				Decrease	
**15049	High sucrose diet	20/28	74.85 (SD 11.08)	71.5 (SD 11.95)			DBP	Clinic BP	6 weeks	Decrease	unclear

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	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
								(mm/Hg)			
	Low sucrose diet	22/24	72.82 (SD 9.02)	69.1 (SD 8.29)		NS				Decrease	
(Poppitt *et al.*, 2002) 15384	Control	7/15	132 (SD 14)	higher			SBP change	Clinic BP (mm/Hg)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	136 (SD 17)	lower	0.01					Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	138 (SD 22)	lower	0.01					No change	
15385	Control	7/15	87 (SD 10)	higher			DBP change	Clinic BP (mm/Hg)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	84 (SD 13)							Decrease	
	Low-fat, high -simple C carbohydrate diet	13/15	86 (SD 13)	lower	0.05					No change	
(Raben *et al.*, 2002) *16479	Sucrose minus Sweetener	Sucrose: 21/21 Sweetener: 20/20				6.9 (CI 2, 11.9)	SBP	Clinic BP (mm/Hg)	10 weeks	Increase in sucrose group, decrease in sweetener group	unclear
**16480	Sucrose minus Sweetener	Sucrose: 21/21 Sweetener: 20/20				5.3 (CI 1.1, 9.6)	DBP	Clinic BP (mm/Hg)	10 weeks	Increase in sucrose group, decrease in sweetener group	unclear
(Black *et al.*, 2006) *16612	High sucrose diet	13/13	127 (SE 3)	122 (SE 3)	NS		SBP	Clinic BP (mm/Hg)	6 weeks	No change	unclear
	Low sucrose diet	13/13	127 (SE 3)	125 (SE 3)						No change	
**16613	High sucrose diet	13/13	69 (SE 3)	71 (SE 2)	NS		DBP	Clinic BP (mm/Hg)	6 weeks	No change	unclear
	Low sucrose diet	13/13	69 (SE 3)	71 (SE 2)						No change	

*This result was used in the meta-analysis for sugar reduction trials and SBP

**This result was used in the meta-analysis for sugar reduction trials and DBP

Blood pressure and carbohydrate rich foods

Summary of cohort results

One publication, reporting results from one cohort study, provided evidence of intake of carbohydrate rich foods and blood pressure (Ascherio *et al.*, 1996). Findings from this study (the NHS) (Ascherio *et al.*, 1996) do not suggest a consistent direction of effect for individual carbohydrate rich foods. Those, namely white bread, French fries, potatoes and total cereals (for SBP only) showed a positive relationship with blood pressure whilst wholemeal bread, breakfast cereals, brown rice and total cereals (for DBP only) indicated a negative relationship (Ascherio *et al.*, 1996). It is important to note, however, that only some of these associations reached statistical significance.

Appropriate confounders were adjusted for including age, cereals and energy intake.

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

No RCTs reported outcomes concerning carbohydrate rich foods and blood pressure.

Table 2.24 Blood pressure and carbohydrate rich foods: 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
(Ascherio *et al.*, 1996) 13932 NHS	USA, Primarily White, Cancer free, No CHD, No T2DM	30-55 %M 0	121700	4 years (0.9)	FFQ (126)	Cereals, total (cold cereal, cooked oats, cooked cereals, white & dark bread, English muffins, muffins, brown & white rice, pasta, other grains, pancakes, potatoes, crackers)	SBP Self-reported	1 g/day	0.143 (0.042)	0.0007	Age, alcohol, BMI, cereals, energy intake
13934 NHS							DBP Self-reported	1 g/day	-0.006 (0.028)	0.8	
13941 NHS						White bread	SBP Self-reported	1 serving/day	0.325 (0.062)	<0.0001	Age, alcohol, BMI, breakfast cereals, brown rice, energy intake, grains, pasta, potatoes, white rice, wholemeal bread
13942 NHS							DBP Self-reported	1 serving/day	0.115 (0.042)	0.006	
13943 NHS						Wholemeal bread	SBP Self-reported	1 serving/day	-0.049 (0.066)	0.5	Age, alcohol, BMI, breakfast cereals, brown rice, energy intake, grains, pasta, potatoes, white bread, white rice
13944 NHS							DBP Self-reported	1 serving/day	-0.167 (0.045)	0.0002	
13945 NHS						Breakfast cereals, unspecified	SBP Self-reported	1 serving/day	-0.016 (0.138)	0.9	Age, alcohol, BMI, brown rice, energy intake, grains, pasta, potatoes, white bread, white rice, wholemeal bread
13946 NHS							DBP Self-reported	1 serving/day	-0.225 (0.093)	0.02	
13953 NHS						Rice, Brown	SBP Self-reported	1 serving/day	-2.981 (0.621)	<0.0001	Age, alcohol, BMI, breakfast cereals, energy intake, grains, pasta, potatoes, white bread, white rice, wholemeal bread
13954 NHS							DBP Self-reported	1 serving/day	-1.223 (0.420)	0.004	
13952 NHS						White rice	SBP Self-reported	1 serving/day	-1.19 (0.343)	0.0005	Age, alcohol, BMI, breakfast cereals, brown rice, energy intake, grains, pasta, potatoes, white bread, wholemeal bread
13952 NHS							DBP Self-reported	1 serving/day	-0.4 (0.233)	0.09	
13949 NHS						French fries	SBP Self-reported	1 serving/day	0.749 (0.251)	0.003	Age, alcohol, BMI, breakfast cereals, brown rice, energy intake, grains, pasta, white bread, white rice, wholemeal bread
13950 NHS							DBP Self-reported	1 serving/day	0.595 (0.170)	0.0004	
13947 NHS						Potatoes (Baked potatoes, excludes French fries)	SBP Self-reported	1 serving/day	0.649 (0.186)	0.0005	As above
13948 NHS							DBP Self-reported	1 serving/day	0.192 (0.126)	0.1	As above

Blood pressure and breakfast cereals

No cohort studies reported results concerning breakfast cereals and continuous blood pressure.

Summary of RCT data

One RCT was identified that reported results from a trial of cereals for breakfast compared to a control group (Kleemola *et al.*, 1999). With six weeks of follow-up, this trial found no evidence for any difference in DBP or SBP between those eating cereal for breakfast and those on a control diet. However, there was some evidence of potential bias as the allocation to intervention groups was unblinded.

Table 2.25 Blood pressure and breakfast cereals: RCT data

Author/ Result ID	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
(Kleemola *et al.*, 1999) 15251	Group 1- Cereal diet first	104/allocated not reported	132 (SD 14.5)	131 (SD 14.4)	-1.0	0.28	SBP	Clinic BP (mm/Hg)	6 weeks	No change	bias
	Group 2- Control diet first	105/allocated not reported	129 (SD 16.6)	127 (SD 15.3)	-2.0					No change	
15252	Group 1- Cereal diet first	104/allocated not reported	83 (SD 9.6)	82 (SD 9.4)	-1.0	0.88	DBP	Clinic BP (mm/Hg)	6 weeks	No change	bias
	Group 2- Control diet first	105/allocated not reported	81 (SD 9.1)	80 (SD 9.2)	-1.0					No change	
15257	Group 1- Control diet second	104/allocated not reported	129 (SD 14)	129 (SD 15)	0.0		SBP	Clinic BP (mm/Hg)	6 weeks	No change	bias
	Group 2- Cereal diet second	105/allocated not reported	126 (SD 14.7)	126 (SD 14.5)	0.0	0.72				No change	
15258	Group 1- Control diet second	104/allocated not reported	81 (SD 9.7)	80 (SD 9.5)	-1.0		DBP	Clinic BP (mm/Hg)	6 weeks	No change	bias
	Group 2- Cereal diet second	105/allocated not reported	79 (SD 9.5)	78 (SD 9.1)	-1.0	0.17				No change	

Blood pressure and legumes

No cohort studies reported results concerning legumes and continuous blood pressure.

Summary of RCT data

One RCT was identified that reported results investigating the effect of legumes on blood pressure measures (Lee *et al.*, 2009). This study followed-up participants for 16 weeks after randomisation to either lupin kernel (a member of the legume family) flour bread or wheat control bread, to record ambulatory SBP and DBP. They found some evidence for a reduction in SBP, but less so for DBP.

Table 2.26 Blood pressure and legumes: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Follow-up	p-value difference between groups	Difference between groups at follow-up	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Lee *et al.*, 2009) 16403	Control bread	37/48	123 (CI 121.1, 124.9)				SBP	Ambulatory BP (mm/Hg)	16 weeks	No change	unclear
	Lupin flour bread	37/40	120 (CI 118.1, 121.9)	0.03						No change	
16405	Lupin flour bread minus control bread	Lupin: 37/40 Control: 37/48			-3 (CI -5.6, -0.3)	0.03	SBP	Ambulatory BP (mm/Hg)	16 weeks	No change in both	unclear
16406	Control bread	37/48	71 (CI 69.9, 72.2)				DBP	Ambulatory BP (mm/Hg)	16 weeks	No change	unclear
	Lupin flour bread	37/40	71.6 (CI 70.5, 72.8)	0.47						No change	
16407	Lupin flour bread minus control bread	Lupin: 37/40 Control: 37/48			0.6 (CI -1, 2.2)	0.47	DBP	Ambulatory BP (mm/Hg)	16 weeks	No change in both	unclear
16408	Control bread	37/48	127.6 (CI 124.9, 130.3)				SBP	Awake ambulatory BP (mm/Hg)	16 weeks	No change	unclear
	Lupin flour bread	37/40	124.6 (CI 121.8, 127.3)	0.08						No change	
16409	Lupin flour bread minus control bread	Lupin: 37/40 Control: 37/48			-3.1 (CI -6.4, 0.3)	0.08	SBP	Awake ambulatory BP (mm/Hg)	16 weeks	No change in both	unclear
16410	Control bread	37/48	74.7 (CI 73, 76.5)				DBP	Awake ambulatory BP (mm/Hg)	16 weeks	No change	unclear
	Lupin flour bread	37/40	76.4 (CI 74.6, 78.2)	0.13						No change	
16411	Lupin flour bread minus control bread	Lupin: 37/40 Control: 37/48			1.6 (CI -0.5, 3.7)	0.13	DBP	Awake ambulatory BP (mm/Hg)	16 weeks	No change in both	unclear
16412	Control bread	37/48	113.6 (CI 110.7, 116.5)				SBP	Asleep ambulatory BP (mm/Hg)	16 weeks	No change	unclear
	Lupin flour bread	37/40	110.5 (CI 107.6, 113.4)	0.06						No change	
16413	Lupin flour bread minus control bread	Lupin: 37/40 Control: 37/48			-3.1 (CI -6.4, 0.2)	0.06	SBP	Asleep ambulatory BP (mm/Hg)	16 weeks	No change in both	unclear
16414	Control bread	37/48	63.9 (CI 61.8, 66.1)				DBP	Asleep ambulatory BP (mm/Hg)	16 weeks	No change	unclear
	Lupin flour bread	37/40	63.1 (CI 61, 65.3)	0.48						No change	
16415	Lupin flour bread minus control bread	Lupin: 37/40 Control: 37/48			-0.8 (CI -3.1, 1.5)	0.48	DBP	Asleep ambulatory BP (mm/Hg)	16 weeks	No change in both	unclear

Blood pressure and wholegrains

No cohort studies reported results concerning wholegrains and continuous blood pressure.

Summary of RCT data

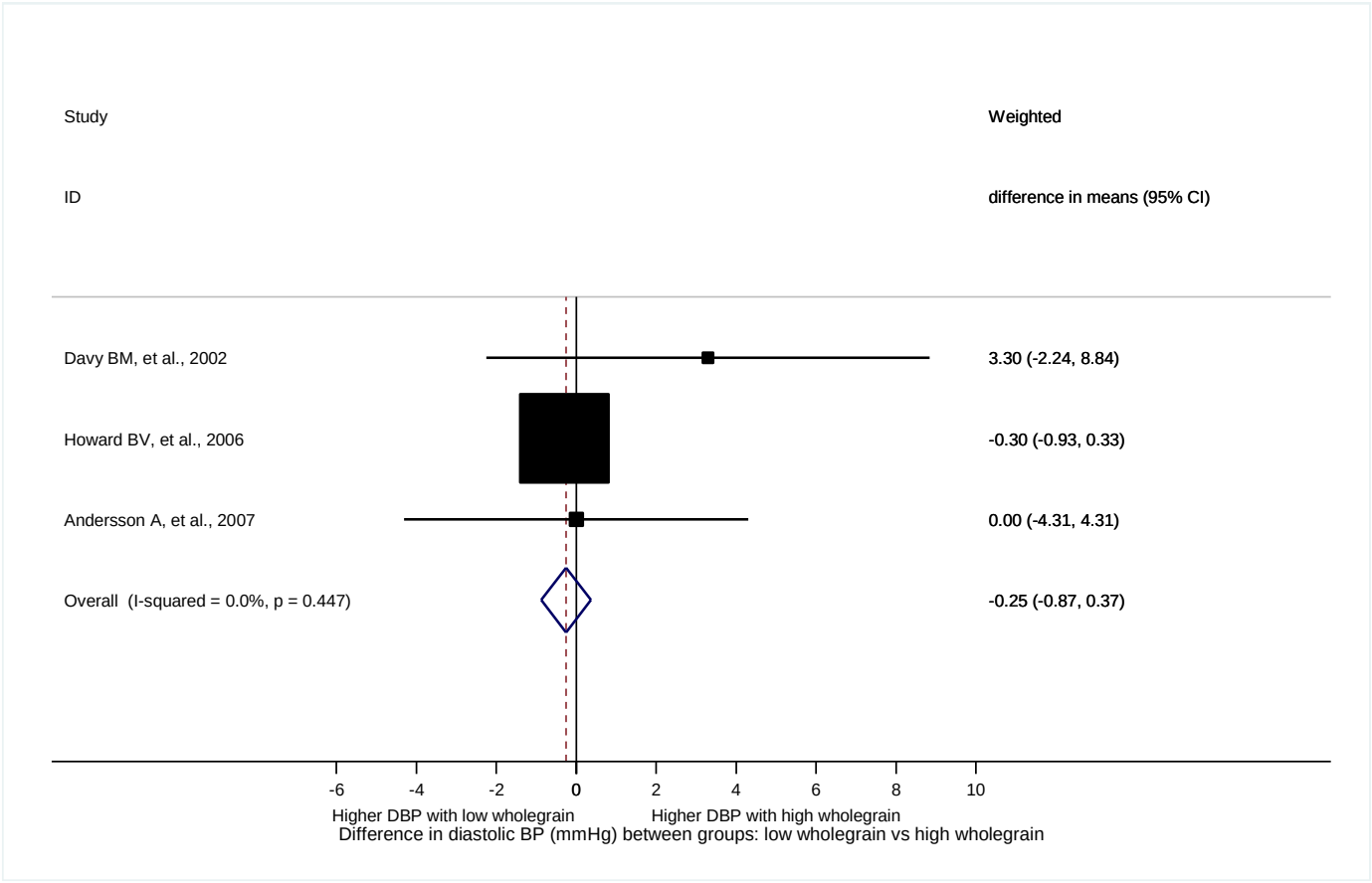
Three trials were identified that explored the effects of wholegrains on blood pressure. Two were conducted in the USA (Tinker *et al.*, 2008;Howard *et al.*, 2006b;Davy *et al.*, 2002), and one in Sweden (Andersson *et al.*, 2007). All were included in a meta-analysis.

All three studies included adults as participants and measured DBP and SBP in mmHg. Results for each are reported separately. The first follow up reported at the end of the intervention was used. This varied from 6 weeks to 3 years. Two publications presented data from the same trial (Tinker *et al.*, 2008;Howard *et al.*, 2006b), so the results from just one were used (Howard *et al.*, 2006b) because this is the result closest to the end of the intervention, which is still ongoing in this trial, so the longest recorded follow-up was selected. This trial compared blood pressure in women advised to reduce fat intake (to 20%), increase fruit, vegetables and wholegrains with a 'no change' group. Compliance with the wholegrain direction was minimal (half a serving per day extra consumed). The study by Davy *et al.* (Davy *et al.*, 2002) included older overweight males, with somewhat raised baseline blood pressure which was reported as casual resting arterial blood pressure and 24-hour ambulatory arterial blood pressure. This study compared a diet with 60g oatmeal and 76g oat bran ready-to-eat cold cereal (containing 14g/day of fibre and providing 5.5g/d beta glucan) with a wheat diet containing 60g wheat cereal and 81g Frosted Mini-Wheats (containing 14g/d of dietary fibre). Andersson *et al.* (Andersson *et al.*, 2007) explored blood pressure differences in men and women consuming their usual diet with whole grain foods (bread, crisp bread, muesli & pasta - minimum 50% wholegrain in provided foods = 112g wholegrain/day) or with refined grain foods (bread, crisp bread, muesli & pasta). There was a marked difference in fibre content between the diets, and body weight increased in both groups possibly due to the test foods supplementing rather than substituting for usual foods.

Diastolic blood pressure (DBP)

The overall pooled estimate indicated that DBP was 0.3mmHg (95% CI, -0.4 to 0.9mmHg) higher with higher consumption of wholegrain. This was not significantly different from zero (p=0.43). Heterogeneity denoted by I^2 was 0% (95% CI, 0 to 87%). It should be noted that one study (Howard *et al.*, 2006b) contributed 97% to the pooled estimate and this dominated the pooled estimate. This study achieved a very small increase in wholegrain consumption (less than one serving per day). However, there were too few remaining studies to exclude this study in a sensitivity analysis. Statistically, there was no evidence of a difference in DBP with differences in consumption of wholegrains.

Figure 2.16 Forest plot for wholegrains and DBP



Systolic blood pressure (SBP)

The overall pooled estimate indicated that SBP was 0.2mmHg (95% CI, -1.6 to 2.0mmHg) higher with higher consumption of wholegrains. This was not significantly different from zero (p=0.85). Heterogeneity denoted by I^2 was 0% (95% CI, 0 to 90%). It should be noted that one study (Howard *et al.*, 2006b) contributed 92% to the pooled estimate, having a dominant influence on the pooled estimate. However, there were too few remaining studies to exclude this study in a sensitivity analysis. Statistically, there was no evidence of a difference in SBP with differences in consumption of wholegrains.

Figure 2.17 Forest plot for wholegrains and SBP

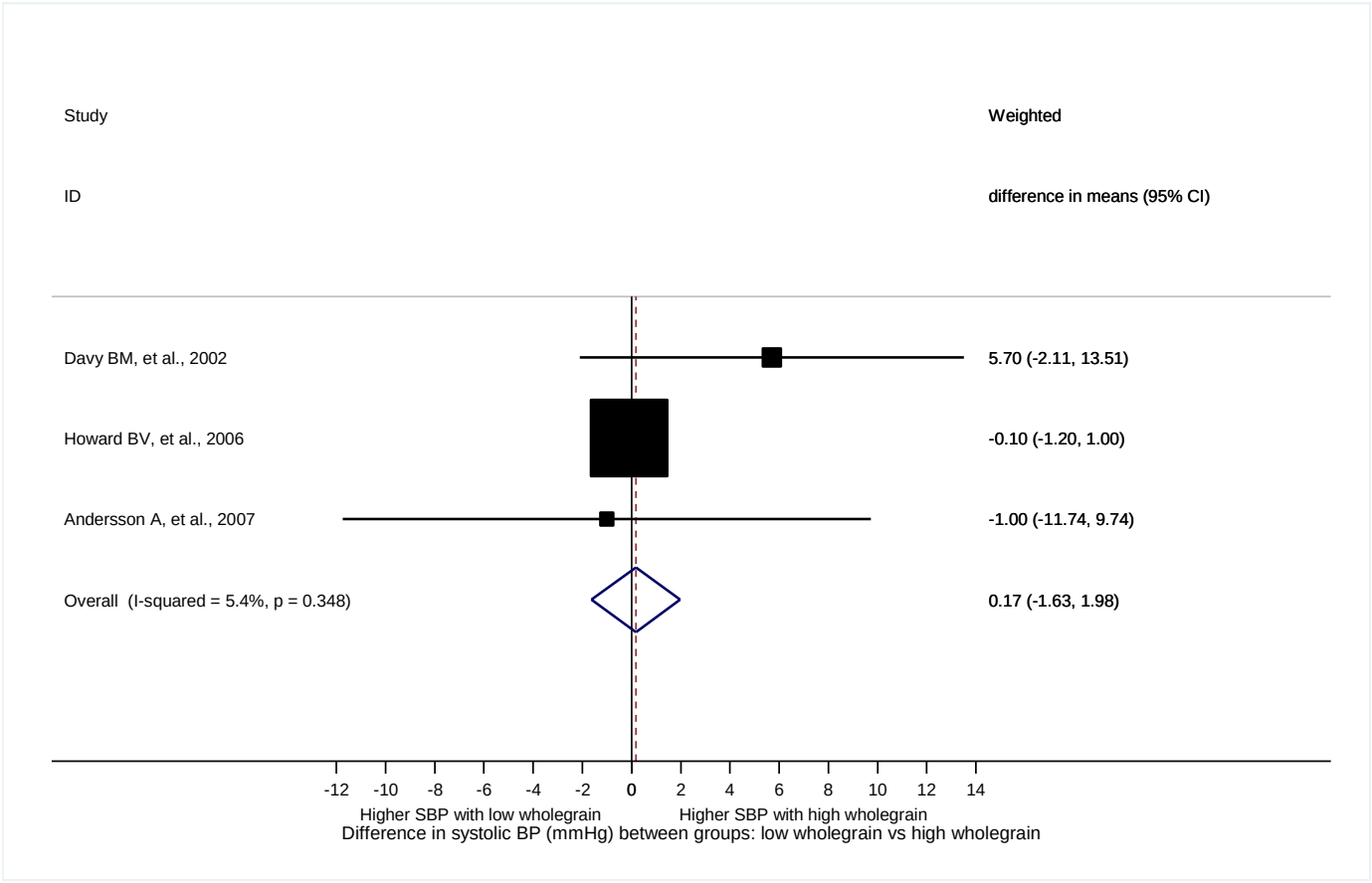


Table 2.27 Blood pressure and wholegrains: RCT data

Author/ Result ID	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
(Davy *et al.*, 2002) 15337	Oat group	18/18	138.2 (SE 2.4)	137 (SE 2)					SBP	Clinic BP Seated (mm/Hg)	8 weeks	Small increase	No bias
	Wheat group	18/18	142.3 (SE 2.4)	141.1 (SE 2.7)			NS					Small increase	
*15338	Oat group	18/18	138.2 (SE 2.4)	134.6 (SE 3.1)					SBP	Clinic BP Seated (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	142.3 (SE 2.4)	140.3 (SE 2.5)			NS					Small increase	
15340	Oat group	18/18	88.5 (SE 1.6)	88.1 (SE 2.1)					DBP	Clinic BP Seated (mm/Hg)	8 weeks	Small increase	No bias
	Wheat group	18/18	90.4 (SE 1.5)	89.7 (SE 1.6)			NS					Small increase	
**15341	Oat group	18/18	88.5 (SE 1.6)	87.6 (SE 2)					DBP	Clinic BP Seated (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	90.4 (SE 1.5)	90.9 (SE 2)			NS					Small increase	
15342	Oat group	18/18	132.6 (SE 2.3)	131.6 (SE 3.1)					SBP	Clinic BP Supine (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	139.2 (SE 2.4)	135.3 (SE 2.8)			NS					Small increase	
15344	Oat group	18/18	83.6 (SE 1.4)	83.6 (SE 1.6)					DBP	Clinic BP Supine (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	85.1 (SE 1.8)	88.3 (SE 4)			NS					Small increase	
15348	Oat group	18/18	133.3 (SE 1.5)	132.8 (SE 2)					DBP	24-hour Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	141.7 (SE 2.2)	143 (SE 2.4)			NS					Small increase	
15349	Oat group	18/18	84.1 (SE 1.6)	84.1 (SE 1.3)					DBP	24-hour Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	87.9 (SE 1.5)	88.3 (SE 1.7)			NS					Small	

Author/ Result ID	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
												increase	
15350	Oat group	18/18	137.5 (SE 1.6)	136.3 (SE 2)					SBP	Ambulatory BP Daytime (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	146.4 (SE 2.2)	147.4 (SE 2.7)			NS					Small increase	
15351	Oat group	18/18	117.4 (SE 2.1)	116.6 (SE 4.7)					SBP	Ambulatory BP Night-time (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	126.6 (SE 2.5)	129.8 (SE 2.6)			NS					Small increase	
15352	Oat group	18/18	86.1 (SE 1.9)	87 (SE 1.4)					DBP	Ambulatory BP Daytime (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	91 (SE 1.9)	91.6 (SE 1.9)			NS					Small increase	
15353	Oat group	18/18	73 (SE 1.9)	75 (SE 2)		0.01			DBP	Ambulatory BP Night-time (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	74.4 (SE 1.7)	78.5 (SE 2.2)			NS					Small increase	
15355	Oat group	18/18	104.1 (SE 1.5)	103.2 (SE 1.6)					Mean arterial pressure	Ambulatory BP Daytime (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	110.1 (SE 1.6)	110.3 (SE 2.3)			NS					Small increase	
15356	Oat group	18/18	88.1 (SE 1.8)	90.2 (SE 2)		0.02			Mean arterial pressure	Ambulatory BP Night-time (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	90.3 (SE 2.9)	96.2 (SE 2.2)			NS					Small increase	
15361	Oat group	18/18	100.7 (SE 1.5)	100 (SE 1.4)					Mean arterial pressure	24-hour Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	106.1 (SE 1.6)	106.7 (SE 2) (SD			NS					Small increase	
15357	Oat group	18/18	13.6 (SE 0.6)	13.6 (SE 1)					SBP variability	Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	12.9 (SE 0.9)	13.5 (SE 0.5)			NS					Small	

Author/ Result ID	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
												increase	
15358	Oat group	18/18	10.2 (SE 0.4)	11.1 (SE 0.7)					DBP variability	Ambulatory BP (mm/Hg)	12 weeks	Small increase	No bias
	Wheat group	18/18	10.9 (SE 0.5)	10.8 (SE 0.6)			NS					Small increase	
15359	Oat group	18/18	14.6 (SE 1.4)	14.7 (SE 3)					SBP nocturnal dip	Ambulatory BP (%)	12 weeks	Small increase	No bias
	Wheat group	18/18	13.7 (SE 1.3)	11.7 (SE 1.7)			NS					Small increase	
15360	Oat group	18/18	14.9 (SE 2.2)	13.9 (SE 1.7)					DBP nocturnal dip	Ambulatory BP (%)	12 weeks	Small increase	No bias
	Wheat group	18/18	18 (SE 1.4)	13.9 (SE 2.5)			NS					Small increase	
(Andersson *et al.*, 2007) *16305	Refined grain products	15/30	130 (SD 16)	130 (SD 15)		NS			SBP	Clinic BP Supine (mm/Hg)	6 weeks	Increase	unclear
	Wholegrain products	15/30	130 (SD 17)	129 (SD 15)		NS	0.35					Increase	
**16306	Refined grain products	30/30	80 (SD 10)	81 (SD 9)		NS			DBP	Clinic BP Supine (mm/Hg)	6 weeks	Increase	unclear
	Wholegrain products	30/30	81 (SD 9)	81 (SD 8)		NS						Increase	
(Tinker *et al.*, 2008) 15368	Control	25173/29294	127.4 (SD 17.1)	125.4 (SD 16.8)					SBP	Not reported (mm/Hg)	1 year	No change	unclear
	Low fat diet	17126/19541	127.1 (SD 17.2)	124.4 (SD 17.1)			0.001					Decrease	
15369	Control	22532/29294	127.4 (SD 17.1)	124.6 (SD 16.3)					SBP	Not reported (mm/Hg)	6 years	No change	unclear
	Low fat diet	14543/19541	127.1 (SD 17.2)	124.5 (SD 16.5)								Decrease	
15370	Control	25169/29294	76 (SD 9)	74.7 (SD 9.1)					DBP	Not reported (mm/Hg)	1 year	No change	unclear
	Low fat diet	17125/19541	75.9 (SD 9.1)	73.9 (SD 9.2)			0.001					Decrease	
15371	Control	22532/29294	76 (SD 9)	71.9 (SD 9.2)					DBP	Not reported (mm/Hg)	6 years	No change	unclear
	Low fat diet	14540/19541	75.9 (SD 9.1)	71.7 (SD 9.2)								Decrease	
(Howard *et al.*, 2006b) 16244	Control	approx 1699 participants included as a	127.9 (SD 17.2)	125.7 (SD 16.8)	-2.1 (SD 16.4)				SBP	Clinic BP (mm/Hg)	3 years	No change	No bias

Author/ Result ID	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
		5.8% sub-sample of 29294 in group											
	Low fat	approx 1132 participants included as a 5.8% sub-sample of 19541 in group	127.5 (SD 17.2)	125.1 (SD 16.9)	-2.2 (SD 16.3)		NS					Decrease	
*17611	Low fat minus control	Low fat: approx 1132 participants included as a 5.8% sub-sample of 19541 in group Control: approx 1699 participants included as a 5.8% sub-sample of 29294 in group						-0.17 (CI -0.49,0.15)	SBP	Clinic BP (mm/Hg)	3 years	No change in control group, decrease in low fat group	No bias
16245	Control	approx 1699 participants included as a 5.8% sub-sample of 29294 in group	76.0 (SD 9.1)	73.6 (SD 9.3)	-2.3 (SD 9.4)				DBP	Clinic BP (mm/Hg)	3 years	No change	No bias
	Low fat	approx 1132 participants included as a 5.8% sub-sample of 19541 in group	75.9 (SD 9.1)	73.1 (SD 9.4)	-2.6 (SD 9.4)		<0.001					Decrease	
**17610	Low fat minus control	Low fat: approx 1132 participants included as a 5.8% sub-sample of 19541 in group Control: approx1699 participants included as a 5.8% sub-sample of 29294 in group						-0.31 (CI -0.5, -0.13)	DBP	Clinic BP (mm/Hg)	3 years	No change in control group, decrease in low fat group	No bias

*This result was used in the meta-analysis for wholegrains and SBP

**This result was used in the meta-analysis for wholegrains and DBP

Blood pressure and glycaemic index and load

No cohort studies reported results concerning glycaemic index or load and continuous blood pressure.

Summary of RCT data

Seven studies conducted in the USA (3), UK, Denmark, France and Spain were identified that provided information on dietary differences in glycaemic index (GI) or glycaemic load (GL) between groups in relation to blood pressure. All seven trials were included in the meta-analysis. All studies included adults as participants. One study was conducted on males only (Philippou *et al.*, 2009), and 2 on women only (Jensen *et al.*, 2008;Bellisle *et al.*, 2007). Mean BMI of participants was greater than 30kg/m^2 in five studies (Abete *et al.*, 2008;Pereira *et al.*, 2004;Maki *et al.*, 2007b;Ebbeling *et al.*, 2005;Bellisle *et al.*, 2007), 28 in one study (Jensen *et al.*, 2008), but was not reported in another study (Philippou *et al.*, 2009).

Definitions of different levels of GI and GL are reported in the trial characteristics table. 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.

Pereira *et al.* (Pereira *et al.*, 2004) provided hypoenergetic high or low GI diets (GI, average of 82 and 50 units respectively) to 39 obese males for about two months. Body composition changes were similar in each diet group, but blood pressure (p=0.07 for both systolic and diastolic) decreased more with the low-glycaemic load diet. In Jensen *et al.* (Jensen *et al.*, 2008) overweight women were allocated to either a high or low GI high carbohydrate, low fat *ad libitum* diet for 10 weeks. Carbohydrate-rich foods such as bread and pasta were provided. Weight losses were similar in both dietary groups, as were blood pressure changes. Bellisle *et al.* (Bellisle *et al.*, 2007) studied the relative efficacy of the Weight Watchers dietary plan with or without a focus on the inclusion of low GI sources of carbohydrate. The diets were both energy restricted, and weight losses were somewhat higher in the standard Weight Watchers diet group, with no beneficial impact of GI on blood pressure.

The Spanish study reported by Abete *et al.* (Abete *et al.*, 2008) was a free-living dietary restriction trial comparing a high GI diet (60-65 units) with low GI (40-45 units) in 32 obese men and women for 8 weeks. The major sources of carbohydrate were potatoes and rice or pasta and legumes in the high and low GI dietary groups respectively. Dietary fibre intake was higher in the low GI group (25 vs. 18.5 g/d). Higher weight losses were experienced by the low GI group compared with the high GI group (7 vs. 5 kg) but the between group differences in blood pressure change were not significantly different. In the UK study conducted by Phillippou *et al.* (Philippou *et al.*, 2009), blood pressure was compared between high and low GI groups in which intake of seeded bread, wholemeal pitta, muesli, porridge, sweet potatoes, pasta, noodles, basmati slow-cook rice, beans, lentils, apples, dried fruit, nuts was encouraged in the low GI group and white/wholemeal bread, cornflakes, weetabix, potatoes, couscous, risotto rice, melon, pineapple, and rice cakes in the high GI group. This resulted in an energy decrease in both groups, and a similar macronutrient intake.

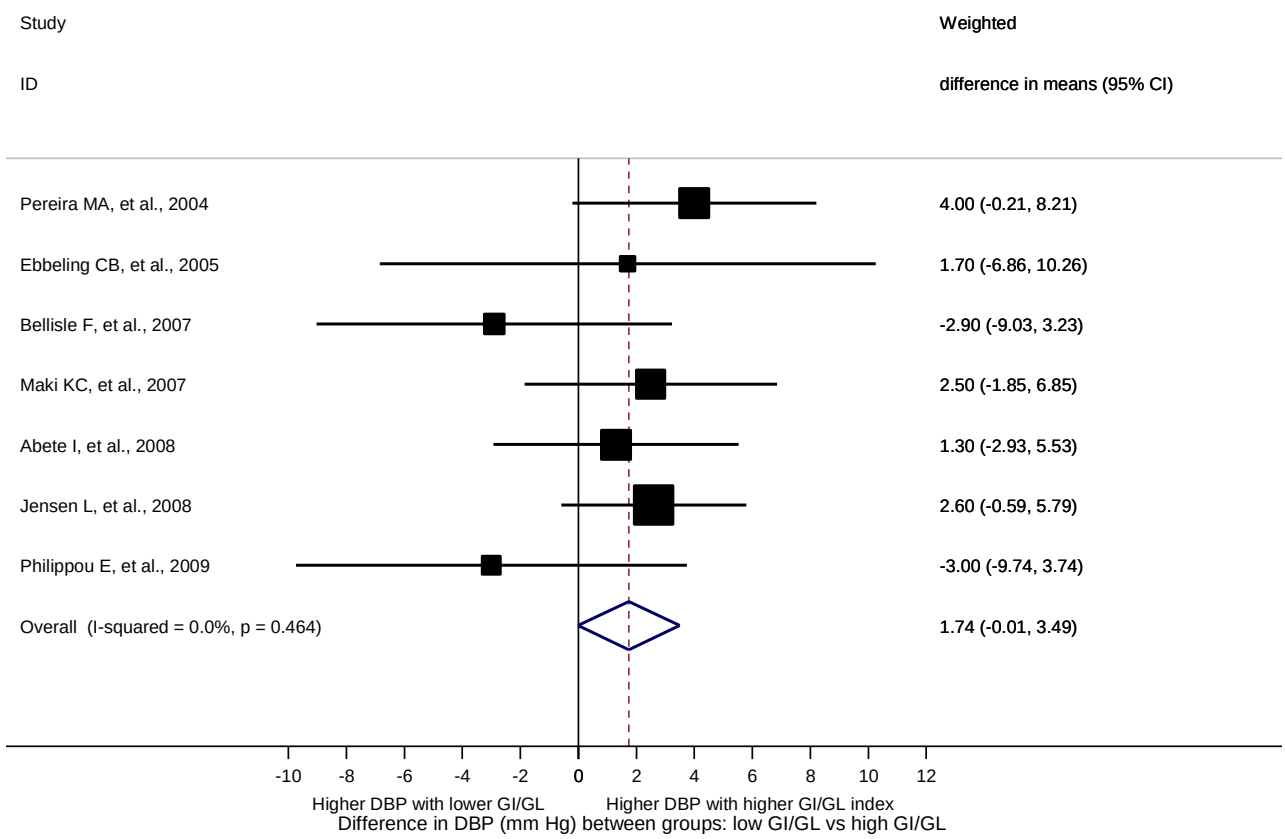
In the study by Maki *et al.* (Maki *et al.*, 2007b) an *ad libitum* reduced-glycaemic-load diet was compared with a low-fat, energy-restricted, portion-controlled diet in overweight and obese adults during a 12-week initial weight-loss phase and a 24-36 week weight-loss maintenance phase. Fibre intakes were similar and average dietary GI did not differ greatly between groups (48 vs. 51), but total carbohydrate was halved in the low GI diet group. Ebbeling *et al.* (Ebbeling *et al.*, 2005) adopted a similar approach since they compared an *ad libitum* energy, low GI diet, which provided 45-50% of energy from carbohydrate and 30-35% from fat with a higher carbohydrate (59%), low fat (23%) energy-restricted meal plan based on an exchange system.

The first follow up reported at the end of the intervention was used. This varied from 2 to 12 months. All trials provided evidence of a decrease in body weight in both high and low GI groups, although there were some differences between high and low GI diet groups in terms of the extent of weight loss. Against a background of decreasing body weight, the impact of the nature of the carbohydrate consumed on blood pressure may be difficult to detect. Since blood pressure may be modified by body weight change, any differences in outcome may therefore not be solely attributable to the nature of the carbohydrate component of the dietary intervention.

Diastolic blood pressure (DBP)

The overall pooled estimate indicated that DBP was 1.7mmHg (95% CI, -0.01 to 3.5) higher with consumption of a high GI or GL diet. This was nearly significantly different from zero (p=0.052). There was no excess heterogeneity, which when denoted by I^2 was 0% (95% CI, 0 to 69%). With borderline statistical significance, results suggest that there is some evidence of higher DBP with diets higher in dietary glycaemic index or glycaemic load. There were too few studies to present a funnel plot.

Figure 2.18 Forest plot for glycaemic index or glycaemic load diets and DBP



Systolic blood pressure (SBP)

The overall pooled estimate indicated that SBP was 0.72mmHg (95% CI, -1.44 to 2.88) higher with consumption of a high GI or GL diet, but this was not significantly different from zero (p=0.51). Heterogeneity denoted by I^2 was 14% (95% CI, 0 to 75%). Statistically, there was no evidence of a difference in SBP with changes in dietary glycaemic index or glycaemic load. There were too few studies to present a funnel plot.

Figure 2.19 Forest plot for glycaemic index or glycaemic load diets and SBP

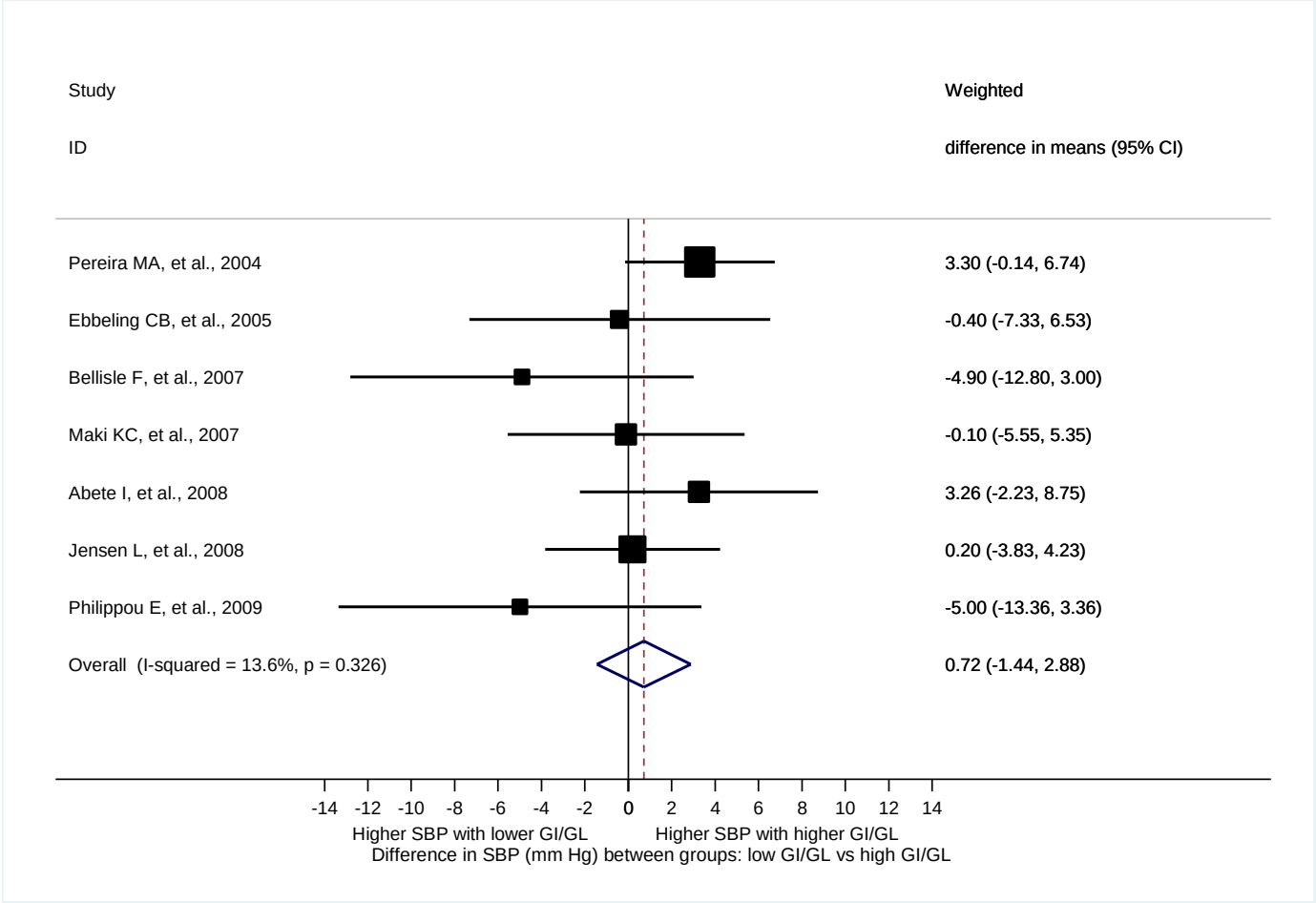


Table 2.28 Blood pressure and glycaemic index and load: RCT data

Author/ Result ID	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
(Philippou *et al.*, 2009) 14665	High GI	16/28	132 (SD 15)	122 (SD 13)	-10 (SD 10)	<0.01		SBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
	Low GI	22/28	130 (SD 13)	126 (SD 12)	-5 (SD 10)	<0.01	NS				Decrease	
14666	High GI	16/28	81 (SD 10)	76 (SD 8)	-5 (SD 7)	<0.01		DBP	Clinic BP (mm/Hg)	6 months	Decrease	unclear
	Low GI	22/28	81 (SD 11)	78 (SD 9)	-2 (SD 9)	<0.01	NS				Decrease	
*14668	High GI	11/28	118 (SD 18)	121 (SD 17)	3 (SD 18)	NS		SBP	Ambulatory BP (mm/Hg)	6 months	Decrease	unclear
	Low GI	20/28	128 (SD 14)	115 (SD 12)	-13 (SD 17)	<0.01	<0.05				Decrease	
**14669	High GI	11/28	79 (SD 8)	79 (SD 5)	-1 (SD 5)	NS		DBP	Ambulatory BP (mm/Hg)	6 months	Decrease	unclear
	Low GI	20/28	83 (SD 7)	77 (SD 7)	-5 (SD 5)	<0.01	<0.01				Decrease	
(Jensen *et al.*, 2008) **15030	High GI diet	22/26	76.2 (SD 1.8)	74.1 (SD 1.4)				DBP	Clinic BP (mm/Hg)	10 weeks	Decrease	unclear
	Low GI diet	22/29	72.5 (SD 7.7)	71.5 (SD 7.5)			0.79				Decrease	
*15031	High GI diet	22/26	123.7 (SD 2.3)	118.7 (SD 1.6)				SBP	Clinic BP (mm/Hg)	10 weeks	Decrease	unclear
	Low GI diet	22/29	121.7 (SD 10.6)	118.5 (SD 9.5)			0.68				Decrease	
(Ebbeling *et al.*, 2005) 15513	Low fat diet	12/17	105 (SE 4)		-0.5% (CI -5.3, 4.4)			SBP	Seated (mm/Hg)	6 months	Decrease	unclear
	Low GI diet	11/17	106 (SE 2)		-0.9% (CI -5.9, 4.2)						Decrease	
*15514	Low fat diet	12/17	105 (SE 4)		0.6% (CI -4.1, 5.5)			SBP	Seated (mm/Hg)	1 year	Decrease	unclear
	Low GI diet	11/17	106 (SE 2)		0.2% (CI -4.7, 5.3)						Decrease	
15515	Low fat diet	12/17	63 (SE 2)		0.3% (CI -4.8, 5.6)			DBP	Seated (mm/Hg)	6 months	Decrease	unclear
	Low GI diet	11/17	64 (SE 3)		-2% (CI -7.2, 3.4)						Decrease	
**15516	Low fat diet	12/17	63 (SE 2)		1.4% (CI -4.4, 7.6)			DBP	Seated (mm/Hg)	1 year	Decrease	unclear
	Low GI diet	11/17	64 (SE 3)		-0.3% (CI -6.2, 6)						Decrease	
(Abete *et al.*, 2008) *15544	Higher GI diet	16/16	114 (SD 9)		-3.7% (SD 5.3%)	NS		SBP	Clinic BP Seated (mm/Hg)	8 weeks	Decrease	unclear

Author/ Result ID	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
	Lower GI diet	16/16	115 (SD 11)		-6.5% (SD 8.2%)	NS	0.275				Decrease	
**15545	Higher GI diet	16/16	76 (SD 9)		-5.7% (SD 8.6%)	0.05		DBP	Clinic BP Seated (mm/Hg)	8 weeks	Decrease	unclear
	Lower GI diet	16/16	75 (SD 6)		-7.5% (SD 7.5%)	NS	0.551				Decrease	
(Bellisle *et al.*, 2007) *16047	Control	30/45	118.6 (SE 3)	113.3 (SE 2.8)				SBP	Clinic BP Supine (mm/Hg)	12 weeks	Decrease	unclear
	Low GI	35/51	120.6 (SE 2.5)	118.2 (SE 2.9)							Decrease	
**16048	Control	30/45	72.8 (SE 2.2)	68.4 (SE 2.2)				DBP	Clinic BP Supine (mm/Hg)	12 weeks	Decrease	unclear
	Low GI	35/51	74.1 (SE 1.8)	71.3 (SE 2.2)							Decrease	
(Pereira *et al.*, 2004) *17030	Hypoenergetic low fat diet	17/23	107.5 (SE 2.9)	104.6 (SE 2.35)	-3.1% (SE 1.32%)			SBP	Seated (mm/Hg)	67 days	Decrease	unclear
	Hypoenergetic low GL diet	22/23	110.4 (SE 2.55)	102.3 (SE 2.06)	-6.4% (SE 1.16%)		0.07				Decrease	
**17031	Hypoenergetic low fat diet	17/23	67.8 (SE 2.03)	66.2 (SE 1.8)	-2.5% (SE 1.61%)			DBP	Seated (mm/Hg)	67 days	Decrease	unclear
	Hypoenergetic low GL diet	22/23	69.2 (SE 1.78)	64.2 (SE 1.58)	-6.5% (SE 1.42%)		0.07				Decrease	
(Maki *et al.*, 2007b) 17294	Ad libitum low GL diet	42/43	112.7 (SE 1.6)		-0.6 (SE 2)			SBP	Seated (mm/Hg)	12 weeks	Decrease	unclear
	Low fat, energy restricted	42/43	114.7 (SE 1.6)		-1.2 (SE 2.3)						Decrease	
*17295	Ad libitum low GL diet	42/43	112.7 (SE 1.6)		0.2 (SE 1.7)			SBP	Seated (mm/Hg)	36 weeks	Decrease	unclear
	Low fat, energy restricted	42/43	114.7 (SE 1.6)		0.1 (SE 2.2)						Decrease	
17296	Ad libitum low GL diet	42/43	74.4 (SE 1.5)		-3.3 (SE 1.8)			DBP	Seated (mm/Hg)	12 weeks	Decrease	unclear
	Low fat, energy restricted	42/43	73.4 (SE 1.1)		-0.6 (SE 1.4)						Decrease	
**17297	Ad libitum low GL diet	42/43	74.4 (SE 1.5)		-4.1 (SE 1.8)			DBP	Seated (mm/Hg)	36 weeks	Decrease	unclear
	Low fat, energy restricted	42/43	73.4 (SE 1.1)		-1.6 (SE 1.3)						Decrease	

*This result was used in the meta-analysis for glycaemic index or load diets and SBP
**This result was used in the meta-analysis for glycaemic index or load diets and DBP

Blood pressure and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and continuous blood pressure.

Summary of RCT data

Two RCTs were identified that reported on trials comparing “complex” carbohydrate with either simple carbohydrate or control diets (Vasilaras *et al.*, 2001;Poppitt *et al.*, 2002). There were therefore insufficient trials to formally combine using meta-analysis.

Definitions of “complex” carbohydrates were not provided by the authors of the included studies, although it is generally recognised that “complex” carbohydrates are composed of complex sugar chains (rather than short chains), with these chains acting as an energy store or fibrous structure in plants (Committee on Medical Aspects of Food Policy, 1989). As such, rich food sources include grains, legumes, fruits and vegetables (Shah *et al.*, 1994;Shah *et al.*, 1996;Poppitt *et al.*, 2002). According to the World Health Organisation (WHO) and as stated in (Farchi *et al.*, 1995), intakes of “complex” carbohydrates should make up 50-70% of total carbohydrate intake.

One very small trial found no evidence for any difference between low-fat high-complex carbohydrate diet, low-fat high-simple carbohydrate diet, or control dietary interventions after six months’ follow-up, in terms of either DBP or SBP (Vasilaras *et al.*, 2001). However, the sample size was too small to detect even large effects. The other small trial (Poppitt *et al.*, 2002), which was liable to potential bias through lack of blinding, suggested that DBP decreased in the high-simple carbohydrate group compared with the control diet after six months. It was unclear whether this also applied to SBP because of substantial imbalance at the start of the trial.

Table 2.29 Blood pressure and "complex" carbohydrates: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	p-value difference between groups	Outcome/Assessment method	Result/Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Vasilaras *et al.*, 2001) 15038	Control diet	7/7				DBP	Clinic BP (mm/Hg)	6 months	Increase	unclear
	Low-fat high-complex carbohydrate diet	9/9			NS				Decrease	
	Low-fat, high-simple carbohydrate diet	8/8			NS				No change	
15040	Control diet	7/7				SBP	Clinic BP (mm/Hg)	6 months	Increase	unclear
	Low-fat high-complex carbohydrate diet	9/9			NS				Decrease	
	Low-fat, high-simple carbohydrate diet	8/8			NS				No change	
(Poppitt *et al.*, 2002) 15385	Control	7/15	87 (SD 10)	higher		DBP change	Clinic BP (mm/Hg)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	84 (SD 13)	lower					Decrease	bias
	Low-fat, high-simple carbohydrate diet	13/15	86 (SD 13)	higher	0.05				No change	bias
15384	Control	7/15	132 (SD 14)	higher		SBP change	Clinic BP (mm/Hg)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	136 (SD 17)	lower	0.01				Decrease	bias
	Low-fat, high-simple carbohydrate diet	13/15	138 (SD 22)	lower	0.01				No change	

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

American Association of Cereal Chemists International. AACC members agree on definition of whole grain. 1999. 11-9-2011.

Andersson A, Tengblad S, Karlstrom B, Kamal-Eldin A, Landberg R, Basu S, Aman P, Vessby B (2007) Whole-grain foods do not affect insulin sensitivity or markers of lipid peroxidation and inflammation in healthy, moderately overweight subjects. *J Nutr* **137** (6): 1401-1407

Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin PH, Karanja N (1997) A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med* **336** (16): 1117-1124, doi:10.1056/NEJM199704173361601 [doi]

Appel LJ, Sacks FM, Carey VJ, Obarzanek E, Swain JF, Miller ER, III, Conlin PR, Erlinger TP, Rosner BA, Laranjo NM, Charleston J, McCarron P, Bishop LM, OmniHeart Collaborative Research Group (2005) Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA* **294** (19): 2455-2464

Ascherio A, Hennekens C, Willett WC, Sacks F, Rosner B, Manson J, Witteman J, Stampfer MJ (1996) Prospective study of nutritional factors, blood pressure, and hypertension among US women. *Hypertension* **27** (5): 1065-1072

Ascherio A, Rimm EB, Giovannucci EL, Colditz GA, Rosner B, Willett WC, Sacks F, Stampfer MJ (1992) A prospective study of nutritional factors and hypertension among US men. *Circulation* **86** (5): 1475-1484

Aucott L, Rothnie H, McIntyre L, Thapa M, Waweru C, Gray D (2009) Long-term weight loss from lifestyle intervention benefits blood pressure?: a systematic review. *Hypertension* **54** (4): 756-762, doi:HYPERTENSIONAHA.109.135178 [pii];10.1161/HYPERTENSIONAHA.109.135178 [doi]

Bell LP, Hectorn KJ, Reynolds H, Hunninghake DB (1990) Cholesterol-lowering effects of soluble-fiber cereals as part of a prudent diet for patients with mild to moderate hypercholesterolemia. *Am J Clin Nutr* **52** (6): 1020-1026

Bellisle F, Dalix AM, De Assis MA, Kupek E, Gerwig U, Slama G, Oppert JM (2007) Motivational effects of 12-week moderately restrictive diets with or without special attention to the Glycaemic Index of foods. *Br J Nutr* **97** (4): 790-798

Birketvedt GS, Aaseth J, Florholmen JR, Ryttig K (2000) Long-term effect of fibre supplement and reduced energy intake on body weight and blood lipids in overweight subjects. *Acta Medica* **43** (4): 129-132

Black RN, Spence M, McMahon RO, Cuskelly GJ, Ennis CN, McCance DR, Young IS, Bell PM, Hunter SJ (2006) Effect of eucaloric high- and low-sucrose diets with identical macronutrient

profile on insulin resistance and vascular risk: a randomized controlled trial. *Diabetes* **55** (12): 3566-3572

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

Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DA (2003) A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab* **88** (4): 1617-1623

Brehm BJ, Spang SE, Lattin BL, Seeley RJ, Daniels SR, D'Alessio DA (2005) The role of energy expenditure in the differential weight loss in obese women on low-fat and low-carbohydrate diets. *J Clin Endocrinol Metab* **90** (3): 1475-1482

British Cardiac Society, British Hypertension Society, Diabetes UK, HEART UK, Primary Care Cardiovascular Society, The Stroke Association (2005) JBS 2: Joint British Societies' guidelines on prevention of cardiovascular disease in clinical practice. *Heart* **91** (suppl 5): v1-v52

Cairella G, Cairella M, Marchini G (1995) Effect of dietary fibre on weight correction after modified fasting. *Eur J Clin Nutr* **49** Suppl-7

Claessens M, van Baak MA, Monsheimer S, Saris WHM (2009) The effect of a low-fat, high-protein or high-carbohydrate ad libitum diet on weight loss maintenance and metabolic risk factors. *Int J Obes* **33** (3): 296-304

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

Collins R, Peto R, MacMahon S, Hebert P, Fiebach NH, Eberlein KA, Godwin J, Qizilbash N, Taylor JO, Hennekens CH (1990) Blood pressure, stroke, and coronary heart disease. Part 2, Short-term reductions in blood pressure: overview of randomised drug trials in their epidemiological context. *Lancet* **335** (8693): 827-838, doi:0140-6736(90)90944-Z [pii]

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

Committee on Medical Aspects of Food Policy (1991) *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*. HMSO: London

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

Davy BM, Melby CL, Beske SD, Ho RC, Davrath LR, Davy KP (2002) Oat consumption does not affect resting casual and ambulatory 24-h arterial blood pressure in men with high-normal blood pressure to stage I hypertension. *J Nutr* **132** (3): 394-398

de Luis DA, Aller R, Izaola O, Gonzalez SM, Conde R (2009a) Modulation of insulin concentrations and metabolic parameters in obese patients by -55CT polymorphism of the UCP3 gene secondary to two hypocaloric diets. *Horm Metab Res* **41** (1): 62-66

de Luis DA, Aller R, Izaola O, Sagrado MG, Conde R (2008) Influence of Ala54Thr polymorphism of fatty acid-binding protein 2 on weight loss and insulin levels secondary to two hypocaloric diets: a randomized clinical trial. *Diabetes Res Clin Pract* **82** (1): 113-118

de Luis DA, Sagrado MG, Conde R, Aller R, Izaola O (2009b) The effects of two different hypocaloric diets on glucagon-like peptide 1 in obese adults, relation with insulin response after weight loss. *J Diabetes Complications* **23** (4): 239-243

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

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

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, 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

Farchi G, Fidanza F, Grossi P, Lancia A, Mariotti S, Menotti A (1995) Relationship between eating patterns meeting recommendations and subsequent mortality in 20 years. *Eur J Clin Nutr* **49** (6): 408-419

Flint AJ, Hu FB, Glynn RJ, Jensen MK, Franz M, Sampson L, Rimm EB (2009) Whole grains and incident hypertension in men. *Am J Clin Nutr* **90** (3): 493-498

Food Standards Agency. Eat well. Be well. Heping you make healthier choices [accessed September 2011]. 27-9-2010.

Forman JP, Choi H, Curhan GC (2009) Fructose and vitamin C intake do not influence risk for developing hypertension. *J Am Soc Nephrol* **20** (4): 863-871

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

Golay A, Allaz AF, Ybarra J, Bianchi P, Saraiva S, Mensi N, Gomis R, de TN (2000) Similar weight loss with low-energy food combining or balanced diets. *Int J Obes* **24** (4): 492-496

He J, Streiffer RH, Muntner P, Krousel-Wood MA, Whelton PK (2004) Effect of dietary fiber intake on blood pressure: a randomized, double-blind, placebo-controlled trial. *J Hypertens* **22** (1): 73-80

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

Howard BV, Van HL, Hsia J, Manson JE, Stefanick ML, Wassertheil-Smoller S, Kuller LH, LaCroix AZ, Langer RD, Lasser NL, Lewis CE, Limacher MC, Margolis KL, Mysiw WJ, Ockene JK, Parker LM, Perri MG, Phillips L, Prentice RL, Robbins J, Rossouw JE, Sarto GE, Schatz IJ, Snetselaar LG, Stevens VJ, Tinker LF, Trevisan M, Vitolins MZ, Anderson GL, Assaf AR, Bassford T, Beresford SA, Black HR, Brunner RL, Brzyski RG, Caan B, Chlebowski RT, Gass M, Granek I, Greenland P, Hays J, Heber D, Heiss G, Hendrix SL, Hubbell FA, Johnson KC, Kotchen JM (2006b) Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA* **295** (6): 655-666

Jacobs DR, Jr., Meyer KA, Kushi LH, Folsom AR (1998) Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. *Am J Clin Nutr* **68** (2): 248-257

Jensen L, Sloth B, Krog-Mikkelsen I, Flint A, Raben A, Tholstrup T, Brunner N, Astrup A (2008) A low-glycemic-index diet reduces plasma plasminogen activator inhibitor-1 activity, but not tissue inhibitor of proteinases-1 or plasminogen activator inhibitor-1 protein, in overweight women. *Am J Clin Nutr* **87** (1): 97-105

Kanda A, Hoshiyama Y, Kawaguchi T (1999) Association of lifestyle parameters with the prevention of hypertension in elderly Japanese men and women: a four-year follow-up of normotensive subjects. *Asia Pac J Public Health* **11** (2): 77-81

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

Kannel WB (2000) Risk stratification in hypertension: new insights from the Framingham Study. *Am J Hypertens* **13** (1 Pt 2): 3S-10S, doi:S0895706199002526 [pii]

Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J (2005) Global burden of hypertension: analysis of worldwide data. *The Lancet* **365** (9455): 217-223

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

Keogh JB, Brinkworth GD, Noakes M, Belobrajdic DP, Buckley JD, Clifton PM (2008) Effects of weight loss from a very-low-carbohydrate diet on endothelial function and markers of cardiovascular disease risk in subjects with abdominal obesity. *Am J Clin Nutr* **87** (3): 567-576

Klag MJ, Whelton PK, Appel LJ (1990) Effect of age on the efficacy of blood pressure treatment strategies. *Hypertension* **16** (6): 700-705

Kleemola P, Puska P, Vartiainen E, Roos E, Luoto R, Ehnholm C (1999) The effect of breakfast cereal on diet and serum cholesterol: a randomized trial in North Karelia, Finland. *Eur J Clin Nutr* **53** (9): 716-721

Landin K, Holm G, Tengborn L, Smith U (1992) Guar gum improves insulin sensitivity, blood lipids, blood pressure, and fibrinolysis in healthy men. *Am J Clin Nutr* **56** (6): 1061-1065

Lee YP, Mori TA, Puddey IB, Sipsas S, Ackland TR, Beilin LJ, Hodgson JM (2009) Effects of lupin kernel flour-enriched bread on blood pressure: a controlled intervention study. *Am J Clin Nutr* **89** (3): 766-772

Lehtimaki T, Metso S, Ylitalo R, Rontu R, Nikkila M, Wuolijoki E, Ylitalo P (2005) Microcrystalline chitosan is ineffective to decrease plasma lipids in both apolipoprotein E epsilon 4 carriers and non-carriers: A long-term placebo-controlled trial in hypercholesterolaemic volunteers. *Basic Clin Pharmacol Toxicol* **97** (2): 98-103

Leidy HJ, Carnell NS, Mattes RD, Campbell WW (2007) Higher protein intake preserves lean mass and satiety with weight loss in pre-obese and obese women. *Obesity* **15** (2): 421-429

Lewington S, Clarke R, Qizilbash N, Peto R, Collins R (2002) Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet* **360** (9349): 1903-1913, doi:S0140673602119118 [pii]

Ley SJ, Metcalf PA, Scragg RKR, Swinburn BA (2004) Long-term effects of a reduced fat diet intervention on cardiovascular disease risk factors in individuals with glucose intolerance. *Diabetes Res Clin Pract* **63** (2): 103-112

Lovejoy JC, Bray GA, Lefevre M, Smith SR, Most MM, Denkins YM, Volaufova J, Rood JC, Eldridge AL, Peters JC, Ole' S (2003) Consumption of a controlled low-fat diet containing olestra for 9 months improves health risk factors in conjunction with weight loss in obese men: the Ole' Study. *Int J Obes* **27** (10): 1242-1249

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

MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, Abbott R, Godwin J, Dyer A, Stamler J (1990) Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet* **335** (8692): 765-774, doi:0140-6736(90)90878-9 [pii]

Maki KC, Galant R, Samuel P, Tesser J, Witchger MS, Ribaya-Mercado JD, Blumberg JB, Geohas J (2007a) Effects of consuming foods containing oat beta-glucan on blood pressure, carbohydrate metabolism and biomarkers of oxidative stress in men and women with elevated blood pressure. *Eur J Clin Nutr* **61** (6): 786-795

Maki KC, Rains TM, Kaden VN, Raneri KR, Davidson MH (2007b) Effects of a reduced-glycemic-load diet on body weight, body composition, and cardiovascular disease risk markers in overweight and obese adults. *Am J Clin Nutr* **85** (3): 724-734

Marett R, Slavin JL (2004) No long-term benefits of supplementation with arabinogalactan on serum lipids and glucose. *J Am Diet Assoc* **104** (4): 636-639

Meckling KA, O'Sullivan C, Saari D (2004) Comparison of a low-fat diet to a low-carbohydrate diet on weight loss, body composition, and risk factors for diabetes and cardiovascular disease in free-living, overweight men and women. *J Clin Endocrinol Metab* **89** (6): 2717-2723

Meckling KA, Sherfey R (2007) A randomized trial of a hypocaloric high-protein diet, with and without exercise, on weight loss, fitness, and markers of the Metabolic Syndrome in overweight and obese women. *Appl Physiol Nutr Metab* **32** (4): 743-752

Morris JN, Marr JW, Clayton DG (1977) Diet and heart: a postscript. *Br Med J* **2** (6098): 1307-1314

National Institute of Clinical Excellence. NICE clinical guideline 34. Essential hypertension: managing adult patients in primary care (partial update of NICE clinical guideline 18). 2004. Centre for Health Services Research Report No 111., Royal College of Physicians.

Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM (2003) Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension* **42** (5): 878-884

Noakes M, Foster PR, Keogh JB, James AP, Mamo JC, Clifton PM (2006) Comparison of isocaloric very low carbohydrate/high saturated fat and high carbohydrate/low saturated fat diets on body composition and cardiovascular risk. *Nutr Metab* **3** 7

O'Brien KD, Brehm BJ, Seeley RJ, Bean J, Wener MH, Daniels S, D'Alessio DA (2005) Diet-induced weight loss is associated with decreases in plasma serum amyloid a and C-reactive protein independent of dietary macronutrient composition in obese subjects. *J Clin Endocrinol Metab* **90** (4): 2244-2249

Olendzki BC, Ma Y, Schneider KL, Merriam P, Culver AL, Ockene IS, Pagoto S (2009) A simple dietary message to improve dietary quality: Results from a pilot investigation. *Nutrition* **25** (7-8): 736-744

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

Pereira MA, Swain J, Goldfine AB, Rifai N, Ludwig DS (2004) Effects of a low-glycemic load diet on resting energy expenditure and heart disease risk factors during weight loss. *JAMA* **292** (20): 2482-2490

Philippou E, Bovill-Taylor C, Rajkumar C, Vampa ML, Ntatsaki E, Brynes AE, Hickson M, Frost GS (2009) Preliminary report: the effect of a 6-month dietary glycemic index manipulation in addition to healthy eating advice and weight loss on arterial compliance and 24-hour ambulatory blood pressure in men: a pilot study. *Metabolism* **58** (12): 1703-1708

Phillips SA, Jurva JW, Syed AQ, Syed AQ, Kulinski JP, Pleuss J, Hoffmann RG, Gutterman DD (2008) Benefit of low-fat over low-carbohydrate diet on endothelial health in obesity. *Hypertension* **51** (2): 376-382

Poppitt SD, Keogh GF, Prentice AM, Williams DE, Sonnemans HM, Valk EE, Robinson E, Wareham NJ (2002) Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome. *Am J Clin Nutr* **75** (1): 11-20

Raben A, Vasilaras TH, Moller AC, Astrup A (2002) Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr* **76** (4): 721-729

Rigaud D, Ryttig KR, Angel LA, Apfelbaum M (1990) Overweight treated with energy restriction and a dietary fibre supplement: a 6-month randomized, double-blind, placebo-controlled trial. *Int J Obes* **14** (9): 763-769

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

Saltzman E, Das SK, Lichtenstein AH, Dallal GE, Corrales A, Schaefer EJ, Greenberg AS, Roberts SB (2001) An oat-containing hypocaloric diet reduces systolic blood pressure and improves lipid profile beyond effects of weight loss in men and women. *J Nutr* **131** (5): 1465-1470

Schroeder TE, Hawkins SA, Hyslop D, Vallejo AF, Jensky NE, Wiswell RA (2007) Longitudinal change in coronary heart disease risk factors in older runners. *Age Ageing* **36** (1): 57-62

Schwab U, Louheranta A, Torronen A, Uusitupa M (2006) Impact of sugar beet pectin and polydextrose on fasting and postprandial glycemia and fasting concentrations of serum total and lipoprotein lipids in middle-aged subjects with abnormal glucose metabolism. *Eur J Clin Nutr* **60** (9): 1073-1080

Sciarrone SE, Strahan MT, Beilin LJ, Burke V, Rogers P, Rouse IR (1993) Ambulatory blood pressure and heart rate responses to vegetarian meals. *J Hypertens* **11** (3): 277-285

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

Shah M, Mcgovern P, French S, Baxter J (1994) Comparison of A Low-Fat, Ad-Libitum Complex-Carbohydrate Diet with A Low-Energy Diet in Moderately Obese Women. *Am J Clin Nutr* **59** (5): 980-984

Smith KN, Queenan KM, Thomas W, Fulcher RG, Slavin JL (2008) Physiological effects of concentrated barley beta-glucan in mildly hypercholesterolemic adults. *J Am Coll Nutr* **27** (3): 434-440

Stamler J, Liu K, Ruth KJ, Pryer J, Greenland P (2002) Eight-year blood pressure change in middle-aged men: relationship to multiple nutrients. *Hypertension* **39** (5): 1000-1006

Steffen LM, Kroenke CH, Yu X, Pereira MA, Slattery ML, Van HL, Gross MD, Jacobs DR, Jr. (2005) Associations of plant food, dairy product, and meat intakes with 15-y incidence of elevated blood pressure in young black and white adults: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Am J Clin Nutr* **82** (6): 1169-1177

Surwit RS, Feinglos MN, McCaskill CC, Clay SL, Babyak MA, Brownlow BS, Plaisted CS, Lin PH (1997) Metabolic and behavioral effects of a high-sucrose diet during weight loss. *Am J Clin Nutr* **65** (4): 908-915

Swain JF, Rouse IL, Curley CB, Sacks FM (1990) Comparison of the effects of oat bran and low-fiber wheat on serum lipoprotein levels and blood pressure. *N Engl J Med* **322** (3): 147-152

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

Uen S, Fimmers R, Brieger M, Nickenig G, Mengden T (2009) Reproducibility of wrist home blood pressure measurement with position sensor and automatic data storage. *BMC Cardiovascular Disorders* **9** (1): 20, doi:10.1186/1471-2261-9-20

United States FDA. FDA provides guidance on "Whole Grain" for manufacturers [accessed September 2001]. 2006.

USDA. Dietary Guidelines for Americans;2010. 2010.

Vasilaras TH, Raben A, Astrup A (2001) Twenty-four hour energy expenditure and substrate oxidation before and after 6 months' ad libitum intake of a diet rich in simple or complex carbohydrates or a habitual diet. *Int J Obes* **25** (7): 954-965

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

Wang L, Gaziano JM, Liu S, Manson JE, Buring JE, Sesso HD (2007) Whole- and refined-grain intakes and the risk of hypertension in women. *Am J Clin Nutr* **86** (2): 472-479

Whelton PK (1994) Epidemiology of hypertension. *The Lancet* **344** (8915): 101-106

Wolever TM, Mehling C (2002) High-carbohydrate-low-glycaemic index dietary advice improves glucose disposition index in subjects with impaired glucose tolerance. *Br J Nutr* **87** (5): 477-487

Wood RJ, Fernandez ML, Sharman MJ, Silvestre R, Greene CM, Zern TL, Shrestha S, Judelson DA, Gomez AL, Kraemer WJ, Volek JS (2007) Effects of a carbohydrate-restricted diet with and without supplemental soluble fiber on plasma low-density lipoprotein cholesterol and other clinical markers of cardiovascular risk. *Metabolism* **56** (1): 58-67

Wright, J. M. and Musini, V. M. First-line drugs for hypertension". *Cochrane Database Syst Rev* (3): CD001841. 2009.

Wursch P, Pi-Sunyer FX (1997) The role of viscous soluble fiber in the metabolic control of diabetes. A review with special emphasis on cereals rich in beta-glucan. *Diabetes Care* **20** (11): 1774-1780