

Chapter 2: Markers of Cardiovascular Disease

The Hyperlipidaemias and Blood Lipids

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Background

Lipids are a diverse group of natural chemicals, which include fats (such as triacylglycerol (TAG)) and sterols (such as cholesterol). They have many important physiological functions including membrane synthesis and maintenance of energy storage, absorption of fat-soluble vitamins, and cell signalling. They are also strongly linked with the development and consequences of cardiovascular disease (CVD) - elevated levels of certain lipid fractions are associated with stroke, peripheral vascular disease and heart attack (Lewington *et al.*, 2007).

Lipoproteins

Within the circulation, lipids such as cholesterol, triacylglycerol (TAG) and phospholipids are associated with proteins (apolipoproteins) which facilitate their transport and metabolism. These lipid-containing particles are defined as lipoproteins. There are a number of different lipoproteins which differ in size, composition and function and include chylomicrons, very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), low density lipoprotein (LDL), and high density lipoprotein (HDL). The liver and intestine are the primary sites of lipoprotein synthesis with VLDL being synthesised by the liver and chylomicrons by the intestine and precursor HDL particles synthesised in both liver and intestine. The mature forms of LDL, IDL and HDL particles are not secreted directly from either the liver or intestine, but are produced by metabolic processes within the circulation. There are a large number of apolipoproteins including: apolipoprotein B which is associated with chylomicrons, VLDL, IDL and LDL particles; apolipoprotein A-1 which is primarily associated with HDL particles and lipoprotein (a) which is bound to apolipoprotein B on LDL-like particles.

LDL particles are the principal carriers of cholesterol and contain cholesterol esters within their core. Elevated concentrations of these lipoproteins, and total cholesterol, have been associated with increased risk of cardiovascular disease. HDL particles principally carry cholesterol esters from the tissues back to the liver. This process is defined as reverse cholesterol transport with high circulating HDL cholesterol concentrations associated with reduced cardiovascular risk. Chylomicrons and VLDL are the largest lipoprotein particles and are the major transporters of TAG (which contains a glycerol backbone and three associated fatty acids). IDL contain appreciable amounts of both TAG and cholesterol esters. Due to the influence of TAG-rich lipoprotein on remodelling of other lipoproteins such as LDL and HDL, elevated TAG is also considered as an independent risk factor for cardiovascular disease. Free or non-esterified fatty acids (NEFA) are transported in the circulation associated with plasma proteins.

Pathophysiology

The underlying pathology of cardiovascular disease is a combination of atherosclerosis and thrombosis. Atherosclerosis is a condition in which the arterial lining is thickened in places by raised plaques as a result of excessive accumulation of modified lipids, and of the proliferation and migration of smooth muscle cells from deeper layers of the arterial wall (Libby *et al.*, 2011; Badimon *et al.*, 2011). Typically these plaques develop at the point of minor injury in the arterial wall, initiating a cascade of chemotactic and cytokine responses, which increase the internalisation of LDL particles. The LDL particles integrate into the extracellular matrix and become oxidised. This is associated with an inflammatory response which increases macrophage infiltration and precipitates vascular remodelling. At a later stage, the plaque becomes sclerosed and calcified. Formation of an atherosclerotic plaque can partially occlude one or more of the arteries, mainly the coronary and cerebral arteries. However it is the rupture of this structure and its consequences that are linked to the clinically apparent endpoints of stroke, heart attack and acute limb ischaemia.

As such, there is a strong association between elevated levels of LDLc, apolipoprotein B and less so lipoprotein (a) and cardiovascular disease (Lewington *et al.*, 2007) and a reciprocal relationship between HDL and apolipoprotein A-1 concentrations and cardiovascular disease (Gordon and Rifkind, 1989). Elevated TAG concentrations are also linked with cardiovascular disease (Miller *et al.*, 2011; Goldberg *et al.*, 2011).

High circulating TAG concentrations are often associated with elevated small dense LDL and low HDL cholesterol concentrations due to remodelling of lipoproteins within the circulation through a process called neutral lipid exchange. For this reason some suggest that the HDL:TAG and LDL:TAG ratios are better predictors of cardiovascular outcomes than TAG concentrations alone (Ballantyne and Hoogeveen, 2003). Various other lipoprotein ratios are thought to reflect CVD risk. These include the ratio of TC:HDL cholesterol, LDL:HDL and non-HDL:HDL. Reductions in these ratios indicate a beneficial effect in terms of CVD risk. For example, Rader *et al.* demonstrated that a 1% decrease in the TC:HDL cholesterol ratio was associated with a 1.3% reduction in CVD risk (Rader *et al.*, 2003). A desirable ratio of TC:HDL cholesterol is thought to be 4.5 or less.

According to the National Cholesterol Education Program, for men a low HDL cholesterol is defined as a level less than 1.03mmol/L, and for women it is low when it is below 1.3mmol/L (Expert Panel on Detection, 2001; Mosca *et al.*, 2004). A normal TAG level is defined as less than 1.7 mmol/L (Miller *et al.*, 2011).

There is both a genetic (primary) and environmental (secondary) component to hyperlipidaemia. The genetic causes of hyperlipidaemia typically are due to mutations that result in abnormal clearance of lipids. Secondary causes of hyperlipidaemia are more common and include a sedentary lifestyle, diabetes and the consumption of saturated fat, trans (polyunsaturated) fat and

cholesterol. Diabetes is particularly important and can be characterised by elevated TAG, low HDLc and high LDLc.

Modification of the lipid profile both for primary prevention and secondary prevention of cardiovascular disease offers the potential for significant reductions in risk of death and cardiovascular disease (LaRosa *et al.*, 1999;Thavendiranathan *et al.*, 2006). Whilst dietary modification of hyperlipidaemia is important to lower overall cardiovascular risk at a population level and appears to be associated with an improved lipid profile (Huang *et al.*, 2011;Jenkins *et al.*, 2003;Stone *et al.*, 2005), typically pharmacological therapies are used, particularly if dietary measures fail to reach the recommended targets (National Institute for Health and Clinical Excellence, 2011;Shepherd, 2001).

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) that concerned the relationship between dietary carbohydrates and hyperlipidaemias and blood lipids. 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 used in the current review.

Papers from COMA reports that did not meet inclusion criteria

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

Table 2.39 Previous studies in COMA reports*: excluded studies

Authors, Year	Intervention description	Intervention duration/ follow up	Exclusion code that would be applied in this review	Exclusion detail
(Burr <i>et al.</i> , 1989)	1) Fat advice 2) Fish advice 3) Fibre advice	2 years	6	Subjects did not fit the definition of 'healthy' – all were diagnosed with acute myocardial infarction.
(Crapo and Kolterman, 1984)	1) Sucrose diet 2) Fructose diet	2 weeks	2	Subjects were not randomly allocated to groups
(Cybulska and Naruszewicz, 1982)	1) Usual diet + fructose 2) No sugar diet	28 days	2	Subjects were not randomly allocated to groups
(Edington <i>et al.</i> , 1989)	1) Low-fat, high-fibre diet (+ consumption of 9 eggs per week) 2) Low-fat, high-fibre diet	3 months	2	Subjects were not randomly allocated to groups.
(Grande <i>et al.</i> , 1965)	1) Sucrose diet 2) Bean diet 3) Sucrose diet + pectin 4) Bean diet + pectin 5) Bread and potato diet 6) Sucrose diet + cellulose 7) Bread and potato diet + cellulose 8) Sucrose and soybean protein diet	7 days	2	Subjects were not randomly allocated to groups.
(Grundy, 1986)	1) High saturated fat diet 2) High monounsaturated fat diet 3) Low fat diet	4 weeks	6	Subjects did not fit the definition of 'healthy' – total cholesterol averaged 6.5mmol/L at baseline.
(Huttunen <i>et al.</i> , 1976)	1) Usual diet + sucrose 2) Usual diet + xylitol 3) Usual diet + fructose	2 years	2	Subjects were not randomly allocated to groups.
(Lewis <i>et al.</i> , 1981)	1) Western diet 2) Fat-modified diet 3) Fat-modified diet + fruit, vegetable and cereal fibre 4) Diet providing 40% energy from fat; P/S ratio 1.0; + supplemented with fibre	5 weeks	2	Subjects were not randomly allocated to groups.
(Lock <i>et al.</i> , 1980)	1) Usual diet + sucrose 2) Usual diet + dried glucose syrup	2 years	2	Subjects were not randomly allocated to groups.
(Lopez <i>et al.</i> , 1966)	Not applicable	Not applicable	1	The publication was a review/ not original research.
(Macdonald, 1967)	1) Sucrose-cream diet 2) Sucrose-sunflower oil diet 3) Glucose-cream diet 4) Glucose-sunflower oil diet	5 days	2	Subjects were not randomly allocated to groups.
(Mann and Truswell, 1972)	1) Basal diet 2) Basal + starch diet 3) Basal + sucrose diet	14 days	6	Subjects did not fit the definition of 'healthy' – all had been admitted to hospital with non-metabolic conditions such as cerebral vascular accident and nerve palsy.
(Mann <i>et al.</i> , 1974)	1) Basal diet 2) Basal diet + sucrose replaced by starch 3) Basal diet + starch removed	14 days	2	Subjects were not randomly allocated to groups.
(McGandy <i>et al.</i> , 1967a) / (McGandy <i>et al.</i> , 1967b)	Not applicable	Not applicable	1	The publication was a review/ not original research.
(Mensink and Katan, 1987)	1) Carbohydrate-rich diet 2) Olive-oil rich diet	36 days	2	Subjects were not randomly allocated to groups.

Authors, Year	Intervention description	Intervention duration/ follow up	Exclusion code that would be applied in this review	Exclusion detail
(Peterson <i>et al.</i> , 1986)	1) Control diet 2) Sucrose diet	6 weeks	6	Subjects did not fit the definition of 'healthy' – all had diabetes.
(Reiser <i>et al.</i> , 1979)	1) Sucrose diet 2) Starch diet	6 weeks	2	Subjects were not randomly allocated to groups.
(Renaud <i>et al.</i> , 1986)	Not applicable	Not applicable	2	The study was not a randomised trial or cohort/prospective study – it was a cross-sectional survey.
(Rifkind <i>et al.</i> , 1966)	1) Sugar-restricted diet	10 weeks	2	Subjects were not randomly allocated to groups.
(Rosenthal <i>et al.</i> , 1985)	1) High-complex-carbohydrate, high-fibre, low-fat, low-cholesterol diet	26 days	2	The study did not have a 'control' group and all subjects received the same intervention.
(Thornton <i>et al.</i> , 1983)	1) Usual diet + refined carbohydrate foods 2) Usual diet + wholegrain foods	6 weeks	6	Subjects did not fit the definition of 'healthy' – all had radiolucent gall stones.
(Vinik and Jenkins, 1988)	Not applicable	Not applicable	1	The publication was a review/ not original research.
(Weisweiler <i>et al.</i> , 1985)	1) Reference diet 2) Polyunsaturated diet 3) Low fat, polyunsaturated diet	6 weeks	2	The three intervention diets were not randomly delivered.
(Werner <i>et al.</i> , 1984)	1) Usual diet + sucrose 2) Usual diet + saccharine	6 weeks	6	Subjects did not fit the definition of 'healthy' – all had radiolucent gall stones.
(Yudkin <i>et al.</i> , 1980)	1) High sugar diet	3 weeks	2	Subjects were not randomly allocated to groups.

Papers from COMA reports that met inclusion criteria

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

Table 2.40 Previous randomised controlled trials (RCT) in COMA reports*: included studies

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

Authors, Study Name	Subject inclusion criteria	Characteristics of participants	Trial Design (washout duration)	Intervention duration	Intervention Style	Total number of participants	Intervention description
(Reiser <i>et al.</i> , 1981)	Generally healthy Exaggerated insulin response to glucose load	US 50% Male Age: (38.6) males (35.1) females	Crossover	6 weeks	All food provided	24	1) Diet containing 5% of total calories as sucrose 2) Diet containing 18% of total calories as sucrose 3) Diet containing 33% of total calories as sucrose

Mann *et al.* (Mann *et al.*, 1970) assessed the effect of a low sugar diet compared with both a reduced starch diet and a control diet on serum lipids and weight loss in men. Serum cholesterol and serum triacylglycerol were measured at two, six, 10-18, 22 weeks as well as one month following the intervention. Subjects in the low sugar diet group experienced a statistically significant reduction in triacylglycerol (statistical significance level not reported) from baseline, whilst the other two groups did not. Furthermore the authors highlight that such a reduction in triacylglycerol in the low sugar diet group may in part be attributed to concurrent weight loss and therefore not necessarily due to the dietary intervention alone (Mann *et al.*, 1970). Serum cholesterol did not statistically significantly differ amongst groups during the study.

Along similar lines, Reiser *et al.* (Reiser *et al.*, 1981) tested the effects of dietary sucrose on blood lipids in a sample of carbohydrate-sensitive subjects (n=24). Triacylglycerol, total, HDL and LDL cholesterol and TC:HDL cholesterol ratio were measured before and once a week during the intervention periods. The authors reported a statistically significant increase in triacylglycerol as the level of sucrose increased, although this was only apparent in males and not females. Total cholesterol, LDL cholesterol and HDL cholesterol also statistically significantly increased as the sucrose content of the diet increased (Reiser *et al.*, 1981). The ratio of TC:HDL cholesterol, on the other hand, decreased when males were fed the 33% sucrose diet compared with the two remaining diets. No change in the ratio was observed in females.

Table 2.41 Previous cohort study in COMA reports*: included study

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

One cohort study of healthy middle aged men investigated intakes of total carbohydrate, sugar and dietary fibre (from fruit, vegetables, potatoes, pulses, wholegrains and cereal foods) and total cholesterol. No association between total cholesterol and the nutrients of interest were observed.

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

Summary of the evidence base

This review includes the following outcomes: incident hyperlipidaemias, total cholesterol, HDL cholesterol, LDL cholesterol, triacylglycerol (sometimes referred to as triglycerides within papers, however within this review the term triacylglycerol (TAG) has been used), non-esterified fatty acids, total cholesterol:HDL ratio (TC:HDL), LDL:HDL cholesterol ratio, TAG:HDL ratio, apolipoproteins A1 and B and lipoprotein (a).

Cohort studies

A description of the cohorts that provided data concerning dietary carbohydrates and blood lipids is provided in table 2.42.

Overall, nine papers reported data on seven cohort studies. Of these, four recruited adults as participants (de Castro *et al.*, 2006; Schroeder *et al.*, 2007; Oxlund and Heitmann, 2006; Iribarren *et al.*, 1997; Ludwig *et al.*, 1999; Archer *et al.*, 1998; Dhingra *et al.*, 2007) and the remaining three either used children aged 9-10 years (Boreham *et al.*, 1999) or adolescents aged 12-15 years at baseline (Twisk *et al.*, 1997; Albertson *et al.*, 2009).

Studies were conducted in The Netherlands (1), Denmark (1) and Northern Ireland (1), although the majority were carried out in the USA (6). All cohorts, bar one, were mixed gender. The exception was the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009), which studied only females.

Dietary assessment was, on the whole, achieved through food frequency questionnaires (FFQ); although dietary histories (Twisk *et al.*, 1997; Oxlund and Heitmann, 2006), food diaries (Schroeder *et al.*, 2007; Albertson *et al.*, 2009) and a dietary recall (Boreham *et al.*, 1999) were also employed in some cohorts.

Length of follow-up ranged from a minimum of 4 years to a maximum of 14 years in the Amsterdam Growth and Health Study (Twisk *et al.*, 1997). Using the longest length of follow-up in multiple papers, the average follow-up duration was 8 years.

Initial cohort sizes ranged from 91 participants in the Middle-aged Runners Study (Schroeder *et al.*, 2007) to 8,997 participants in The Framingham Heart Study (Dhingra *et al.*, 2007).

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. Please interpret observational data with caution: the bias could be large in size, and act in either direction, either towards or away from the null.

Table 2.42 Characteristics of cohort studies (studies with grey shading are on children or adolescents)

Cohort Name	Authors/ Reference	Population characteristics	Recruitment of participants	Dietary assessment methods	Length of follow up (years)	Initial cohort size	Losses to follow-up (%)
Amsterdam Growth and Health Study	(Twisk <i>et al.</i> , 1997)	Adolescents/ young adults Mean age: 13 (12-15) % Male: 46 Country: The Netherlands Ethnicity: Not reported	Community cohort	Diet was assessed once using a dietary history. This method was reported as validated.	14	233	Not reported
Japanese-Brazilian Diabetes Study Group	(de Castro <i>et al.</i> , 2006)	First and second generation Japanese Brazilians Mean age 57 (40-79) % Male: 48 Country: Brazil Ethnicity: Japanese	Community cohort	Diet assessed with validated FFQs	7	647	19.7%
Middle-aged Runners Study	(Schroeder <i>et al.</i> , 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	91	Not reported
MONICA	(Oxlund and Heitmann, 2006)	Mean age: 45 (30-60) %Male: 48.9 Country: Denmark Ethnicity: Primarily White	Population-based cohort	Diet was assessed using a dietary history interview, administered once by a registered dietician. Average daily intake was based on intakes during the previous month. This study was not reported to be validated.	6	3608	Not reported

Cohort Name	Authors/ Reference	Population characteristics	Recruitment of participants	Dietary assessment methods	Length of follow up (years)	Initial cohort size	Losses to follow-up (%)
National Heart, Lung, and Blood Institute Growth and Health Study	(Albertson <i>et al.</i> , 2009)	10-year longitudinal study of girls aged 9-10 at baseline from locations in Berkeley, Cincinnati and Washington areas. Mean age: 9-10 %Male: 0 Country: USA Ethnicity: Multi-Ethnic	Community cohort	Diet was assessed from a 3-day food diary (2 weekdays and 1 weekend day) administered 8 times and it was reported to be validated.	10	2379	Not reported
The CARDIA Study	(Iribarren <i>et al.</i> , 1997)	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.	7	5115	19
	(Ludwig <i>et al.</i> , 1999)	As above	As above		10	5115	Not reported
	(Archer <i>et al.</i> , 1998)	As above	As above		7	5115	Not reported

Cohort Name	Authors/ Reference	Population characteristics	Recruitment of participants	Dietary assessment methods	Length of follow up (years)	Initial cohort size	Losses to follow-up (%)
The Framingham Heart Study	(Dhingra <i>et al.</i> , 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	8997	Not reported
The Northern Ireland Young Hearts Project	(Boreham <i>et al.</i> , 1999)	Representative sample of adolescents from Northern Ireland. Mean age: 12-15 %Male: 49.5 Country: Northern Ireland Ethnicity: Primarily White	Population-based cohort	Diet was assessed by dietary recall over the previous month and it was administered twice and reported to be validated.	4	509	1.7

Trial design

One hundred and forty one papers from 132 studies provided data on the relationship between blood lipids and aspects of dietary carbohydrate. Data from one study are not included in the tables or the meta-analysis due to convincing evidence of poor study quality (Singh *et al.*, 1992).

Details concerning the design, participants, duration and nature of the interventions are included in Table 2.43. Twenty four studies employed a crossover design (Appel *et al.*, 2005;Furtado *et al.*, 2008;Sharman *et al.*, 2004;Dreon *et al.*, 1994;Campos *et al.*, 1995;Clevidence *et al.*, 1992;Ginsberg *et al.*, 1998;Segal-Isaacson *et al.*, 2004;Turley *et al.*, 1998;Haskell *et al.*, 1992;Landin *et al.*, 1992;Ryle *et al.*, 1990;Garcia *et al.*, 2007;Garcia *et al.*, 2006;Mee and Gee, 1997;Lehtimäki *et al.*, 2005;Kleemola *et al.*, 1999;Andersson *et al.*, 2007;Kesaniemi *et al.*, 1990;Swain *et al.*, 1990;Bantle *et al.*, 2000;Black *et al.*, 2006;Davidson *et al.*, 1998;Panlasigui *et al.*, 2003;Peterson and Jovanovic-Peterson, 1995;Letexier *et al.*, 2003), one used a factorial design (Dale *et al.*, 2009) and the majority used a parallel group approach. Thirty one studies were double-blind, 9 were single-blind and 19 were open. More than a third of trials did not state clearly the extent of blinding.

Multiple papers by de Luis *et al.* were published on the same study (de Luis *et al.*, 2009a;de Luis *et al.*, 2008;de Luis *et al.*, 2009b), as well as by Appel *et al.* (Appel *et al.*, 2005;Furtado *et al.*, 2008), Wolever *et al.* (Wolever and Mehling, 2003;Wolever and Mehling, 2002), Wood *et al.* (Wood *et al.*, 2007;Wood *et al.*, 2006), Garcia *et al.* (Garcia *et al.*, 2007;Garcia *et al.*, 2006), Due *et al.* (Due *et al.*, 2005;Due *et al.*, 2004), Dreon *et al.* (Dreon *et al.*, 1994;Campos *et al.*, 1995) and Noakes *et al.* (Noakes *et al.*, 2005;Clifton *et al.*, 2008).

Studies were conducted in a variety of countries, such as the USA (62), the UK (9), Australia (7), Spain (5), Denmark (5), Canada (4), France (4), The Netherlands (3), Finland (4), New Zealand (3), Switzerland (3), Scotland (3), Sweden (3), Italy (3), Europe (3), Germany (2), Mexico (2), Israel (1), the Philippines (1), Argentina (1), Norway (1), Brazil (1), Korea (1) and the UK and USA collectively.

For the most part, trials were conducted on adults. However, five also used children or adolescents (Demol *et al.*, 2009;Rosado *et al.*, 2008;Sondike *et al.*, 2003;Vido *et al.*, 1993;Williams *et al.*, 1995). Twenty eight trials included females only, 22 used males only and the remaining were mixed gender. Of the studies that provided data on age, approximately 20% had a mean participant age of over 50 years. In addition, the majority of studies that reported blood lipids recruited participants with a body mass index (BMI) of greater than 30kg/m² (42%). Approximately 23% of trials reported a mean BMI of 25-30kg/m² and only 4% used participants with a BMI of less than 25kg/m². A large proportion of the trials were intentionally designed to effect weight loss and in others, a reduction in weight was an inadvertent consequence of the nature of the intervention.

Weight loss can beneficially affect the blood-lipid profile (Aucott *et al.*, 2011; Dattilo and Kris-Etherton, 1992). In one systematic review, which included adult participants with a mean baseline BMI less than 35kg/m², the authors reported that a maintained weight loss of 1kg could be expected to reduce total cholesterol, TAG and LDL cholesterol by 1.3%, 1.6% and 0.34% respectively and increase HDL cholesterol by 4%, at 2-3 years follow-up (Aucott *et al.*, 2011). Such improvements in blood lipids owing to weight loss are also likely to be apparent in the short term. A meta-analysis which focused on studies that mostly had durations of less than 52 weeks indicated statistically significant reductions in total cholesterol (-0.05mmol/L), LDL cholesterol (-0.02mmol/L) and TAG (-0.015mmol/L) for active weight loss and a significant increase in HDL cholesterol (0.009) during stabilized weight loss (Dattilo and Kris-Etherton, 1992). Given such results, changes in blood lipids, when accompanied by body weight loss, may not necessarily be solely attributed to a dietary carbohydrate intervention.

Excluding the Women's Health Initiative Study (Howard *et al.*, 2006) which had a sample size of over 48,000 (of which 5.8% gave blood), the average number of participants in each paper was 101 and the median was 51.

Risk of bias

A summary of the risk of bias assessment is provided in Table 2.44. 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' in terms of allocation sequence generation or allocation concealment. Two were judged to be 'biased' with regard to allocation concealment and allocation sequence generation ((Brehm *et al.*, 2003) and (Drummond *et al.*, 2003) respectively). Blinding of participants and researchers to the various dietary approaches was more difficult to achieve, as might be anticipated with dietary intervention trials. However, 34 papers were judged to have 'no bias' in respect of participants' awareness of the dietary intervention, and 37 trials were judged to have 'no bias' in respect of researchers' awareness (these generally overlapped). There was some evidence of incomplete outcome reporting in 41 publications.

Table 2.43 Trial characteristics (studies shaded in grey were conducted on children or adolescents)

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 <i>et al.</i> , 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. Energy restricted. Individually prescribed diet within a strict dietary framework repeated on a 3 day rotation basis. 84% of CHO provided by rice and potatoes. GI 60 - 65 2. Energy restricted. Individually prescribed diet within a strict dietary framework repeated on a 3 day rotation basis. 84% of CHO provided by pasta and legumes. GI 40-45	1. %E: C 47.8 P 19.6 F 32.6 Fibre g/d:18.5 2. %E: C 50.2 P 18.3 F 31.5 Fibre g/d:24.9	Yes	Government funding
(Aller <i>et al.</i> , 2004)	Age 18-70y Generally healthy No HTN, T2DM, statins or steroids Not hyperlipidaemic/hypercholesterolaemic Weight stable	Spain 36% Male Age: (47) BMI: (25)	Parallel Group	3 months	Free living diet plan	53	1. High fibre 2. Low fibre	1. Fibre 30.5g/d: 4.11g soluble fibre (pectins, gums and mucilages) and 25.08g insoluble (hemicellulose, cellulose and lignins). High fibre intake reached through breakfast cereal consumption 60g/d plus 2 apples/d 2. Fibre 10.4g/d: 1.97g soluble fibre (pectins, gums and mucilages) and 8.13g insoluble fibre (hemicellulose, cellulose and lignins)	1. g/d: F 72.6 Energy 1707 kcal/d Fibre g/d:25.95 2. g/d: F 73.4 Energy 1633 kcal/d Fibre g/d:9.06	Yes	Not reported
(Andersson <i>et al.</i> , 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 + wholegrain 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	Swedish Diabetes Association and Government and research institute 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
(Appel <i>et al.</i> , 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
(Bantle <i>et al.</i> , 2000)	Age >18y BMI <32 No CHD Normal glucose tolerance Not hyperlipidaemic/hypercholesterolaemic	USA 50% Male Age: (41) BMI: (25)	Crossover (washout not reported)	6 weeks	All food provided	24	1. High-fructose diet 2. High-glucose diet	1. 55% of energy as carbohydrate, 15% of energy as protein, and 30% of energy as fat (17% total energy as fructose). Crystalline fructose was added to diet. 2. 55% of energy as carbohydrate, 15% of energy as protein, and 30% of energy as fat (3% total energy as fructose). Crystalline glucose was added to diet.	1. g/d: C 276 P 76 F 66 Energy 2004 kcal/d Fibre g/d:23 2. g/d: C 276 P 76 F 66 Energy 2001 kcal/d Fibre g/d:23	No, intended diet only	NIH
(Bell <i>et al.</i> , 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. Estimated approx. 3g/d pectin 3. Step 1 diet with 57g of cornflakes containing oat bran, sugar-beet fibre, white wheat bran and psyllium		Yes	General Mills Inc.

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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
								consumed each morning. 50% total soluble fibre in cereal was from psyllium. Estimated approx. 3 g/d psyllium			
(Bellisle <i>et al.</i> , 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
(Bhargava, 2006) The Women's Health Trial: Feasibility Study in Minority Populations	Age 50-80y Post-menopausal Women	UK and USA 0% Male Age: 50 - 79 BMI: 29	Parallel Group	12 months	Free living diet plan	2208	1. Low fat 2. Control	1. Reduce fat intake to 20% and increase fruit, vegetable and grain consumption. 2. No intervention	1. 5430 kJ, E%: F 20, 13g/d sat fat, 13g/d fibre 2. 6149 kJ, 20g/d sat fat, 12g/d fibre	Yes	National Cancer Institute
(Birketvedt <i>et al.</i> , 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 <i>et al.</i> , 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	Government funding and The Sugar Bureau and Suikerstichting

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 <i>et al.</i> , 2003) American LC study I	Age >18y BMI 30-35 Familial CVD/CHD Generally healthy No HTN or T2DM Weight stable	USA	Parallel Group	6 months	Free living diet plan	53	1. Low carbohydrate	1. Ad libitum food intake. Max CHO intake 20g/d. CHO increased to 40-60g/d if ketosis was induced after 2 weeks.	1. %E: C 30 P 23 F 46 Energy 1302 kcal/d Fibre g/d:8.4	Yes	American Heart Association, research institute funding and NIH
		0% Male Age: (44) BMI: (34)					2. Moderate fat	2. American Heart Association Step 1 diet + restrict to 1200kcal/d. Intended intake: 55% CHO, 15% PRO, 30% FAT	2. %E: C 53 P 18 F 29 Energy 1247 kcal/d Fibre g/d:12.35		
(Brehm <i>et al.</i> , 2005) American LC study II	<10% Δ body weight in previous 6m Age >18y BMI 30-35 Free of chronic disease	USA	Parallel Group	4 months	Free living diet plan	50	1. Low carbohydrate	1. Ad libitum food intake. Max CHO intake 20g/d. CHO increased to 40-60g/d if ketosis was induced after 2 weeks.	1. %E: C 15 P 28 F 57 Energy 1288 kcal/d	Yes	American Heart Association, research institute funding and NIH
		0% Male Age: 44 BMI: (34)					2. Moderate fat	2. American Heart Association Step 1 diet + restrict to 1200kcal/d. Intended intake: 55% CHO, 15% PRO, 30% FAT	2. %E: C 53 P 18 F 29 Energy 1339 kcal/d		
(Cairella <i>et al.</i> , 1995)	BMI >30 No CHD Sedentary occupation	Italy	Parallel Group	60 days	Supplement	30	1. Balanced diet + fibre tablets	1. Fibre tablets (vegetable, citrus, cereal fibre, 6g/d) + balanced diet following 2 week VLCD	1. Fibre g/d:6	Yes	Not reported
		27% Male Age: (36) BMI:31 - 47(37)					2. Balanced diet + placebo tablets	2. Placebo tablets, plus balanced diet following 2 week VLCD			
(Campos <i>et al.</i> , 1995) American Fat & Carbohydrate Study	<130% ideal body weight Familial CVD/CHD Generally healthy Not taking lipid lowering drugs Resting BP < 160/105 mmHg TC <260 mg/dl TG <500 mg/dl	USA	Crossover (washout not reported)	6 weeks	Free living diet plan	43	1. Low-fat higher CHO	1. 60% carbohydrate, with equal amounts of simple and “complex” carbohydrates. 24% total fat (6% saturated and 4% polyunsaturated). Dietary cholesterol (150 mg/1000 kcal), fibre (4-5 g/1000 kcal), protein (16%)	1. %E: C 58.8 P 16.8 F 24.2: Energy 2781 kcal/d	Yes	NIH
		100% Male Age: (50) BMI: (26)					2. High-fat low CHO	2. 38% carbohydrate and 46% total fat (18% saturated 12% polyunsaturated). dietary cholesterol (150 mg/1000 kcal), fibre (4-5 g/1000 kcal), protein (16%)	2. %E: C 39.2 P 16.3 F 45.2 Energy 2866 kcal/d		

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
(Chen <i>et al.</i> , 2006) American Fibre Study	Age 30-65y Good compliance during run-in No antihypertensive / cholesterol lowering No CHD/CVD, T2DM or HTN Not hyperlipidaemic/ hypercholesterolaemic	USA 40% Male Age: (48) BMI: (29)	Parallel Group	12 weeks	Substitution	110	1. High fibre 2. Low fibre	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	Yes	NIH and research institute funding
(Claessens <i>et al.</i> , 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
(Clevidence <i>et al.</i> , 1992)	Body weight 80-130% of ideal Generally healthy No chronic illness No medication	USA 100% Male Age: 19 - 56(34) BMI: mean not reported	Crossover	10 weeks	All food provided	46	1. High fat diet 2. Low fat diet	1. Fibre consumption was 8.4g/1000kcal 2. Emphasis on obtaining fibre from legumes, cereals and fruits. Fibre consumption was 19.3g/1000kcal	1. %E: C 45.8 P 14.8 F 40.7 2. %E: C 67.3 P 17.1 F 18.9	Yes	Not reported
(Clifton <i>et al.</i> , 2008) Australian Protein Study	27-40 Female adults	Australia 0% Male Age: (49) BMI: (33)	Parallel Group	12 weeks intensive , plus 12 mo follow up	Free living diet plan	119	1. High protein diet 2. High carbohydrate diet	1. 46% CHO, 34% PRO, 20% FAT 2. 64% CHO, 17% PRO, 20% FAT	1. %E: C 46.4 P 23.2 F 28.5 g/d: C 179 P 94.6 F 51.4 Energy: 6583kJ/d Fibre g/d:3.9 2. %E: C 50.8 P 19.6 F 27.5 g/d: C 189.5 P 77 F 48.4 Energy: 6391kJ/d Fibre g/d:4.3	Yes	Meat and Livestock Australia

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
(Clifton <i>et al.</i> , 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	1. 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	Yes	Meadow Lea Foods, Australia
		2. High MUFA					2. Diet was closely prescribed and key foods were provided	2. %E: C 43.7 P 21.3 F 35.3 Energy: 5972kJ/d Fibre g/d:32			
(Colette <i>et al.</i> , 2003)	BMI >25 No medications which influence outcomes No T2DM	France 28% Male Age: (48) BMI: (35)	Parallel Group	8 weeks	Free living diet plan	52	1. High carbohydrate diet	1. Hypocaloric diet (-30% energy intake). 55%CHO, 20%PRO, 25% FAT (10%MUFA, (7.5%SFA, 7.5%PUFA)	1. %E: C 52.4 P 20.9 F 25.8 Energy: 6000kJ/d Fibre g/d:17	Yes	Not reported
		2. High MUFA diet					2. Hypocaloric diet (-30% energy intake). 40%CHO, 20%PRO, 40%FAT (25%MUFA, 7.5%SFA 7.5%PUFA).	2. %E: C 40.3 P 20.2 F 39.4 Energy: 7200kJ/d Fibre g/d:18			
(Cornier <i>et al.</i> , 2005)	Normoglycaemic	USA 0% Male Age: 23 - 53(42) BMI:30 - 35(32)	Parallel Group	16 weeks	All food provided	21	1. High carbohydrate, low fat	1. 60%CHO, 20%PRO, 20%FAT	1. %E: C 60 P 20 F 20	No, intended diet only	Research institute funding, American Diabetes Association and American Heart Association
		2. Low carbohydrate, high fat					2. 40%CHO, 20%PRO, 40%FAT	2. %E: C 40 P 20 F 40			
(Couture <i>et al.</i> , 2003)	No endocrine disease No CHD No medications which influence outcomes Weight stable	Canada 100% Male Age: (38) BMI: (29)	Parallel Group	6 weeks	All food provided	65	1. High carbohydrate diet	Participants in both groups received food in quantities that met 150% of their habitual energy intake/day and 200-kcal snacks provided on demand.	1. %E: C 58.3 P 15.9 F 25.8	Yes	Research institute funding, the International Olive Oil Council and Knoll Pharmaceuticals
		2. High MUFA diet					2. %E: C 44.7 P 15.2 F 40.1				
(Crujeiras <i>et al.</i> , 2007)	<3kg Δ weight in previous 3m Generally healthy No medication	Spain 56.6% Male Age: (36)	Parallel Group	8 weeks	Free living diet plan	30	1. Hypocaloric diet + legumes	1. Energy deficit of 30%. Intended diet: 50%CHO, 20% PRO, 30% FAT. Nonsoybean legume servings 4 days/week	1. %E: C 50.2 P 18.9 F 33.4 Energy 2479 kcal/d	Yes	Government funding and University funding
		2. Hypocaloric control diet					2. Energy deficit of 30%.	2. %E: C 50.7 P			

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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: (32)						Intended diet: 50%CHO, 20% PRO, 30% FAT	18.9 F 30.8 Energy 2479 kcal/d		
(Dale <i>et al.</i> , 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 g/d: C 183 P 77 F 46 Energy: 6192kJ/d Fibre g/d:23	Yes	Health Research Council of New Zealand
(Dansinger <i>et al.</i> , 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. 2. Macronutrient balance. 3. Calorie restriction. 4. Fat restriction. 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

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
(Das <i>et al.</i> , 2007) CALERIE	BMI 25-30 Generally healthy No medications which influence outcomes Not extremely athletic/active Weight stable	USA % Male: not reported Age: (35) BMI: (28)	Parallel Group	12 months	All food provided	34	1. Energy restricted high GL diet 2. Energy restricted low GL diet	1. 30% calorie restriction. fibre 15 g/1000kcal. Estimated GI=86, GL=116 g/1000 kcal 2. 30% calorie restriction. fibre 15 g/1000 kcal. Estimated GI=53, GL=45 g/1000kcal	1. %E: C 60 P 20 F 20 2. %E: C 40 P 30 F 30	Yes	NIH and Government funding
(Davidson <i>et al.</i> , 1998)	Age 30-75y Mild to moderate lipidaemias (LDL-C 3.63-5.17mmol/L)	USA 48% Male Age: 30 - 75(60) BMI: (28)	Crossover (washout 6 weeks)	6 weeks	Substitution	25	1. Inulin 2. Control	1. Low fat diet + inulin food products (chocolate, spread, sweeteners). 18g inulin/d as Raftiline (Orafti) – average degree of polymerisation 10 (2-65) 2. Low fat diet + non-supplemented food products	NCEP Step 1 diet advocated throughout (high carbohydrate, low fat)	Yes	Not reported
(Davy <i>et al.</i> , 2002) American Cereal Study	50-75 year old men BMI 25-35 DBP 85-99mmHg Fibre <30g/d No CHD, T2DM No medical conditions which influence outcomes Non smokers Normal glucose tolerance Not extremely athletic/active SBP 130-15	USA 100% Male Age: 50 - 75(59) BMI: (29)	Parallel Group	12 weeks	Supplement	36	1. Wholegrain oat cereal 2. Wheat cereal	1. 60g oatmeal plus 76g oat bran ready-to-eat cold cereal (14g fibre/d, 5.5 g beta-glucan) 2. 60g whole wheat cereal plus 81g Frosted Mini-Wheats (14g fibre/d)	1. g/d: C 95 P 21 F 8 Energy 513 kcal/d Fibre g/d:14 2. g/d: C 112 P 14 F 3 Energy 480 kcal/d Fibre g/d:14	Yes	Quaker Oats
(de Luis <i>et al.</i> , 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
(de Luis <i>et al.</i> , 2009b) Spanish	BMI >30 No CHD, T2DM or HTN	Spain 28% Male	Parallel Group	3 months	Free living diet plan	118	1. Low carbohydrate	1. Intended diet: 1507kcal/d. 38% CHO, 26% PRO, 36% FAT	1. %E: C 30.8 Energy 1548 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
Hypocaloric Diet Study		Age: (46) BMI: (35)					2. Low fat	2. Intended diet: 1500 kcal/d. 52% CHO, 20% PRO, 27% FAT	2. %E: F 25.3 Energy 1613 kcal/d		
(de Luis <i>et al.</i> , 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
(Delbridge <i>et al.</i> , 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 and Livestock Australia
(Demol <i>et al.</i> , 2009)	BMI >95th centile No medications which influence outcomes No recent weight loss program Without chronic disease	Israel 38% Male Age: 12 - 18(14) BMI: mean not reported	Parallel Group	12 weeks (9 mo Follow up)	Free living diet plan	55	1. Low carbohydrate, high protein 2. Low carbohydrate, high fat 3. High carbohydrate, low fat	All groups prescribe energy restriction to 1200-1500 kcal/d 1. Low-carbohydrate, low-fat, protein-rich diet containing 60 g carbohydrate (up to 20%), 30% fat and 50% protein. 2. Low-carbohydrate, high-fat diet containing: 60 g carbohydrate (up to 20%), 60% fat and 20% protein 3. High-carbohydrate, low-fat diet containing: 50–60% carbohydrate, 30% fat and 20% protein	1. %E: C 20 P 50 F 30 g/d: C 60 2. %E: C 20 P 20 F 60 g/d: C 60 3. %E: C 50 P 20 F 30	No, intended diet only	Not reported
(Dreon <i>et al.</i> , 1994) American Fat &	Age >20y Body weight <130% of ideal Free of chronic	USA 100% Male	Crossover	6 weeks	Free living diet plan	105	1. High-fat low CHO	1. 38% carbohydrate and 46% total fat (18% saturated 12% polyunsaturated). dietary cholesterol (150 mg/1000	1. %E: C 39 P 16 F 46 Energy 2866 kcal/d	Yes	National Dairy Promotion and Research Board

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Carbohydrate Study	disease in the past 5 yrs No medications which influence outcomes Non smokers Resting BP < 160/105 mmHg TC <260 mg/dl TG <500 mg/dl	Age: (49) BMI: (26)					2. Low-fat higher CHO	kcal), fibre (4-5 g/1000 kcal), protein (16%) 2. 60% carbohydrate, with equal amounts of simple and “complex” carbohydrates. 24% total fat (6% saturated and 4% polyunsaturated). Dietary cholesterol (150 mg/1000 kcal), fibre (4-5 g/1000 kcal), protein (16%)	Fibre g/d:13 2. %E: C 59 P 17 F 25 Energy 2781 kcal/d Fibre g/d:14		
(Drummond <i>et al.</i> , 2003)	Free of chronic disease No medication	Scotland 100% Male Age: >40 BMI: mean not reported	Parallel Group	8 weeks	Free living diet plan	30	1. Reduced fat 2. Reduced fat and sugar	1. Dietician advised to reduce fat intake. Fat intake did not actually decrease. 2. Dietician advised to reduce fat and sugar intake. Fat intake did not actually decrease. Reduced NMES and increased starch.	1. %E: C 47.4 P F 35.2 Energy: 9210kJ/d 2. %E: C 48.7 P F 33.1 Energy: 8030kJ/d	Yes	The Sugar Bureau
(Due <i>et al.</i> , 2008) MonoUnsaturated Fatty acids in Obesity trial	<3kg Δ weight in previous 2m Age 18-35y BMI 28-36 Non smokers No T2DM Pre-menopausal Recently involved in weight loss trial	Denmark 42% Male Age: (28) BMI: (31)	Parallel Group	6 months	Free living diet plan	154	1. High MUFA 2. Low fat 3. Control	1. Dietary counselling and food provided from study supermarket. Prescribed 35-45%FAT, >20%MUFA This diet <i>also</i> included more wholegrains, legumes and nuts. SFA:MUFA:PUFA% 7:20:8 2. Dietary counselling. Food provided from study supermarket. Prescribed 20-30%FAT. SFA:MUFA:PUFA% 8:8:5 3. Dietary counselling. Food provided from study supermarket. Moderate fat (35% energy) with >15% SFA. SFA:MUFA:PUFA% 15:10:4.	1. %E: C 43.3 P 15.3 F 38.4 Energy: 11500kJ/d 2. %E: C 57.6 P 15.8 F 23.6 Energy: 10500kJ/d 3. %E: C 49.8 P 15.9 F 32.1 Energy: 10900kJ/d	Yes	HA Foundation, The Danish Heart Association, The Danish Diabetes Association, The Danish Pork Council and research institute funding
(Due <i>et al.</i> , 2004) The Danish Protein Swap Study	Previously overweight/obese	Denmark 24% Male Age: (40) BMI: (30)	Parallel Group	6 months strict, 6-12 mo less strict, plus 24 mo	All food provided	50	1. High protein 2. Moderate protein	1. 25%PRO, <30%FAT 2. 12%PRO, <30%FAT	1. %E: C 48.9 P 21.2 F 30 Energy: 8400kJ/d 2. %E: C 54.7 P 13.9 F 31.4 Energy: 8200kJ/d	Yes	Research institute funding, The Federation of Danish Pig Producers and Slaughterhouse

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				follow up							and The Danish Livestock and Meat Board
(Due <i>et al.</i> , 2005)	Overweight/ Obese	Denmark	Parallel Group	6 months	All food provided	50	1. High protein 2. Moderate protein	1. 25%PRO, <30%FAT 2. 12%PRO, <30%FAT	1. %E: C 48.9 P 21.2 F 30 Energy: 8400kJ/d 2. %E: C 54.7 P 13.9 F 31.4 Energy: 8200kJ/d	Yes	Research institute funding, The Federation of Danish Pig Producers and Slaughterhouse , Danish Dairy Research Foundation and The Danish Livestock and Meat Board
The Danish Protein Swap Study		28% Male Age: (40) BMI: (30)									
(Dyson <i>et al.</i> , 2007)	Age >18y BMI >25 No T2DM Weight stable	UK	Parallel Group	3 months	Free living diet plan	13	1. Low carbohydrate diet 2. Healthy eating diet	1. Healthy eating advice plus reduction in CHO to <40g/d 2. Dietary guidelines of Diabetes UK plus energy restriction.		Yes	Medisense UK, Abbott Laboratories
		23% Male Age: (51) BMI: (36)									
(Ebbeling <i>et al.</i> , 2007)	Age 18-35y BMI >30 Generally healthy No medication No recent weight loss program Non smokers No T2DM	USA	Parallel Group	6 months intensive , 12 mo follow up. Monthly group workshops throughout 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. GI 46, GL53 2. General healthy eating advice. Target: 55% CHO, 25% PRO, 20% FAT. Ad libitum consumption. GI 53, GL77		Yes	National Institute of Diabetes & Digestive & Kidney Diseases, Charles H. Hood Foundation and research institute funding
		21% Male Age: 18 - 35(27) BMI: mean not reported									
(Ebbeling <i>et al.</i> , 2005)	Age 18-35y BMI >27.5 Healthy	USA	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	1. %E: C 47.2 P 21.1 F 33 Energy 1391 kcal/d Fibre g/d:20.7	Yes	National Institute of Diabetes & Digestive & Kidney
		12% Male Age: 28									

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		BMI: obese						exchange system, energy deficit of 250-500kcal/d. GL 77 g/1000 kcal	2. %E: C 59.4 P 18.7 F 23.4 Energy 1409 kcal/d Fibre g/d:17.8		Diseases, Charles H. Hood Foundation and research institute funding
(Forcheron and Beylot, 2007)	Not extremely athletic/active	France 35% Male Age: mean not reported BMI: mean not reported	Parallel Group	6 months	Supplement	20	1. Fructans 2. Placebo	1. 10g mix of inulin and oligofructose 2. Maltodextrin 10g/d		Yes	Orafti
(Foster <i>et al.</i> , 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 1 st 2 wks, rising until desired wt. achieved. 60% ppts 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 <i>et al.</i> , 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 1 st 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 and the Institute for Applied Telemedicine

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(Furtado <i>et al.</i> , 2008) OMNI-Heart	Age >30y Generally healthy No CVD or T2DM No medications which influence outcomes Not hyperlipidaemic/ hypercholesterolaemic prehypertension/ stage 1 HTN Weight < 160kg	USA 56% Male Age: (53) 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	No, intended diet only	NIH
(Garcia <i>et al.</i> , 2007) The Arabinoxylan and Glucose Metabolism study	Age 20-70y BMI >26 Free of chronic disease Generally healthy Impaired glucose tolerance No medication	Germany 36% Male Age: 48 - 70(56) BMI: 26 - 46(30)	Crossover (washout 6 weeks)	6 weeks	Supplement	14	1. Arabinoxylan 2. Placebo	1. Arabinoxylan 15g/d (10g within bread, 5g as powder). 2. Placebo powder and bread rolls		Yes	Federal Ministry of Education and Research Germany
(Garcia <i>et al.</i> , 2006) The Arabinoxylan and Glucose Metabolism study	Age 20-70y BMI >26 Generally healthy Impaired glucose tolerance No chronic illness No medication	Germany 36% Male Age: 48 - 70(56) BMI:26 - 46(30)	Crossover (washout 6 weeks)	6 weeks	Supplement	14	1. Arabinoxylan 2. Placebo	1. Arabinoxylan 15g/d (10g within bread, 5g as powder). 2. Placebo powder and bread rolls		Yes	Federal Ministry of Education and Research Germany
(Gardner <i>et al.</i> , 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	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

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				12mo post randomisation							
(Genta <i>et al.</i> , 2009)	BMI >30 Generally healthy History of constipation Mild lipidaemias Pre-menopausal	Argentina 0% Male Age: (41) BMI: (34)	Parallel Group	120 days	Supplement	55	1. Fructooligo-saccharide (Yacon) syrup low dose 2. Placebo 3. Fructooligo-saccharide (Yacon) syrup high dose.	1. Provided 0.14 g fructooligosaccharides/ kg body weight/d from yacon syrup. 2. Placebo syrup 3. Provided 0.29 g fructooligosaccharides/ kg body weight/d from yacon syrup. No data were presented for this group as significant undesirable gastrointestinal side effects were observed.	1. %E: C 67.04 P 2.16 F 0.14	Yes	Research institute funding
(Ginsberg <i>et al.</i> , 1998)	Age 22-65y Generally healthy No medications which influence outcomes Normal lipid profile	USA 45% Male Age: (38) BMI: (24)	Crossover (washout 5 weeks)	8 weeks	All food provided	118	1. Average American Diet 2. Step 1 diet 3. Low saturated fat diet	1. 16%SFA, 14%MUFA, 7%PUFA 2. 9%SFA, 14%MUFA, 7%PUFA 3. 5%SFA, 14%MUFA, 7%PUFA	1. %E: C 48 F 37 2. %E: C 55 F 30 3. %E: C 59 F 26	No, intended diet only	Research institute funding
(Golay <i>et al.</i> , 1996)	BMI >30 No endocrine disease	Switzerland 21% Male Age: (43) BMI: (40)	Parallel Group	6 weeks	All food provided	43	1. Low carbohydrate diet 2. Moderate carbohydrate diet	1. Hypocaloric diet (1000kcal/d) 15%CHO, plus aerobic exercise 1h/d 2. Hypocaloric diet (1000kcal/d) 45%CHO plus aerobic exercise 1h/d	1. %E: C 15 P 32 F 53 g/d: C 37 P 79 F 60 Energy: 4214kJ/d 2. %E: C 45 P 29 F 26 g/d: C 115 P 73 F 30 Energy: 4296kJ/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
(Golay <i>et al.</i> , 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 simultaneously	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 P 72 F 38 Energy: 4600kJ/d	No, intended diet only	Not reported
(Haskell <i>et al.</i> , 1992) Study# 1 reported in this reference	Age 20-75y Generally healthy Mild to moderate lipidaemias No fibre supplement use No medication Normal glucose tolerance	USA 43% Male Age: (52) BMI:<130% ideal body weight	Parallel Group	12 weeks	Substitution	62	1. Study1 Soluble fibre mix 2. Study1 Placebo	1. 17.2g/d soluble fibre (3.9g Pectin, 6.3g Psyllium husk, 3.3g Guar gum, 1.5g Locust bean gum). 45g of fructose/d 2. Placebo – fructose carrier product only- 45g of fructose/d	approximately 70 kcal/serving of fibre and placebo products	Yes	Shaklee U.S., Inc.
(Helge, 2002)	Generally healthy Stable activity level	Denmark 100% Male Age: (27) BMI: (25)	Parallel Group	7 weeks	Free living diet plan	41	1. High fat + exercise 2 . High carbohydrate + exercise 3. High fat	1. 21%CHO, 17%PRO, 62%FAT 2. 65% CHO, 15%PRO, 20%FAT 3. Data for this group will not be included, the lack of exercise element means it is not an appropriate comparison group	1. %E: C 21.8 P 16.6 F 61.6 Energy 3367 kcal/d 2. %E: C 64.9 P 14.6 F 20.3 Energy 3487 kcal/d	Yes	Research institute funding
(Howard <i>et al.</i> , 2006) The Women's Health Initiative Dietary Modification Trial	Age 50-79y Fat intake >32% Post-menopausal	USA 0% Male Age: (62) BMI: (29)	Parallel Group	6 years	Free living diet plan	48835 (5.8% gave blood)	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

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(Hunninghake <i>et al.</i> , 1994)	Age 18-70y Generally healthy Mild to moderate lipidaemias No medications which influence outcomes Within 30% of ideal weight	USA 76% Male Age: 52 BMI: 26	Parallel Group	15 weeks	Supplement	161	1. Placebo 2. Fibre 10g 3. Fibre 20g	1. Placebo sachet before breakfast and dinner while consuming a step 1 diet. 2. Placebo sachet and 10 of fibre supplement before dinner, while consuming a step 1 diet. Fibre = guar gum, pectin, soy, corn bran, pea fibre taken with milk or water 3. 10g of fibre supplement before breakfast and again before dinner, while consuming a step 1 diet. Fibre = guar gum, pectin, soy, corn bran, pea fibre taken with milk or water		Yes	Sandoz Pharmaceutical Department
(Jackson <i>et al.</i> , 1999)	Mild to moderate lipidaemias No CHD or T2DM No medications which influence outcomes Not extremely athletic/active	UK % Male not reported Age: 35 - 65(52) BMI:20 - 32(26)	Parallel Group	8 weeks	Supplement	54	1. Inulin 2. Placebo	1. Inulin powder added to usual diet 10g/d 2. Maltodextrin powder added to usual diet 10g/d		Yes	Raffinerie Tirlemontoise (ORAFIT)
(Jensen <i>et al.</i> , 1997)	Age 25-65y Generally healthy Mild to moderate lipidaemias Moderately hypercholesterolemic No medications which influence outcomes	USA 53% Male Age: (52) BMI: (26)	Parallel Group	24 weeks	Supplement	58	1. Water soluble dietary fibre (WSDF) 2. Acacia gum	Low fat, low cholesterol (Step 1) diet, plus: 1. A mixture of psyllium (2.1 g WSDF/serving), pectin (1.3 g WSDF/serving), guar gum (1.1 g WSDF/serving), and locust bean gum (0.5 g WSDF/serving) prepared as a powder in a carbohydrate base (ca 15g fructose/serving). 2. 5.0 g WSDF/serving, prepared as a powder in the same fructose base.		Yes	Shaklee Corporation

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(Jensen <i>et al.</i> , 2008) The Danish GI study	Age 20-40y BMI 25-30 Generally healthy Moderate alcohol No HTN No medical conditions which influence outcomes No medication Non 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. GI of provided foods 72 2. Received high GI test foods in place of their usual CHO rich foods. GI of provided foods 95	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	Yes	Research institute funding
(Johnston <i>et al.</i> , 2004)	Generally healthy	USA 10% Male Age: 19 - 54 BMI: (29)	Parallel Group	6 weeks	All food provided	20	1. High protein, low fat 2. High carbohydrate, low fat	1. Low fat, energy restricted, 30%PRO 2. Low fat, energy restricted, 60%CHO	1. g/d: C 170 P 134 F 53 Energy 1700 kcal/d Fibre g/d:23 2. g/d: C 280 P 64 F 39 Energy 1700 kcal/d Fibre g/d:25	No, intended diet only	University funding and research institute funding
(Johnston <i>et al.</i> , 2006)	No medications which influence outcomes	USA 21% Male Age: 20 - 60 BMI: (34)>25	Parallel Group	6 weeks	All food provided	20	1. Low carbohydrate diet 2. Very low-carbohydrate diet	1. Nonketogenic low carbohydrate diet. 40%CHO, 30%PRO, 30%FAT (SFA 9%) 2. 5%CHO (increased by 5g/wk in weeks 3-6), 30%PRO, 60%FAT (SFA 21%)	1. %E: C 42 P 31 F 30 g/d: C 157 P 117 F 50 Energy: 6250kJ/d Fibre g/d:30 2. %E: C 9 P 33 F 60 g/d: C 33 P 125 F 100 Energy: 6250kJ/d Fibre g/d:15	Yes	Research institute funding

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(Johnston, 1998)	Age 40-70y Body weight <140% of ideal Mild to moderate lipidaemias No CVD No medications which influence outcomes No metabolic disease	USA 63% Male Age: (57) BMI: mean not reported	Parallel Group	6 weeks	Substitution	135	1. Control cereal 2. Wholegrain oat cereal	1. Cornflakes 90g/d, delivering 2g fibre (0.1g soluble, 1.9g insoluble) 2. Oat Cheerios 90g/d delivering 9g fibre (2.9g soluble, 6.1g insoluble)	1. g/d: C 78 P 5.4 F 1.4 Energy: 338kJ/d Fibre g/d:2 2. g/d: C 67.6 P 9.9 F 5.2 Energy: 321kJ/d Fibre g/d:9	Yes	General Mills Inc.
(Keenan <i>et al.</i> , 2007)	49% with metabolic syndrome Age 25-73y Elevated LDL cholesterol Mild to moderate lipidaemias No chronic illness No medications which influence outcomes No T2DM Weight stable	USA 48% Male Age: (55) BMI: (29)	Parallel Group	6 weeks	Supplement	155	 1. Low-dose, LMW barley beta-glucan 2. High-dose LMW barley beta-glucan 3. Low-dose, HMW barley beta-glucan 4. High-dose, HMW barley beta-glucan 5. Placebo	Fibre incorporated into two functional food products: a ready-to-eat cereal and a reduced-calorie fruit juice beverage 1. 3g/d low molecular weight barley beta-glucan 2. 5g/d low molecular weight barley beta-glucan 3. 3g/d high molecular weight barley beta-glucan 4. 5g/d high molecular weight barley beta-glucan 5. Placebo - no fibre incorporation		Yes	Not reported
(Keogh <i>et al.</i> , 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 dietician 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

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
(Keogh <i>et al.</i> , 2008)	≥ 1 metabolic syndrome risk factor Abdominal obesity No CHD or T2DM	Australia % Male: not reported 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
(Kesaniemi <i>et al.</i> , 1990)	Some with mild HTN, Some with gallstones No heart failure or thyroid, liver, renal, GI diseases	Finland 100% Male Age: 47 - 55(50) BMI:18 - 35(26)	Crossover (washout 0 weeks)	8 weeks	Free living diet plan	34	1. Low fibre 2. High fibre	1. Advise: avoid unpurified cereals, vegetables, salads, fruit and berries. Low fibre products were recommended, purified wheat products, filtered berry soups and processed juices. Wheat flour hot cereal porridge and fibre-free biscuits provided 2. Advise: eat large quantities of unpurified corn, fruit, vegetables, salads & berries. Given 200ml/day hot porridge: oat flakes, bran, guar gum (9.4g/100g dry) and pectin (2.3g/100g dry) plus graham biscuits fortified with carrots and bran. Mix of soluble and insoluble fibre sources	1. g/d: C 273 P 101 F 109 Energy 2557 kcal/d Fibre g/d:11.6 2. g/d: C 252 P 90 F 105 Energy 2557 kcal/d Fibre g/d:26.2	Yes	Juho Vainio Foundation, Sigrid Juselius Foundation, Medical Council of the Academy of Finland and the Finnish Life Insurance Companies
(Kim <i>et al.</i> , 2008)	BMI 25-35 No chronic illness Normal glucose tolerance Normal lipid profile	Korea 0% Male Age: 20 - 35 BMI:25 - 35	Parallel Group	6 weeks	All food provided	47	1. White rice meal replacement 2. Brown & black rice meal replacement	1. Energy restricted diet (258kJ/d), three meals per day replaced with supplement containing white rice plus soybean, seaweed, laver, vegetables. Cooked with milk. 2. Energy restricted diet (258kJ/d), three meals per		Yes	Research institute funding

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								day replaced with supplement containing brown and black rice plus soybean, seaweed, laver, vegetables. Cooked with milk.			
(Kirk <i>et al.</i> , 2009)	Impaired glucose tolerance Insulin resistant No chronic illness which influence outcomes No T2DM Weight stable	USA 18% Male Age: (44) BMI: (37)	Parallel Group	11 weeks	All food provided	22	1. High carbohydrate	1. Energy deficit 1000kcal/d until 7% body weight loss (~6 weeks) followed by weight maintenance. CHO>180g/d	1. %E: C 65 P 15 F 20	No, intended diet only	NIH
							2. Very low carbohydrate	2. Energy deficit 1000kcal/d until 7% body weight loss (~6 weeks) followed by weight maintenance. CHO <50g/d	2. %E: C 10 P 15 F 75		
(Kirkwood <i>et al.</i> , 2007)	Age 30-50y BMI 25-40 Generally healthy Not on weight loss diet	Scotland 0% Male Age: (41) BMI: (32)	Parallel Group	12 weeks	Free living diet plan	109	1. Group 1: No advice	1. Comparison for group 2	1. %E: C 49.6 P 17 F 33.1 Energy: 8100kJ/d	Yes	The Sugar Bureau
							2. Group 2: Conventional weight loss diet	2. Low fat, high carbohydrate, including sucrose, energy reduced diet	2. %E: C 50.1 P 19.1 F 30.2 Energy: 7100kJ/d		
							3. Group 3: Exercise	3. Intervention was exercise-based (comparison for group 4)	3. %E: C 44.2 P 18.9 F 36.7 Energy: 7400kJ/d		
							4. Group 4: Conventional weight loss diet + exercise	4. Low fat, high carbohydrate, including sucrose, energy reduced diet plus exercise	4. %E: C 52.3 P 17.8 F 29 Energy: 7100kJ/d		
(Kleemola <i>et al.</i> , 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	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	Yes	Not reported
							2. Group 2- Control diet first		2. %E: C 49 P 16.3 F 34.6 Energy 2063 kcal/d Fibre g/d:21.3		
							3. Group 1- Control diet second		3. %E: C 50.5 P 16.6 F 32.9		

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							4. Group 2- Cereal diet second		Energy 2004 kcal/d Fibre g/d:22.3 4. %E: C 55.4 P15.7 F 28.8 Fibre g/d: 21.3 Energy 1963 kcal/d		
(Knopp <i>et al.</i> , 1999)	Age 18-70y Generally healthy Mild to moderate lipidaemias No medications which influence outcomes Specific diet during trial run-in	USA 65% Male Age: 26 - 69(52) BMI: (26)	Parallel Group	15 weeks	Supplement	169	1. Fibre supplementation 2. Placebo	1. Step 1 diet plus fibre supplementation (15g/d of guar gum and pectin and 5g/d of a mixture of soy fibre, pea fibre and corn bran) 2. Step 1 diet plus placebo (non-water soluble fibre from cellulose)		Yes	Novartis Consumer Health
(Krauss <i>et al.</i> , 2006)	BMI 26-35 No chronic illness No CVD, HTN No medications which influence outcomes Not hyperlipidaemic/hypercholesterolaemic	USA 100% Male Age: mean not reported BMI: mean not reported	Parallel Group	12 weeks	Free living diet plan	224	1. 54% CHO Low saturated fat 2. 39% CHO Low saturated fat 3. 26% CHO Low saturated fat 4. 26% CHO High saturated fat	1. 7%SFA, 13%MUFA, 8%PUFA 2. 8%SFA, 13%MUFA, 8%PUFA 3. 9%SFA, 27%MUFA, 5%PUFA 4. 15%SFA, 20%MUFA, 6%PUFA	1. %E: C 54 P 16 F 30 2. %E: C 39 P 29 F 31 3. %E: C 26 P 29 F 46 4. %E: C 26 P 29 F 45	No, intended diet only	National Dairy Council
(Landin <i>et al.</i> , 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 and Goteborg Medical Society.

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(Landry <i>et al.</i> , 2003)	Generally healthy No CHD Normal glucose tolerance Weight stable	Canada	Parallel Group	7 weeks	All food provided	37	1. High carbohydrate	1. Ad libitum consumption of plentifully supplied foods.	1. %E: C 60 P 16 F 27 Energy: 12000kJ/d	Yes	Knoll Pharmaceutical Company and the International Life Sciences Institute
		100% Male Age: (34) BMI: (28)					2. Low carbohydrate, high fat diet	2. Ad libitum consumption of plentifully supplied foods.	2. %E: C 46 P 16 F 41 Energy: 13000kJ/d		
(Lasker <i>et al.</i> , 2008)	BMI >25 No medications which influence outcomes Non smokers	USA	Parallel Group	4 months	Free living diet plan	65	1. High carbohydrate	1. Energy restriction 500kcal/d	1. g/d: C 215.4 P 66.7 F 39.2 Energy: 5875kJ/d Fibre g/d:24.3	Yes	National Cattlemen's Beef Association, The Beef Board and Kraft Foods
		38% Male Age: (47) BMI: (34)					2. High protein	2. Energy restriction 500kcal/d	2. g/d: C 152.6 P 121.4 F 56.2 Energy: 6607kJ/d Fibre g/d:21.1		
(Layman <i>et al.</i> , 2005)	BMI >26 Body weight <140% of ideal No medical conditions which influence outcomes No medications which influence outcomes	USA	Parallel Group	16 weeks	Free living diet plan	48	1. High protein diet	1. Carbohydrate:protein ratio designed to be <1.5.	1. g/d: C 141 P 110 F 52 Energy: 6062kJ/d Fibre g/d:18.6	Yes	Illinois Council on Food and Agricultural Research, National Cattlemen's Beef Association, The Beef Board and Kraft Foods
		0% Male Age: 40 - 56(47) BMI: (33)					2. High protein diet + exercise	2. Carbohydrate:protein ratio designed to be <1.5. Exercise recommendations were minimum of 30minutes of walking 5d/week	2. g/d: C 127 P 102 F 46 Energy: 5540kJ/d Fibre g/d:16		
							3. High carbohydrate diet	3. Carbohydrate:protein ratio designed to be >3.5	3. g/d: C 197 P 58 F 34 Energy: 5377kJ/d Fibre g/d:23		
							4. High carbohydrate diet + exercise	4. Carbohydrate:protein ratio designed to be >3.5. Exercise recommendations were minimum of 30minutes of walking 5d/week			

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(Layman <i>et al.</i> , 2009)	BMI >25 Non-smokers No lipid-lowering medication/steroids/antidepressants	USA 45% Male Age: 40 - 56(45) BMI: (33)	Parallel Group	12 months 4 months weight loss followed by 8 mo weight maintenance. Weekly meetings for 12 mo	Free living diet plan	130	1. High carbohydrate, low protein diet 2. Low carbohydrate, high protein diet	1. Protein provided ~15% energy intake, with carbohydrate:protein ratio >3.2 and lipids ~30% energy intake. Protein provided 0.8g.kg/d. Kcal and fibre were similar between groups 2. Protein provided ~30% energy intake, with carbohydrate:protein ratio <1.5 and lipids ~30% energy intake. Protein provided 1.6g.kg/d. Kcal and fibre similar between groups	1. g/d: C 232 P 70 F 51 Energy: 6800kJ/d Fibre g/d:25 2. g/d: C 168 P 116 F 67 Energy: 7180kJ/d Fibre g/d:20	Yes	The National Cattlemen's Beef Association, Beef Checkoff, and Kraft Foods
(Lehtimäki <i>et al.</i> , 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 funding and the Finnish Cultural Foundation
(Leidy <i>et al.</i> , 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 and the National Pork Board
(Letexier <i>et al.</i> , 2003)	Generally healthy No medications which influence outcomes No T2DM	France 50% Male Age: 23 - 32 BMI:19 - 25	Crossover (washout 4 months)	6 weeks	Supplement	8	 1. Inulin 2. Placebo	High-carbohydrate, low-fat diet (55% of total energy) plus 1. Inulin 10g/d 2. Maltodextrin 10g/d		Yes	European Union

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(Ley <i>et al.</i> , 2004) New Zealand Diabetic Workforce Study	Age >40y Impaired glucose tolerance	New Zealand 74% Male Age: >40 (53) BMI: mean not reported	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 institute funding and the Lotteries Medical Board
(Lofgren <i>et al.</i> , 2005)	Age 20-50y BMI >30 No chronic illness No medications which influence outcomes	Sweden 0% Male Age: (36) BMI: (37)	Parallel Group	10 weeks	Not stated	40	1. High fat, moderate carbohydrate 2. High carbohydrate, low fat	1. Hypoenergetic (-600 kcal/d). 40-45%CHO, 15-20%PRO, 40-45%FAT. No alcohol permitted 2. Hypoenergetic (-600 kcal/d). 60-65%CHO, 15-20%PRO, 20-25%FAT. No alcohol permitted.	1. %E: C 38.9 P 19.6 F 41.5 2. %E: C 52.4 P 21.1 F 26.5	Yes	European Community
(Lovejoy <i>et al.</i> , 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 and Procter & Gamble Co.
(Mahon <i>et al.</i> , 2007)	Age 50-80y BMI 25-35 Generally healthy No T2DM Post-menopausal	USA 0% Male Age: (58) BMI: (30)	Parallel Group	9 weeks	All food provided	57	1. Control 2. Energy restriction + beef 3. Energy restriction + chicken 4. Energy restriction + carbohydrate/fat	1. Habitual diet 2. Energy restricted diet (1000 kcal/day) lacto-ovo vegetarian diet plus 250kcal/d from beef 3. Energy restricted diet (1000 kcal/day) lacto-ovo vegetarian diet plus 250kcal/d from chicken 4. Energy restricted diet (1000 kcal/day) lacto-ovo vegetarian diet plus 250kcal/d from carbohydrate/fat foods (shortbread cookies and sugar coated chocolates)	1. %E: C 47 P 20 F 33 Energy: 1570 kcal/d 2. %E: C 46 P 24 F 30 Energy: 1114 kcal/d 3. %E: C 51 P 25 F 24 Energy: 1098 kcal/d 4. %E: C 59 P 17 F 24 Energy: 1158	Yes	Cattlemen's Beef Board and the National Cattlemen's Beef Association, research institute funding and University funding

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(Maki <i>et al.</i> , 2007)	<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	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
(McMillan-Price <i>et al.</i> , 2006)	<150 kg <5kg Δ weight in the previous 2m Age 18-40y BMI >25 Maintain current PA levels No chronic illness No medication	Australia 24% Male Age: (32) BMI: (31)	Parallel Group	12 weeks	All food provided	129	1. High CHO, high GI diet 2. High CHO, low GI diet 3. High protein, high GI diet 4. High protein, low GI diet	All groups: 1400 kcal/d women and 1900 kcal/d men. 1. 55% CHO, 15% PRO, <30% FAT, fibre 30g/d. Diet based on high-GI wholegrains, fibre-rich cereals/breads. GI 70, GL 127g 2. 55% CHO, 15% PRO, <30% FAT, fibre 30g/d. Diet based on low-GI food. GI 45, GL 89g 3. 45% CHO, 25% PRO, <30%FAT, fibre 30g/d. Diet based on lean red meat and	1. %E: C 60 P 18 F 19 Energy: 9630kJ/d Fibre g/d:23 2. %E: C 56 P 19 F 22 Energy: 9030kJ/d Fibre g/d:20 3. %E: C 42 P 28 F 27 Energy: 9220kJ/d Fibre g/d:19	Yes	National Heart Foundation of Australia and Meat and Livestock Australia

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								high-GI CHO wholegrains. GI 59, GL 75g 4. 45% CHO, 25% PRO, <30%FAT, fibre 30g/d. Diet based on lean red meat and low-GI CHO foods.GI 54, GL 59g	4. %E: C 40 P 26 F 29 Energy: 8890kJ/d Fibre g/d:21		
(Meckling <i>et al.</i> , 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 intake >30%). 4. Hypoenergetic (-500kcal/day). Target PRO:CHO ratio 1:1 (Fat intake >30%).	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 3. %E: C 36.6 P 24.3 F 38.6 g/d: C 127 P 84 F 60 Energy: 5787kJ/d	Yes	Not reported
(Mee and Gee, 1997)	Mild hypercholesterolaemia No metabolic disease No recent change in smoking status Not taking lipid lowering drugs	USA 100% Male Age: mean not reported BMI: mean not reported	Crossover	6 weeks	Supplement	27	1. Gum arabic-supplemented apple juice 2. Filtered apple juice	1. 20 ounces/day unfiltered apple juice + gum arabic, containing 80% soluble fibre and 20% insoluble fibre. With 200mg vitamin C added as an antibrowning agent. 2. 20 ounces of commercial filtered apple juice	1. Energy: 300 kcal/d Fibre g/d:10	Yes	Tree Top, Inc.

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(Morgan <i>et al.</i> , 2009)	Age 18-70y BMI >25 Generally healthy	UK	Parallel Group	6 months	Free living diet plan	300	1. Control	1. No intervention	1. %E: C 43 P 16 F 36 Energy: 7947kJ/d	Yes	The British Broadcasting Corporation
		30% Male					2. Atkins	2. Atkins Diet - very low carbohydrate	2. %E: C 12 P 28 F 57 Energy: 6809kJ/d		
		Age: 21 - 60(40)					3. Weight Watchers	3. Weight Watchers Pure Points programme (an energy-controlled low-fat healthy eating diet)	3. %E: C 47 P 19 F 29 Energy: 6084kJ/d		
		BMI: (32)					4. Slim Fast	4. The Slim-Fast Plan (a low-fat meal replacement approach)	4. %E: C 50 P 19 F 28 Energy: 6076kJ/d		
							5. Rosemary Conley	5. Rosemary Conley's 'Eat yourself Slim' Diet and Fitness Plan (energy controlled, low-fat healthy eating diet and weekly group exercise class) Group not included as a comparison as it includes an exercise component			
(Nelson <i>et al.</i> , 1995)	Age 20-35y BMI 19-25 No chronic illness	USA	Crossover (washout 0 days)	50 days	All food provided	12	1. Low fat diet	1. Low fat diet. [for 20d prior to randomisation, all participants had stabilisation diet (39%FAT)]	1. %E: C 61.9 P 15.9 F 22.2	Yes	Not reported
		100% Male Age: (33) BMI: (23)					2. High fat diet	2. High fat diet. [for 20d prior to randomisation, all participants had stabilisation diet (39%FAT)]	2. %E: C 45.7 P 15.7 F 38.7		
(Noakes <i>et al.</i> , 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

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(Noakes <i>et al.</i> , 2005) Australian Protein Study	Age 20-65y BMI 27-40 No metabolic disease No T2DM	Australia 0% Male Age: (49) BMI: (32)	Parallel Group	12 weeks	Free living diet plan	119	1. High protein diet 2. High carbohydrate diet	1. 46%CHO, 34%PRO, 20%FAT (<10%SFA). Advise: 200g/d red meat + 100g/d lunch meat/chicken/fish 2. 64%CHO, 17%PRO, 20%FAT (<10%SFA). Advise: 80g/d chicken or pork plus bread.	1. %E: C 44.2 P 31.3 F 22.1 Energy: 5310kJ/d Fibre g/d:27.6 2. %E: C 60.8 P 17.8 F 20.1 Energy: 5219kJ/d Fibre g/d:26.1	Yes	Meat and Livestock Australia
(O'Brien <i>et al.</i> , 2005) American LC study IV	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	NIH, University funding and American Heart Association Grant-in-Aid
(Olendzki <i>et al.</i> , 2009)	Age 18-70y BMI >25	USA 16% Male Age: (48) BMI: (31)	Parallel Group	3 months	Free living diet plan	31	1. Hypoenergetic high fibre 2. Hypoenergetic low saturated fat 3. Hypoenergetic 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
(Panlasigui <i>et al.</i> , 2003)		Philippines 20% Male Age: 28 - 61(41)	Crossover (washout 2 weeks)	8 weeks	Substitution	20	1. Usual diet	1. No intervention - usual diet consumed	1. g/d: C 273.1 P 58.2 F 40.1 Fibre g/d:10.7 Energy: 1685 kcal/d	Yes	Research institute funding

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		BMI: (25)					2. Carageenan-added test foods	2. Typical Philipino test foods with carrageenan partly substituting similar items in the usual diet	2. g/d: C 315.8 P 67.4 F 38.7 Energy: 1881 kcal/d Fibre g/d:39.9		
(Pasman <i>et al.</i> , 1997a)	BMI >30 Energy restriction during trial run-in Weight loss >5kg during run-in	The Netherlands 0% Male Age: (41) BMI: (33)	Parallel Group	14 months	Supplement	39	1. Guar gum - High compliance 2. Control 3. Guar Gum - Low compliance	1. 20g 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 guar gum in 2x10g dose. 50-80% compliant	Nb. groups 1 and 3 are post-hoc defined – subjects not randomised to these groups initially	Yes	Sandoz Nutrition Ltd (Novartis Nutrition)
(Pasman <i>et al.</i> , 1997b)	BMI >30 Energy restriction during trial run-in Good compliance during run-in	The Netherlands 0% Male Age: (35) BMI: (31)	Parallel Group	14 months	Supplement	33	1. CHO/Cr-Pic (Chromium III)/Fibre/Caffeine 2. Carbohydrate supplement 3. Control	1. Group not comparable, multi-ingredient supplement. Data not extracted 2. 50g carbohydrate daily, dissolved in 250ml water (42% glucose, 58% maltodextrin) 3. No supplement	1. data not extracted 2. %E: C 50 P 13 F 36 Energy: 8100kJ/d Fibre g/d:12 3. %E: C 42 P 15 F 37 Energy: 7600kJ/d Fibre g/d:15	Yes	Novartis Nutrition Ltd
(Pelkman <i>et al.</i> , 2004)	Age 20-67y Body weight 120-135% of ideal Generally healthy Not hyperlipidaemic/hypercholesterolaemic	USA 30% Male Age: mean not reported BMI: mean not reported	Parallel Group	10 weeks	All food provided	52	1. Low fat, high carbohydrate diet 2. Moderate fat, lower carbohydrate diet	1. Energy restriction weeks1-6, weight maintenance weeks 7-10. SFA replaced by CHO 2. Energy restriction weeks1-6, weight maintenance weeks 7-10. SFA replaced by MUFA (peanuts/peanut oil)	1. %E: C 63.9 P 17.8 F 18.3 Fibre g/d:17.2 2. %E: C 50.5 P 16.8 F 32.8 Fibre g/d:17.6	No, intended diet only	The Peanut Institute
(Pereira <i>et al.</i> , 2004)	Age 18-35y BMI >25 Generally healthy No medications which influence outcomes No recent weight	USA 23.7% Male Age: (31) BMI: mean not	Parallel Group	Mean interval from baseline to follow-up = 65d in	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	1. %E: C 43 P 27 F 30 Energy: 1500 kcal/d Fibre g/d:32	Yes	National Institute of Diabetes, NIH, Digestive and Kidney Diseases, Charles H.

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	loss program Non smokers Not extremely athletic/active Weight stable	reported		low GL group and 69d in low fat				requirements). 18%FAT. GI 82, GL 205. NCEP Step 1 diet	2. %E: C 65 P 17 F 18 Energy: 1500 kcal/d Fibre g/d:20		Hood Foundation and General Mills
(Petersen <i>et al.</i> , 2006) NUGENOB	Age 20-50y BMI >30 No HTN or T2DM Not hyperlipidaemic/hypercholesterolaemic Weight stable	Europe 25% Male Age: (38) BMI: (35)	Parallel Group	10 weeks	Free living diet plan	771	1. Hypoenergetic high carbohydrate, low fat diet 2. Hypoenergetic low carbohydrate, high fat diet	1. Hypoenergetic (-600 kcal/d) 60-65% CHO, 15% PRO, 20-25% FAT 2. Hypoenergetic (-600 kcal/d) 40-45% CHO, 15% PRO, 40-45% FAT	1. %E: C 57 P 18 F 25 Energy: 1561kJ/d Fibre g/d:23 2. %E: C 43 P 17 F 40 Energy: 1620kJ/d Fibre g/d:19	Yes	European Community
(Peterson and Jovanovic-Peterson, 1995)	130-200% ideal body weight No HTN Normal glucose tolerance during pregnancies Postpartum 1-4 yrs	USA 0% Male Age: 21 - 50(36) BMI: mean not reported	Crossover	6 weeks	Substitution Meal replacement study	25	1. 40% CHO supplement bar 1st 2. 40% CHO supplement bar 2nd 3. 55% CHO supplement bar 1st 4. 55% CHO supplement bar 2nd	Bars provided to replace all meals/snacks other than evening meal. Energy restriction prescription to 1500 kcal/d 1. 180 kcal/bar. 20% protein, 40% CHO. 2. 180 kcal/bar. 20% protein, 40% CHO. 3. 180 kcal/bar. 20% protein, 55% CHO. 4. 180 kcal/bar. 20% protein, 55% CHO.		Yes	Bio-Foods Inc.
(Philippou <i>et al.</i> , 2008)	≥1 CHD risk factor Age 35-65y No chronic illness	UK 38% Male Age: mean not reported BMI: mean not reported	Parallel Group	12 weeks	Free living diet plan	18	1. Low GI 2. High GI	1. Healthy eating advice plus low GI diet (median GI: 51.3) 2. Healthy eating advice plus high GI diet (median GI: 59.3)	1. %E: C 46 P 17.1 F 32.8 Energy: 1773 kcal/d 2. %E: C 49.4 P 19.6 F 29.2 Energy: 1308 kcal/d	Yes	British Heart Foundation
(Philippou <i>et al.</i> , 2009b)	Age 18-65y BMI 27-45 Generally healthy	UK % Male: not	Parallel Group	4 months	Free living diet plan	43	1. High GI	1. 4 month GI=64, GL=137. High GI foods at each meal (white/wholemeal bread,	1. %E: C 50 P 19 F 31 Energy: 1604	Yes	Not reported

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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
	Recently involved in weight loss trial and lost at least 5% body weight	reported Age: mean not reported BMI: mean not reported					2. Low GI	cornflakes, Weetabix, potatoes, couscous, melon, pineapple and rice cakes) 2. 4 month GI=50 GL=90. Low GI food at each meal (seeded bread, brown pitta, muesli, sweet potatoes, pasta, noodles, basmati slow-cook rice, beans, lentils, apples and dried fruit)	kcal/d Fibre g/d:11 2. %E: C 48 P 20 F 32 Energy: 1604 kcal/d Fibre g/d:13		
(Philippou <i>et al.</i> , 2009a)	≥1 cardiac risk factor (BMI 27-35 kg/m ² , waist ≥94 cm, total cholesterol to high-density lipoprotein ratio ≥5.0, raised BP up to a maximum of 140/90 mm Hg) 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). GI=63, GL=175 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). GI=50.6, GL=114	Both groups decreased EI (greater in low GI group). CHO in hi GI group = 278g/d, in low GI group =224g/d but no other macronutrient differences between groups	Yes	British Heart Foundation
(Phillips <i>et al.</i> , 2008)	Age 18-50y BMI 29-39 Generally healthy No CHD, T2DM or HTN Non smokers Not hyperlipidaemic/ hypercholesterolaemic	USA 25% Male Age: mean not reported BMI: 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. American Heart Association low fat diet (30% total energy from fat). 750kcal/d energy deficit weeks 1-4.	1. g/d: C 20 2.%E: F 30	No, intended diet only	NIH and the Medical College of Wisconsin Cardiovascular centre
(Poppitt <i>et al.</i> , 2002)	≥3 metabolic syndrome risk factors Age >38y No intention to	Europe 31% Male Age: (46)	Parallel Group	6 months	Free living diet plan	46	1. Low-fat, high-simple carbohydrate diet	1. 60-70% of the diet was provided. 17.6% energy from simple CHO, 35.5% energy from complex CHO	1. %E: F 26 Energy: 7316kJ/d	Yes	EU-FAIR program and European Sugar Industries

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	begin a weight loss program Not on weight loss diet Overweight/ Obese	BMI: (32)					2. Low-fat high-complex carbohydrate diet	2. 60-70% of the diet was provided. 28.9% energy from simple CHO, 28.5% energy from complex CHO	2. %E: F 19.6 Energy: 9790kJ/d		
							3. Control diet	3. 60-70% of the diet was provided. 20.6% energy from simple CHO, 28.6% energy from complex CHO	3. %E: F 31.2 Energy: 8281kJ/d		
(Raatz <i>et al.</i> , 2005)	Age 18-70y BMI 30-40 No medical conditions which influence outcomes No medication	USA 17.2% Male Age: mean not reported BMI: (36)	Parallel Group	36 weeks	Free living diet plan	42	1. High GI diet	1. GI=63, GL=272 during feeding phase (12wk feeding phase, 24wk free living). Hypocaloric diet, feeding phase: 60%CHO, 15%PRO, 25%FAT.		Yes	NIH and research institute funding
							2. Low GI diet	2. GI=33, GL=178 during feeding phase (12wk feeding phase, 24wk free living). Hypocaloric diet, feeding phase: 60%CHO, 15%PRO, 25%FAT.			
							3. High fat diet	3. GI=59, GL=182 during feeding phase (12wk feeding phase, 24wk free living). Hypocaloric diet, feeding phase: 45%CHO, 15%PRO, 40%FAT.			
(Reppas <i>et al.</i> , 2009)	Mild to moderate lipidaemias	USA 50% Male Age: (40) BMI: mean not reported	Parallel Group	8 weeks	Supplement	40	1. Low dose Hydroxypropylmethylcellulose	1. 5g/d Hydroxypropylmethylcellulose during week 1 and weeks 3-8. Within drinks (week1) and cookies (week 1 and 3-8)		Yes	University funding and Dow Chemical Company
							2. High dose Hydroxypropylmethylcellulose	2. 15g/d Hydroxypropylmethylcellulose during week 1 and weeks 3-8. Within drinks (week1) and cookies (week 1 and 3-8)			
							3. Placebo	3. Placebo drinks			
(Romero <i>et al.</i> , 1998)	No CHD or T2DM Not	Mexico 100% Male	Parallel Group	8 weeks	Supplement	36	1. Wheat bran cookies	1. 100g/day. Equivalent to 0.6g wheat bran/d.	1. %E: C 61.1 P 9.3 F 21.2 Fibre g/d:1.9	Yes	Not reported

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	hyperlipidaemic/ hypercholesterolaemic Not taking lipid lowering drugs Sedentary only	Age: 20 - 45 BMI: 26 - 27					2. Psyllium cookies 3. Oat bran cookies	2. 100 g/day of cookies containing 87.3% total fibre and 11.2% soluble fibre. Equivalent to 1.7g psyllium/d. 3. 100 g/day of cookies containing 14.3% total dietary fibre and 4.3%. Equivalent to 2.8g of oat bran/d soluble fibre	2. %E: C 52.6 P 11.6 F 14.4 Fibre g/d: 13.1 3. %E: C 49.3 P 6.8 F 17.5 Fibre g/d: 9.6		
(Rosado <i>et al.</i> , 2008)	BMI >85th centile Generally healthy	Mexico 49% Male Age: 6 - 12 BMI: (24)	Parallel Group	12 weeks		256	1. One serving breakfast cereal/d 2. Two servings breakfast cereal/d 3. One serving breakfast cereal + nutrition education 4. Control	1. 33g breakfast cereal/d 2. 66g breakfast cereal/d 3. 33g breakfast cereal + nutrition education 4. No intervention (Choice of 3 RTEC groups 1-3)	1. g/d: C 35 P 5.8 F 0.5 Energy: 165 kcal/d 2. g/d: C 70 P 11.6 F 1 Energy: 330 kcal/d 3. g/d: C 35 P 5.8 F 0.5 Energy: 165 kcal/d	Yes	Kellogg's Company
(Ryle <i>et al.</i> , 1990)	No diabetes	UK 64% Male Age: (26) BMI: (22)	Crossover	6 weeks	All food provided	11	1. High glucose low soluble fibre 2. Low glucose high soluble fibre diet (guar gum)	1. High glucose and low soluble fibre. 75g supplement of high glucose drink (Lucozade) 2. low glucose high soluble fibre diet with 15g supplement of guar gum.		Yes	Not reported
(Sacks <i>et al.</i> , 2009)	Age 30-70y BMI 25-40 No CVD or T2DM	USA 36% Male Age: (51) BMI: (33)	Parallel Group	2 years Contact throughout 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	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	Yes	NIH

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								35% CHO	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		
(Salas-Salvado <i>et al.</i> , 2008)	Age 18-70y BMI >25 Generally healthy Highly motivated to lose weight No medication No recent weight loss program	Spain 22% Male Age: 18 - 70(48) BMI: (31)	Parallel Group	16 weeks		200	1. Mixed soluble fibre twice a day 2. Mixed soluble fibre 3 times a day 3. Placebo	1. Mixed fibre dose (3g Plantago ovata husk and 1g glucomannan) added to hypoenergetic diet (-2.5MJ/d) twice a day. 2. Mixed fibre dose (3g Plantago ovata husk and 1g glucomannan) added to hypoenergetic diet (-2.5MJ/d) three times a day. 3. 3g microcrystalline cellulose added to an energy restricted diet (reduced by 2.5MJ/d)	1. %E: C 45 P 25 F 35 2. %E: C 45 P 25 F 35 3. %E: C 45 P 25 F 35	No, intended diet only	MADAUS, S.A. and the Carlos III Health Institute funding
(Saltzman <i>et al.</i> , 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	Government funding, NIH Quaker Oats Company
(Saris <i>et al.</i> , 2000) CARMEN	Age 20-55y BMI 26-35 Generally healthy Moderate alcohol intake No weight loss >5kg in past 6m Not extremely athletic/active Not on weight loss	Denmark 49.1% Male Age: (39) BMI: (30)	Parallel Group	6 months	All food provided	398	1. Low-fat, high-simple carbohydrate diet 2. Low-fat high-complex carbohydrate diet 3. Control diet	For all groups, diets ad libitum. 60-70% food provided via study supermarket. 3. Control diet corresponds to	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	Yes	EU-FAIR and European Sugar industries

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	diet							average national intake.	Energy: 9.6kJ/d		
(Schwab <i>et al.</i> , 2006)	Abnormal glucose metabolism Age 30-65y BMI <35 No CHD No insulin treatment Not taking lipid lowering drugs Plasma glucose <8mmol/L TC <7.5mmol/L TG <4mmol/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. Polydextrose, 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
(Segal-Isaacson <i>et al.</i> , 2004)	BMI >25 No CHD or T2DM No medications which influence outcomes Post-menopausal Weight stable	USA 0% Male Age: (52) BMI: (33)	Crossover (washout 0 days)	6 weeks	All food provided	4	1. Low fat diet 2. Very low carbohydrate	1. High protein, low fat diet. Resting energy expenditure - 200kcal = approx 1400 kcal/d. Carbohydrates were provided as low GI starches and fruit. 2. Atkins type diet. Resting energy expenditure -200kcal = approx 1400 kcal/d	1. %E: C 50 P 30 F 20 2. %E: C 5 P 30 F 65	No, intended diet only	The Robert C. Atkins Foundation and research institute funding
(Seshadri <i>et al.</i> , 2005)	Age >18y BMI >35 Free of severe chronic disease No medications which influence outcomes No uncontrolled diabetes	USA 85% Male Age: mean not reported BMI: mean not reported	Parallel Group	6 months	Free living diet plan	132	1. Low carbohydrate diet 2. Standard diet, energy restricted	1. Limit CHO intake to <30g/d 2. National Heart, Lung and Blood Institute obesity management guidelines. Calorie restriction 500kcal/d.	1. %E: C 31 P 25 F 44 Energy: 1343 kcal/d 2. %E: C 51 P 16 F 32 Energy: 1590 kcal/d	Yes	Veteran Affairs Healthcare Network Competitive Pilot Project Grant

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(Sharman <i>et al.</i> , 2004) American VLC study	Generally healthy No medications which influence outcomes Non smokers Not extremely athletic/active Not on weight loss diet Weight stable	USA 100% Male Age: (33) BMI: (34)	Crossover (washout 0 days)	6 weeks	Free living diet plan	15	1. Low fat 2. Very low carbohydrate	1. <30%FAT, hypoenergetic (-500 kcal/d) <10% SAFA, <300mg cholesterol 2. <10%CHO, hypoenergetic (-500 kcal/d)	1. %E: C 56 P 20 F 23 Energy: 6540kJ/d Fibre g/d:17 2. %E: C 8 P 28 F 63 Energy: 7770kJ/d Fibre g/d:8	Yes	The Robert C. Atkins Foundation
(Sichieri <i>et al.</i> , 2007)	Age 25-45y BMI 23-30 Generally healthy No T2DM Parity ≥1 Pre-menopausal	Brazil 0% Male Age: (37) BMI: (27)	Parallel Group	18 months Monthly contact	Substitution	203	1. Low GI/GL diet 2. High GI/GL diet	1. Energy restriction 100-300kcal/d. Staple foods provided. At 18m, GI=30, GL=104 2. Energy restriction 100-300kcal/d. Staple foods provided. At 18m, GI=72, GL=280	1. %E: C 60 P F 27 Energy: 11200kJ/d Fibre g/d:36 2. %E: C 62 P F 26 Energy: 14000kJ/d Fibre g/d:45	Yes	NIH and research institute funding
(Singh <i>et al.</i> , 1992)	Data not included in review – concerns about veracity										
(Smith <i>et al.</i> , 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

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(Sondike <i>et al.</i> , 2003)	Age 12-18y BMI >95th centile Generally healthy No familial hypercholesterolaemia No T2DM	USA % Male: not reported Age: (14) BMI: (36)	Parallel Group	12 weeks	Free living diet plan	39	1. Very low carbohydrate 2. Low fat	1. Instructed to consume <20g/d CHO for 2wk then <40g/d for 10wk. Ad lib PRO and FAT 2. <30%FAT (<40g/d), plus 5 servings of starch per day (5x15g CHO per serving) and ad libitum fat-free dairy foods, fruits, vegetables.	1. %E: C 8 F 60 g/d: C 37 F 121 Energy: 1830kJ/d 2. %E: C 56 F 12 g/d: C 154 F 15 Energy: 1100kJ/d	Yes	Not reported
(Sorensen <i>et al.</i> , 2005) 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	Research institute funding and Danisco Sugar
(Stoernell <i>et al.</i> , 2008)	Mild to moderate lipidaemias No recent weight loss program No T2DM Not hyperlipidaemic/hypercholesterolaemic	USA 46-50% Male Age: Low fat group 57, Low CHO group 48 BMI: Low fat group 30, low CHO group 35	Parallel Group	8 weeks	Free living diet plan	28	1. Low carbohydrate diet 2. Low fat diet	1. Similar to Atkins but not as restrictive on quantity of carbohydrates. Goal 15% E CHO 2. Low fat diet was based on the standard dietary approach to lower elevated triacylglycerol (including weight loss)	1. %E: C 20 P 25 F 55 Energy: 5475kJ/d 2. %E: C 48 P 20 F 33 Energy: 6898kJ/d	Yes	No funding: Master of Science thesis
(Surwit <i>et al.</i> , 1997)	Generally healthy No medications which influence outcomes Non smokers Sedentary only	UK 0% Male Age: mean not reported BMI: mean not reported	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, The Sugar Association, Inc and the Kellogg Company, Inc

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(Swain <i>et al.</i> , 1990)	No HTN Not hyperlipidaemic/hypercholesterolaemic Not obese 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.		Yes	National, Heart, lung and Blood Institute and NIH
(Thompson <i>et al.</i> , 2005)	BMI 30-40 No medications which influence outcomes No supplement use Weight stable	USA 14% Male Age: mean not reported BMI: mean not reported	Parallel Group	48 weeks	Free living diet plan	90	1. Energy restricted diet 2. Energy restriction + dairy 3. Energy restriction + dairy + fibre	1. Calorie deficit of 500kcal/d. 50%CHO, 20%PRO, 30%FAT. Dairy 2 servings/d 2. Calorie deficit of 500kcal/d. 50%CHO, 20%PRO, 30%FAT. Dairy 4 servings/d (at least 2 fluid milk). 3. Calorie deficit of 500kcal/d. 50%CHO, 20%PRO, 30%FAT. Dairy 4 servings/d, high fibre	1. %E: C 54.5 P 18.8 F 26.3 Energy: 1437.1 kcal/d Fibre g/d:18.8 2. %E: C 53.6 P 21.5 F 24.6 Energy: 1490.1 kcal/d Fibre g/d:17.6 3. %E: C 58.1 P 20.9 F 20.6 Energy: 1510.2 kcal/d Fibre g/d:28.9	Yes	National Dairy Council and research institute funding
(Turley <i>et al.</i> , 1998)	Generally healthy Mild to moderate lipidaemias	New Zealand 100% Male Age: (37) BMI: (26)	Crossover (washout 1 weeks)	6 weeks	Free living diet plan	38	1. Western diet 2. Low fat, high carbohydrate diet	1. Western diet high in saturated fat, 20% TE 2. 5%TE from saturated fat	1. %E: C 43 P 16 F 36 Energy: 11400kJ/d Fibre g/d:22 2. %E: C 59 P 15 F 22 Energy: 9500kJ/d Fibre g/d:40	Yes	New Zealand Lottery Health Research
(Vido <i>et al.</i> , 1993)	Age <15y	Italy 55% Male Age: (11) BMI: mean not reported	Parallel Group	2 months	Supplement	60	1. Glucomannan supplement 2. Placebo	1. 2 glucomannan capsules one hour before every meal. Equivalent to 2g/day. 2. 2 capsules one hour before every meal.		Yes	Dicofarm

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(Williams <i>et al.</i> , 1995)	Age 2-11y Generally healthy LDL-C >110mg/dL Serum cholesterol >170mg/cL	USA % Male not reported Age: (7) BMI: (20)	Parallel Group	12 weeks	Substitution	58	1. Low soluble fibre cereal plus Step 1 diet 2. High soluble fibre cereal plus Step 1 diet	1. 28g cereal + 0.5 g soluble fibre (1/d for ages 2-5, 2/d for ages 5-11). Parents were counselled on Step 1 diet plan: 30% calories from total fat; <10% from saturated fat. 2. 28g cereal + 3.2g soluble fibre (1/d for ages 2-5, 2/d for ages 5-11). Parents were counselled on Step 1 diet plan: 30% calories from total fat; <10% from saturated fat.		Yes	Not reported
(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. GI=59.3 2. Ad libitum diet. 55%CHO, 30%FAT. At least one serving of a low GI food with each meal. GI=54.4 3. Ad libitum diet. 45%CHO, 40%FAT (20%MUFA). GI=58.6	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
(Wolever and Mehling, 2003) American GI & carbohydrate study	≥1 diabetes risk factor Age 30-65y BMI <40 Impaired glucose tolerance Not hyperlipidaemic/hypercholesterolaemic	USA % Male: not reported Age: (56) BMI: (30)	Parallel Group	4 months	Free living diet plan	37	1. High carbohydrate, high GI 2. High carbohydrate, low GI	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. GI=59.3 2. Ad libitum diet. 55%CHO, 30%FAT. At least one serving of a low GI food with each meal. GI=54.4	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: 1693 kcal/d Fibre g/d:36.2	Yes	Canadian Diabetes Association and the International Olive Oil Council

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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
							3. Low carbohydrate, high MUFA	3. Ad libitum diet. 45%CHO, 40%FAT (20%MUFA). GI=58.6	3. %E: C 47.4 P 16.4 F 35.4 Energy: 1877 kcal/d Fibre g/d:23.7		
(Wood <i>et al.</i> , 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	Yes	University funding and Nutraquest
(Wood <i>et al.</i> , 2006) American Soluble Fibre Study	DBP <90mmHg Weight loss <2.5kg in the past 6m No CHD or T2DM Not on CHO restricted diet 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 + Soluble fibre 2. Low carbohydrate diet + placebo	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: 1632 kcal/d Fibre g/d:12.7 2. %E: C 13.3 P 27.1 F 59.6 Energy: 1632 kcal/d Fibre g/d:9.6	Yes	Not reported
(Zamboni <i>et al.</i> , 1999)	Generally healthy No medication Normal glucose tolerance Normal lipid profile Pre-menopausal Weight stable	Italy 0% Male Age: (30) BMI: (31)	Parallel Group	6 months	Free living diet plan	20	1. High carbohydrate, energy restriction 2. Olive oil enriched energy restriction diet	1. Diet designed to provide 24kcal/Kg of ideal body weight. 7% of total energy from MUFA 2. Three tablespoons per day extra virgin olive oil. Diet designed to provide 24kcal/Kg of ideal body weight. 27% of total energy from MUFA	1. %E: C 60 P 15 F 25 Fibre g/d:20 2. %E: C 40 P 15 F 45 Fibre g/d:20	No, intended diet only	Research institute funding
(Zaveri and Drummond, 2009)	Age 25-50y BMI 25-35 Free of chronic disease Generally healthy Not on weight loss diet	Scotland 100% Male Age: [39.6] BMI: [29.8]	Parallel Group	12 weeks	Supplement	45	1. Control 2. Cereal bar 3. Almond snack	1. Healthy eating advice 2. Healthy eating advice plus 2 cereal bars daily (30g each) 3. Healthy eating advice plus 28g almonds/day. Group not relevant to this review so results not extracted.	Cereal bars provided: g/d C 44 P 3.0 F 4.7 Energy: 227 kcal/d	No, intended diet only	Kellogg Group

Table 2.44 RCT sources of bias

Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Abete <i>et al.</i> , 2008)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Aller <i>et al.</i> , 2004)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Andersson <i>et al.</i> , 2007)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Appel <i>et al.</i> , 2005)	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Bantle <i>et al.</i> , 2000)	No Bias	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Bell <i>et al.</i> , 1990)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Bellisle <i>et al.</i> , 2007)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Bhargava, 2006)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Birketvedt <i>et al.</i> , 2000)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Black <i>et al.</i> , 2006)	No Bias	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Brehm <i>et al.</i> , 2003)	No Bias	Bias	Bias	Unclear	Unclear	No Bias	No Bias
(Brehm <i>et al.</i> , 2005)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Cairella <i>et al.</i> , 1995)	No Bias	Unclear	No Bias	No Bias	Unclear	Bias	Bias
(Campos <i>et al.</i> , 1995)	Unclear	Unclear	Bias	No Bias	Bias	No Bias	No Bias
(Chen <i>et al.</i> , 2006)	No Bias	No Bias	No Bias	No Bias	No Bias	Bias	Bias
(Claessens <i>et al.</i> , 2009)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Clevidence <i>et al.</i> , 1992)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Clifton <i>et al.</i> , 2008)	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Clifton <i>et al.</i> , 2004)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Colette <i>et al.</i> , 2003)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Cornier <i>et al.</i> , 2005)	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Couture <i>et al.</i> , 2003)	Unclear	Unclear	Bias	No Bias	No Bias	No Bias	No Bias
(Crujeiras <i>et al.</i> , 2007)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Dale <i>et al.</i> , 2009)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Dansinger <i>et al.</i> , 2005)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Das <i>et al.</i> , 2007)	No Bias	Unclear	Bias	Unclear	No Bias	Bias	No Bias
(Davidson <i>et al.</i> , 1998)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias

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Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Davy <i>et al.</i> , 2002)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(de Luis <i>et al.</i> , 2008)	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(de Luis <i>et al.</i> , 2009b)	No Bias	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(de Luis <i>et al.</i> , 2009a)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Delbridge <i>et al.</i> , 2009)	No Bias	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Demol <i>et al.</i> , 2009)	Unclear	Unclear	Bias	Bias	No Bias	Bias	Bias
(Dreon <i>et al.</i> , 1994)	Unclear	Unclear	Bias	No Bias	Bias	No Bias	No Bias
(Drummond <i>et al.</i> , 2003)	Bias	Unclear	Bias	Unclear	Unclear	Bias	Bias
(Due <i>et al.</i> , 2008)	No Bias	No Bias	Bias	Bias	Bias	No Bias	No Bias
(Due <i>et al.</i> , 2004)	No Bias	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Due <i>et al.</i> , 2005)	No Bias	Unclear	Unclear	Unclear	Unclear	Unclear	Unclear
(Dyson <i>et al.</i> , 2007)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Ebbeling <i>et al.</i> , 2007)	No Bias	No Bias	Bias	No Bias	No Bias	No Bias	No Bias
(Ebbeling <i>et al.</i> , 2005)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Forcheron and Beylot, 2007)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Foster <i>et al.</i> , 2003)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Frisch <i>et al.</i> , 2009)	No Bias	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Furtado <i>et al.</i> , 2008)	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Garcia <i>et al.</i> , 2007)	Unclear	Unclear	No Bias	Bias	No Bias	No Bias	No Bias
(Garcia <i>et al.</i> , 2006)	Unclear	Unclear	No Bias	Bias	No Bias	No Bias	No Bias
(Gardner <i>et al.</i> , 2007)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias
(Genta <i>et al.</i> , 2009)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Ginsberg <i>et al.</i> , 1998)	No Bias	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Golay <i>et al.</i> , 1996)	Unclear	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias
(Golay <i>et al.</i> , 2000)	Unclear	Unclear	Unclear	Bias	Bias	No Bias	No Bias
(Haskell <i>et al.</i> , 1992)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Helge, 2002)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Howard <i>et al.</i> , 2006)	No Bias	Unclear	Bias	No Bias	No Bias	No Bias	No Bias

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Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Hunninghake <i>et al.</i> , 1994)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Jackson <i>et al.</i> , 1999)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Jensen <i>et al.</i> , 1997)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Jensen <i>et al.</i> , 2008)	No Bias	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Johnston <i>et al.</i> , 2004)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Johnston <i>et al.</i> , 2006)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Johnston, 1998)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Keenan <i>et al.</i> , 2007)	No Bias	Unclear	No Bias	No Bias	No Bias	Bias	Bias
(Keogh <i>et al.</i> , 2007)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Keogh <i>et al.</i> , 2008)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Kesaniemi <i>et al.</i> , 1990)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Kim <i>et al.</i> , 2008)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Kirk <i>et al.</i> , 2009)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Kirkwood <i>et al.</i> , 2007)	Unclear	Unclear	Unclear	Unclear	Unclear	Bias	Bias
(Kleemola <i>et al.</i> , 1999)	Unclear	Unclear	Bias	Bias	Unclear	No Bias	No Bias
(Knopp <i>et al.</i> , 1999)	Unclear	Unclear	No Bias	Unclear	No Bias	Bias	Bias
(Krauss <i>et al.</i> , 2006)	No Bias	No Bias	Bias	Unclear	Bias	No Bias	No Bias
(Landin <i>et al.</i> , 1992)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Landry <i>et al.</i> , 2003)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Lasker <i>et al.</i> , 2008)	No Bias	Unclear	Bias	Unclear	No Bias	Bias	Bias
(Layman <i>et al.</i> , 2005)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Layman <i>et al.</i> , 2009)	Unclear	Unclear	Bias	Unclear	No Bias	Unclear	Unclear
(Lehtimäki <i>et al.</i> , 2005)	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Leidy <i>et al.</i> , 2007)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Letexier <i>et al.</i> , 2003)	Unclear	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Ley <i>et al.</i> , 2004)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Lofgren <i>et al.</i> , 2005)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Lovejoy <i>et al.</i> , 2003)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias

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Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Mahon <i>et al.</i> , 2007)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Maki <i>et al.</i> , 2007)	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
(McMillan-Price <i>et al.</i> , 2006)	No Bias	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Meckling <i>et al.</i> , 2004)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Meckling and Sherfey, 2007)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Mee and Gee, 1997)	Unclear	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias
(Nelson <i>et al.</i> , 1995)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Noakes <i>et al.</i> , 2006)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Noakes <i>et al.</i> , 2005)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(O'Brien <i>et al.</i> , 2005)	Unclear	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Olendzki <i>et al.</i> , 2009)	No Bias	Unclear	Bias	Bias	Bias	Unclear	Unclear
(Panlasigui <i>et al.</i> , 2003)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Pasman <i>et al.</i> , 1997a)	Unclear	Unclear	Bias	Unclear	Bias	Bias	Bias
(Pasman <i>et al.</i> , 1997b)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Pekman <i>et al.</i> , 2004)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Pereira <i>et al.</i> , 2004)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Petersen <i>et al.</i> , 2006)	No Bias	No Bias	Bias	Bias	Unclear	No Bias	No Bias
(Peterson and Jovanovic-Peterson, 1995)	Unclear	Unclear	No Bias	Bias	Bias	No Bias	No Bias
(Philippou <i>et al.</i> , 2008)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Philippou <i>et al.</i> , 2009b)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Philippou <i>et al.</i> , 2009a)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Phillips <i>et al.</i> , 2008)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Poppitt <i>et al.</i> , 2002)	Unclear	Unclear	Bias	Bias	Unclear	No Bias	No Bias
(Raatz <i>et al.</i> , 2005)	Unclear	Unclear	Bias	Unclear	Bias	Bias	Bias
(Reppas <i>et al.</i> , 2009)	Unclear	Unclear	No Bias	No Bias	Unclear	Unclear	Unclear
(Romero <i>et al.</i> , 1998)	Unclear	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias
(Rosado <i>et al.</i> , 2008)	No Bias	No Bias	Bias	Unclear	No Bias	No Bias	No Bias

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Authors	Allocation sequence generation	Allocation concealment	Participant blinding	Researcher Blinding	Incomplete outcome reporting	Selective outcome reporting	Any other bias
(Ryle <i>et al.</i> , 1990)	Unclear	Unclear	Unclear	Unclear	No Bias	Unclear	Unclear
(Sacks <i>et al.</i> , 2009)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Salas-Salvado <i>et al.</i> , 2008)	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias	No Bias
(Saltzman <i>et al.</i> , 2001)	No Bias	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Saris <i>et al.</i> , 2000)	No Bias	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Schwab <i>et al.</i> , 2006)	Unclear	Unclear	No Bias	No Bias	Unclear	No Bias	No Bias
(Segal-Isaacson <i>et al.</i> , 2004)	Unclear	Unclear	Bias	Bias	No Bias	No Bias	No Bias
(Seshadri <i>et al.</i> , 2005)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Sharman <i>et al.</i> , 2004)	Unclear	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Sichieri <i>et al.</i> , 2007)	No Bias	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Smith <i>et al.</i> , 2008)	No Bias	Unclear	No Bias	No Bias	No Bias	Bias	Bias
(Sondike <i>et al.</i> , 2003)	Unclear	Unclear	Bias	Bias	Bias	No Bias	No Bias
(Sorensen <i>et al.</i> , 2005)	No Bias	Unclear	Unclear	Unclear	No Bias	No Bias	No Bias
(Stoernell <i>et al.</i> , 2008)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Surwit <i>et al.</i> , 1997)	Unclear	Unclear	Unclear	Unclear	Bias	No Bias	No Bias
(Swain <i>et al.</i> , 1990)	Unclear	Unclear	No Bias	No Bias	Bias	No Bias	No Bias
(Thompson <i>et al.</i> , 2005)	No Bias	No Bias	Bias	Bias	No Bias	No Bias	No Bias
(Turley <i>et al.</i> , 1998)	Unclear	Unclear	Bias	Unclear	No Bias	No Bias	No Bias
(Vido <i>et al.</i> , 1993)	No Bias	Unclear	No Bias	No Bias	No Bias	Bias	Bias
(Williams <i>et al.</i> , 1995)	Unclear	Unclear	Unclear	No Bias	Unclear	No Bias	No Bias
(Wolever and Mehling, 2003)	No Bias	Unclear	Bias	Unclear	Unclear	No Bias	No Bias
(Wolever and Mehling, 2002)	No Bias	Unclear	Bias	Unclear	Unclear	Bias	Bias
(Wood <i>et al.</i> , 2007)	No Bias	Unclear	No Bias	No Bias	No Bias	No Bias	No Bias
(Wood <i>et al.</i> , 2006)	No Bias	Unclear	No Bias	No Bias	Unclear	No Bias	No Bias
(Zambon <i>et al.</i> , 1999)	Unclear	Unclear	Bias	Unclear	Bias	No Bias	No Bias
(Zaveri and Drummond, 2009)	No Bias	Unclear	Bias	Unclear	No Bias	No Bias	No Bias

Results – Total carbohydrate and high carbohydrate diets

Information on the carbohydrate, fat and protein content of the diets investigated is provided in the Trial Characteristics table, but also in summary tables provided in Appendix 1. In these summary tables, all studies that manipulated the percentage of energy from the macronutrients and that provided outcome data on blood lipids are listed according to whether the dominant dietary change was in carbohydrate and fat, carbohydrate and protein or whether the changes involved all three macronutrients. Studies are listed by publication year, as in the Forest plots. Trials were separated into three main types on the basis of the proportion of energy derived from the macronutrients. For inclusion in a meta-analysis a 5% difference in energy from carbohydrate was taken as meaningful. Actual consumption was used rather than the intended diet unless otherwise stated – see the Trial Characteristics table.

If a trial tested the effects of diets which differed by 5% or more of energy from carbohydrate it was then further categorised into one of three categories. Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat also differed by 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein differed by 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more, but protein intakes were also different by more than 2%.

Incident hyperlipidaemias and total carbohydrate

Summary of cohort results

Data on the relationship between baseline carbohydrate intakes and risk of incident hypercholesterolaemia were provided by 2 cohort studies.

Data were extracted from one study of young adults. In the CARDIA study (Iribarren *et al.*, 1997) the baseline consumption of total carbohydrates in those with total cholesterol above (cases) or below (non-cases) the 10th centile at follow-up was reported. No carbohydrate differences were found by gender or race in those who became cases compared with those whose cholesterol remained below the 10th centile. This study reported carbohydrate intake as a percentage of total energy and this was assessed using a 700 item FFQ. The mean intakes presented were unadjusted for covariates.

One cohort study (the Amsterdam Growth and Health Study) of children initially aged 13-16 years also provided the odds of having a raised total serum cholesterol in association with increasing total carbohydrate intake as assessed by the dietary history method (Twisk *et al.*, 1997). This publication did not provide odds ratios for associations that were not statistically significant, but the authors stated that there was no association between carbohydrate intake and total serum cholesterol.

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

No RCTs reported outcomes concerning total carbohydrate intake and incident hyperlipidaemia.

Table 2.45 Incident hyperlipidaemias and total carbohydrate: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range %Male	Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group Details	Exposure Units	Mean exposure in cases and non-cases
(Iribarren <i>et al.</i> , 1997) 15102 The CARDIA Study	USA, Not hyperlipidaemic	18-30 %M 47	5115	7 years (19)	FFQ (700)	Carbohydrate (% energy)	Achieving low total cholesterol (≤10th centile) Experimenter/ clinic assessed	Race - Black Men	% Energy	Cases: 46.4 Non-cases: 46.1
15705 The CARDIA Study								Race - White Men	% Energy	Cases: 47.4 Non-cases: 47.3
15706 The CARDIA Study								Race - Black Women	% Energy	Cases: 48.3 Non-cases: 49.9
15707 The CARDIA Study								Race - White Women	% Energy	Cases: 47.8 Non-cases: 46.3

Table 2.46 Incident hyperlipidaemia and total carbohydrate: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub- group Details	Contrast	Exposure Units	RR (CI)	p	Adjustments
(Twisk <i>et al.</i> , 1997) Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 46	233	14 years	Dietary history	Carbohydrate, total (% energy)	Total cholesterol* Serum		Continuous risk estimate	Per 1% carbohydrate energy	Not reported	NS	age, gender, sum of skinfolds, VO2 max

*Odds of total cholesterol >4.2mmol/L (participants aged 13-16 years) >5.2mmol/l (participants aged 21-27 years)

Total cholesterol, total carbohydrate and high carbohydrate diets

Summary of cohort results

Data were extracted from two cohort studies that reported total cholesterol expressed as a continuous variable in relation to baseline carbohydrate intake (Schroeder *et al.*, 2007; Boreham *et al.*, 1999). Neither study provided evidence of an association between total carbohydrate expressed as grams per day (Schroeder *et al.*, 2007) or per cent energy (Boreham *et al.*, 1999) and total cholesterol levels in the blood or serum.

The Middle-aged Runners Study (Schroeder *et al.*, 2007) presented total carbohydrates in grams per day as assessed by a food diary whilst The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) used a dietary history method to assess total carbohydrate as percentage energy. The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) adjusted for socio-economic status and sexual maturity but the Middle-aged Runners Study (Schroeder *et al.*, 2007) only adjusted for age.

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

Summary of RCT data

Fifty eight studies, presented in sixty two papers, explored the effects of dietary variation in the carbohydrate proportion of diets – replacing carbohydrate with fat, protein or both – on total blood cholesterol.

Nine studies employed a crossover design (Furtado *et al.*, 2008; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Dreon *et al.*, 1994; Ginsberg *et al.*, 1998; Nelson *et al.*, 1995; Segal-Isaacson *et al.*, 2004; Turley *et al.*, 1998), one implemented a factorial design (Dale *et al.*, 2009) and the remaining used a parallel group design.

Trials were conducted in a variety of countries, which included: Australia (6), the UK (3), Canada (3), New Zealand (3), Switzerland (2), Spain (1), Israel (1), France (1), Denmark (1), Germany (1), Sweden (1), Italy (1), the Netherlands (1) and one was a European trial. The majority of studies, however, were carried out in the USA (30).

The majority of trials used adults as participants, although two studies of adolescents were identified (Demol *et al.*, 2009; Sondike *et al.*, 2003). Fourteen studies recruited females only (Brehm *et al.*, 2005; O'Brien *et al.*, 2005; Brehm *et al.*, 2003; Layman *et al.*, 2005; Leidy *et al.*, 2007; Mahon *et al.*, 2007; Meckling and Sherfey, 2007; Clifton *et al.*, 2004; Cornier *et al.*, 2005; Dale *et al.*, 2009; Kirkwood *et al.*, 2007; Lofgren *et al.*, 2005; Segal-Isaacson *et al.*, 2004; Zambon *et al.*, 1999; Howard *et al.*, 2006; Noakes *et al.*, 2005) and ten studied males (Krauss *et al.*, 2006; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Couture *et al.*, 2003; Dreon *et al.*, 1994; Lovejoy *et al.*, 2003; Nelson *et al.*, 1995; Turley *et al.*, 1998; Drummond *et al.*, 2003).

Most of the studies that reported total cholesterol recruited participants with a BMI greater than 25kg/m². In fact, only (Ginsberg *et al.*, 1998) and (Nelson *et al.*, 1995) used participants with a BMI indicative of a healthy weight (mean BMI: 24kg/m² and 23kg/m², respectively).

The sample sizes ranged from 4 to 811 (Segal-Isaacson *et al.*, 2004) and (Sacks *et al.*, 2009). However, the Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006) had an extremely large sample size of 48,835 (only 5.8% provided a blood sample).

Two studies were not included in the meta-analysis as participants were adolescents aged 12-18 years (Demol *et al.*, 2009; Sondike *et al.*, 2003). The study reported by Demol *et al.* compared the effects of a high carbohydrate, low fat diet with lower carbohydrate diets that varied in the proportion of energy derived from fat or protein using obese adolescents (Demol *et al.*, 2009). Total cholesterol, measured at 12 weeks and 1 year, was not significantly different between diet groups. Likewise, Sondike *et al.* (Sondike *et al.*, 2003) explored the effects of a low carbohydrate diet and a low fat diet on serum lipids in obese adolescents. After 12 weeks, total cholesterol had decreased from baseline in the low fat group ($p < 0.05$) but not in the low carbohydrate group ($p > 0.05$). This difference between groups was not statistically significant.

A number of studies provided data for multiple papers. This necessitated the selection of one paper for inclusion in the meta-analysis. This was generally the paper that provided the most complete dataset, with expression that facilitated inclusion in the meta-analysis. Data from the OmniHeart trial were reported by Appel *et al.* (Appel *et al.*, 2005) and by Furtado *et al.* (Furtado *et al.*, 2008). Data from the Appel paper, with higher participant numbers, were included in the meta-analysis. This 6-week crossover trial compared the effects of a high carbohydrate diet, a high protein diet or a high PUFA diet on lipids in participants with pre-hypertension. Total cholesterol levels at the end of the intervention had statistically significantly decreased from baseline on all three diets ($p = 0.01$ for all). The decreases in total cholesterol were greatest in the high protein and high monounsaturated fat phases relative to the high carbohydrate phase. This suggests that partial substitution of carbohydrate with either protein or monounsaturated fat can further improve lipid levels on an energy restricted diet.

Papers from Campos *et al.* (Campos *et al.*, 1995) and Dreon *et al.* (Dreon *et al.*, 1994) are from the same study. The results from Campos *et al.* (Campos *et al.*, 1995) are included in the meta-analysis.

Five studies had four groups (Mahon *et al.*, 2007;Sacks *et al.*, 2009;Dansinger *et al.*, 2005;McMillan-Price *et al.*, 2006;Krauss *et al.*, 2006). Three studies compared lowest and highest carbohydrate intakes (Mahon *et al.*, 2007;Dansinger *et al.*, 2005;Krauss *et al.*, 2006). One study compared high and low carbohydrate with medium and high protein levels (Sacks *et al.*, 2009) and one study compared higher and lower carbohydrate each on high and low glycaemic index (GI) diets (McMillan-Price *et al.*, 2006). Four studies had three groups and compared the lowest and highest carbohydrate intakes (Due *et al.*, 2008;Furtado *et al.*, 2008;Noakes *et al.*, 2006;Ginsberg *et al.*, 1998).

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

Seven studies could not be included in the meta-analysis for a variety of reasons.

Two studies were not included in the meta-analysis as the between group difference in carbohydrate energy intake was less than 5% (Dale *et al.*, 2009;Drummond *et al.*, 2003). Dale *et al.* (Dale *et al.*, 2009) reported that total cholesterol was statistically significantly lower in the high carbohydrate group compared with the high-monounsaturated fat diet. Drummond *et al.* (Drummond *et al.*, 2003), on the other hand, did not find a difference in total cholesterol in their comparison of male groups with elevated blood cholesterol receiving advice to reduce dietary fat or to reduce dietary fat and sugar.

Peterson *et al.* (Peterson and Jovanovic-Peterson, 1995) conducted a 12-week crossover trial to explore the effects of a 40% carbohydrate calorically restricted diet compared with a 55% carbohydrate calorically restricted diet using obese women. Of the 25 women who participated, 13 had gestational diabetes in their previous pregnancy. Diets were administered as nutritional supplement bars (one bar containing 40% carbohydrate, the other 55% carbohydrate) which were consumed for breakfast, lunch and as snacks. After 6 weeks on each diet, subjects in both groups tended to experience a small decrease in total cholesterol levels. However any observed changes were not statistically significant and it was concluded that there was no differential effect of the diets. These data were not included in the meta-analysis since they were reported in the paper only as four groups according to order of dietary presentation.

Four studies could not be included in the meta-analysis as insufficient data were available (because data were provided in a figure only or measures of variance were lacking, or results were expressed as percentage change only or results were described in text only with no numerical data provided). In a 12-week study, Kirkwood *et al.* randomised individuals to a low-fat, high-carbohydrate (including sucrose) energy-reduced diet or a 'no dietary change' diet or the same diets with the addition of an exercise regimen. The carbohydrate difference between the non-exercise groups was very small, but was 52 versus 44% energy in the high carbohydrate and 'no dietary advice' exercise groups respectively. No data were provided in the paper, but the authors reported that there were no differences between groups in total cholesterol after 12 weeks (Kirkwood *et al.*, 2007).

(Wolever and Mehling, 2002) compared 4-month high carbohydrate (55%) diets that were high or low GI with a lower carbohydrate (45%), high monounsaturated fat diet. Data were not provided, however the authors reported no statistically significant differences in total cholesterol between diets.

Foster *et al.* (Foster *et al.*, 2003) provided total cholesterol data as a percent change, which could not be incorporated into a meta-analysis. The authors investigated the effects of a low carbohydrate and a conventional diet in 63 obese males and females. Total cholesterol, measured at three months, was statistically significantly lower in the conventional diet group compared with the low carbohydrate diet group ($p=0.03$). This difference, however, was not apparent at six months or one year.

Johnston *et al.* compared a ketogenic low-carbohydrate (5% carbohydrate) diet with a nonketogenic low-carbohydrate (40% carbohydrate) diet in 20 obese adults for six weeks. Both diets were equally effective in terms of weight loss, but the authors reported that there were no differences between the diets in terms of cholesterol reduction (follow-up data not provided in the paper) (Johnston *et al.*, 2006).

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 lipids. However, it was included in the meta-analysis as the carbohydrate differences between the groups met the inclusion criteria of more than 5% of energy.

Nineteen studies were included in the meta-analysis comparing different carbohydrate and fat intakes and changes in total cholesterol reported as mmol/L. Nine of these studies had an energy restriction goal for at least one group (Clifton *et al.*, 2004; Colette *et al.*, 2003; Cornier *et al.*, 2005; Frisch *et al.*, 2009; Golay *et al.*, 2000; Lofgren *et al.*, 2005; Pelkman *et al.*, 2004; Petersen *et al.*, 2006; Segal-Isaacson *et al.*, 2004). The carbohydrate percentage difference between groups ranged from 6 to 45, with the lowest carbohydrate intakes being in (Segal-Isaacson *et al.*, 2004) (5% energy). Other than the latter study, the average difference in carbohydrate percentage between the highest and lowest carbohydrate study arms was in the region of 13%.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. The pooled estimate indicated that total cholesterol was 0.16mmol/L (95% CI 0.04 to 0.28) lower with consumption of a higher carbohydrate, low fat diet. This was significantly different from zero ($p=0.01$). Overall heterogeneity denoted by I^2 was 70% (95% CI 53 to 81%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was evidence that higher carbohydrate, lower fat diets are associated with lower levels of total cholesterol than lower carbohydrate, higher fat diets.

Figure 2.21 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and total cholesterol (mmol/L)

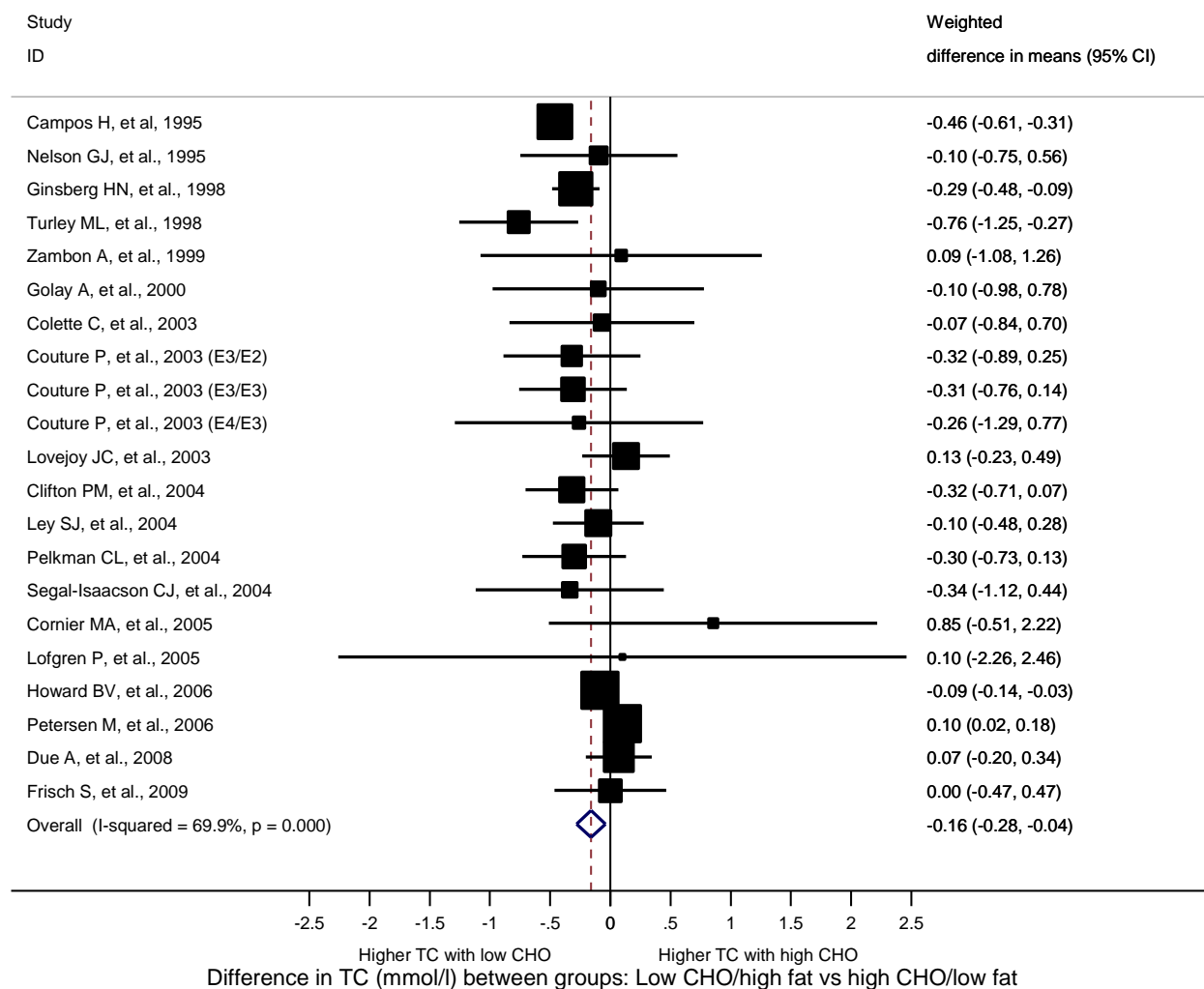
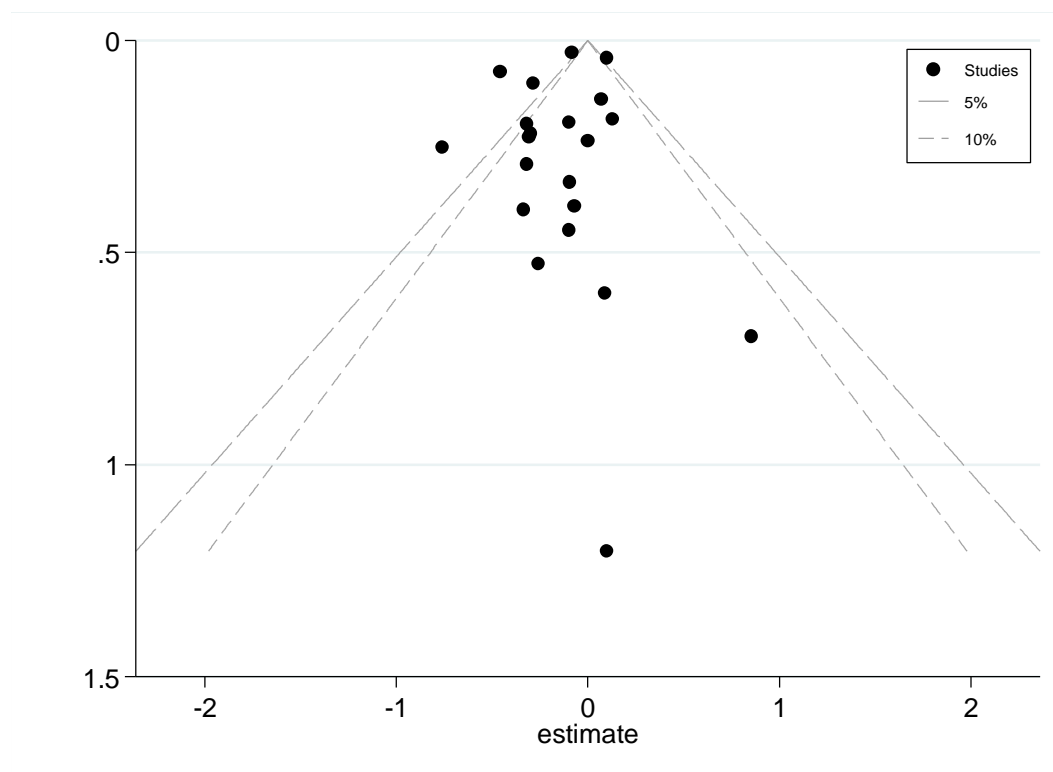


Figure 2.22 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat versus lower carbohydrate, higher fat diets and total cholesterol



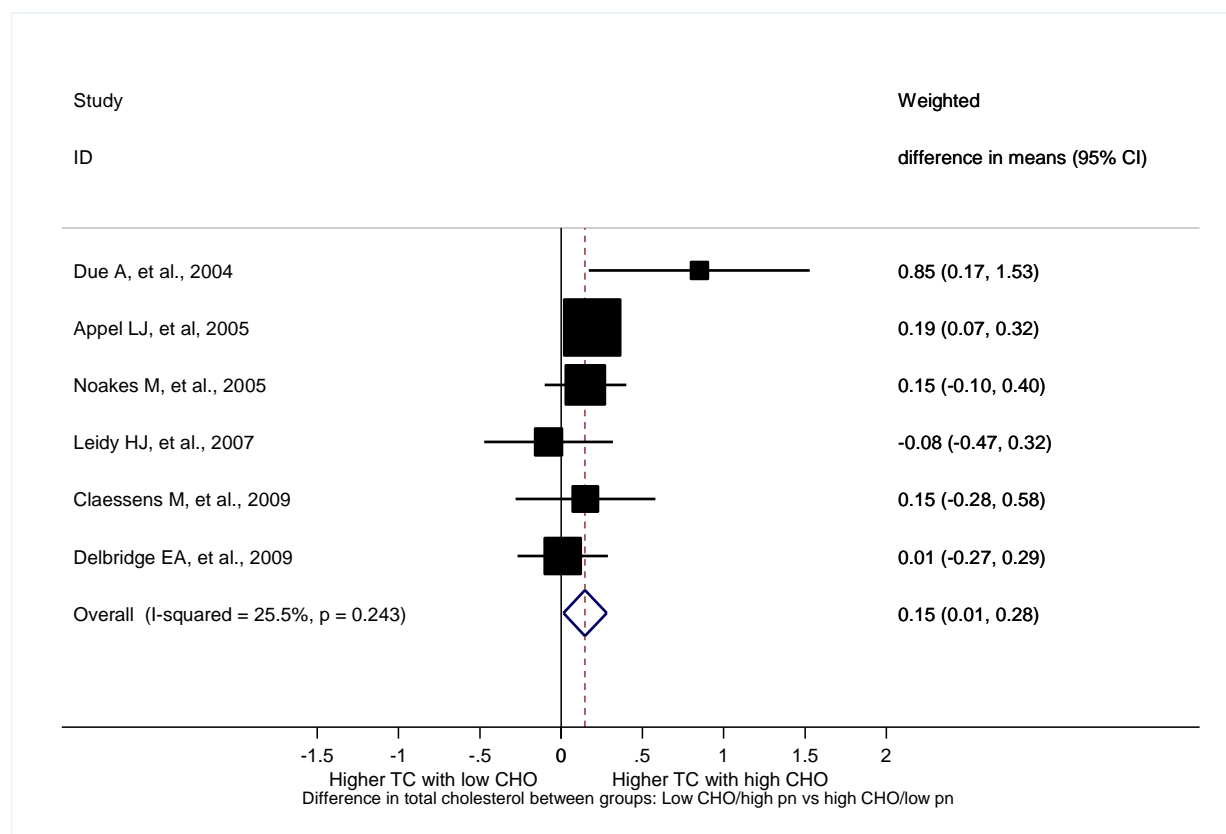
Comparison of higher carbohydrate, lower protein diets with lower carbohydrate, higher protein diets

All eligible studies were included in a meta-analysis; however there were multiple papers for some studies in which case the former one of each pair was selected for inclusion - (Appel *et al.*, 2005;Furtado *et al.*, 2008), (Due *et al.*, 2004;Due *et al.*, 2005), and (Noakes *et al.*, 2005;Clifton *et al.*, 2008).

The percentage carbohydrate in the highest intake groups ranged from 55 to 63%, and in the lower carbohydrate groups from 40 to 49. Corresponding differences in protein were 14 to 18% and 21 to 31%. Three studies prescribed an energy restriction goal (Noakes *et al.*, 2005;Leidy *et al.*, 2007;Appel *et al.*, 2005). There was a lack of consistency between the studies in terms of weight change within the high and low carbohydrate groups. Body weights were unchanged in one study (Appel *et al.*, 2005), increased in one study (Delbridge *et al.*, 2009), and decreased in three studies (Due *et al.*, 2004;Leidy *et al.*, 2007;Noakes *et al.*, 2005). In one study body weights increased in the high carbohydrate group, and decreased in the low carbohydrate group (Claessens *et al.*, 2009). This may have acted as the driver for change in cholesterol.

Six studies were included in the meta-analysis comparing different carbohydrate and protein intakes and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. The pooled estimate indicated that total cholesterol was 0.15mmol/L (95% CI 0.01 to 0.28) higher with consumption of a higher carbohydrate, low protein diet. This was significantly different from zero ($p=0.03$). Overall heterogeneity denoted by I^2 was 40% (95% CI 0 to 69%). There was evidence that higher carbohydrate, lower protein diets are associated with higher total cholesterol.

Figure 2.23 Forest plot for higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets and total cholesterol (mmol/L)



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

Data from three adult studies could not be included in the meta-analysis as insufficient data were available. One study could not be included in the meta-analysis as no measures of variation were available (Dyson *et al.*, 2007). No changes in total cholesterol between diet groups were observed.

Data from (O'Brien *et al.*, 2005) and (Layman *et al.*, 2009) could not be included due to the presentation style of the data. In Layman *et al.* (Layman *et al.*, 2009), total cholesterol was reported to be statistically significantly lower in the high carbohydrate, low protein group compared with the low carbohydrate, high protein group. However, O'Brien *et al.* did not find a differential effect of a high carbohydrate, low fat diet and a very low carbohydrate diet on total cholesterol.

Data from two papers (de Luis *et al.*, 2009a; de Luis *et al.*, 2008) which explored the dietary impact of high compared with low carbohydrate diets in individuals with different genetic profiles were not included in the meta-analysis as it was considered that these were from the same study as de Luis *et al.* (de Luis *et al.*, 2009b). Changes in total cholesterol were similar in both diet groups overall (de Luis *et al.*, 2009b). In individuals with different polymorphisms of the fatty acid binding protein 2 (FABP2) gene (de Luis *et al.*, 2008) some differences were reported. In the wild type group, total cholesterol decreased with both high and low carbohydrate diets, but no significant changes occurred in the mutant-type group.

In individuals with different polymorphisms of the uncoupling protein-3 gene (UCP-3; a gene with influence on energy expenditure and fat storage) (de Luis *et al.*, 2009a), separating participants according to genetic subgroups also showed differences in total cholesterol response. A significant improvement in total cholesterol – that is, a decrease in total cholesterol from baseline in probands with the wild type allele of the UCP-3 gene treated with the low carbohydrate diet ($p < 0.05$) was reported. In carriers of the T variant total cholesterol was unaffected by either diet.

*Nb. As there has been no evidence from the authors to suggest otherwise, it is assumed that (de Luis *et al.*, 2008; de Luis *et al.*, 2009b; de Luis *et al.*, 2009a; de Luis *et al.*, 2007) are the same study given the identical diets, same ethical submission dates (for two out of the four studies) and use of similar participants and sample sizes.*

Twenty three studies were included in the meta-analysis comparing different carbohydrate, fat and protein intakes and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. The pooled estimate indicated that total cholesterol was 0.26mmol/L (95% CI 0.12 to 0.40) lower with consumption of a higher carbohydrate diet. This was significantly different from zero ($p < 0.001$). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Overall heterogeneity denoted by I^2 was 71% (95% CI 57 to 81%). There was evidence that higher carbohydrate diets are associated with lower levels of total cholesterol.

Figure 2.24 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and total cholesterol (mmol/L)

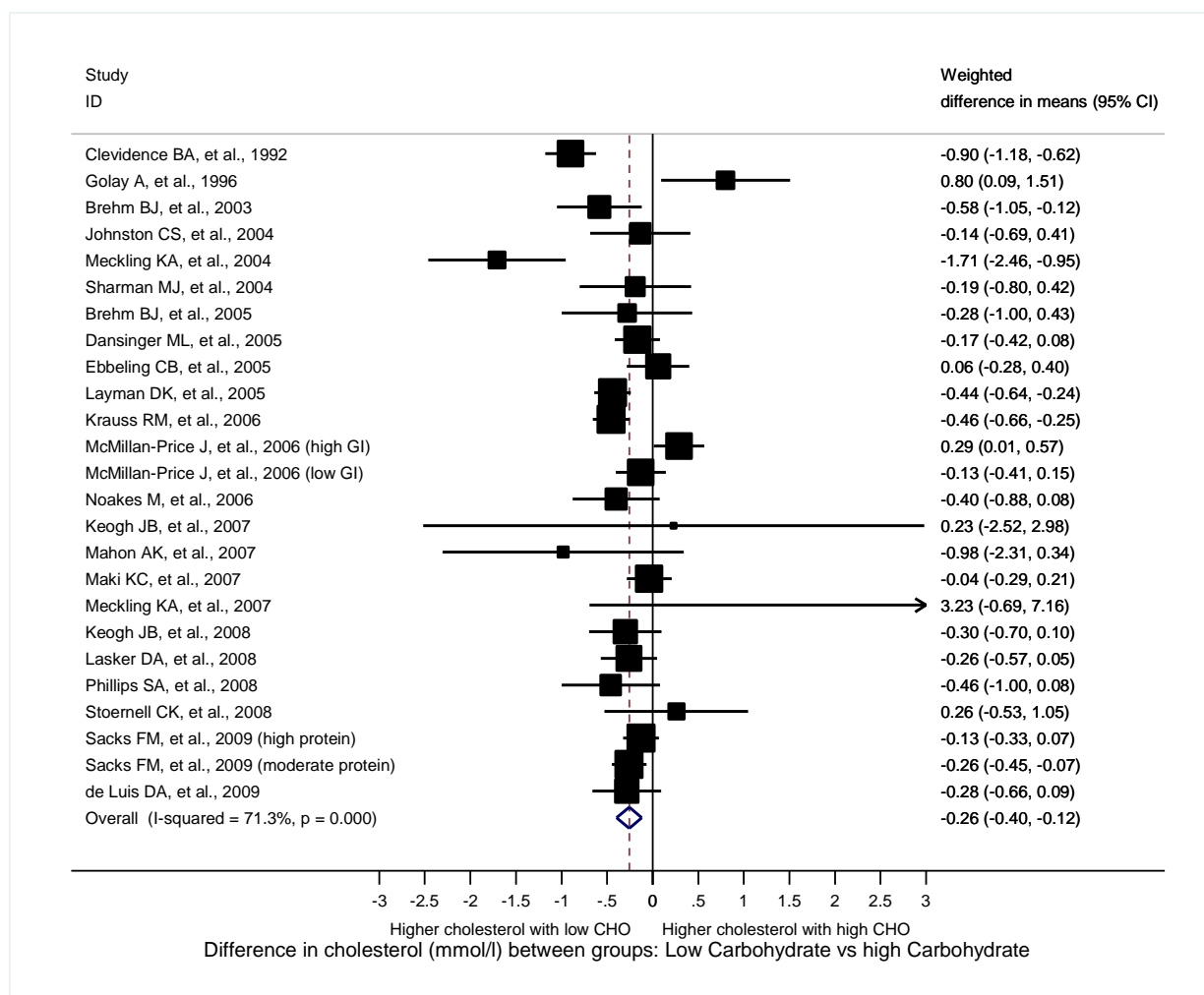


Figure 2.25 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat and protein versus lower carbohydrate, higher fat and protein diets and total cholesterol

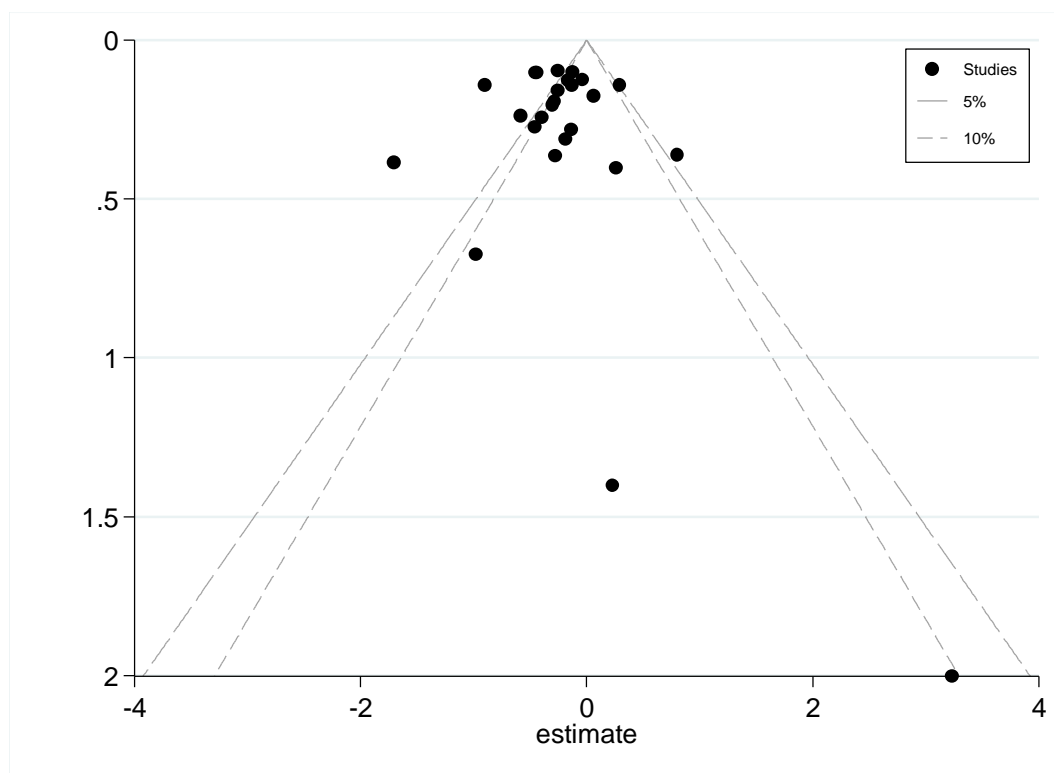


Table 2.47 Total cholesterol and carbohydrates: cohort studies in children and adults

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 Details	Exposure Units	Beta coefficient (SE)/(CI)	P trend	Adjustments
Adolescent study												
(Boreham <i>et al.</i> , 1999) 14166 The Northern Ireland Young Hearts Project	Northern Ireland, Primarily White	12-15 %M 49.3	509	4 years (1.7)	Dietary history	Carbohydrate, total (% energy)	Total cholesterol Serum	Male	1 %energy/ day	Not reported	NS	SES, sexual maturity
17605 The Northern Ireland Young Hearts Project								Female	1 %energy/ day	Not reported	NS	SES, sexual maturity
Adult study												
(Schroeder <i>et al.</i> , 2007) 14175 Middle-aged Runners Study	USA, Active people only, No CHD, No hypertension	(51) %M 62	91	10 years	Food diary	Carbohydrate, total (grams/day)	Total cholesterol Fasting		1 g/day	No effect on regression direction		Age

Table 2.48 Total cholesterol (TC) and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ	p-value within group Δ from baseline	p- diff. betwe en groups	Diff. between groups at follow- up	Diff. between groups Δ from baseline	p-value diff. between groups	Out- come	Outcome details	Result- specific follow- up	Weight change	Outcome Assess- ment Bias
Adolescent studies																
(Demol <i>et al.</i> , 2009) 15401		High carbohydrate, low fat	20/20	163.5 (SD 6.4)	147.8 (SD 6.9)							TC	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Low carbohydrate, high fat	17/17	170.5 (SD 6.9)	152.8 (SD 7.6)			NS							Decrease	
		Low carbohydrate, high protein	18/18	171.9 (SD 6.7)	148.7 (SD 7.1)			NS							Decrease	
15402		High carbohydrate, low fat	20/20	163.5 (SD 6.4)	150.4 (SD 8.0)							TC	Fasting (mg/dL)	1 year	Decrease	unclear
		Low carbohydrate, high fat	17/17	170.5 (SD 6.9)	140.7 (SD 8.9)			NS							Decrease	
		Low carbohydrate, high protein	18/18	171.9 (SD 6.7)	166.4 (SD 7.7)			NS							Decrease	
(Sondike <i>et al.</i> , 2003) 15989		Low fat	14/19			-17.3 (SD 15.8)	<0.05					TC	Fasting serum (mg/dL)	12 weeks	Decrease	bias

*Actual figure reported in paper, although this is clearly a mistake.

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ	p-value within group Δ from baseline	p- diff. betwe en groups	Diff. between groups at follow- up	Diff. between groups Δ from baseline	p-value diff. between groups	Out- come	Outcome details	Result- specific follow- up	Weight change	Outcome Assess- ment Bias	
		Very low carbohydrate	12/20			-3.7 (SD 18.0)	>0.05	NS								Decrease	
Adult studies																	
(Appel <i>et al.</i> , 2005)* Omni-Heart Study		High carbohydrate	164/164	203.7 (SD 35.7)		-12.4 (CI - 15.7, -9.1)						TC	Fasting serum (mg/dL)	6 weeks	No change	No bias	
		High protein	164/164	203.7 (SD 35.7)		-19.9 (CI - 23.5, -16.4)		<0.05							No change		
		High PUFA	164/164	203.7 (SD 35.7)		-15.4 (CI - 19.1, -11.8)		<0.05							No change		
(Brehm <i>et al.</i> , 2003) *15723		Low carbohydrate	22/22	206.32 (SE 6.63)	185.68 (SE 5.64)		<0.01	NS				TC	Fasting (mg/dL)	3 months	Decrease	unclear	
		Moderate fat	20/20	184.45 (SE 6.07)	176.25 (SE 5.87)		<0.01								Decrease		
15724		Low carbohydrate	22/22	206.32 (SE 6.63)	205.46 (SE 6.79)							TC	Fasting (mg/dL)	6 months	Decrease	unclear	
		Moderate fat	20/20	184.45 (SE 6.07)	182.85 (SE 6.21)			NS							Decrease		
(Brehm <i>et al.</i> , 2005) 16365		Low carbohydrate	20/25	205.05 (SE 9.58)	193.9 (SE 7.07)			NS				TC	Fasting plasma (mg/dL)	2 months	Decrease	No bias	
		Moderate fat	20/25	196.21 (SE 7.93)	180.65 (SE 8.74)										Decrease		
*16368		Low carbohydrate	20/25	205.05 (SE 9.58)	199.7 (SE 10.36)			NS				TC	Fasting plasma (mg/dL)	4 months	Decrease	No bias	
		Moderate fat	20/25	196.21 (SE 7.93)	188.85 (SE 9.59)										Decrease		
(Campos <i>et al.</i> , 1995) 17089		High-fat minus low- fat higher CHO	43/allocated not reported							18 (SD 19)	0.0001	TC	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear	
17094		High-fat	43/allocated not reported	206 (SD 27)	212 (SD 38)			0.0001				TC	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear	
		Low-fat higher CHO	43/allocated not reported	206 (SD 27)	194 (SD 36)										Not reported		
(Claessens <i>et al.</i> , 2009) *16821		High carbohydrate supplement	16/allocated not reported	4.15 (SE 0.27)	4.88 (SE 0.27)	0.73 (SE 0.18)	<0.05	NS				TC	Fasting (mmol/L)	12 weeks	Increase	unclear	
		High protein supplement - casein	14/allocated not reported	4.1 (SE 0.13)	4.6 (SE 0.16)	0.5 (SE 0.13)	<0.05	NS							Decrease		

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Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ	p-value within group Δ from baseline	p- diff. betwe en groups	Diff. between groups at follow- up	Diff. between groups Δ from baseline	p-value diff. between groups	Out- come	Outcome details	Result- specific follow- up	Weight change	Outcome Assess- ment Bias
		High protein supplement - whey	18/allocated not reported	4.2 (SE 0.21)	4.85 (SE 0.22)	0.64 (SE 0.14)	<0.05	NS								Decrease
(Clevidence <i>et al.</i> , 1992) *16605		High fat diet	42/46	4.73 (SE 0.09)	5.17 (SE 0.1)	0.44	<0.001	<0.001				TC	Fasting plasma (mmol/L)	10 weeks	No change	unclear
		Low fat diet	42/46	4.73 (SE 0.09)	4.27 (SE 0.1)	-0.47	<0.001								No change	
(Clifton <i>et al.</i> , 2004) 16741		High MUFA	31/35	5.45 (SD 0.83)	4.77 (SD 0.67)							TC	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		Very low fat	31/35	5.45 (SD 0.84)	4.59 (SD 0.62)										Decrease	
*16742		High MUFA	31/35	5.45 (SD 0.83)	4.95 (SD 0.77)		<0.001	Unclear				TC	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		Very low fat	31/35	5.45 (SD 0.84)	4.63 (SD 0.78)		<0.001								Decrease	
(Colette <i>et al.</i> , 2003) *17410		High carbohydrate diet	15/15	5.57 (SE 0.31)	5.15 (SE 0.33)		0.045					TC	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		High MUFA diet	17/17	5.79 (SE 0.23)	5.22 (SE 0.21)		0.0015	NS							Decrease	
(Cornier <i>et al.</i> , 2005) *16345	Insulin sensitive	Low carbohydrate, high fat	6/11	184 (SE 16)	163 (SE 14)							TC	Fasting (mg/dL)	16 weeks	Decrease	unclear
		High carbohydrate, low fat	6/10	210 (SE 22)	196 (SE 23)			NS							Decrease	
(Couture <i>et al.</i> , 2003) *15871	Genetics - Apo E genotype E3/E2	High carbohydrate diet	3/3	4.09 (SD 0.80)	2.93 (SD 0.18)		0.09	Not reported/ unclear				TC	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	5/5	3.78 (SD 0.41)	3.25 (SD 0.47)										Decrease	
*15873	Genetics - Apo E genotype E3/E3	High carbohydrate diet	22/22	4.27 (SD 1.06)	3.72 (SD 0.85)		<0.01					TC	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	21/21	4.8 (SD 0.9)	4.03 (SD 0.65)		<0.01								Decrease	
*15874	Genetics - Apo E genotype E3/E4	High carbohydrate diet	8/8	4.99 (SD 1.02)	3.98 (SD 0.88)		<0.01					TC	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	6/6	4.91 (SD	4.24 (SD		0.01								Decrease	

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Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ	p-value within group Δ from baseline	p- diff. betwe en groups	Diff. between groups at follow- up	Diff. between groups Δ from baseline	p-value diff. between groups	Out- come	Outcome details	Result- specific follow- up	Weight change	Outcome Assess- ment Bias
				1.23)	1.2)											
(Dale <i>et al.</i> , 2009) 15982		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100							0.17 (CI 0.01, 0.33)	0.04	TC	Fasting (mmol/L)	2 years	Decrease in both	unclear
17397		High carbohydrate diet	89/100	5.1 (SD 1.0)	4.82 (SD 0.87)							TC	Fasting (mmol/L)	1 year	Decrease	unclear
		High MUFA diet	85/100	5.1 (SD 1.0)	5.08 (SD 0.96)										Decrease	
17368		High carbohydrate diet	89/100	5.1 (SD 1.0)	4.98 (SD 0.93)							TC	Fasting (mmol/L)	2 years	Decrease	unclear
		High MUFA diet	85/100	5.1 (SD 1.0)	5.12 (SD 0.93)										Decrease	
(Dansinger <i>et al.</i> , 2005) 15695		Atkins	40/40			-1.8 (SD 24)	NS	Unclear				TC	Fasting serum (mg/dL)	2 months	Decrease	No bias
		Ornish	40/40			-19 (SD 28)	0.01								Decrease	
		Weight watchers	40/40			-14.8 (SD 26)	0.01								Decrease	
		Zone	40/40			-18.4 (SD 25)	0.01								Decrease	
15696		Atkins	40/40			-0.9 (SD 18)	NS					TC	Fasting serum (mg/dL)	6 months	Decrease	No bias
		Ornish	40/40			-11.4 (SD 26)	0.01								Decrease	
		Weight watchers	40/40			-8.1 (SD 21)	0.05								Decrease	
		Zone	40/40			-6.2 (SD 19)	0.05								Decrease	
*15697		Atkins	40/40			-4.3 (SD 23)	NS					TC	Fasting serum (mg/dL)	1 year	Decrease	No bias
		Ornish	40/40			-10.8 (SD 21)	0.01								Decrease	
		Weight watchers	40/40			-8.2 (SD 24)	0.05								Decrease	
		Zone	40/40			-10.1 (SD 35)	NS								Decrease	
(de Luis <i>et al.</i> , 2008) 16143	Genetics - wild-type Ala54/Ala 54	Low carbohydrate	55/105	206.8 (SD 28)	191.2 (SD 34)		<0.05	NS				TC	Fasting (mg/dL)	2 months	Decrease	unclear
		Low fat	55/99	189.4 (SD 45)	177.6 (SD 42)		<0.05								Decrease	
16161	Genetics - mutant-	Low carbohydrate	50/105	211.4 (SD 48.4)	198.7 (SD 36.4)			NS				TC	Fasting (mg/dL)	2 months	Decrease	unclear

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	type Ala54/Thr 54 or Thr54/Thr 54	Low fat	44/99	202.4 (SD 48.4)	196.7 (SD 36.4)										Decrease	
(de Luis <i>et al.</i> , 2009b) *16082		Low carbohydrate	52/52	211 (SD 34)	195 (SD 31)		<0.05	NS				TC	Fasting (mg/dL)	3 months	Decrease	unclear
		Low fat	66/66	197 (SD 44)	184.3 (SD 44)		<0.05								Decrease	
(de Luis <i>et al.</i> , 2009a) 16695	Genetics - UCP3 Gene - 55CC polymorp hism	Low carbohydrate	54/67	197.3 (SD 28.1)	183.2 (SD 34.1)		<0.05	Unclea r				TC	Serum (mg/dL)	2 months	Decrease	unclear
		Low fat	40/64	199.5 (SD 36.0)	196.6 (SD 42.0)		NS								Decrease	
16696	Genetics - UCP3 Gene - 55CT/TT polymorp hism	Low carbohydrate	13/67	186.4 (SD 48.4)	181.8 (SD 36.4)		NS					TC	Serum (mg/dL)	2 months	Decrease	unclear
		Low fat	24/64	203.4 (SD 37.0)	195.7 (SD 36.4)		NS								Decrease	
(Delbridge <i>et al.</i> , 2009) *15322		Low fat, high carbohydrate weight maintenance diet	70/70			0.41 (SE 0.11)						TC	Fasting (mmol/L)	1 year	Increase	unclear
		Low fat, high protein weight maintenance diet	68/71			0.4 (SE 0.09)		0.945							Increase	
(Dreon <i>et al.</i> , 1994) 15636	Larger LDL particles	High-fat low CHO	87/105	208.1 (SD 29.3)	208.9 (SD 4.0)		<0.0001					TC	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	208.1 (SD 29.3)	195.2 (SD 3.8)		<0.0001								Not reported	
17040	Smaller and denser LDL particles	High-fat low CHO	87/105	208.1 (SD 29.3)	225.5 (SD 9.3)		<0.0001					TC	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	208.1 (SD 29.3)	201.9 (SD 7.6)		<0.0001								Not reported	

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17047	Larger LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 87/105						-13.7 (SD 6)		<0.0001	TC	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17053	Smaller and denser LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 18/105						-23.6 (SD 4)		<0.0001	TC	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17059	LDL particles remained large during study	High-fat low CHO	87/105	208.1 (SD 29.3)	198.6 (SD 5.4)		<0.0001					TC	Derived by calculatio n Not reported, (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	208.1 (SD 29.3)	186.3 (SD 5.2)		<0.0001								Not reported	
17063	LDL particles changed from large to small and dense during study	High-fat low CHO	36/105	208.1 (SD 29.3)	223.5 (SD 5.1)		<0.001					TC	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	36/105	208.1 (SD 29.3)	207.8 (SD 4.9)		<0.001								Not reported	
17067	LDL particles remained large during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 51/105						-12.3 (SD 3)		<0.0001	TC	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17073	LDL particles changed from large to small and dense during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 36/105						-15.7 (SD 4)		<0.001	TC	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Drummond <i>et al.</i> ,		Reduced fat	completers not				NS	Not reporte				TC	Not reported	12 weeks	Not reported	unclear

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2003) 15105		Reduced fat and sugar	reported/~22 completers not reported/~22				NS	d					(mmol/L)		Not reported	
(Due <i>et al.</i> , 2004) 17536		High protein	23/23	4.86 (CI 4.2, 5.3)	4.55 (CI 4.1, 4.7)			NS				TC	Fasting (mmol/L)	6 months	Decrease	unclear
		Moderate protein	23/18	5.13 (CI 4.6, 5.5)	5.16 (CI 4.3, 5.6)										Decrease	
*17537		High protein	23/23	4.86 (CI 4.2, 5.3)	4.96 (CI 4.4, 5.5)			NS				TC	Fasting (mmol/L)	1 year	Decrease	unclear
		Moderate protein	18/18	5.13 (CI 4.6, 5.5)	5.81 (CI 5.3, 6.1)										Decrease	
(Due <i>et al.</i> , 2008) *15298		Control	24/25	4.37 (CI 4.1, 4.6)	4.57 (CI 4.1, 5.0)	0.17 (CI - 0.1, 0.4)						TC	Fasting plasma (mmol/L)	6 months	Increase	unclear
		High MUFA	39/52	4.44 (CI 4.2, 4.7)	4.38 (CI 4.2, 4.6)	-0.06 (CI - 0.3, 0.2)		NS					L)		Increase	
		Low fat	43/48	4.52 (CI 4.2, 4.8)	4.53 (CI 4.2, 4.9)	0.01 (CI - 0.2, 0.2)		NS							Increase	
(Dyson <i>et al.</i> , 2007) 16350		Healthy eating diet	4/~6	5.5	5.4	-0.1						TC	(mmol/L)	3 months	Decrease	bias
		Low carbohydrate diet	6/~6	5.5	5.7	0.2		NS							Decrease	
(Ebbeling <i>et al.</i> , 2005) 15421		Low fat diet	12/17	186 (SE 9)		-2.1% (CI - 9.2, 5.5)						TC	Fasting (mg/dL)	6 months	Decrease	unclear
		Low GI diet	11/17	191.2 (SE 9.4)		-9.9% (CI - 16.7, -2.5)		NS							Decrease	
*15476		Low fat diet	12/17	186 (SE 9)		-6.2%(CI - 15, 3.5)						TC	Fasting (mg/dL)	1 year	Decrease	unclear
		Low GI diet	11/17	191.2 (SE 9.4)		-8.5% (CI - 17.4, 1.5)		NS							Decrease	
(Foster <i>et al.</i> , 2003) 15208		Conventional diet plan	30/30			-5.4 (SD 10.1)	<0.05					TC	Fasting serum (%)	3 months	Decrease	unclear
		Low carbohydrate diet	33/33			1.7 (SD 15.0)	NS	0.03							Decrease	
15210		Conventional diet plan	30/30			-2.4 (SD 9.5)	NS					TC	Fasting serum (%)	6 months	Decrease	unclear
		Low carbohydrate diet	33/33			2.4 (SD 9.3)	NS	0.06							Decrease	
15211		Conventional diet plan	30/30			-2.9 (SD 8.0)	NS					TC	Fasting serum	1 year	Decrease	unclear

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		Low carbohydrate diet	33/33			0.1 (SD 9.8)	NS	0.27					(%)		Decrease	
(Frisch <i>et al.</i> , 2009) *15166		High carbohydrate diet	100/100			-0.07 (SD 0.5)	NS					TC	Fasting serum (mmol/L)	6 months	Decrease	unclear
		Moderate carbohydrate diet	100/100			-0.07 (SD 0.56)	NS	0.926							Decrease	
15167		High carbohydrate diet	100/100			0.13 (SD 0.61)	0.05					TC	Fasting serum (mmol/L)	1 year	Decrease	unclear
		Moderate carbohydrate diet	100/100			0.03 (SD 0.75)	NS	0.259							Decrease	
(Furtado <i>et al.</i> , 2008) 16329		High carbohydrate	111/164	213 (SD 46)		-17 (SD 29)	0.01					TC	Fasting serum (mg/dL)	6 weeks	No change	No bias
		High protein	111/164	213 (SD 46)		-25 (SD 30)	0.01	0.01							No change	
		High PUFA	111/164	213 (SD 46)		-22 (SD 30)	0.01	0.06							No change	
16330		High protein minus high carbohydrate	Crossover: 111/164							-7.7 (SD 24)	0.01	TC	Fasting serum (mg/dL)	6 weeks	No change in both	No bias
16331		High PUFA minus high carbohydrate	Crossover: 111/164							-4.6 (SD 26)	0.06	TC	Fasting serum (mg/dL)	6 weeks	No change in both	No bias
(Ginsberg <i>et al.</i> , 1998) *17247		Average American Diet	103/118		202.1 (SE 2.8)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		183.4 (SE 2.7)			<0.01							Not reported	
		Step 1 diet	103/118		191.0 (SE 2.7)			<0.01							Not reported	
17256	Men	Average American Diet	46/118		202.3 (SE 4.1)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	46/118		184.4 (SE 3.9)			<0.01							Not reported	
		Step 1 diet	46/118		191.3 (SE 4.2)			<0.01							Not reported	
17257	Women	Average American Diet	57/118		201.9 (SE 3.8)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	57/118		182.7 (SE 3.7)			<0.01							Not reported	

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		Step 1 diet	57/118		190.7 (SE 3.7)			<0.01								Not reported
17298	Black	Average American Diet	26/118		195.5 (SE 5.4)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	26/118		176.2 (SE 4.7)		<0.01								Not reported	
		Step 1 diet	26/118		184.6 (SE 4.8)		<0.01								Not reported	
17299	Non black	Average American Diet	77/118		204.3 (SE 3.2)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	77/118		185.6 (SE 3.2)		<0.01								Not reported	
		Step 1 diet	77/118		193.1 (SE 3.3)		<0.01								Not reported	
17314	Pre- menopau sal	Average American Diet	39/118		118.7 (SE 2.9)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	39/118		169.5 (SE 2.6)		<0.01								Not reported	
		Step 1 diet	39/118		177.9 (SE 2.9)		<0.01								Not reported	
17315	Post- menopau sal	Average American Diet	18/118		230.5 (SE 6.2)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	18/118		211.3 (SE 6.1)			<0.01								
		Step 1 diet	18/118		218.4 (SE 6.3)		<0.01									
17330	Men <40y	Average American Diet	30/118		192.8 (SE 4.3)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	30/118		174.5 (SE 4.3)		<0.01									
		Step 1 diet	30/118		181.6 (SE 4.5)		<0.01									
17331	Men >40y	Average American Diet	16/118		220.2 (SE 6.9)							TC	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	16/118		202.9 (SE 5.7)		<0.01									
		Step 1 diet	16/118		209.5 (SE 6.5)		<0.01									
(Golay <i>et al.</i> , 1996)		Low carbohydrate diet	completers not	5.7 (SE 0.3)	4.5 (SE 0.2)		<0.001	Not reporte				TC	Fasting plasma	6 weeks	Decrease	unclear

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*16625			reported/22					d/ unclear					(mmol/L)			
		Moderate carbohydrate diet	completers not reported/21	6.1 (SE 0.4)	5.3 (SE 0.3)		<0.01								Decrease	
(Golay <i>et al.</i> , 2000) *14852		Higher carbohydrate, macronutrients not eaten simultaneously	26/26	6.1 (SE 0.4)	5.2 (SE 0.4)		<0.01	Unclea r				TC	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Lower carbohydrate, macronutrients eaten simultaneously	28/28	6.0 (SE 0.3)	5.3 (SE 0.2)		<0.001								Decrease	
(Howard <i>et al.</i> , 2006) 16246		Control	approx n=1699 (5.8% sub-sample of 29294)	224.2 (SD 39.2)	216.6 (SD 35.9)	-6.9 (SD 31.9)						TC	Fasting (mg/dL)	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub-sample of 19541)	224.0 (SD 36.5)	214.1 (SD 35.3)	-10.2 (SD 32.0)									Decrease	
*17612		Low fat minus control	As above							-3.26 (CI - 6.53, 0.00)	<0.05	TC	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Johnston <i>et al.</i> , 2004) *14860		High carbohydrate, low fat	7/10	5.04 (SE 0.21)		-12.2% (SE 4.4% SD)	NS					TC	Serum (mmol/L)	6 weeks	Decrease	unclear
		High protein, low fat	9/10	5.07 (SE 0.31)		-9.5% (SE 3.4%)	NS	0.691							Decrease	
14868		High carbohydrate, low fat	7/10	5.04 (SE 0.21)	4.75 (SE 0.26)		0.05					TC	Serum (mmol/L)	10 weeks	Decrease	unclear
		High protein, low fat	9/10	5.07 (SE 0.31)	5.11 (SE 0.53)		0.05								Decrease	
(Johnston <i>et al.</i> , 2006) 17516		Low carbohydrate diet	10/10	5.28 (SE 0.30)								TC	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low-	9/9	5.67 (SE				NS							Decrease	

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		carbohydrate diet		0.28)												
(Keogh <i>et al.</i> , 2007) 15616		High carbohydrate diet	12/12	5.73 (SE 1.31)	4.78 (SE 1.38)		0.01					TC	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	5.32 (SE 0.88)	4.52 (SE 0.84)		0.01	NS							Decrease	
*15617		High carbohydrate diet	12/12	5.73 (SE 1.31)	4.82 (SE 1.1)		0.01					TC	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	5.32 (SE 0.88)	4.59 (SE 0.87)		0.01	NS							Decrease	
15618		High carbohydrate diet	completers not reported/12	6.08 (SE 0.52)	4.94 (SE 0.51)		0.01					TC	Fasting plasma (mmol/L)	1 year	Decrease	unclear
		Low carbohydrate diet	completers not reported/13	5.41 (SE 0.23)	4.62 (SE 0.25)		0.01	NS							Decrease	
(Keogh <i>et al.</i> , 2008) *16720		High carbohydrate, low SFA	47/50	5.3 (SD 0.8)	4.8 (SD 0.7)		<0.001					TC	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low carbohydrate, high SFA	52/57	5.4 (SD 1.1)	5.1 (SD 1.2)		<0.001	<0.05							Decrease	
(Kirkwood <i>et al.</i> , 2007) 15666		Group 1: No advice	18/allocated not reported				NS					TC	Fasting (mmol/L)	12 weeks	No change	unclear
		Group 2: Conventional weight loss diet	16/allocated not reported				NS	NS							Decrease	
15667		Group 3: Exercise	19/allocated not reported				NS	NS				TC	Fasting (mmol/L)	12 weeks	Decrease	unclear
		Group 4: Conventional weight loss diet + exercise	16/allocated not reported	4.91	4.46	0.45	0.05								Decrease	
(Krauss <i>et al.</i> , 2006) *17472		26% CHO High saturated fat	40/52	203.0 (SD 34.8)		2.2 (SE 3.3)		NS				TC	Fasting plasma (mg/dL)	12 weeks	Decrease	unclear
		26% CHO Low saturated fat	47/59	201.1 (SD 31.7)		7.0 (SE 3.1)		<0.0001							Decrease	
		39% CHO Low saturated fat	42/56	202.1 (SD 23.2)		-2.1 (SE 2.9)		NS							Decrease	
		54% CHO Low saturated fat	49/57	203.2 (SD 34.6)		-10.6 (SE 2.5)									Decrease	
(Lasker <i>et al.</i> , 2008)		High carbohydrate	25/33			-0.39 (SE 0.09)						TC	Fasting plasma	4 months	Decrease	unclear

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*15906		High protein	25/32			-0.13 (SE 0.13)		0.09					(mmol/L)		Decrease	
(Layman <i>et al.</i> , 2005)		High carbohydrate diet	12/12	5.46 (SD 0.24)	4.91 (SD 0.22)		<0.05					TC	Fasting serum (mmol/L)	16 weeks	Decrease	unclear
*16173		High protein diet	12/12	5.59 (SD 0.26)	5.35 (SD 0.28)		NS	0.06							Decrease	
16174		High carbohydrate diet + exercise	12/12	5.09 (SD 0.18)	4.63 (SD 0.16)		<0.05					TC	Fasting serum (mmol/L)	16 weeks	Decrease	unclear
		High protein diet + exercise	12/12	5.0 (SD 0.23)	4.80 (SD 0.22)		NS	0.7							Decrease	
(Layman <i>et al.</i> , 2009)		High carbohydrate, low protein diet	51/66		lower							TC	Fasting plasma	4 months	Decrease	unclear
14957		Low carbohydrate, high protein diet	52/64		higher			<0.01							Decrease	
(Leidy <i>et al.</i> , 2007)		High protein, energy restricted	21/27	190 (SE 6)	158 (SE 5)	-29 (SE 5)		0.05				TC	Fasting serum (mg/dL)	12 weeks	Decrease	unclear
*16839		Moderate protein, energy restricted	25/27	206 (SE 6)	176 (SE 5)	-32 (SE 6)									Decrease	
(Ley <i>et al.</i> , 2004)		Control	70/70			-0.07 (SE 0.18)						TC	Fasting serum (mmol/L)	6 months	No change	unclear
15929		Low fat	66/66			-0.37 (SE 0.07)		0.01							Decrease	
*15930		Control	70/70			-0.05 (SE 0.17)						TC	Fasting serum (mmol/L)	1 year	No change	unclear
		Low fat	66/66			-0.15 (SE 0.09)		NS							Decrease	
15931		Control	57/70			0.14 (SE 0.19)						TC	Fasting serum (mmol/L)	2 years	No change	unclear
		Low fat	47/66			-0.06 (SE 0.11)		NS							Decrease	
15932		Control	51/70			-0.06 (SE 0.18)						TC	Fasting serum (mmol/L)	3 years	No change	unclear
		Low fat	48/66			-0.12 (SE 0.11)		NS							Decrease	

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15933		Control	52/70			-0.15 (SE 0.18)						TC	Fasting serum (mmol/L)	5 years	No change	unclear
		Low fat	51/66			-0.2 (SE 0.11)		NS							Decrease	
(Lofgren <i>et al.</i> , 2005) *17274		High carbohydrate, low fat	20/20	4.8 (SE 0.6)	4.3 (SE 0.8)		0.0003	NS				TC	Fasting plasma (mmol/L)	10 weeks	Decrease	unclear
		High fat, moderate carbohydrate	20/20	4.4 (SE 0.7)	4.2 (SE 0.9)		0.08								Decrease	
(Lovejoy <i>et al.</i> , 2003) 14970		Control	13/15	4.58 (SE 0.2)		-0.09 (SE 0.14)		Unclea r				TC	Fasting (mmol/L)	3 months	Decrease	unclear
		Fat reduced	13/15	4.57 (SE 0.25)		0.06 (SE 0.1)									Decrease	
14972		Control	13/15	4.58 (SE 0.2)		0.19 (SE 6.04)						TC	Fasting (mmol/L)	6 months	Decrease	unclear
		Fat reduced	13/15	4.57 (SE 0.25)		0.11 (SE 0.15)									Decrease	
*14973		Control	13/15	4.58 (SE 0.2)		0.23 (SE 0.15)						TC	Fasting (mmol/L)	9 months	Decrease	unclear
		Fat reduced	13/15	4.57 (SE 0.25)		0.36 (SE 0.11)									Decrease	
(Mahon <i>et al.</i> , 2007) *15067		Control	11/11	300 (SD 70)	294 (SD 73)	-6 (SD 56)	<0.05					TC	Fasting serum (mg/dL)	9 weeks	No change	unclear
		Energy restriction + beef	14/14	241 (SD 57)	218 (SD 53)	-23 (SD 36)	<0.05	NS							Decrease	
		Energy restriction + carbohydrate/fat	14/14	284 (SD 87)	240 (SD 42)	-44 (SD 66)	<0.05	NS							Decrease	
		Energy restriction + chicken	15/15	218 (SD 37)	198 (SD 42)	-19 (SD 48)	<0.05	NS							Decrease	
(Maki <i>et al.</i> , 2007) 17278		Ad libitum low GL diet	39/43	199.3 (SE 4.5)		-12.2 (SE 2.7)		NS				TC	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	206.5 (SE 6.5)		-8.3 (SE 3.7)									Decrease	
*17279		Ad libitum low GL diet	39/43	199.3 (SE 4.5)		-1.5 (SE 3.9)		NS				TC	Fasting (mg/dL)	36 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	206.5 (SE 6.5)		-3 (SE 2.9)									Decrease	
(McMillan -Price <i>et al.</i> , 2006)		High CHO, high GI diet	32/32	4.79 (SE 0.19)		0.05 (SE 0.10)						TC	Fasting mmol/L)	12 weeks	Decrease	unclear
		High CHO, low GI	32/32	4.71 (SE		-0.18 (SE									Decrease	

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*16220		diet		0.19)		0.10)										
		High protein, high GI diet	32/32	5.15 (SE 0.18)		0.24 (SE 0.10)		0.03 (compa red with high CHO, low GI diet)							Decrease	
		High protein, low GI diet	33/33	4.83 (SE 0.14)		-0.05 (SE 0.10)									Decrease	
(Meckling and Sherfey, 2007) *16375		Hypocaloric control diet	8/15	205 (SD 56)	246 (SD 215)		NS					TC	Fasting serum (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet	10/15	178 (SD 31)	121 (SD 42)		<0.05	NS							Decrease	
16376		Hypocaloric control diet + exercise	11/15	154 (SD 55)	127 (SD 55)		<0.05					TC	Fasting serum (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet + exercise	14/15	193 (SD 58)	174 (SD 31)		NS	NS							Decrease	
(Meckling <i>et al.</i> , 2004) *14872		Low carbohydrate	15/20	230 (SE 12)	232 (SE 11)		NS	NS				TC	Fasting (mg/dL)	10 weeks	Decrease	No bias
		Low fat	16/20	228 (SE 14)	166 (SE 10)		0.05								Decrease	
(Nelson <i>et al.</i> , 1995) *16937		High fat diet	11/11	176.3 (SD 33.1)	176.9 (SD 32.9)			NS				TC	Fasting plasma (mg/dL)	50 days	Not reported	unclear
		Low fat diet	11/11	176.3 (SD 33.1)	173.2 (SD 27.3)										Not reported	
(Noakes <i>et al.</i> , 2005) 16989		High carbohydrate diet	48/48	5.88 (SE 0.14)	5.26 (SE 0.15)							TC	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		High protein diet	52/52	5.75 (SE 0.16)	5.14 (SE 0.14)			NS							Decrease	
*16990		High carbohydrate diet	48/48	5.88 (SE 0.14)	5.54 (SE 0.15)	-0.33 (SE 0.08)						TC	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	52/52	5.75 (SE 0.16)	5.26 (SE 0.15)	-0.48 (SE 0.10)		0.164							Decrease	
(Noakes <i>et al.</i> , 2006)		High unsaturated fat	21/27	6.09 (SE 0.23)	5.27 (SE 0.26)			Unclea r				TC	Fasting plasma	8 weeks	Decrease	unclear

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16573													(mmol/L)			
		Very low carbohydrate	24/28	5.92 (SE 0.21)	5.68 (SE 0.29)										Decrease	
		Very low fat	22/28	5.64 (SE 0.23)	4.94 (SE 0.23)										Decrease	
*16574		High unsaturated fat	21/27	6.09 (SE 0.23)	5.62 (SE 0.24)	-0.47 (SE 0.15)						TC	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Very low carbohydrate	24/28	5.92 (SE 0.21)	5.82 (SE 0.26)	-0.09 (SE 0.2)									Decrease	
		Very low fat	22/28	5.64 (SE 0.23)	5.15 (SE 0.26)	-0.49 (SE 0.14)									Decrease	
(O'Brien <i>et al.</i> , 2005) 16952		Low carbohydrate	22/22					0.12				TC	Fasting (mg/dL)	3 months	Decrease	unclear
		Moderate fat	19/19												Decrease	
(Pelkman <i>et al.</i> , 2004) 16875		Low fat, high carbohydrate diet	25/25	5.6 (SE 0.16)	4.61 (SE 0.16)		<0.05	Not reporte d/ unclear				TC	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	5.64 (SE 0.15)	4.89 (SE 0.15)		<0.05								Decrease	
*16876		Low fat, high carbohydrate diet	25/25	5.6 (SE 0.16)	4.85 (SE 0.16)		<0.05					TC	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	5.64 (SE 0.15)	5.15 (SE 0.15)		<0.05								Decrease	
16898	Weight stable during maintena nce	Low fat, high carbohydrate diet	12/25	5.59 (SE 0.23)	4.50 (SE 0.23)		<0.05					TC	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	17/27	5.83 (SE 0.19)	5.02 (SE 0.19)		<0.05								Decrease	
16899	Weight stable during maintena nce	Low fat, high carbohydrate diet	12/25	5.59 (SE 0.23)	4.99 (SE 0.16)		<0.05					TC	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	17/27	5.83 (SE 0.19)	5.34 (SE 0.15)		<0.05								Decrease	
(Petersen <i>et al.</i> ,	Women	Hypoenergetic high carbohydrate, low	251/292	4.92 (SD 0.87)		-0.32 (SD 0.61)						TC	Fasting plasma	10 weeks	Decrease	bias

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2006) 17196		fat diet											(mmol/L)			
		Hypoenergetic low carbohydrate, high fat diet	235/287	4.85 (SD 0.93)		-0.2 (SD 0.54)									Decrease	
17197	Men	Hypoenergetic high carbohydrate, low fat diet	85/97	5.01 (SD 0.83)		-0.48 (SD 0.68)						TC	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
		Hypoenergetic low carbohydrate, high fat diet	77/95	5.03 (SD 0.92)		-0.41 (SD 0.57)									Decrease	
17198		Hyperenergetic low carbohydrate, high fat diet	312/382	4.9 (SD 0.93)		-0.25 (SD 0.55)						TC	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
		Hypoenergetic high carbohydrate, low fat diet	336/389	4.94 (SD 0.86)		-0.36 (SD 0.63)									Decrease	
*17214		Hypoenergetic low carbohydrate, high fat diet minus hypoenergetic high carbohydrate, low fat diet	Low CHO: 312/383 High CHO: 336/389							0.1 (CI 0.02, 0.18)	0.016	TC	Fasting plasma (mmol/L)	10 weeks	Decrease in both	bias
(Peterson and Jovanovic-Peterson, 1995) 17471	BMI - Obese (130-200% ideal BW)	40% CHO supplement bar 1st**	4/13	188 (SD 50)	176 (SD 30)		NS	Not reported				TC	Fasting serum (mg/dL)	6 weeks	Decrease	bias
		55% CHO supplement bar 1st**	6/12	217 (SD 25)	192 (SD 15)		NS								Decrease	
17476	BMI - Obese (130-200% ideal BW)	40% CHO supplement bar 2nd**	6/12	217 (SD 25)	196 (SD 25)							TC	Fasting serum (mg/dL)	6 weeks	No change	bias
		55% CHO supplement bar 2nd**	4/13	188 (SD 50)	204 (SD 25)										No change	
17481	Previous gestational DM in last pregnancy	40% CHO supplement bar 1st**	5/13	193 (SD 85)	168 (SD 26)		NS					TC	Fasting serum (mg/dL)	6 weeks	Decrease	bias

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	y	55% CHO supplement bar 1st**	4/12	206 (SD 31)	200 (SD 24)		NS									Decrease
17486	Previous gestational DM in last pregnancy	40% CHO supplement bar 2nd**	2/12	206 (SD 31)	199 (SD 30)		NS					TC	Fasting serum (mg/dL)	6 weeks	No change	bias
		55% CHO supplement bar 2nd**	5/13	193 (SD 85)	136 (SD 31)		NS									No change
(Phillips <i>et al.</i> , 2008) *17419		Low carbohydrate diet	10/~14	157.9 (SE 4.2)	163 (SE 6.1)		NS	NS				TC	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
		Low fat diet	8/~14	152.7 (SE 8.7)	145.25 (SE 8.7)		NS									Decrease
(Sacks <i>et al.</i> , 2009) 15569		High-fat, average- protein	ITT: /204	203 (SD 37)	195 (SD 39)	-3.7%		NS				TC	Fasting serum (mg/dL)	6 months	Decrease	No bias
		High-fat, high- protein	ITT: /201	204 (SD 35)	199 (SD 35)	-2.3%		NS								Decrease
		Low-fat, average- protein	ITT: /204	199 (SD 38)	188 (SD 36)	-5.9%		NS								Decrease
		Low-fat, high- protein	ITT: /202	203 (SD 36)	193 (SD 39)	-4.9%		NS								Decrease
*15570		High-fat, average- protein	ITT: /204		202 (SD 39)	-0.3%		NS				TC	Fasting serum (mg/dL)	2 years	Decrease	No bias
		High-fat, high- protein	ITT: /201		202 (SD 38)	-0.8%		NS								Decrease
		Low-fat, average- protein	ITT: /204		192 (SD 37)	-3.7%		NS								Decrease
		Low-fat, high- protein	ITT: /202		197 (SD 40)	-2.9%		NS								Decrease
(Segal- Isaacson <i>et al.</i> , 2004) *14982		Low fat diet	4/4	212 (SD 17)	130 (SD 7)		<0.05					TC	Fasting whole blood (mg/dL)	6 weeks	Decrease	unclear
		Very low carbohydrate	4/4	212 (SD 17)	143 (SD 30)		<0.05	0.378								Decrease

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ	p-value within group Δ from baseline	p- diff. betwe en groups	Diff. between groups at follow- up	Diff. between groups Δ from baseline	p-value diff. between groups	Out- come	Outcome details	Result- specific follow- up	Weight change	Outcome Assess- ment Bias
(Sharman <i>et al.</i> , 2004) *14749		Low fat	15/15	4.98 (SD 0.83)	4.25 (SD 0.75)		0.05					TC	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15	4.98 (SD 0.83)	4.44 (SD 0.95)		0.05	NS							Decrease	
(Stoernell <i>et al.</i> , 2008) *16520		Low carbohydrate diet	10/14	4.74 (SD 0.78)	4.47 (SD 0.45)			NS				TC	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low fat diet	13/14	4.77 (SD 1.10)	4.73 (SD 1.19)										Decrease	
(Turley <i>et al.</i> , 1998) *15207		Low fat, high carbohydrate diet	36/38	5.51 (SD 0.93)	4.76 (SD 1.1)			0.001				TC	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Western diet	36/38	5.51 (SD 0.93)	5.52 (SD 1.04)										Decrease	
(Wolever and Mehling, 2002) 17009		High carbohydrate, high GI	11/11					NS				TC	Fasting	16 weeks	Decrease	unclear
		High carbohydrate, low GI	13/13					NS							Decrease	
		Low carbohydrate, high MUFA	11/11					NS							Increase	
(Zambon <i>et al.</i> , 1999) 16259		High carbohydrate, energy restriction	11/11	5.35 (SD 0.68)	5.02 (SD 0.66)		NS					TC	Fasting plasma (mmol/L)	3 months	Decrease	unclear
		Olive oil enriched energy restriction diet	9/9	5.00 (SD 0.88)	4.99 (SD 1.14)		NS	NS							Decrease	
*16260		High carbohydrate, energy restriction	5/11	5.35 (SD 0.68)	4.88 (SD 1.05)		NS					TC	Fasting plasma (mmol/L)	6 months	Decrease	unclear
		Olive oil enriched energy restriction diet	7/9	5.0 (SD 0.88)	4.79 (SD 0.82)		NS	NS							Decrease	

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

**This study did not present data for groups combined

HDL cholesterol, total carbohydrate and high carbohydrate diets

Summary of cohort results

Data were extracted from three studies providing evidence concerning the association between HDL cholesterol and total carbohydrate in grams per day (Schroeder *et al.*, 2007) and as a percentage of energy intake (Boreham *et al.*, 1999; Ludwig *et al.*, 1999). Since two studies were on adults and one on children it was not appropriate to pool the studies in a meta-analysis. In the adult studies, no significant association was seen between total carbohydrate and HDL cholesterol for either the Middle-aged Runners Study (Schroeder *et al.*, 2007) or the CARDIA study (Ludwig *et al.*, 1999). The Northern Ireland Young Hearts Project which studied boys and girls initially aged 12-15 years observed a small but statistically significant decrease in HDL for each percentage increase in total energy from carbohydrates in girls, but not in boys (Boreham *et al.*, 1999). These studies therefore provide inconsistent results concerning the relationship between dietary carbohydrate and HDL cholesterol.

Exposure definition and assessment

The Middle-aged Runners Study (Schroeder *et al.*, 2007) presented total carbohydrates in grams per day as assessed by a food diary. The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) used a dietary history to assess total carbohydrates as percentage energy from carbohydrate. The CARDIA study (Ludwig *et al.*, 1999) used a FFQ with 700 food items to assess carbohydrate and sucrose as a percentage of total energy.

Adjustment for appropriate confounders

The CARDIA study (Ludwig *et al.*, 1999) adjusted for an appropriate number of variables including age, gender, smoking and physical activity. The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) adjusted for socio-economic status and sexual maturity and the Middle-aged Runners Study (Schroeder *et al.*, 2007) only adjusted for age.

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

Summary of RCT data

Sixty two studies, presented in sixty seven papers, explored the effects of dietary variation in carbohydrate diets - replacing carbohydrate with fat, protein or both - on HDL cholesterol. Details of these studies can be found in the Trial Characteristics table and in Appendix 1.

Fifty two of the sixty two studies implemented a parallel group design, nine used a crossover approach (Appel *et al.*, 2005; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Dreon *et al.*, 1994; Ginsberg *et al.*, 1998; Nelson *et al.*, 1995; Segal-Isaacson *et al.*,

2004;Turley *et al.*, 1998) and one a factorial design (Dale *et al.*, 2009). Most of the studies did not state the extent of blinding of participants and/or researchers, although 11 were open (Demol *et al.*, 2009;Dyson *et al.*, 2007;Layman *et al.*, 2009;Dale *et al.*, 2009;Due *et al.*, 2008;Foster *et al.*, 2003;Petersen *et al.*, 2006;Phillips *et al.*, 2008;Segal-Isaacson *et al.*, 2004;Sondike *et al.*, 2003;Maki *et al.*, 2007), four were double blind (Appel *et al.*, 2005;Sacks *et al.*, 2009;Ginsberg *et al.*, 1998;Lovejoy *et al.*, 2003) and four classed as single blind (Couture *et al.*, 2003;Gardner *et al.*, 2007;Ebbeling *et al.*, 2007;Howard *et al.*, 2006).

Studies were conducted in Australia (7), New Zealand (3), Canada (3), Switzerland (2), Denmark (2), the UK (2), Spain, Israel, France, Germany, Scotland, Italy, the Netherlands, Europe and the UK and USA collectively. However, the majority of studies in this evidence base were carried out in the USA (33). All studies, bar two, recruited adult participants (mean age of adult trials was 44 years); the exceptions being the trials of (Demol *et al.*, 2009) and (Sondike *et al.*, 2003) which used adolescents. Most studies included both males and females, but 15 were restricted to females (Brehm *et al.*, 2005;Layman *et al.*, 2005;Leidy *et al.*, 2007;Mahon *et al.*, 2007;Meckling and Sherfey, 2007;Bhargava, 2006;Clifton *et al.*, 2004;Cornier *et al.*, 2005;Dale *et al.*, 2009;Gardner *et al.*, 2007;Kirkwood *et al.*, 2007;Segal-Isaacson *et al.*, 2004;Zambon *et al.*, 1999;Howard *et al.*, 2006;Noakes *et al.*, 2005) and nine to males only (Krauss *et al.*, 2006;Sharman *et al.*, 2004;Campos *et al.*, 1995;Clevidence *et al.*, 1992;Couture *et al.*, 2003;Dreon *et al.*, 1994;Lovejoy *et al.*, 2003;Nelson *et al.*, 1995;Turley *et al.*, 1998).

The sample sizes of the studies ranged from four to 48,335. Of these studies, two were particularly large with 2208 and 48,335 participants ((Bhargava, 2006) and (Howard *et al.*, 2006), respectively).

Six studies had four groups (Mahon *et al.*, 2007;Sacks *et al.*, 2009;Dansinger *et al.*, 2005;McMillan-Price *et al.*, 2006;Krauss *et al.*, 2006;Morgan *et al.*, 2009). Four studies compared lowest and highest carbohydrate intakes (Mahon *et al.*, 2007;Dansinger *et al.*, 2005;Krauss *et al.*, 2006;Morgan *et al.*, 2009). One study compared high and low carbohydrate with medium and high protein levels (Sacks *et al.*, 2009) and one study compared higher and lower carbohydrate on high and low GI diets (McMillan-Price *et al.*, 2006). Four studies had three groups and compared the lowest and highest carbohydrate intakes (Due *et al.*, 2008;Noakes *et al.*, 2006;Ginsberg *et al.*, 1998;Appel *et al.*, 2005).

Two studies were not included in the meta-analysis as the participants used were adolescents aged 12-18 years (Demol *et al.*, 2009;Sondike *et al.*, 2003). The study reported by Demol *et al.* compared the effects of a high carbohydrate low fat diet, with lower carbohydrate diets that varied in the proportion of energy derived from fat or protein using obese adolescents (Demol *et al.*, 2009). Likewise, Sondike *et al.* (Sondike *et al.*, 2003) explored the effects of a low carbohydrate diet and a low fat diet on serum lipids in obese adolescents. No differences in HDL cholesterol in either study were observed.

Papers from Campos *et al.* (Campos *et al.*, 1995) and Dreon *et al.* (Dreon *et al.*, 1994) are from the same study. The results from Campos *et al.* (Campos *et al.*, 1995) are included in the meta-analysis.

Trials were separated into three main types on the basis of the proportion of energy derived from the macronutrients. For inclusion in a meta-analysis a 5% difference in energy from carbohydrate was taken as meaningful. Actual consumption was used rather than the intended diet unless otherwise stated – see the Trial Characteristics table.

If a trial tested the effects of diets which differed by 5% or more of energy from carbohydrate it was then further categorised into one of three categories. Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat also differed by 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein differed by 2% or more, and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more, but protein intakes were also different by more than 2%.

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

Three studies could not be included in the meta-analysis; however, these tend to support the outcome of the meta-analysis. The RCT reported by Dale *et al.* (Dale *et al.*, 2009) could not be included in the meta-analysis as the between group difference in carbohydrate was less than 5%. HDL cholesterol did not statistically significantly differ between a high carbohydrate diet and the high monounsaturated fatty acid (MUFA) diet in this two-year study.

In a 12-week study, Kirkwood *et al.* randomised individuals to a low-fat, high-carbohydrate (including sucrose) energy-reduced diet, a 'no dietary change' diet or these diets with the addition of an exercise regimen. The carbohydrate difference between the non-exercise groups was very small, but was 52 versus 44 % energy in the high carbohydrate and 'no dietary advice' exercise groups respectively. No data were provided in the paper, but the authors reported that there were no statistically significant differences in HDL cholesterol between groups after 12 weeks (Kirkwood *et al.*, 2007).

Johnston *et al.* compared a ketogenic low-carbohydrate (5% carbohydrate) diet with a nonketogenic low-carbohydrate (40% carbohydrate) diet in 20 obese adults for six weeks. Both diets were equally effective in terms of weight loss, but the authors reported that there was no difference between the diets in terms of HDL cholesterol reduction (follow-up data not provided in the paper) (Johnston *et al.*, 2006).

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 lipids. However, it was included in the meta-analysis as the carbohydrate differences between the groups met our inclusion criteria of >5% of energy.

Twenty-two studies were included in the meta-analysis comparing different carbohydrate and fat intake and changes in HDL cholesterol reported as mmol/L. The carbohydrate percentage in the high intake groups ranged from 65 to 50%, and in the low intake groups from 5 to 51%, with the lowest carbohydrate intakes being in (Segal-Isaacson *et al.*, 2004) (5% energy). Other than the latter study, the average difference in carbohydrate percentage between the highest and lowest carbohydrate study groups was in the region of 13%. Ten studies prescribed an energy restriction goal for at least one of the dietary groups (Segal-Isaacson *et al.*, 2004; Petersen *et al.*, 2006; Pelkman *et al.*, 2004; Johnston *et al.*, 2006; Golay *et al.*, 2000; Frisch *et al.*, 2009; Foster *et al.*, 2003; Cornier *et al.*, 2005; Colette *et al.*, 2003; Clifton *et al.*, 2004).

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from five weeks to three years. The pooled estimate indicated that HDL cholesterol was 0.03mmol/L (95% CI -0.01 to 0.06) lower with consumption of a higher carbohydrate diet. This was not significantly different from zero ($p=0.11$). Overall heterogeneity denoted by I^2 was 60% (95% CI 38 to 75%). There is a suggestion of asymmetry in the funnel plot, but this could be the result of chance. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was no evidence that higher carbohydrate lower fat diets are associated with lower levels of HDL cholesterol.

Figure 2.26 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and HDL cholesterol (mmol/L)

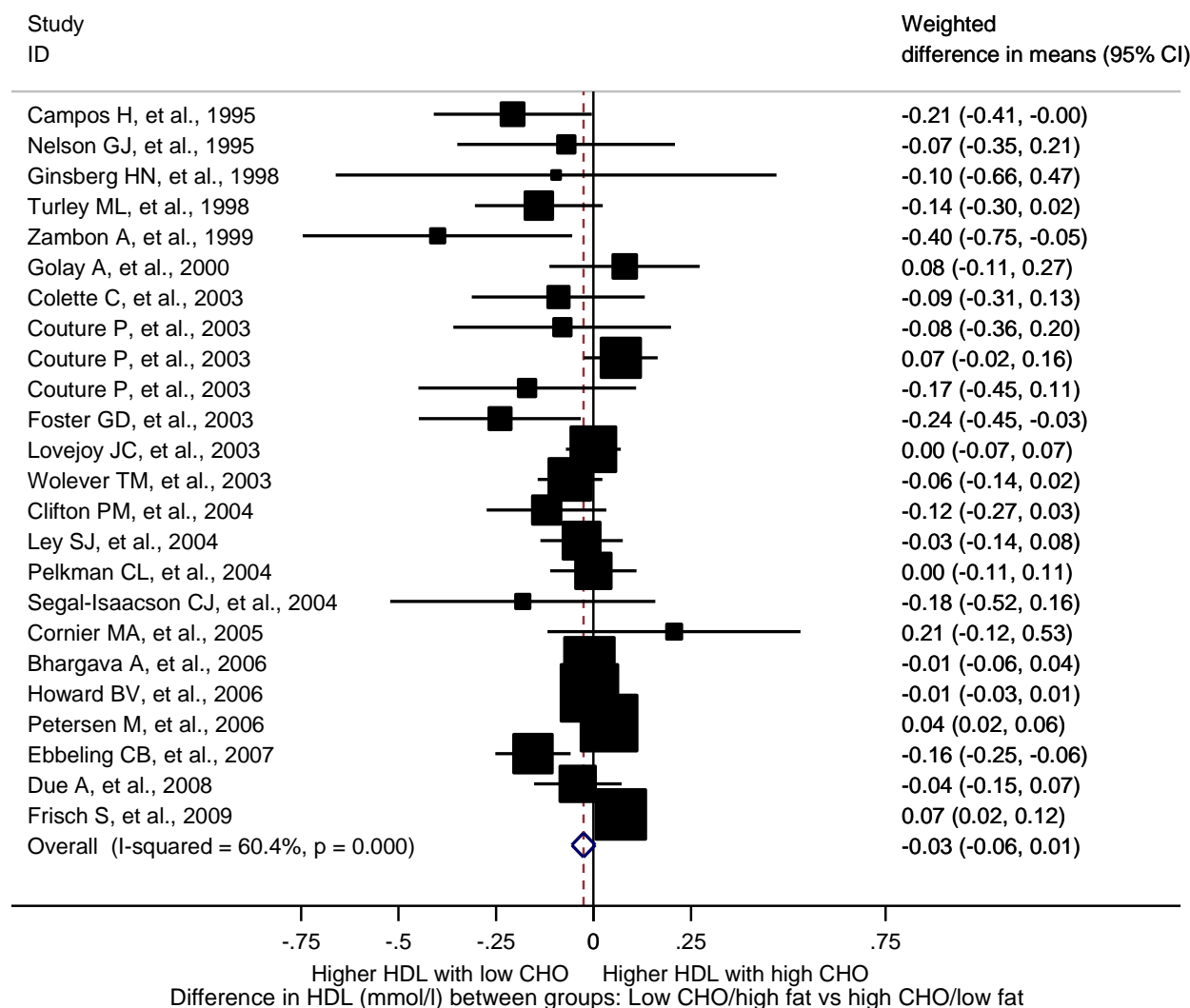
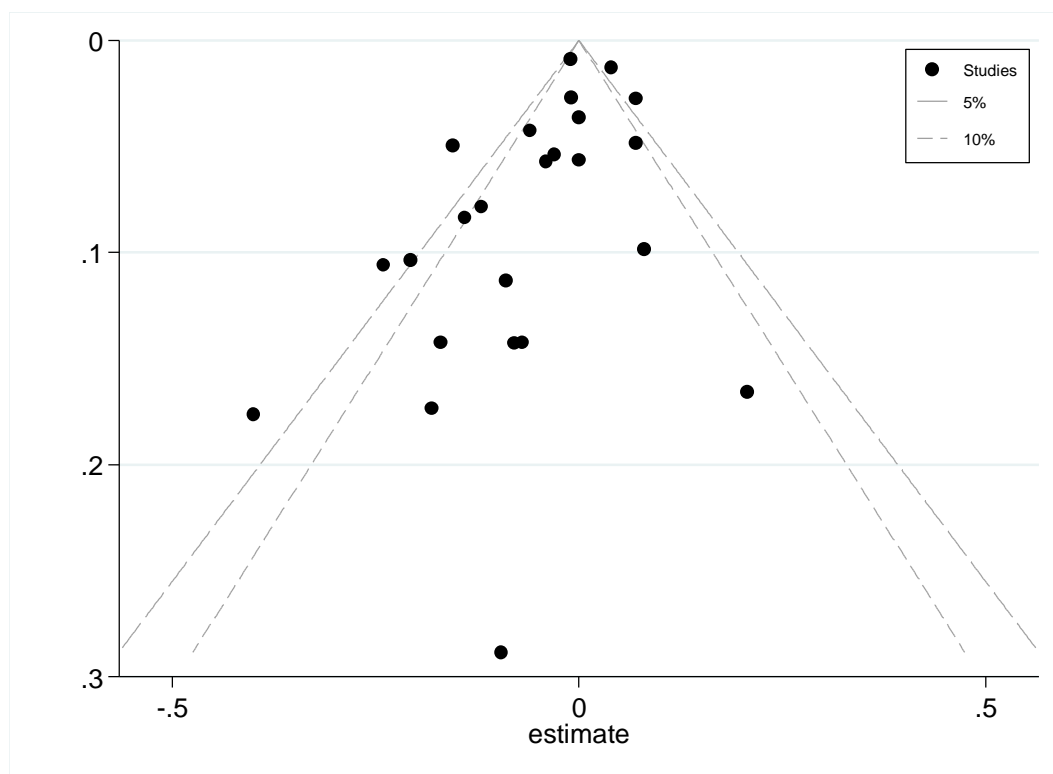


Figure 2.27 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat versus lower carbohydrate, higher fat diets and HDL cholesterol



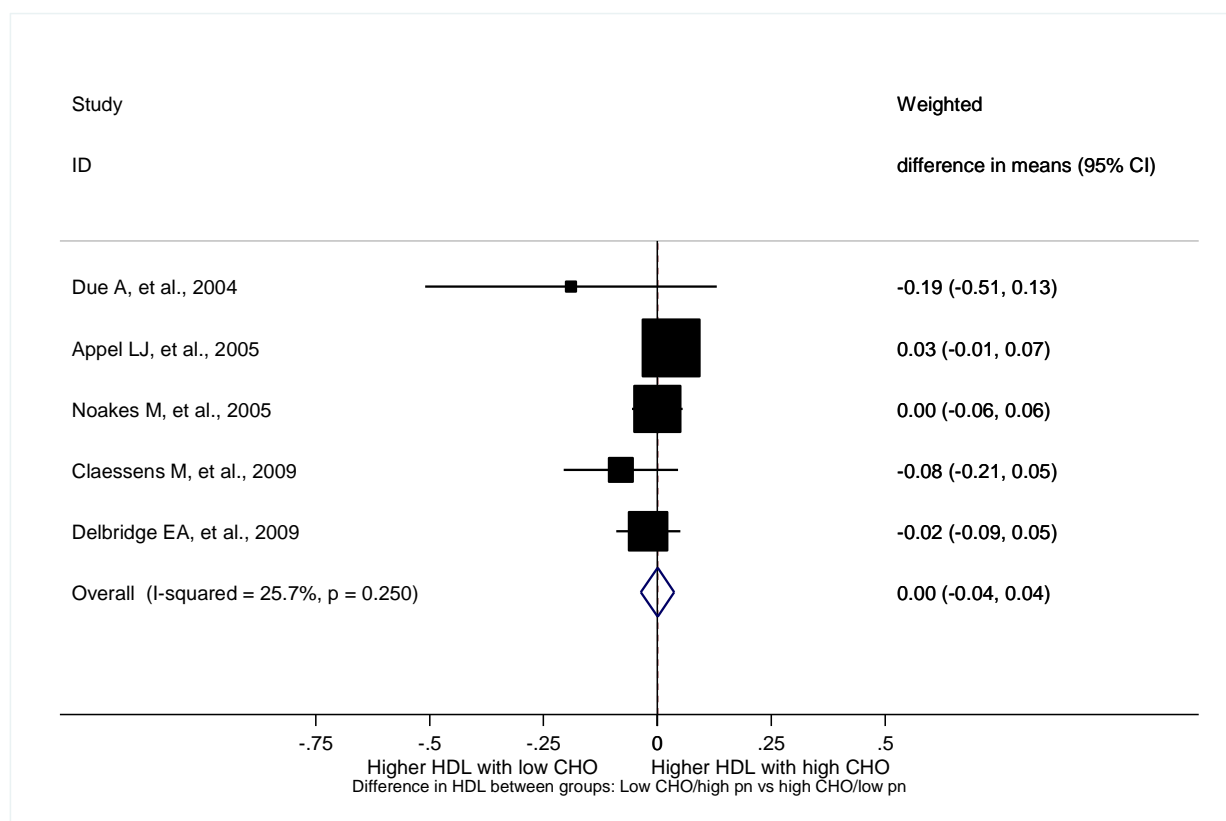
Comparison of higher carbohydrate, lower protein diets with lower carbohydrate, higher protein diets

All eligible studies were included in a meta-analysis; however there were multiple papers for some studies in which case the former one of each pair was selected for inclusion, (Appel *et al.*, 2005;Furtado *et al.*, 2008), and (Due *et al.*, 2004;Due *et al.*, 2005), and (Noakes *et al.*, 2005;Clifton *et al.*, 2008).

Five studies were included in the meta-analysis comparing different carbohydrate and protein intakes and changes in HDL cholesterol reported as mmol/L. The percentage carbohydrate in the highest intake groups ranged from 55 to 63%, and in the lower carbohydrate groups from 40 to 49%. Corresponding differences in protein were 14 to 18% and 21 to 31%. Three studies prescribed an energy restriction goal (Noakes *et al.*, 2005;Leidy *et al.*, 2007;Appel *et al.*, 2005). There was a lack of consistency between the studies in terms of weight change within the high and low carbohydrate groups. Body weights were unchanged in one study (Appel *et al.*, 2005), increased in one study (Delbridge *et al.*, 2009), and decreased in three studies (Due *et al.*, 2004;Leidy *et al.*, 2007;Noakes *et al.*, 2005). In one study body weights increased in the high carbohydrate group, and decreased in the low carbohydrate group (Claessens *et al.*, 2009). This may have acted as the driver for change in cholesterol.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from five weeks to three years. The pooled estimate indicated that HDL cholesterol was 0.0mmol/L (95% CI -0.04 to 0.04) lower with consumption of a higher carbohydrate, low protein diet. This was not significantly different from zero ($p=0.98$). Overall heterogeneity denoted by I^2 was 26% (95% CI 0 to 70%). Statistically, there was no evidence that high carbohydrate, low protein diets are associated with changes in levels of HDL cholesterol.

Figure 2.28 Forest plot for higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets and HDL cholesterol (mmol/L)



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

The papers by O'Brien *et al.* (O'Brien *et al.*, 2005) and Gray *et al.* (Gray *et al.*, 2008) were not included since they are multiple publications from the same study, which was also reported in Brehm *et al.* (Gray *et al.*, 2008). The latter was included in the meta-analysis.

Data from two adult studies could not be included in the meta-analysis as insufficient data were available. Lasker *et al.* (Lasker *et al.*, 2008) reported that in a four-month study, HDL cholesterol increased in a low carbohydrate, high protein diet group, but decreased slightly in a high carbohydrate diet group ($p=0.045$). No measures of variance around the averages were provided, so the data could not be included in the meta-analysis. Similarly, one further study could not be included in the meta-analysis as no measures of variation were available (Dyson *et al.*, 2007). HDL cholesterol increased in both high and low carbohydrate groups, but there was no significant difference between the groups in the extent of change experienced.

Data from two papers (de Luis *et al.*, 2009a; de Luis *et al.*, 2008) which explored the dietary impact of high compared with low carbohydrate diets in individuals with different genetic profiles were not included in the meta-analysis as it was considered that these were from the same study as de Luis *et al.* (de Luis *et al.*, 2009b). Changes in HDL cholesterol were similar in both diet groups overall (de Luis *et al.*, 2009b). In individuals with different polymorphisms of the fatty acid binding protein 2 (FABP2) gene (de Luis *et al.*, 2008) no differences in response to either diet were reported. Similarly, in individuals with different polymorphisms of the uncoupling protein-3 gene (a gene with influence on energy expenditure and fat storage) (de Luis *et al.*, 2009a), separating participants according to genetic subgroups also showed no differences in response.

*Nb. As there has been no evidence from the authors to suggest otherwise, it is assumed that (de Luis *et al.*, 2008; de Luis *et al.*, 2009b; de Luis *et al.*, 2009a; de Luis *et al.*, 2007) are the same study given the identical diets, same ethical submission dates (for two out of the four studies) and use of similar participants and sample sizes.*

Twenty seven studies were included in the meta-analysis comparing different carbohydrate, fat and protein intakes and changes in HDL cholesterol reported as mmol/L. There was considerable variation in the carbohydrate contents of the comparison diets. The higher carbohydrate diets ranged from 47 to 67%, and the lower carbohydrate diets from 5 to 47%. The majority of studies prescribed an energy restriction goal for at least one diet group (Brehm *et al.*, 2003; Brehm *et al.*, 2005; de Luis *et al.*, 2009b; Ebbeling *et al.*, 2005; Gardner *et al.*, 2007; Golay *et al.*, 1996; Keogh *et al.*, 2007; Keogh *et al.*, 2008; Krauss *et al.*, 2006; Layman *et al.*, 2005; Layman *et al.*, 2009; Mahon *et al.*, 2007; McMillan-Price *et al.*, 2006; Meckling *et al.*, 2004; Meckling and Sherfey, 2007; Morgan *et al.*, 2009; Noakes *et al.*, 2006; Pereira *et al.*, 2004; Phillips *et al.*, 2008; Sacks *et al.*, 2009; Sharman *et al.*, 2004; Stoernell *et al.*, 2008). Accordingly, almost all studies reported decreases in body weight in all diet groups.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from five weeks to three years. The pooled estimate indicated that HDL cholesterol was 0.06mmol/L (95% CI 0.02 to 0.10) lower with consumption of a higher carbohydrate diet. This was significantly different from zero ($p=0.006$). Overall heterogeneity denoted by I^2 was 72% (95% CI 59 to 81%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was evidence that high carbohydrate diets are associated with lower levels of HDL cholesterol.

Figure 2.29 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and HDL cholesterol (mmol/L)

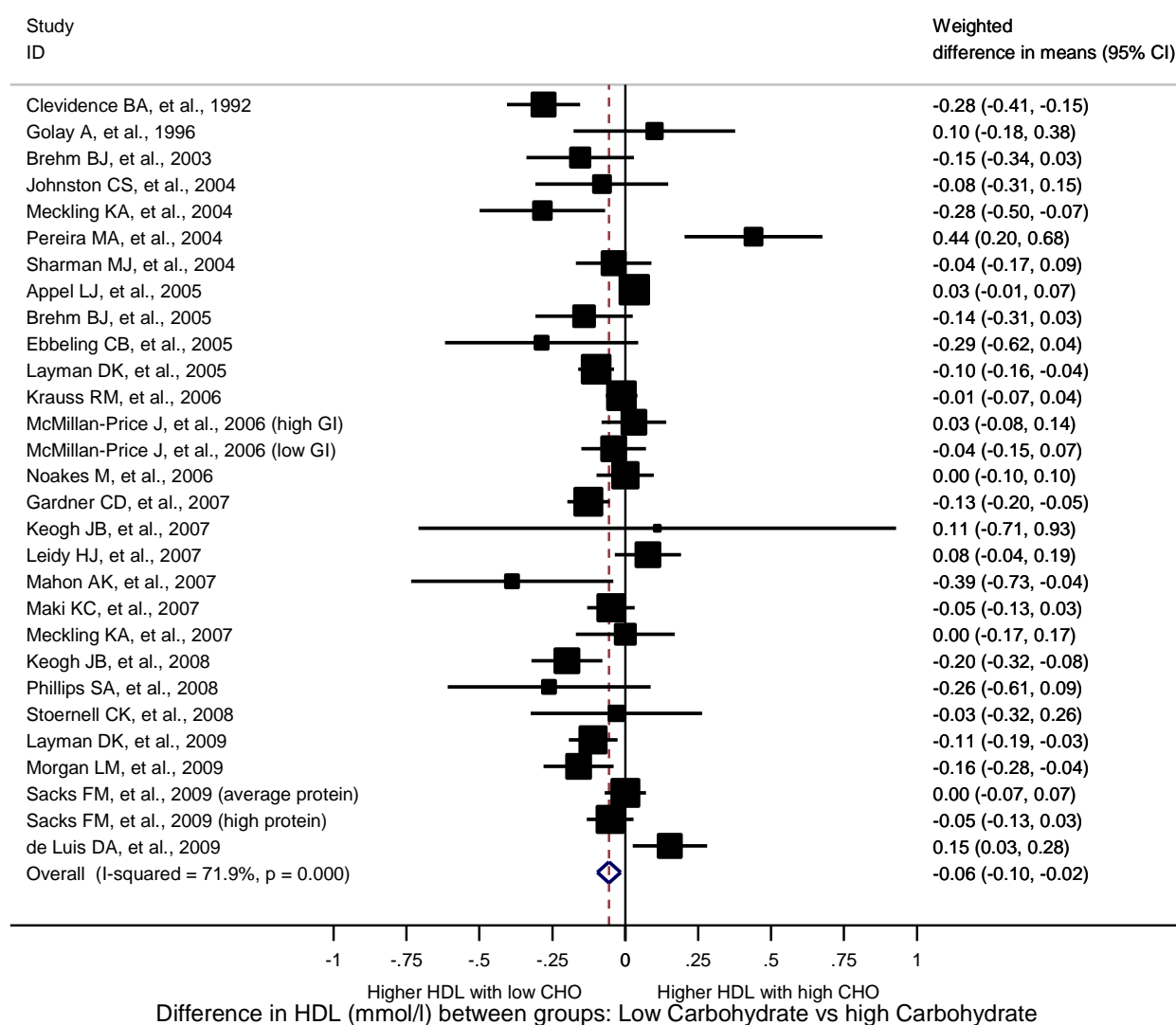


Figure 2.30 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat and protein versus lower carbohydrate, higher fat and protein diets and HDL cholesterol

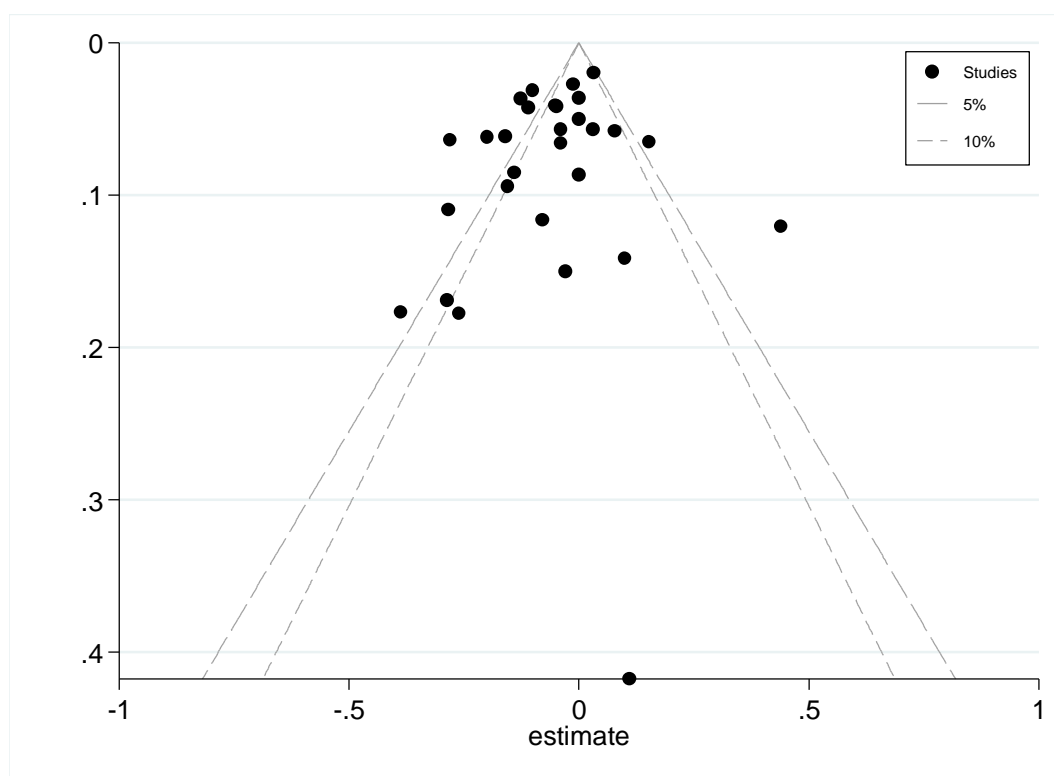


Table 2.49 HDL cholesterol and total carbohydrate: cohort studies in children and adults

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 Details	Contrast	Exposure Units	Mean Outcome	Beta coefficient (SE)/(CI)	p	P trend	Adjustments
Adolescent study															
(Boreham <i>et al.</i> , 1999) 17606 The Northern Ireland Young Hearts Project	Northern Ireland, Primarily White	12-15 %M 49.3	509	4 years (1.7)	Dietary history	Carbohydrate, total (% energy)	HDL-C Serum	Male		1 % energy/ day		Not reported		NS	SES/Class, sexual maturity
14192 The Northern Ireland Young Hearts Project	Northern Ireland, Primarily White	12-15 %M 49.3	509	4 years (1.7)	Dietary history	Carbohydrate, total (% energy)	HDL-C Serum	Female		1 % energy/ day		-0.22 (0.09)	0.014		SES/Class, sexual maturity
Adult studies															
(Ludwig <i>et al.</i> , 1999) 13696 The CARDIA Study	USA, Multi- ethnic, Generally healthy, No hypertension, No T2DM	18-30 %M 45.9	5115	10 years	FFQ (700)	Carbohydrate, total (% energy)	HDL-C Fasting, mg/dL	Race - White	(51.9) vs (33.5)	% Energy	48.9 vs. 48.4			0.59	age, alcohol, centre, education, energy intake, HDL-C, physical activity, gender, smoking, vitamin intake
13697 The CARDIA Study								Race - Black	(51.9) vs (33.5)	% Energy	50.8 vs. 52.3			0.11	As above
(Schroeder <i>et al.</i> , 2007) 14177 Middle-aged Runners Study	USA, Active people only, No CHD, No hypertension	(51) %M 62	91	10 years	Food diary	Carbohydrate, total (grams/day)	HDL-C Fasting			1 g/day		No effect on regression direction			age

Table 2.50 HDL cholesterol and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
Adolescent studies																
(Demol <i>et al.</i> , 2009) 15403		High carbohydrate, low fat	20/20	48.3 (SD 2)	46.0 (SD 2.1)							HDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Low carbohydrate, high fat	17/17	46.3 (SD 2.1)	43.0 (SD 2.4)			NS							Decrease	
		Low carbohydrate, high protein	18/18	45.0 (SD 2)	44.4 (SD 2.2)			NS							Decrease	
15404		High carbohydrate, low fat	20/20	48.3 (SD 2.0)	44.7 (SD 2.5)							HDL-C	Fasting (mg/dL)	1 year	Decrease	unclear
		Low carbohydrate, high fat	17/17	46.3 (SD 2.1)	38.3 (SD 2.8)			NS							Decrease	
		Low carbohydrate, high protein	18/18	45.0 (SD 2.0)	44.8 (SD 2.4)			NS							Decrease	
(Sondike <i>et al.</i> , 2003) 15991		Low fat	14/19			1.8 (SD 7.7)	NS					HDL-C	Fasting serum (mg/dL)	12 weeks	Decrease	bias
		Very low carbohydrate	12/20			3.8 (SD 7.2)	NS	NS							Decrease	
Adult studies																
(Appel <i>et al.</i> , 2005) *16321		High carbohydrate	164/164	50 (SD 16.1)		-1.4 (CI - 2.5, -0.3)						HDL - C	Fasting serum (mg/dL)	6 weeks	No change	No bias
		High protein	164/164	50 (SD 16.1)		-2.6 (CI - 3.6, -1.6)		0.02							No change	
		High PUFA	164/164	50 (SD 16.1)		-0.3 (CI - 1.3, 0.7)		0.03							No change	
(Bhargava, 2006) *16870		Control	379/allocated not reported	1.43 (SD 0.39)	1.42 (SD 0.38)		NS					HDL-C	Fasting plasma (mmol/L)	1 year	Decrease	unclear
		Low fat	615/allocated not reported	1.45 (SD 0.41)	1.41 (SD 0.38)		0.05	0.05							Decrease	
(Brehm <i>et al.</i> , 2003)		Low carbohydrate	22/22	51.77 (SE 2.82)	54.09 (SE 2.77)		<0.01	NS				HDL-C	Fasting (mg/dL)	3 months	Decrease	unclear

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
15729		Moderate fat	20/20	48.75 (SE 2.23)	51.05 (SE 3.49)		<0.01									Decrease
*15730		Low carbohydrate	22/22	51.77 (SE 2.82)	58.83 (SE 2.57)		<0.01	NS				HDL-C	Fasting (mg/dL)	6 months	Decrease	unclear
		Moderate fat	20/20	48.75 (SE 2.23)	52.85 (SE 2.58)		<0.01									Decrease
(Brehm <i>et al.</i> , 2005) 16387		Low carbohydrate	20/25	44.4 (SE 2.11)	48.1 (SE 2.71)			0.01				HDL-C	Fasting plasma (mg/dL)	2 months	Decrease	No bias
		Moderate fat	20/25	44.21 (SE 1.69)	43.5 (SE 2.02)											Decrease
*16388		Low carbohydrate	20/25	44.4 (SE 2.11)	51.65 (SE 2.55)			0.01				HDL-C	Fasting plasma (mg/dL)	4 months	Decrease	No bias
		Moderate fat	20/25	44.21 (SE 1.69)	46.2 (SE 2.08)											Decrease
(Campos <i>et al.</i> , 1995) *17091		High-fat minus low-fat higher CHO	Crossover: 43/allocated not reported						8 (SD 4)		0.0001	HDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17096		High-fat	43/allocated not reported	45 (SD 9)	47 (SD 10)			0.0001				HDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	43/allocated not reported	45 (SD 9)	39 (SD 80)											Not reported
(Claessens <i>et al.</i> , 2009) *16822		High carbohydrate supplement	16/allocated not reported	1 (SE 0.06)	1.1 (SE 0.06)	0.1 (SE 0.04)	<0.05	NS				HDL-C	Fasting (mmol/L)	12 weeks	Increase	unclear
		High protein supplement - casein	14/allocated not reported	0.99 (SE 0.06)	1.22 (SE 0.09)	0.23 (SE 0.05)	<0.05	NS								Decrease
		High protein supplement - whey	18/allocated not reported	1.02 (SE 0.06)	1.2 (SE 0.08)	0.18 (SE 0.05)	<0.05	NS								Decrease
(Clevence <i>et al.</i>)		High fat diet	42/46	1.22 (SE 0.04)	1.39 (SE 0.04)	0.18	<0.001	<0.001				HDL-C	Fasting plasma	10 weeks	No change	unclear

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
<i>al., 1992</i> *16607		Low fat diet	42/46	1.22 (SE 0.04)	0.05) 1.11 (SE 0.04)	-0.1	<0.001						(mmol/L)			No change
(Clifton <i>et al., 2004</i>) 16750		High MUFA	31/35	1.53 (SD 0.35)	1.45 (SD 0.36)			Unclear				HDL-C	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Very low fat	31/35	1.55 (SD 0.34)	1.33 (SD 0.26)											Decrease
*16751		High MUFA	31/35	1.53 (SD 0.35)	1.49 (SD 0.35)		<0.01					HDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
		Very low fat	31/35	1.55 (SD 0.34)	1.37 (SD 0.26)		<0.01									Decrease
(Clifton <i>et al., 2008</i>) *16008		High carbohydrate diet	38/38			0.31 (SD 0.23)						HDL-C	Fasting (mmol/L)	1.25 years	Decrease	unclear
		High protein diet	40/41			0.36 (SD 0.22)		NS								Decrease
(Colette <i>et al., 2003</i>) *17411		High carbohydrate diet	15/15	1.22 (SE 0.10)	1.21 (SE 0.08)		NS					HDL-C	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		High MUFA diet	17/17	1.42 (SE 0.10)	1.30 (SE 0.08)		0.010	NS								Decrease
(Cornier <i>et al., 2005</i>) *16688	Insulin sensitive	High carbohydrate, low fat	6/10	59 (SE 4)	54 (SE 5)			NS				HDL - C	Fasting (mg/dL)	16 weeks	Decrease	unclear
		Low carbohydrate, high fat	6/11	47 (SE 4)	46 (SE 4)											Decrease
(Couture <i>et al., 2003</i>) *15879	Genetics - Apo E genotype E3/E2	High carbohydrate diet	3/3	1.13 (SD 0.2)	1.0 (SD 0.13)		0.16	Not reported/unclear				HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	5/5	1.07 (SD 0.16)	1.08 (SD 0.21)		0.89									Decrease

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
*15880	Genetics - Apo E genotype E3/E3	High carbohydrate diet	22/22	1.07 (SD 0.18)	0.98 (SD 0.16)		0.01					HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	21/21	0.95 (SD 0.14)	0.91 (SD 0.16)		0.11								Decrease	
*15881	Genetics - Apo E genotype E3/E4	High carbohydrate diet	8/8	1.09 (SD 0.24)	0.96 (SD 0.28)		0.14					HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	6/6	1.12 (SD 0.23)	1.13 (SD 0.29)		0.98								Decrease	
(Dale <i>et al.</i> , 2009) 15983		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.01 (CI - 0.04, 0.06)		NS	HDL-C	Fasting (mmol/L)	2 years	Decrease in both	unclear
17398		High carbohydrate diet	89/100	1.3 (SD 0.3)	1.28 (SD 0.36)							HDL-C	Fasting (mmol/L)	1 year	Decrease	unclear
		High MUFA diet	85/100	1.3 (SD 0.3)	1.30 (SD 0.33)										Decrease	
17369		High carbohydrate diet	89/100	1.3 (SD 0.3)	1.29 (SD 0.36)							HDL-C	Fasting (mmol/L)	2 years	Decrease	unclear
		High MUFA diet	85/100	1.3 (SD 0.3)	1.27 (SD 0.37)										Decrease	
(Dansinger <i>et al.</i> , 2005) 15701		Atkins	40/40			3.2 (SD 6.2)	0.01	Unclear				HDL-C	Fasting serum (mg/dL)	2 months	Decrease	No bias
		Ornish	40/40			-3.6 (SD 7.3)	0.01								Decrease	
		Weight watchers	40/40			-0.2 (SD 11.8)	NS								Decrease	
		Zone	40/40			1.8 (SD 7.6)	NS								Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
15702		Atkins	40/40			3.8 (SD 6.4)	0.01					HDL-C	Fasting serum (mg/dL)	6 months	Decrease	No bias
		Ornish	40/40			-1.5 (SD 7)	NS								Decrease	
		Weight watchers	40/40			2.4 (SD 9)	NS								Decrease	
		Zone	40/40			3.6 (SD 10.5)	0.05								Decrease	
15703		Atkins	40/40			3.4 (SD 7.1)	0.01					HDL-C	Fasting serum (mg/dL)	1 year	Decrease	No bias
		Ornish	40/40			-0.5 (SD 6.5)	NS								Decrease	
		Weight watchers	40/40			3.4 (SD 9.9)	0.05								Decrease	
		Zone	40/40			3.3 (SD 10.3)	0.05								Decrease	
(de Luis <i>et al.</i> , 2008) 16146	Genetics - wild-type Ala54/Ala54	Low carbohydrate	55/105	54.7 (SD 19.2)	53.2 (SD 15.4)			NS				HDL-C	Fasting (mg/dL)	2 months	Decrease	unclear
		Low fat	55/99	52.6 (SD 10.7)	51.4 (SD 9.7)										Decrease	
16163	Genetics - mutant-type Ala54/Thr54 or Thr54/Thr54	Low carbohydrate	50/105	52.9 (SD 12.6)	51.8 (SD 14.6)			NS				HDL-C	Fasting (mg/dL)	2 months	Decrease	unclear
		Low fat	44/99	51.3 (SD 10.6)	52.8 (SD 11.6)										Decrease	
(de Luis <i>et al.</i> , 2009b) *16084		Low carbohydrate	52/52	53.1 (SD 13)	51.4 (SD 12.5)			NS				HDL-C	Fasting (mg/dL)	3 months	Decrease	unclear
		Low fat	66/66	56.8 (SD 10.4)	57.3 (SD 13)										Decrease	
(de Luis <i>et al.</i> , 2009a) 16699	Genetics - UCP3 Gene -55CC polymorphi	Low carbohydrate	54/67	55.6 (SD 19.2)	56.2 (SD 15.4)		NS	Unclear				HDL-C	(mg/dL)	2 months	Decrease	unclear

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
16700	sm															
	Genetics - UCP3 Gene -55CC polymorphism	Low fat	40/64	55.1 (SD 10.7)	54.1 (SD 9.7)		NS								Decrease	
	Genetics - UCP3 Gene -55CT/TT polymorphism	Low carbohydrate	13/67	58.9 (SD 12.6)	56.8 (SD 14.6)		NS					HDL-C	(mg/dL)	2 months	Decrease	unclear
(Delbridge <i>et al.</i> , 2009) *15323	Genetics - UCP3 Gene -55CT/TT polymorphism	Low fat	24/64	54.4 (SD 10.6)	52.8 (SD 11.6)		NS								Decrease	
		Low fat, high carbohydrate weight maintenance diet	70/70			0.13 (SE 0.02)						HDL-C	Fasting (mmol/L)	1 year	Increase	unclear
		Low fat, high protein weight maintenance diet	68/71			0.15 (SE 0.03)		0.611							Increase	
(Dreon <i>et al.</i> , 1994) 15638	Larger LDL particles	High-fat low CHO	87/105	47.5 (SD 8.9)	50.6 (SD 1.1)		<0.0001					HDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	47.5 (SD 8.9)	43.2 (SD 0.9)		<0.0001								Not reported	
17042	Smaller and denser LDL particles	High-fat low CHO	87/105	47.5 (SD 8.9)	41.4 (SD 1.8)		<0.0001					HDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	47.5 (SD 8.9)	36.3 (SD 1.8)		<0.0001								Not reported	
17049	Larger LDL particles	Low-fat higher CHO minus high-	Crossover: 87/105						-7.4 (SD 1)		<0.0001	HDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
fat low CHO																
17055	Smaller and dense LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 18/105						-5.2 (SD 1)		<0.0001	HDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17061	LDL particles remained large during study	High-fat low CHO	87/105	47.5 (SD 8.9)	51.4 (SD 1.4)		<0.0001					HDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	47.5 (SD 8.9)	45.4 (SD 1.2)		<0.0001								Not reported	
17065	LDL particles changed from large to small and dense during study	High-fat low CHO	87/105	47.5 (SD 8.9)	49.4 (SD 1.7)		<0.0001					HDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	47.5 (SD 8.9)	40 (SD 1.1)		<0.0001								Not reported	
17069	LDL particles remained large during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 51/105						-6 (SD 1)		<0.0001	HDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17076	LDL particles changed from large to small and dense during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 36/105						-9.4 (SD 1)		<0.0001	HDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Due <i>et al.</i> , 2004)		High protein	23/23	1.35 (CI 1.1, 1.6)	1.32 (CI 1.1, 1.4)			NS				HDL-C	Fasting (mmol/L)	6 months	Decrease	unclear

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17538		Moderate protein	23/18	1.37 (CI 1.1, 1.5)	1.16 (CI 1.0, 1.4)											Decrease
*17539		High protein	23/23	1.35 (CI 1.1, 1.6)	1.47 (CI 1.1, 1.6)			NS				HDL-C	Fasting (mmol/L)	1 year	Decrease	unclear
		Moderate protein	18/18	1.37 (CI 1.1, 1.5)	1.28 (CI 1.2, 1.6)											Decrease
(Due <i>et al.</i> , 2008)		Control	24/25	1.15 (CI 1.0, 1.3)	1.24 (CI 1.1, 1.4)	0.09 (CI -0.1, 0.4)						HDL-C	Fasting plasma (mmol/L)	6 months	Increase	unclear
*15300		High MUFA	39/52	1.22 (CI 1.1, 1.3)	1.31 (CI 1.2, 1.4)	0.09 (CI 0, 0.2)		NS								Increase
		Low fat	43/48	1.23 (CI 2.1, 1.3)	1.27 (CI 1.2, 1.4)	0.05 (CI 0, 0.1)		NS								Increase
(Dyson <i>et al.</i> , 2007)		Healthy eating diet	4/~6	1.32	1.38	0.06						HDL-C	(mmol/L)	3 months	Decrease	bias
16349		Low carbohydrate diet	6/~6	1.32	1.4	0.08		NS								Decrease
(Ebbelin <i>g et al.</i> , 2005)		Low fat diet	12/17	53.8 (SE 2.7)		-0.3% (CI -8.1, 8.2)						HDL-C	Fasting (mg/dL)	6 months	Decrease	unclear
15493		Low GI diet	11/17	49 (SE 2.9)		2.3% (CI -6, 11.3)		NS								Decrease
*15508		Low fat diet	12/17	53.8 (SE 2.7)		1.1% (CI -6.9, 9.8)						HDL-C	Fasting (mg/dL)	1 year	Decrease	unclear
		Low GI diet	11/17	49 (SE 2.9)		12.2% (CI 2.9, 22.3)		NS								Decrease
(Ebbelin <i>g et al.</i> , 2007)		Low fat diet	37/37			-4.4 (SE 1.3)						HDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
*15451		Low GL diet	ITT: 36/36			1.6 (SE 1.4)		0.02								Decrease
15452		Low fat diet	37/37			-8.2 (SE 1.5)						HDL-C	Fasting plasma (mg/dL)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-3.7 (SE 1.5)		0.3								Decrease

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(Foster <i>et al.</i> , 2003) *15216		Conventional diet plan	30/30			1.4 (SD 16.1)	NS					HDL-C	Fasting serum (%)	3 months	Decrease	unclear
		Low carbohydrate diet	33/33			9.6 (SD 19.1)	<0.05	0.04							Decrease	
15217		Conventional diet plan	30/30			2.5 (SD 12.0)	NS					HDL-C	Fasting serum (%)	6 months	Decrease	unclear
		Low carbohydrate diet	33/33			14.7 (SD 20.5)	<0.05	0.007							Decrease	
*15218		Conventional diet plan	30/30			1.6 (SD 11.1)	NS					HDL-C	Fasting serum (%)	1 year	Decrease	unclear
		Low carbohydrate diet	33/33			11.0 (SD 19.4)	<0.05	0.04							Decrease	
(Frisch <i>et al.</i> , 2009) *15170		High carbohydrate diet	100/100			-0.09 (SD 0.19)	0.05					HDL-C	Fasting serum (mmol/L)	6 months	Decrease	unclear
		Moderate carbohydrate diet	100/100			-0.02 (SD 0.2)	NS	0.005							Decrease	
15171		High carbohydrate diet	100/100			-0.03 (SD 0.17)	NS					HDL-C	Fasting serum (mmol/L)	1 year	Decrease	unclear
		Moderate carbohydrate diet	100/100			-0.02 (SD 0.21)	NS	0.668							Decrease	
(Gardner <i>et al.</i> , 2007) *15111		Atkins: low carbohydrate	70/77			-0.4 (SD 7.7)		NS				HDL-C	Fasting plasma (mg/dL)	2 months	Decrease	No bias
		Ornish: high carbohydrate	64/76			-5.3 (SD 9)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			-0.5 (SD 5.4)									Decrease	
15112		Atkins: low carbohydrate	70/77			5.1 (SD 9.6)		NS				HDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
		Ornish: high carbohydrate	64/76			0 (SD 9.2)		NS							Decrease	
		Zone: moderate	65/79			3.3 (SD 6.9)									Decrease	

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		carbohydrate														
15113		Atkins: low carbohydrate	70/77			4.9 (SD 9.1)		NS				HDL-C	Fasting plasma (mg/dL)	1 year	Decrease	No bias
		Ornish: high carbohydrate	64/76			0 (SD 6.3)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			2.2 (SD 6.1)									Decrease	
(Ginsberg <i>et al.</i> , 1998) *17249		Average American Diet	103/118		52.2 (SE 1.1)							HDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		46.2 (SE 1.0)			<0.01							Not reported	
		Step 1 diet	103/118		48.5 (SE 11.1)			<0.01							Not reported	
17260	Men	Average American Diet	46/118		46.5 (SE 1.3)							HDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	46/118		40.6 (SE 1.1)			<0.01							Not reported	
		Step 1 diet	46/118		42.8 (SE 1.4)			<0.01							Not reported	
17261	Women	Average American Diet	57/118		56.2 (SE 1.4)							HDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	57/118		50.1 (SE 1.3)			<0.01							Not reported	
		Step 1 diet	57/118		52.5 (SE 1.3)			<0.01							Not reported	
17302	Black	Average American Diet	26/118		51.5 (SE 2.0)							HDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	26/118		46.1 (SE 1.8)			<0.01							Not reported	

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17303	Non black	Step 1 diet	26/118		48.3 (SE 2.1)			NS								Not reported	
		Average American Diet	77/118		52.0 (SE 1.3)							HDL-C	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	77/118		45.7 (SE 1.2)			<0.01								Not reported	
		Step 1 diet	77/118		48.1 (SE 1.2)			<0.01								Not reported	
17318	Pre-menopausal	Average American Diet	39/118		56.3 (SE 1.7)							HDL-C	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	39/118		50.2 (SE 1.6)			<0.01								Not reported	
		Step 1 diet	39/118		52.9 (SE 1.7)			<0.01								Not reported	
17319	Post-menopausal	Average American Diet	18/118		55.8 (SE 2.5)							HDL-C	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	18/118		49.7 (SE 2.2)			<0.01								Not reported	
		Step 1 diet	18/118		51.6 (SE 2.3)			<0.01								Not reported	
17334	Men <40y	Average American Diet	30/118		48.1 (SE 1.6)							HDL-C	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	30/118		41.7 (SE 1.3)			<0.01								Not reported	
		Step 1 diet	30/118		43.8 (SE 1.4)			<0.01								Not reported	
17335	Men >40y	Average American Diet	16/118		43.5 (SE 2.4)							HDL-C	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	16/118		38.6 (SE 2.1)			<0.01								Not reported	
		Step 1 diet	16/118		40.8 (SE 2.8)			NS								Not reported	

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(Golay <i>et al.</i> , 1996) *16626		Low carbohydrate diet	completers not reported/22	1.1 (SE 0.1)	0.9 (SE 0.1)		<0.001	Not reported/unclear				HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Moderate carbohydrate diet	completers not reported/21	1.1 (SE 0.1)	1.0 (SE 0.1)		<0.05								Decrease	
(Golay <i>et al.</i> , 2000) *14853		Higher carbohydrate, macronutrients not eaten simultaneously	26/26	1.26 (SE 0.08)	1.02 (SE 0.09)		<0.01	NS				HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Lower carbohydrate, macronutrients eaten simultaneously	28/28	1.12 (SE 0.06)	0.94 (SE 0.04)		<0.001								Decrease	
(Howard <i>et al.</i> , 2006) 16248		Control	approx n=1699 (5.8% sub-sample of 29294)	58.4 (SD 15.4)	58.2 (SD 15.5)	-0.3 (SD 10.2)						HDL-C	Fasting (mg/dL)	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub-sample of 19541)	60.1 (SD 16.1)	59.7 (SD 15.8)	-0.7 (SD 9.4)		NS							Decrease	
*17614		Low fat minus control	As above						-0.43 (CI - 1.42, 0.57)		NS	HDL-C	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Johnston <i>et al.</i> , 2004) *14862		High carbohydrate, low fat	7/10	1.32 (SE 0.13)		-19.1% (SE 7.1%)	NS					HDL-C	Whole blood (mmol/L)	6 weeks	Decrease	unclear
		High protein, low fat	9/10	1.55 (SE 0.16)		-13.5% (SE 3.9%)	0.05	0.780							Decrease	
(Johnston		Low	10/10	1.33 (SE		decrease						HDL-C	Fasting serum	6 weeks	Decrease	unclear

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n <i>et al.</i> , 2006) *17518		carbohydrate diet		0.07)									(mmol/L)			
		Very low-carbohydrate diet	9/9	1.27 (SE 0.10)		decrease		NS							Decrease	
(Keogh <i>et al.</i> , 2007) 15619		High carbohydrate diet	12/12	1.33 (SE 0.31)	1.26 (SE 0.33)		0.05					HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	1.26 (SE 0.31)	1.12 (SE 0.24)		0.05	NS							Decrease	
*15620		High carbohydrate diet	12/12	1.33 (SE 0.31)	1.34 (SE 0.31)		NS					HDL-C	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	1.26 (SE 0.31)	1.23 (SE 0.28)		NS	NS							Decrease	
15621		High carbohydrate diet	completers not reported/12	1.3 (SE 0.08)	1.34 (SE 0.07)		NS					HDL-C	Fasting plasma (mmol/L)	1 year	Decrease	unclear
		Low carbohydrate diet	completers not reported/13	1.33 (SE 0.08)	1.44 (SE 0.14)		NS	NS							Decrease	
(Keogh <i>et al.</i> , 2008) *16721		High carbohydrate, low SFA	47/50	1.3 (SD 0.4)	1.3 (SD 0.3)		NS					HDL-C	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low carbohydrate, high SFA	52/57	1.4 (SD 0.3)	1.5 (SD 0.3)		<0.001	<0.001							Decrease	
(Kirkwood <i>et al.</i> , 2007) 15670		Group 1: No advice	18/allocated not reported				NS					HDL-C	Fasting (mmol/L)	12 weeks	No change	unclear
		Group 2: Conventional weight loss diet	16/allocated not reported				NS	NS							Decrease	
15671		Group 3: Exercise	19/allocated not reported	1.28	1.46	0.18	0.09	NS				HDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear

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		Group 4: Conventional weight loss diet + exercise	16/allocated not reported				NS									Decrease
(Krauss <i>et al.</i> , 2006) *17479		26% CHO High saturated fat	40/52	41.0 (SD 11.1)		2.5 (SE 0.9)		NS				HDL-C	Fasting plasma (mg/dL)	12 weeks	Decrease	unclear
		26% CHO Low saturated fat	47/59	43.1 (SD 12.4)		2.4 (SE 0.8)		NS								Decrease
		39% CHO Low saturated fat	42/56	41.6 (SD 9.0)		2.0 (SE 0.7)		NS								Decrease
		54% CHO Low saturated fat	49/57	41.7 (SD 8.7)		1.9 (SE 0.7)										Decrease
(Lasker <i>et al.</i> , 2008) 15913		High carbohydrate	25/33			-1.7						HDL-C	Fasting plasma (%)	4 months	Decrease	unclear
		High protein	25/32			6.9		0.045								Decrease
(Layman <i>et al.</i> , 2005) *16177		High carbohydrate diet	12/12	1.30 (SD 0.06)	1.2 (SD 0.04)		<0.05					HDL-C	Fasting serum (mmol/L)	16 weeks	Decrease	unclear
		High protein diet	12/12	1.33 (SD 0.09)	1.30 (SD 0.1)		NS	<0.05								Decrease
16178		High carbohydrate diet + exercise	12/12	1.36 (SD 0.07)	1.28 (SD 0.07)		NS					HDL-C	Derived by calculation Fasting (mmol/L)	16 weeks	Decrease	unclear
		High protein diet + exercise	12/12	1.2 (SD 0.06)	1.25 (SD 0.09)		NS	0.19								Decrease
(Layman <i>et al.</i> , 2009) 14959		High carbohydrate, low protein diet	51/66		lower		<0.05					HDL-C	Fasting plasma	4 months	Decrease	unclear
		Low carbohydrate, high protein diet	52/64		higher		<0.05	<0.01								Decrease
*14964		High carbohydrate,	30/66		0.15 (SE		<0.05					HDL-C	Fasting plasma	1 year	Decrease	unclear

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		low protein diet			0.03)								(mmol/L)			
		Low carbohydrate, high protein diet	41/64		0.26 (SE 0.03)		<0.05	0.025							Decrease	
(Leidy <i>et al.</i> , 2007) *16840		High protein, energy restricted	21/27	65 (SE 2)	56 (SE 2)	-9 (SE 1)		NS				HDL-C	Fasting serum (mg/dL)	12 weeks	Decrease	unclear
		Moderate protein, energy restricted	25/27	63 (SE 3)	57 (SE 3)	-6 (SE 2)									Decrease	
(Ley <i>et al.</i> , 2004) 15941		Control	70/70			-0.03 (SE 0.05)						HDL-C	Fasting serum (mmol/L)	6 months	No change	unclear
		Low fat	66/66			0.01 (SE 0.02)		NS							Decrease	
*15942		Control	70/70			0.01 (SE 0.05)						HDL-C	Fasting serum (mmol/L)	1 year	No change	unclear
		Low fat	66/66			-0.02 (SE 0.02)		NS							Decrease	
15943		Control	57/70			0.06 (SE 0.05)						HDL-C	Fasting serum (mmol/L)	2 years	No change	unclear
		Low fat	47/66			0.08 (SE 0.04)		NS							Decrease	
15944		Control	51/70			-0.01 (SE 0.05)						HDL-C	Fasting serum (mmol/L)	3 years	No change	unclear
		Low fat	48/66			-0.03 (SE 0.03)		NS							Decrease	
15945		Control	52/70			0.06 (SE 0.05)						HDL-C	Fasting serum (mmol/L)	5 years	No change	unclear
		Low fat	51/66			0.01 (SE 0.02)		NS							Decrease	
(Lovejoy <i>et al.</i> , 2003) 14977		Control	13/15	1 (SE 0.04)		-0.03 (SE 0.03)						HDL-C	Fasting (mmol/L)	3 months	Decrease	unclear
		Fat reduced	13/15	1.06 (SE 0.03)		-0.01 (SE 0.02)		NS							Decrease	
14978		Control	13/15	1 (SE 0.04)		0.06 (SE 0.02)						HDL-C	Fasting (mmol/L)	6 months	Decrease	unclear
		Fat reduced	13/15	1.06 (SE 0.03)		0.04 (SE 0.03)		NS							Decrease	

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*14980		Control	13/15	1 (SE 0.04)		0.09 (SE 0.02)						HDL-C	Fasting (mmol/L)	9 months	Decrease	unclear
		Fat reduced	13/15	1.06 (SE 0.03)		0.09 (SE 0.03)		NS							Decrease	
(Mahon <i>et al.</i> , 2007) *15072		Control	11/11	68 (SD 15)	71 (SD 13)	3 (SD 15)	NS					HDL-C	Fasting serum (mg/dL)	9 weeks	No change	unclear
		Energy restriction + beef	14/14	59 (SD 15)	57 (SD 13)	-2 (SD 11)	NS	NS							Decrease	
		Energy restriction + carbohydrate /fat	14/14	73 (SD 19)	61 (SD 13)	-12 (SD 17)	NS	NS							Decrease	
		Energy restriction + chicken	15/15	50 (SD 10)	50 (SD 12)	0 (SD 16)	NS	NS							Decrease	
(Maki <i>et al.</i> , 2007) 17282		Ad libitum low GL diet	39/43	56.2 (SE 2)		-0.2 (SE 1.2)		NS				HDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	56.4 (SE 2)		-2.1 (SE 0.9)									Decrease	
*17283		Ad libitum low GL diet	39/43	56.2 (SE 2)		3.8 (SE 1.4)		0.037				HDL-C	Fasting (mg/dL)	36 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	56.4 (SE 2)		1.9 (SE 0.8)									Decrease	
(McMillan-Price <i>et al.</i> , 2006) *16221		High CHO, high GI diet	32/32	1.29 (SE 0.07)		0.08 (SE 0.04)		NS				HDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
		High CHO, low GI diet	32/32	1.17 (SE 0.05)		0.03 (SE 0.04)		NS							Decrease	
		High protein, high GI diet	32/32	1.16 (SE 0.05)		0.05 (SE 0.04)		NS							Decrease	
		High protein, low GI diet	33/33	1.36 (SE 0.08)		0.07 (SE 0.04)		NS							Decrease	
(Mecklin <i>et al.</i> , 2004) *14874		Low carbohydrate	15/20	49 (SE 2)	55 (SE 3)		0.05	0.05				HDL-C	Fasting (mg/dL)	10 weeks	Decrease	No bias
		Low fat	16/20	52 (SE 3)	44 (SE 3)			0.05							Decrease	
(Mecklin <i>et al.</i> and Sherfey, 2007)		Hypocaloric control diet	8/15	34 (SD 10)	30 (SD 9)		NS					HDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich	10/15	32 (SD 7)	30 (SD 3)		NS	NS							Decrease	

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*16379		diet														
16380		Hypocaloric control diet + exercise	11/15	55 (SD 22)	31 (SD 9)		NS					HDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet + exercise	14/15	40 (SD 24)	35 (SD 14)		NS	NS							Decrease	
(Morgan <i>et al.</i> , 2009) 14709		Atkins	33/57	1.22 (SD 0.23)	1.24 (SD 0.25)		NS	Unclear				HDL-C	Fasting Whole blood (mmol/L)	8 weeks	Decrease	unclear
		Control	37/61	1.19 (SD 0.22)	1.22 (SD 0.24)		NS								No change	
		Slim Fast	44/59	1.25 (SD 0.27)	1.15 (SD 0.28)		0.01								Decrease	
		Weight Watchers	46/58	1.16 (SD 0.24)	1.04 (SD 0.21)		0.01								Decrease	
*14710		Atkins	33/57	1.22 (SD 0.23)	1.14 (SD 0.32)		NS					HDL-C	Fasting Whole blood (mmol/L)	24 weeks	Decrease	unclear
		Control	37/61	1.19 (SD 0.22)	1.04 (SD 0.2)		0.01								No change	
		Slim Fast	44/59	1.25 (SD 0.27)	1.09 (SD 0.27)		0.01								Decrease	
		Weight Watchers	46/58	1.16 (SD 0.24)	0.98 (SD 0.15)		0.01								Decrease	
(Nelson <i>et al.</i> , 1995) *16939		High fat diet	11/11	46.3 (SD 14.0)	43.2 (SD 13.4)			NS				HDL-C	Fasting plasma (mg/dL)	50 days	Not reported	unclear
		Low fat diet	11/11	46.3 (SD 14.0)	40.5 (SD 12.4)										Not reported	
(Noakes <i>et al.</i> , 2005)		High carbohydrate diet	48/48	1.32 (SE 0.04)	1.17 (SE 0.04)							HDL-C	Fasting serum (mmol/L)	8 weeks	Decrease	unclear

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
16999		High protein diet	52/52	1.33 (SE 0.05)	1.21 (SE 0.04)			NS								Decrease
*17000		High carbohydrate diet	48/48	1.32 (SE 0.04)	1.22 (SE 0.04)	-0.09 (SE 0.02)						HDL-C	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	52/52	1.33 (SE 0.05)	1.25 (SE 0.04)	-0.09 (SE 0.02)		0.657								Decrease
(Noakes <i>et al.</i> , 2006) 16581		High unsaturated fat	21/27	1.26 (SE 0.05)	1.15 (SE 0.05)			Unclear				HDL-C	Fasting plasma (mmol/L)	8 weeks	Decrease	unclear
		Very low carbohydrate	24/28	1.26 (SE 0.05)	1.26 (SE 0.05)											Decrease
		Very low fat	22/28	1.31 (SE 0.07)	1.15 (SE 0.06)											Decrease
*16582		High unsaturated fat	21/27	1.26 (SE 0.05)	1.19 (SE 0.04)	-0.06 (SE 0.03)						HDL-C	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Very low carbohydrate	24/28	1.26 (SE 0.05)	1.32 (SE 0.05)	-0.06 (SE 0.03)										Decrease
		Very low fat	22/28	1.31 (SE 0.07)	1.25 (SE 0.06)	-0.06 (SE 0.04)										Decrease
(O'Brien <i>et al.</i> , 2005) 16955		Low carbohydrate	22/22					0.98				HDL-C	Fasting (mg/dL)	3 months	Decrease	unclear
		Moderate fat	19/19													Decrease
(Pereira <i>et al.</i> , 2004) *14580		Hypoenergetic low fat diet	11/23	49.4 (SE 3.61)	44.1 (SE 2.41)	8.1% (SE 3.49%)						HDL-C	Fasting serum (mg/dL)	67 days	Decrease	unclear
		Hypoenergetic low GL diet	14/23	46.9 (SE 3.2)	42.2 (SE 2.14)	-8.9% (SE 3.09%)		0.87								Decrease
(Pelkman <i>et al.</i> , 2004) 16877		Low fat, high carbohydrate diet	25/25	1.24 (SE 0.04)	1.09 (SE 0.04)		<0.05	Not reported/unclear				HDL-C	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower	27/27	1.14 (SE 0.04)	1.10 (SE 0.04)		NS									Decrease

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
*16878		carbohydrate diet			0.04)											
		Low fat, high carbohydrate diet	25/25	1.24 (SE 0.04)	1.12 (SE 0.04)		<0.05					HDL-C	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	1.14 (SE 0.04)	1.12 (SE 0.04)		NS								Decrease	
(Peterse n <i>et al.</i> , 2006) 17205	Women	Hypoenergetic high carbohydrate, low fat diet	251/292	1.19 (SD 0.31)		-0.11 (SD 0.18)						HDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
		Hypoenergetic low carbohydrate, high fat diet	235/287	1.16 (SD 0.3)		-0.05 (SD 0.17)									Decrease	
17206	Men	Hypoenergetic high carbohydrate, low fat diet	85/97	0.94 (SD 0.21)		0.00 (SD 0.14)						HDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
		Hypoenergetic low carbohydrate, high fat diet	77/95	0.96 (SD 0.22)		0.00 (SD 0.14)									Decrease	
17207		Hypoenergetic high carbohydrate, low fat diet	336/389	1.12 (SD 0.31)		-0.08 (SD 0.18)						HDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
		Hypoenergetic low carbohydrate, high fat diet	312/382	1.11 (SD 0.29)		-0.04 (SD 0.16)									Decrease	
*17217		Hypoenergetic low carbohydrate, high fat diet minus hypoenergetic high carbohydrate, low fat diet	Low CHO: 312/383 High CHO: 336/389						0.04 (CI 0.02, 0.07)		<0.001	HDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease in both	bias

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Phillips <i>et al.</i> , 2008) *17422		Low carbohydrate diet	10/~14	54.6 (SE 5.3)	54.5 (SE 5)		NS	NS				HDL-C	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
		Low fat diet	8/~14	49.9 (SE 4.29)	44.4 (SE 4.71)		NS								Decrease	
(Sacks <i>et al.</i> , 2009) 15583 *15584		High-fat, average-protein	ITT: /204		49 (SD 13)	2.9%						HDL-C	Fasting serum (mg/dL)	6 months	Decrease	No bias
		High-fat, high-protein	ITT: /201		53 (SD 15)	4%									Decrease	
		Low-fat, average-protein	ITT: /204		49 (SD 13)	-0.4%									Decrease	
		Low-fat, high-protein	ITT: /202		51 (SD 13)	2.7%									Decrease	
		High-fat, average-protein	ITT: /204	48 (SD 12)	51 (SD 13)	6.3%						HDL-C	Fasting serum (mg/dL)	2 years	Decrease	No bias
		High-fat, high-protein	ITT: /201	51 (SD 16)	55 (SD 17)	8.8%		0.02 (compared with low-fat, average-protein)							Decrease	
		Low-fat, average-protein	ITT: /204	49 (SD 15)	51 (SD 15)	5.6%									Decrease	
		Low-fat, high-protein	ITT: /202	49 (SD 13)	53 (SD 15)	6.5%									Decrease	
(Segal-Isaacson <i>et al.</i> , 2004) *14985		Low fat diet	4/4	55 (SD 6)	34 (SD 6)		<0.05					HDL-C	Fasting Whole blood (mg/dL)	6 weeks	Decrease	unclear
		Very low carbohydrate	4/4	55 (SD 6)	41 (SD 12)		<0.05	0.123							Decrease	
(Sharma <i>et al.</i> , 2004) *14751		Low fat	15/15	1.02 (SD 0.16)	0.95 (SD 0.16)		NS					HDL-C	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15	1.02 (SD 0.16)	0.99 (SD 0.2)		NS	NS							Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Stoerndal <i>et al.</i> , 2008) *16528		Low carbohydrate diet	10/14	1.16 (SD 0.21)	1.13 (SD 0.2)			NS				HDL-C	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low fat diet	13/14	1.14 (SD 0.47)	1.10 (SD 0.43)										Decrease	
(Turley <i>et al.</i> , 1998) *15215		Low fat, high carbohydrate diet	18/38	1.26 (SD 0.31)	1.07 (SD 0.23)			0.057				HDL-C	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Western diet	18/38	1.26 (SD 0.31)	1.21 (SD 0.27)										Decrease	
(Wolever and Mehling, 2003) *17136		High carbohydrate, high GI	11/13			0.09 (SE 0.04)		Significant compared with high carb, low GI but p value not reported				HDL-C	Fasting serum (mmol/L)	4 months	Decrease	unclear
		High carbohydrate, low GI	13/13			-0.01 (SE 0.03)									Decrease	
		Low carbohydrate, high MUFA	11/12			0.05 (SE 0.03)									Increase	
(Zamboni <i>et al.</i> , 1999) 16263		High carbohydrate, energy restriction	11/11	1.48 (SD 0.46)	1.42 (SD 0.29)		NS					HDL-C	Fasting plasma (mmol/L)	3 months	Decrease	unclear
		Olive oil enriched energy restriction diet	9/9	1.44 (SD 0.38)	1.48 (SD 0.44)		NS	NS							Decrease	
	*16264	High carbohydrate, energy restriction	5/11	1.48 (SD 0.46)	1.35 (SD 0.32)		NS					HDL-C	Fasting plasma (mmol/L)	6 months	Decrease	unclear
		Olive oil enriched energy restriction	7/9	1.44 (SD 0.38)	1.75 (SD 0.23)		<0.05	<0.05							Decrease	

Author/ Result ID	Subgrou p detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessmen t Bias
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diet

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

LDL cholesterol, total carbohydrate and high carbohydrate diets

Summary of cohort results

Data were extracted from one publication, reporting results from one US study of young adults (Ludwig *et al.*, 1999). The CARDIA study reported total carbohydrate intake as a percentage of total energy and the association with continuous LDL cholesterol in black and white ethnic subgroups (Ludwig *et al.*, 1999). Similar LDL cholesterol results were reported in participants in the highest and lowest quintile of total carbohydrate intake, in both black and white subgroups. This study adjusted for an appropriate number of variables including age, gender, alcohol intake and smoking status, but not BMI.

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

Summary of RCT data

Sixty studies, presented in sixty four papers, explored the effects of dietary variation in carbohydrate - replacing carbohydrate with fat, protein or both - on LDL cholesterol. Of these studies, four also provided data on non-HDL cholesterol (Sondike *et al.*, 2003; Gardner *et al.*, 2007; Pelkman *et al.*, 2004; Howard *et al.*, 2006). Details of these studies can be found in the Trial Characteristics table.

Of the included studies, 50 used a parallel group design, nine used a crossover approach (Appel *et al.*, 2005; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Dreon *et al.*, 1994; Ginsberg *et al.*, 1998; Nelson *et al.*, 1995; Segal-Isaacson *et al.*, 2004; Turley *et al.*, 1998) and one used a factorial design (Dale *et al.*, 2009). The majority did not state the extent of blinding of participants and/or researchers, but 11 were open (Demol *et al.*, 2009; Dyson *et al.*, 2007; Layman *et al.*, 2009; Dale *et al.*, 2009; Due *et al.*, 2008; Foster *et al.*, 2003; Petersen *et al.*, 2006; Phillips *et al.*, 2008; Segal-Isaacson *et al.*, 2004; Sondike *et al.*, 2003; Maki *et al.*, 2007), four were double blind (Appel *et al.*, 2005; Sacks *et al.*, 2009; Ginsberg *et al.*, 1998; Lovejoy *et al.*, 2003) and four were single blind (Couture *et al.*, 2003; Gardner *et al.*, 2007; Ebbeling *et al.*, 2007; Howard *et al.*, 2006). Intervention durations ranged from six weeks to five years.

Studies were primarily conducted in the USA (33) but were also carried out in Australia (8), Canada (3), New Zealand (3), the UK (2), Denmark (2), Spain, Israel, France, Germany, Scotland, Italy, the Netherlands, Europe and the UK and USA collectively. All studies, bar two, recruited adult participants (mean age of adult trials was 42 years); the exceptions being Demol *et al.* (Demol *et al.*, 2009) and Sondike *et al.* (Sondike *et al.*, 2003) who used adolescents in their trials. Most studies used both males and females, although 16 were restricted to females only (Brehm *et al.*, 2005; Brehm *et al.*, 2003; O'Brien *et al.*, 2005; Layman *et al.*, 2005; Leidy *et al.*, 2007; Mahon *et*

et al., 2007; Meckling and Sherfey, 2007; Bhargava, 2006; Clifton *et al.*, 2004; Cornier *et al.*, 2005; Dale *et al.*, 2009; Gardner *et al.*, 2007; Kirkwood *et al.*, 2007; Segal-Isaacson *et al.*, 2004; Zambon *et al.*, 1999; Howard *et al.*, 2006; Clifton *et al.*, 2008; Noakes *et al.*, 2005) and nine to males only (Krauss *et al.*, 2006; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Couture *et al.*, 2003; Dreon *et al.*, 1994; Lovejoy *et al.*, 2003; Nelson *et al.*, 1995; Turley *et al.*, 1998). In the studies that reported mean BMI, participants were mostly overweight or obese.

The final sample sizes ranged from four to 48,335. Of these studies, two were particularly large with 2208 and 48,335 participants ((Bhargava, 2006) and (Howard *et al.*, 2006) respectively).

Two studies were not included in the meta-analysis as the participants used were adolescents aged 12-18 years (Demol *et al.*, 2009; Sondike *et al.*, 2003). The study reported by Demol *et al.* compared the effects of a high carbohydrate low fat diet, with lower carbohydrate diets that varied in the proportion of energy derived from fat or protein using obese adolescents (Demol *et al.*, 2009). LDL cholesterol, measured at 12 weeks and one year, did not statistically significantly differ between diet groups. Likewise, Sondike *et al.* (Sondike *et al.*, 2003) explored the effects of a low carbohydrate diet and a low fat diet on serum lipids in obese adolescents. After 12 weeks, LDL cholesterol had decreased from baseline in the low fat group ($p < 0.05$) but not in the low carbohydrate group ($p > 0.05$). This outcome also differed between conditions as the low fat group experienced a substantial significant decrease in LDL cholesterol compared with the low carbohydrate group ($p = 0.006$).

Six studies had four groups (Mahon *et al.*, 2007; Sacks *et al.*, 2009; Dansinger *et al.*, 2005; McMillan-Price *et al.*, 2006; Krauss *et al.*, 2006; Morgan *et al.*, 2009). Four studies compared lowest and highest carbohydrate intakes (Mahon *et al.*, 2007; Dansinger *et al.*, 2005; Krauss *et al.*, 2006; Morgan *et al.*, 2009). One study compared high and low carbohydrate with medium and high protein levels (Sacks *et al.*, 2009) and one study compared higher and lower carbohydrate on high and low GI diets (McMillan-Price *et al.*, 2006). Four studies had three groups and compared the lowest and highest carbohydrate intakes (Due *et al.*, 2008; Noakes *et al.*, 2006; Ginsberg *et al.*, 1998; Appel *et al.*, 2005).

Papers from Campos *et al.* (Campos *et al.*, 1995) and Dreon *et al.* (Dreon *et al.*, 1994) are from the same study. The results from Campos *et al.* (Campos *et al.*, 1995) are included in the meta-analysis.

Trials were separated into three main types on the basis of the proportion of energy derived from the macronutrients. For inclusion in a meta-analysis a 5% difference in energy from carbohydrate was taken as meaningful. Actual consumption was used rather than the intended diet unless otherwise stated – see the Trial Characteristics table.

If a trial tested the effects of diets which differed by 5% or more of energy from carbohydrate it was then further categorised into one of three categories. Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat also differed by 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein differed by 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more, but protein intakes were also different by more than 2%.

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

Three studies did not report data that could be incorporated into a meta-analysis (Kirkwood *et al.*, 2007; Wolever and Mehling, 2002): one of which provided baseline data only (Johnston *et al.*, 2006). None of these found statistically significant differences in LDL cholesterol over time or between groups. One further study could not be included in the meta-analysis because it had differences in carbohydrate of less than 5% between groups (Dale *et al.*, 2009). Dale *et al.* (Dale *et al.*, 2009) did not show changes in LDL cholesterol in the treatment groups.

Twenty studies were included in the meta-analysis comparing different carbohydrate and fat intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Overall heterogeneity denoted by I^2 was 76% (95% CI 64 to 84%).

Figure 2.31 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and LDL cholesterol (mmol/L)

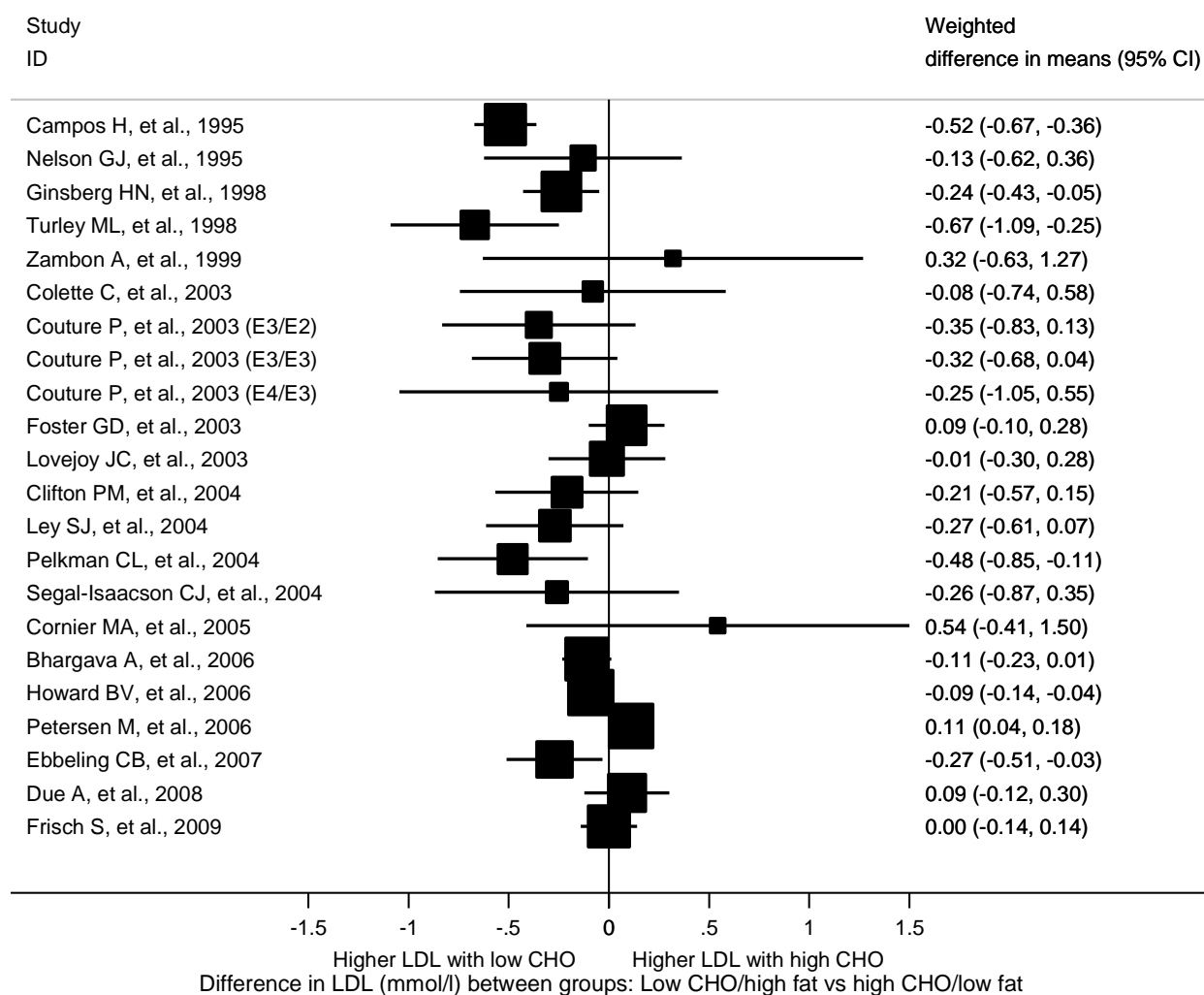
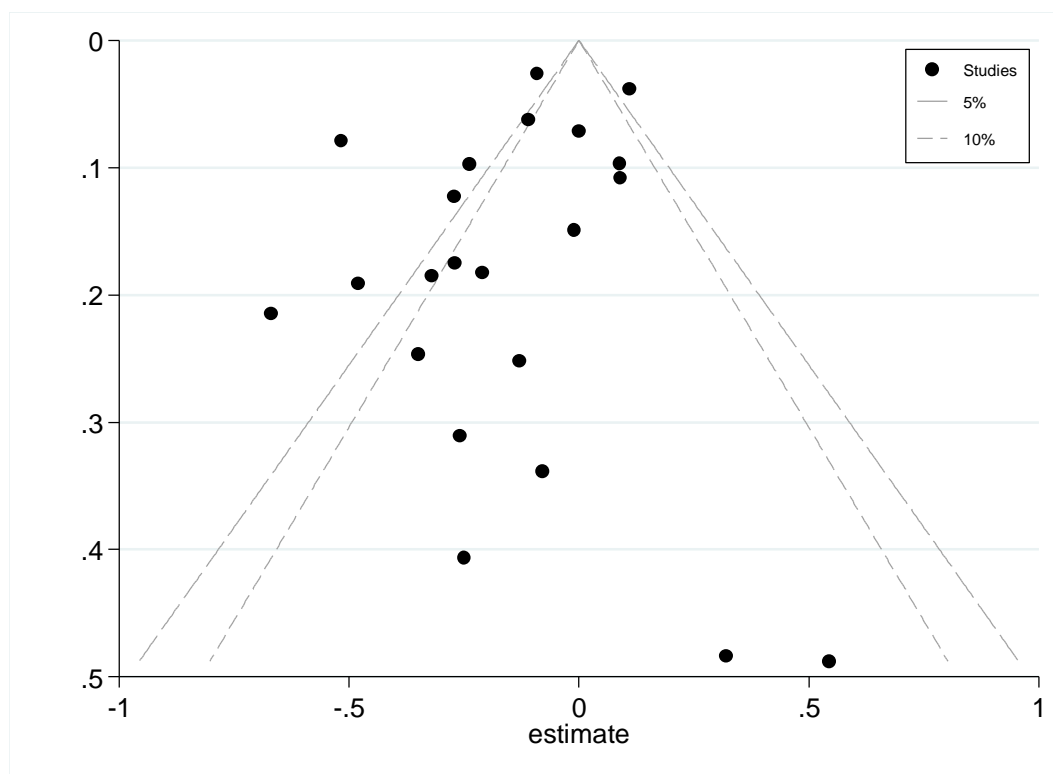


Figure 2.32 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat versus lower carbohydrate, higher fat diets and LDL cholesterol



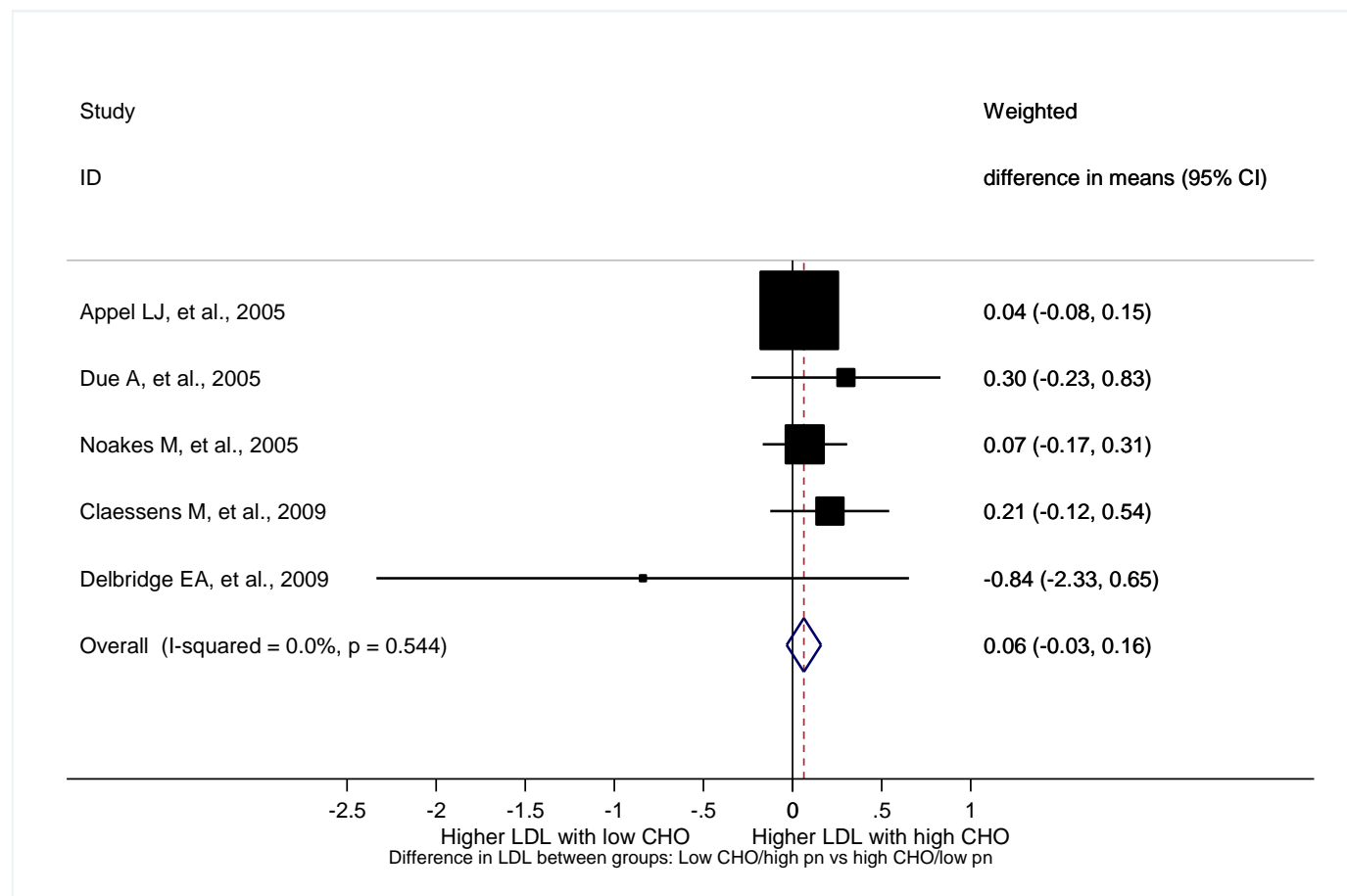
Comparison of higher carbohydrate, lower protein diets with lower carbohydrate, higher protein diets

All eligible studies were included in a meta-analysis; however there were multiple papers for some studies in which case the former one of each pair was selected for inclusion - (Appel *et al.*, 2005;Furtado *et al.*, 2008), (Due *et al.*, 2004;Due *et al.*, 2005), and (Noakes *et al.*, 2005;Clifton *et al.*, 2008).

Five studies were included in the meta-analysis comparing different carbohydrate and protein intakes and changes in LDL cholesterol reported as mmol/L. The percentage carbohydrate in the highest intake groups ranged from 55 to 63%, and in the lower carbohydrate groups from 40 to 49. Corresponding differences in protein were 14 to 18% and 21 to 31%. Three studies prescribed an energy restriction goal (Noakes *et al.*, 2005;Leidy *et al.*, 2007;Appel *et al.*, 2005). There was a lack of consistency between the studies in terms of weight change within the high and low carbohydrate groups. Body weights were unchanged in one study (Appel *et al.*, 2005), increased in one study (Delbridge *et al.*, 2009), and decreased in three studies (Due *et al.*, 2004;Leidy *et al.*, 2007;Noakes *et al.*, 2005). In one study body weights increased in the high carbohydrate group, and decreased in the low carbohydrate group (Claessens *et al.*, 2009). This may have acted as the driver for change in cholesterol.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. The pooled estimate indicated that LDL cholesterol was 0.06mmol/L (95% CI -0.03 to 0.16) higher with consumption of a higher carbohydrate, low protein diet. This was not significantly different from zero ($p=0.20$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 73%). Statistically, there was no evidence that higher carbohydrate lower protein diets are associated with differences in LDL cholesterol.

Figure 2.33 Forest plot for higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets and LDL cholesterol (mmol/L)



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

The papers by O'Brien *et al.* (O'Brien *et al.*, 2005) and Gray *et al.* (Gray *et al.*, 2008) were not included since they are multiple publications from the same study, which was also reported in Brehm *et al.* (Gray *et al.*, 2008). The latter was included in the meta-analysis.

Two studies did not report data that could be incorporated into a meta-analysis (Lasker *et al.*, 2008; Layman *et al.*, 2009). In the 12-month randomised parallel group trial by Layman *et al.* (Layman *et al.*, 2009), overweight and obese men and women received a high carbohydrate, low protein diet or a low carbohydrate, high protein diet. At four months, LDL cholesterol in the low carbohydrate, high protein group and the high carbohydrate, low protein group had statistically significantly increased and decreased, respectively ($p < 0.05$). LDL cholesterol also differed between conditions as the high carbohydrate, low protein group had lower LDL cholesterol compared with the low carbohydrate, high protein group ($p < 0.05$). These differences were not apparent at the 12-month follow-up, however. Similarly, Lasker *et al.* (Lasker *et al.*, 2008), using a comparable parallel group design, found that LDL cholesterol had reduced by 6.5% in the high carbohydrate diet group, but increased by 4.9% in the high protein group ($p = 0.046$). Differences over time were not reported.

Data from two papers (de Luis *et al.*, 2009a; de Luis *et al.*, 2008) which explored the dietary impact of high compared with low carbohydrate diets in individuals with different genetic profiles were not included in the meta-analysis as it was considered that these were from the same study as de Luis *et al.* (de Luis *et al.*, 2009b). Changes in LDL cholesterol were similar in both diet groups overall (de Luis *et al.*, 2009b). In individuals with different polymorphisms of the fatty acid binding protein 2 (FABP2) gene (de Luis *et al.*, 2008) no differences in response to either diet were reported. Similarly, in individuals with different polymorphisms of the uncoupling protein-3 gene (a gene with influence on energy expenditure and fat storage) (de Luis *et al.*, 2009a), separating participants according to genetic subgroups also showed no differences in response.

Finally, one study could not be included in the meta-analysis as no measure of variation was available (Dyson *et al.*, 2007). This study did not show a statistically significant difference in LDL cholesterol between high and low carbohydrate diets.

Twenty five studies were included in the meta-analysis comparing different carbohydrate, fat and protein intakes and changes in LDL cholesterol reported as mmol/L. There was considerable variation in the carbohydrate content of the comparison diets. The higher carbohydrate diets ranged from 43 to 67%, and the lower carbohydrate diets from 5 to 47%. The majority of studies prescribed an energy restriction goal for at least one diet group, the exceptions to this being just three studies (Clevidence *et al.*, 1992; Johnston *et al.*, 2004; Maki *et al.*, 2007). Accordingly, almost all studies reported decreases in body weight in all diet groups.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. The pooled estimate indicated that LDL cholesterol was 0.27mmol/L (95% CI 0.18 to 0.36) lower with consumption of a higher carbohydrate diet. This was significantly different from zero ($p < 0.001$). Overall heterogeneity denoted by I^2 was 55% (95% CI 31 to 71%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was evidence that higher carbohydrate, lower fat and protein diets are associated with lower levels of LDL cholesterol.

Figure 2.34 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and LDL cholesterol (mmol/L)

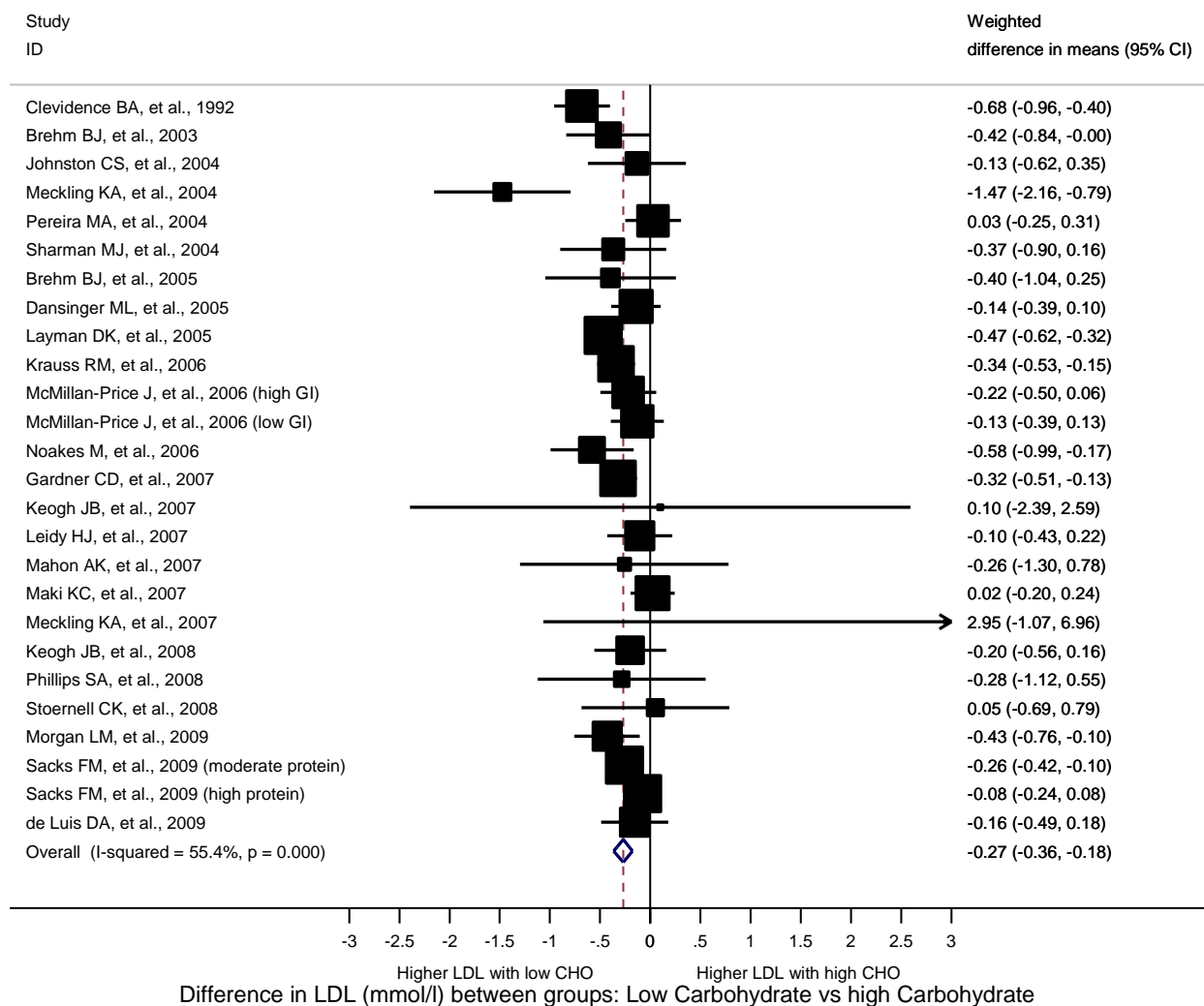
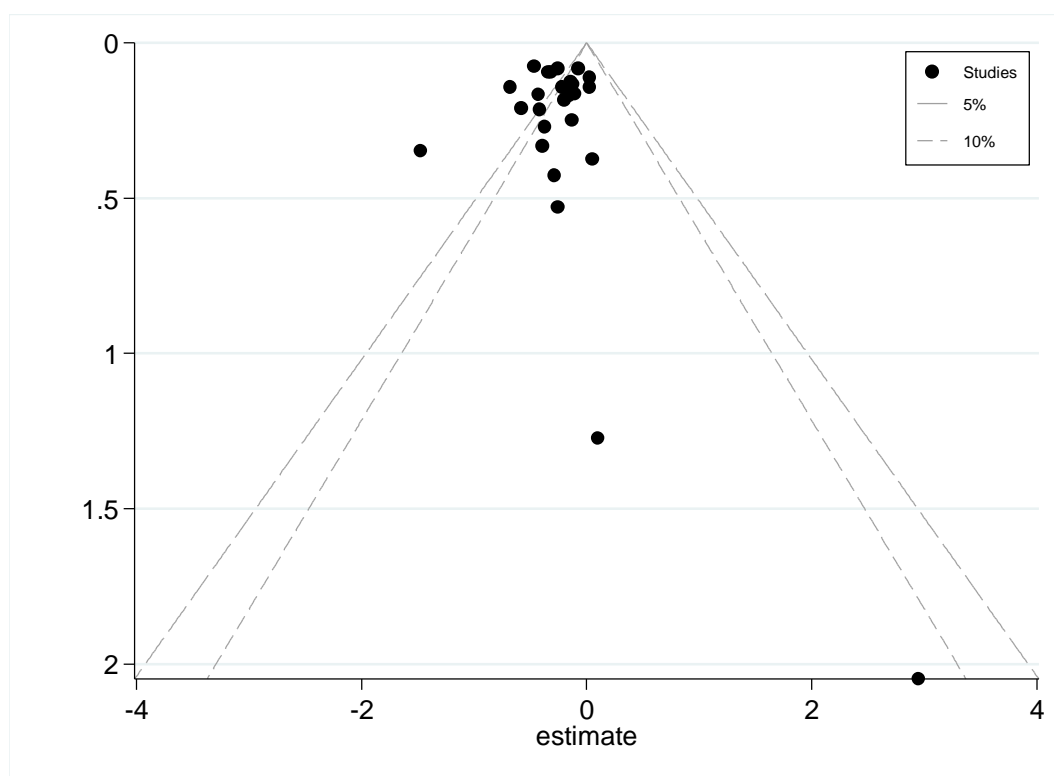


Figure 2.35 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat and protein versus lower carbohydrate, higher fat and protein diets and LDL cholesterol



Non-HDL cholesterol, total carbohydrate and high carbohydrate diets

Summary of cohort results

No cohort studies provided data.

Summary of RCT data

One study of adolescents (Sondike *et al.*, 2003) and three of adults (Pelkman *et al.*, 2004; Gardner *et al.*, 2007; Howard *et al.*, 2006) reported data on non-HDL cholesterol responses to manipulation of dietary carbohydrate intake.

Sondike *et al.* (Sondike *et al.*, 2003) conducted a parallel group trial using adolescents, in which a very low carbohydrate diet (<20g/day carbohydrate for two weeks then <40g/day for the remaining 10 weeks) plus an *ad libitum* intake of protein, fat and energy was compared with a low fat diet (<40g/day fat and 5 x 15g carbohydrate per serving) and *ad libitum* intake of fat-free dairy foods, fruits and vegetables. Following the intervention, non-HDL cholesterol had decreased from baseline in both dietary groups ($p < 0.05$ for both) but was generally lower in the very low carbohydrate group than the low fat group, with a statistically significant difference at 12 weeks (difference between groups, $p = 0.036$).

All three studies were included in the meta-analysis comparing high and low carbohydrate diets and changes in non-HDL cholesterol reported as mmol/L. One study had three groups (Gardner *et al.*, 2007) and compared the lowest and highest carbohydrate intakes. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from 10 weeks to three years. The pooled estimate indicated that non-HDL cholesterol was 0.03mmol/L (95% CI 0 to 0.06) lower with consumption of a higher carbohydrate diet. This was significantly different from zero ($p = 0.04$). Overall heterogeneity denoted by I^2 was 6% (95% CI 0 to 90%). Statistically, there was evidence that higher carbohydrate diets are associated with lower levels of non-HDL cholesterol. However it should be noted that one large study (Howard *et al.*, 2006) dominated the analysis and contributed 87% to the pooled estimate.

Figure 2.36 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and non-HDL cholesterol

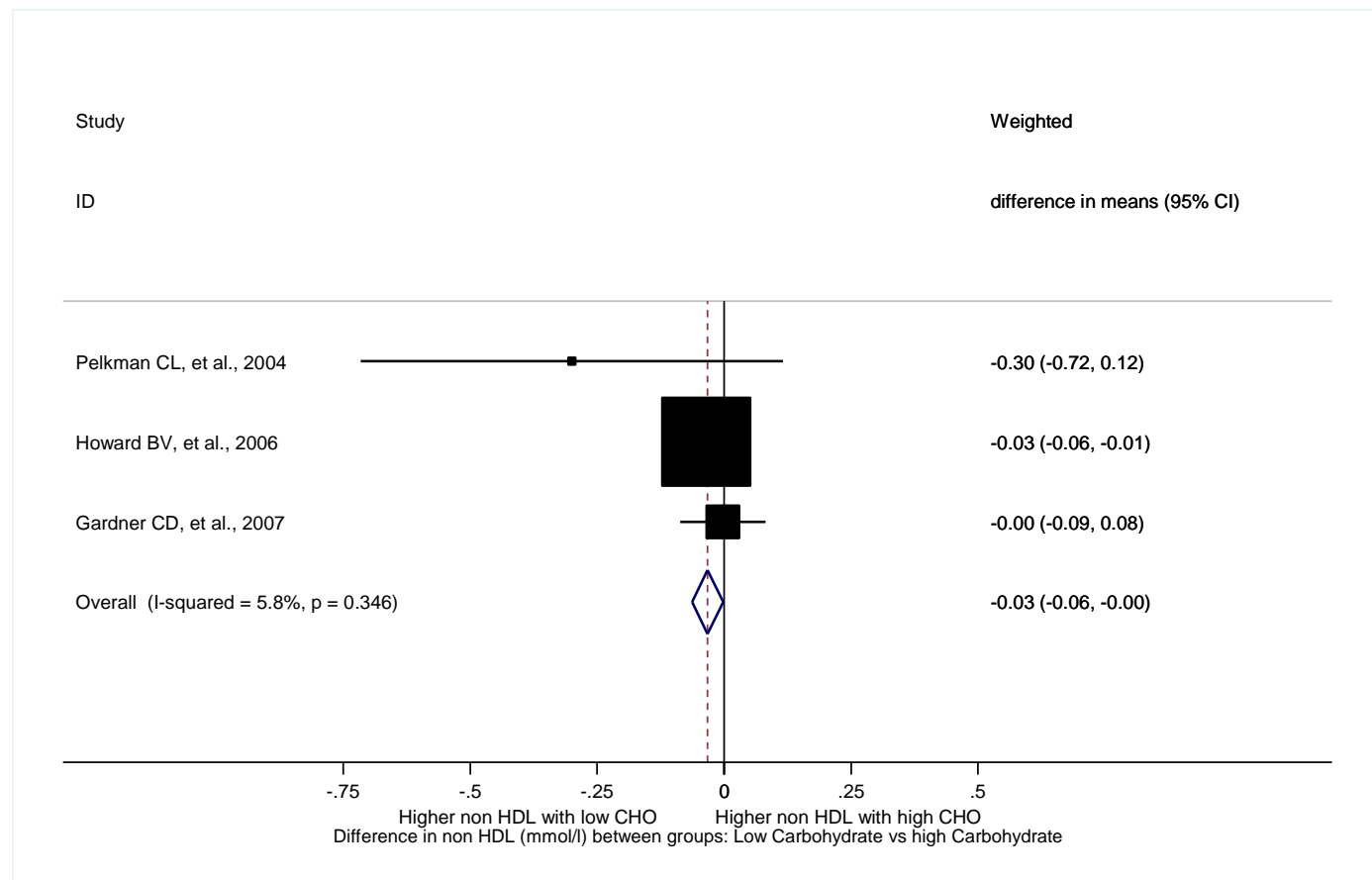


Table 2.51 LDL cholesterol and total carbohydrate: cohort study in adults

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 Details	Contrast	Exposure Units	Mean outcome	P trend	Adjustments
(Ludwig <i>et al.</i> , 1999) 13700 The CARDIA Study	USA, Multi- ethnic, Generally healthy, No hypertension, No T2DM	18-30 %M 45.9	5115	10 years	FFQ (700)	Carbohydrate, total (% energy)	LDL-C Fasting, mg/dL	Race - White	(51.9) vs (33.5)	% Energy	109.0 vs.109.0	0.56	age, alcohol, centre, education, energy intake, LDL-C, physical activity, gender, smoking, vitamin intake
13701 The CARDIA Study								Race - Black	(51.9) vs (33.5)	% Energy	106.9 vs. 109.2	0.57	As above

Table 2.52 LDL cholesterol and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
Adolescent studies																
(Demol <i>et al.</i> , 2009) 15405		High carbohydrate, low fat	20/20	94.4 (SD 5.3)	83.9 (SD 5.6)							LDL-C	Derived by calculation Fasting, (mg/dL)	12 weeks	Decrease	unclear
		Low carbohydrate, high fat	17/17	98.9 (SD 5.7)	89.0 (SD 6.2)			NS							Decrease	
		Low carbohydrate, high protein	18/18	103.1 (SD 5.5)	88.4 (SD 5.8)			NS							Decrease	
15406		High carbohydrate, low fat	20/20	94.4 (SD 5.3)	89.8 (SD 6.5)							LDL-C	Derived by calculation Fasting, (mg/dL)	1 year	Decrease	unclear
		Low carbohydrate, high fat	17/17	98.9 (SD 5.7)	82.1 (SD 7.2)			NS							Decrease	
		Low carbohydrate,	18/18	103.1 (SD 5.5)	96.8 (SD			NS							Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
		high protein			6.2)											
(Sondike <i>et al.</i> , 2003) *15990		Low fat	14/19			-25.1 (SD 25.3)	<0.05					LDL-C	Fasting serum (mg/dL)	12 weeks	Decrease	bias
		Very low carbohydrate	12/20			3.8 (SD 13.0)	>0.05	0.006							Decrease	
Adult studies																
(Appel <i>et al.</i> , 2005) *16315		High carbohydrate	161/164	129.2 (SD 32.4)		-11.6 (CI - 14.6, - 8.6)						LDL-C	Derived by calculation Fasting, Serum (mg/dL)	6 weeks	No change	No bias
		High protein	161/164	129.2 (SD 32.4)		-14.2 (CI - 17.5, - 10.9)		0.01							No change	
		High PUFA	161/164	129.2 (SD 32.4)		-13.1 (CI - 16.4, - 9.8)		NS							No change	
16317	LDL ≥130mg/dl	High carbohydrate	75/164	156.7 (SD 21)		-19.8 (CI - 24.2, - 15.5)						LDL-C	Derived by calculation Fasting, Serum (mg/dL)	6 weeks	No change	No bias
		High protein	75/164	156.7 (SD 21)		-23.6 (CI - 28.5, - 18.8)		NS							No change	
		High PUFA	75/164	156.7 (SD 21)		-21.9 (CI - 26.9, - 16.8)		NS							No change	
16319	LDL <130mg/dl	High carbohydrate	86/164	105.2 (SD 18.5)		-4.4 (CI - 7.8, -0.9)						LDL-C	Derived by calculation Fasting, Serum (mg/dL)	6 weeks	No change	No bias
		High protein	86/164	105.2 (SD 18.5)		-6.1 (CI - 9.9, -2.2)		NS							No change	
		High PUFA	86/164	105.2 (SD 18.5)		-5.4 (CI - 9.1, -1.8)		NS							No change	
(Bhargava, 2006) *16869		Control	379/all located not report	3.53 (SE SD 0.96	3.43 (SD 0.87)		0.05					LDL-C	Fasting plasma (mmol/L)	1 year	Decrease	unclear

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		Low fat	615/allocated not reported	3.5 (SD 0.94)	3.32 (SD 0.85)		0.05	0.05								Decrease	
(Brehm <i>et al.</i> , 2003) 15727		Low carbohydrate	22/22	124.86 (SE 5.39)	113.00 (SE 5.34)		<0.01	NS				LDL-C	Fasting (mg/dL)	3 months	Decrease	unclear	
		Moderate fat	20/20	113.80 (SE 6.36)	104.90 (SE 5.97)		<0.01									Decrease	
*15728		Low carbohydrate	22/22	124.86 (SE 5.39)	124.00 (SE 5.81)			NS				LDL-C	Fasting (mg/dL)	6 months	Decrease	unclear	
		Moderate fat	20/20	113.80 (SE 6.36)	107.80 (SE 5.86)											Decrease	
(Brehm <i>et al.</i> , 2005) 16385		Low carbohydrate	20/25	134.85 (SE 8.26)	130.1 (SE 7.16)			NS				LDL-C	Fasting plasma (mg/dL)	2 months	Decrease	No bias	
		Moderate fat	20/25	125.28 (SE 5.95)	111.15 (SE 7.35)											Decrease	
*16386		Low carbohydrate	20/25	134.85 (SE 8.26)	131.9 (SE 9.93)			NS				LDL-C	Fasting plasma (mg/dL)	4 months	Decrease	No bias	
		Moderate fat	20/25	125.28 (SE 5.95)	116.6 (SE 8.08)											Decrease	
(Campos <i>et al.</i> , 1995) *17090		High-fat minus low-fat higher CHO	43/allocated not reported						20 (SD 20)		0.0001	LDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear	
17095		High-fat	43/allocated not reported	134 (SD 24)	145 (SD 32)			0.0001				LDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear	
		Low-fat higher CHO	43/allocated not	134 (SD 24)	124 (SD 35)											Not reported	

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
			reported													
(Claessens <i>et al.</i> , 2009) *16823		High carbohydrate supplement	16/allocated not reported	2.52 (SE 0.2)	2.95 (SE 0.18)	0.43 (SE 0.13)	<0.05	NS				LDL-C	Fasting (mmol/L)	12 weeks	Increase	unclear
		High protein supplement - casein	14/allocated not reported	2.56 (SE 0.14)	2.81 (SE 0.15)	0.22 (SE 0.11)	NS	NS								Decrease
		High protein supplement – whey	18/allocated not reported	2.62 (SE 0.17)	3.09 (SE 0.2)	0.48 (SE 0.12)	<0.05	NS								Decrease
(Clevidence <i>et al.</i> , 1992) *16606		High fat diet	42/46	3.1 (SE 0.11)	3.39 (SE 0.1)	0.28	0.004	<0.001				LDL-C	Fasting plasma (mmol/L)	10 weeks	No change	unclear
		Low fat diet	42/46	3.1 (SE 0.11)	2.71 (SE 0.1)	-0.39	<0.001									No change
(Clifton <i>et al.</i> , 2004) 16747		High MUFA	31/35	3.25 (SD 0.76)	2.77 (SD 0.66)			Unclear				LDL-C	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Very low fat	31/35	3.25 (SD 0.9)	2.69 (SD 0.67)											Decrease
*16748		High MUFA	31/35	3.25 (SD 0.76)	2.93 (SD 0.65)		<0.0.1					LDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
		Very low fat	31/35	3.25 (SD 0.9)	2.72 (SD 0.78)		<0.01									Decrease
(Clifton <i>et al.</i> , 2008) *16006		High carbohydrate diet	38/38			-0.48 (SD 0.75)		NS				LDL-C	Fasting (mmol/L)	1.25 years	Decrease	unclear
		High protein diet	40/41			-0.57 (SD 0.87)										Decrease
(Colette <i>et al.</i> , 2003) *17413		High carbohydrate diet	15/15	3.61 (SE 0.26)	3.30 (SE 0.28)		NS					LDL-C	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		High MUFA diet	17/17	3.68 (SE	3.38		0.01	NS								Decrease

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				0.21)	(SE 0.19)											
(Cornier <i>et al.</i> , 2005) *16711	Insulin sensitive	High carbohydrate, low fat	6/10	124 (SE 12)	118 (SE 16)			NS				LDL-C	Fasting (mg/dL)	16 weeks	Decrease	unclear
		Low carbohydrate, high fat	6/11	110 (SE 14)	97 (SE 10)										Decrease	
(Couture <i>et al.</i> , 2003) *15876	Genetics - Apo E genotype E3/E2	High carbohydrate diet	3/3	2.51 (SD 0.69)	1.58 (SD 0.15)		0.10	Not reported/ unclear				LDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
	Genetics - Apo E genotype E3/E2	High MUFA diet	5/5	2.33 (SD 0.44)	1.93 (SD 0.4)		0.17								Decrease	
*15877	Genetics - Apo E genotype E3/E3	High carbohydrate diet	22/22	2.81 (SD 0.88)	2.34 (SD 0.71)		<0.01					LDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
	Genetics - Apo E genotype E3/E3	High MUFA diet	21/21	3.28 (SD 0.77)	2.66 (SD 0.5)		<0.01								Decrease	
*15878	Genetics - Apo E genotype E3/E4	High carbohydrate diet	8/8	3.36 (SD 0.86)	2.5 (SD 0.66)		<0.01					LDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
	Genetics - Apo E genotype E3/E4	High MUFA diet	6/6	3.29 (SD .99)	2.75 (SD .94)		0.3								Decrease	
(Dale <i>et al.</i> , 2009) 15984		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0.16 (CI 0.01, 0.31)	0.039		LDL-C	Fasting (mmol/L)	2 years	Decrease in both	unclear
17400		High carbohydrate diet	89/100	3.3 (SD 0.9)	3.04 (SD 0.77)							LDL-C	(mmol/L)	1 year	Decrease	unclear
		High MUFA diet	85/100	3.4 (SD 0.9)	3.27 (SD)										Decrease	

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17370	High carbohydrate diet	89/100	3.3 (SD 0.9)	3.18 (SD 0.81)	0.87)							LDL-C	Fasting (mmol/L)	2 years	Decrease	unclear
	High MUFA diet	85/100	3.4 (SD 0.9)	3.34 (SD 0.88)											Decrease	
(Dansinger <i>et al.</i> , 2005) 15698	Atkins	40/40				1.3 (SD 18)	NS	Unclear				LDL-C	Derived by calculation Fasting, Serum (mg/dL)	2 months	Decrease	No bias
	Ornish	40/40				-16.5 (SD 25)	0.01								Decrease	
	Weight watchers	40/40				-12.1 (SD 25)	0.01								Decrease	
	Zone	40/40				-9.7 (SD 27)	0.05								Decrease	
15699	Atkins	40/40				-2.7 (SD 14)	NS					LDL-C	Derived by calculation Fasting, Serum (mg/dL)	6 months	Decrease	No bias
	Ornish	40/40				-10.5 (SD 22)	0.01								Decrease	
	Weight watchers	40/40				-7 (SD 24)	NS								Decrease	
	Zone	40/40				-6.7 (SD 22)	NS								Decrease	
*15700	Atkins	40/40				-7.1 (SD 24)	NS					LDL-C	Derived by calculation Fasting, Serum (mg/dL)	1 year	Decrease	No bias
	Ornish	40/40				-12.6 (SD 19)	0.01								Decrease	
	Weight watchers	40/40				-9.3 (SD 27)	0.05								Decrease	
	Zone	40/40				-11.8 (SD 34)	0.05								Decrease	
(Delbridge <i>et al.</i> , 2009)	Low fat, high carbohydrate weight	70/70				-0.6 (SE 0.76)						LDL-C	Fasting (mmol/L)	1 year	Increase	unclear

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*15324		maintenance diet														
		Low fat, high protein weight maintenance diet	68/71			0.24 (SE 0.06)		0.273							Increase	
(de Luis <i>et al.</i> , 2008) 16145	Genetics - wild-type Ala54/Ala54	Low carbohydrate	55/105	123.7 (SD 29)	116.9 (SD 38)			NS				LDL-C	Fasting (mg/dL)	2 months	Decrease	unclear
		Low fat	55/99	116.7 (SD 38)	105.8 (SD 46)										Decrease	
16162	Genetics - mutant- type Ala54/Thr54 or Thr54/Thr54	Low carbohydrate	50/105	121.8 (SD 49)	114.7 (SD 38.2)			NS				LDL-C	Fasting (mg/dL)	2 months	Decrease	unclear
		Low fat	44/99	131.8 (SD 49)	120.7 (SD 37.2)										Decrease	
(de Luis <i>et al.</i> , 2009b) *16083		Low carbohydrate	52/52	125 (SD 36)	115 (SD 31)			NS				LDL-C	Fasting (mg/dL)	3 months	Decrease	unclear
		Low fat	66/66	121 (SD 38)	109 (SD 36)										Decrease	
(de Luis <i>et al.</i> , 2009a) 16697	Genetics - UCP3 Gene - 55CC polymorphism	Low carbohydrate	54/67	118.7 (SD 29.1)	108.9 (SD 38.0)		NS	Unclear				LDL-C	Serum (mg/dL)	2 months	Decrease	unclear
		Low fat	40/64	114.7 (SD 38.1)	115.5 (SD 46)		NS								Decrease	
16698	Genetics - UCP3 Gene - 55CT/TT polymorphism	Low carbohydrate	13/67	123.8 (SD 49.0)	117.6 (SD 38.2)		NS					LDL-C	Serum (mg/dL)	2 months	Decrease	unclear
		Low fat	24/64	127.8 (SD 49.0)	123.0 (SD 37.2)		NS								Decrease	
(Dreon <i>et al.</i> , 1994) 15637	Larger LDL particles	High-fat low CHO	87/105	135.9 (SD 26.6)	141.1 (SD 3.7)		<0.0001					LDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	135.9 (SD 26.6)	127.3 (SD 3.4)		<0.0001								Not reported	
17041	Smaller and denser LDL particles	High-fat low CHO	87/105	135.9 (SD 26.6)	150.8 (SD 7.5)		<0.0001					LDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	135.9 (SD 26.6)	120.7 (SD 3.4)		<0.0001								Not reported	

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7.6)																
17048	Larger LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 87/105						-13.7 (SD 2)		<0.0001	LDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17054	Smaller and denser LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 18/105						-30.1 (SD 5)		<0.0001				Both not reported	
17060	LDL particles remained large during study	High-fat low CHO	87/105	135.9 (SD 26.6)	131.8 (SD 4.9)		<0.001					LDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	135.9 (SD 26.6)	121.6 (SD 4.9)		<0.001								Not reported	
17064	LDL particles changed from large to small and dense during study	High-fat low CHO	87/105	135.9 (SD 26.6)	154.2 (SD 4.7)		<0.001					LDL-C	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	135.9 (SD 26.6)	135.5 (SD 4.3)		<0.001								Not reported	
17068	LDL particles remained large during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 51/105						-10.3 (SD 2)		<0.001	LDL-C	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17074	LDL particles changed from large to small and dense during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 36/105						-18.7 (SD 4)		<0.0001				Both not reported	
(Due <i>et al.</i> , 2005) *17545		High protein	23/23	2.9 (CI 2.5, 3.3)	2.8 (CI 2.4, 3.2)			NS				LDL-C	Fasting (μmol/L)	6 months	Decrease	unclear
		Moderate protein	23/18	3.2 (CI	3.1 (CI										Decrease	

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				2.9, 3.6)	2.8, 3.5)											
(Due <i>et al.</i> , 2008) *15299		Control	24/25	2.71 (CI 2.4, 3)	2.89 (CI 2.4, 3.3)	0.14 (CI - 0.1, 0.4)						Change in LDL- C	Derived by calculation Fasting plasma (mmol/L)	6 months	Increase	unclear
		High MUFA	39/52	2.75 (CI 2.5, 3)	2.67 (CI 2.5, 2.9)	-0.08 (CI - 0.2, 0.1)		NS							Increase	
		Low fat	43/48	2.78 (CI 2.5, 3)	2.79 (CI 2.5, 3.1)	0.01 (CI - 0.1, 0.2)		NS							Increase	
(Dyson <i>et al.</i> , 2007) 16348		Healthy eating diet	4/~6	3.57	3.4	-0.17						LDL-C	(mmol/L)	3 months	Decrease	bias
		Low carbohydrate diet	6/~6	3.46	3.6	0.16		NS							Decrease	
(Ebbeling <i>et al.</i> , 2005) 15491		Low fat diet	12/17	109.4 (SE 7.6)		-2.6% (CI -12.3, 8.2)						LDL-C	Fasting (mg/dL)	6 months	Decrease	unclear
		Low GI diet	11/17	113.1 (SE 6.1)		-9.1% (CI -18.6, 1.4)		NS							Decrease	
15492		Low fat diet	12/17	109.4 (SE 7.6)		-7.4% (CI -19.1, 6)						LDL-C	Fasting (mg/dL)	1 year	Decrease	unclear
		Low GI diet	11/17	113.1 (SE 6.1)		-9.7% (CI -21.6, 3.9)		NS							Decrease	
(Ebbeling <i>et al.</i> , 2007) *15449		Low fat diet	37/37			-16.3 (SE 3.3)						Change in LDL- C	Fasting Plasma, (mg/dL)	6 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-5.8 (SE 3.4)		0.3							Decrease	
15450		Low fat diet	37/37			-10.6 (SE 3.3)						Change in LDL- C	Fasting Plasma, (mg/dL)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-0.3 (SE 3.4)		0.3							Decrease	
(Foster <i>et</i>		Conventional diet	30/30			-7.4 (SD	<0.05					Change	Fasting	3 months	Decrease	unclear

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<i>al., 2003)</i> 15212		plan				16.6)						in LDL- C	serum (%)			
		Low carbohydrate diet	33/33			5.4 (SD 19.2)	NS	0.007							Decrease	
15213		Conventional diet plan	30/30			-1.5 (SD 15.8)	NS					Change in LDL- C	Fasting serum (%)	6 months	Decrease	unclear
		Low carbohydrate diet	33/33			2.7 (SD 12.8)	NS	0.34							Decrease	
*15214		Conventional diet plan	30/30			-3.1 (SD 12.0)	NS					Change in LDL- C	Fasting serum (%)	1 year	Decrease	unclear
		Low carbohydrate diet	33/33			0.31 (SD 16.6)	NS	0.52							Decrease	
(Frisch <i>et al.</i> , 2009) *15168		High carbohydrate diet	100/100			-0.03 (SD 0.51)	NS					LDL-C	Fasting serum (mmol/L)	6 months	Decrease	unclear
		Moderate carbohydrate diet	100/100			-0.03 (SD 0.5)	NS	0.921							Decrease	
15169		High carbohydrate diet	100/100			0.06 (SD 0.59)	NS					LDL-C	Fasting serum (mmol/L)	1 year	Decrease	unclear
		Moderate carbohydrate diet	100/100			0.02 (SD 0.65)	NS	0.564							Decrease	
(Gardner <i>et al.</i> , 2007) *15108		Atkins: low carbohydrate	70/77			2.3 (SD 23.5)		NS				LDL-C	Fasting plasma (mg/dL)	2 months	Decrease	No bias
		Ornish: high carbohydrate	64/76			-10.1 (SD 19.8)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			-5.3 (SD 17.8)									Decrease	
15109		Atkins: low carbohydrate	70/77			1.7 (SD 22.3)		NS				LDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
		Ornish: high carbohydrate	64/76			-3.2 (SD 19.9)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			0.5 (SD 14.9)									Decrease	
15110		Atkins: low	70/77			0.8 (SD		NS				LDL-C	Fasting	1 year	Decrease	No bias

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		carbohydrate				22.6)							plasma (mg/dL)			
		Ornish: high carbohydrate	64/76			-3.8 (SD 19)		NS								Decrease
		Zone: moderate carbohydrate	65/79			0 (SD 17.6)										Decrease
(Ginsberg <i>et al.</i> , 1998) *17248		Average American Diet	103/118		131.4 (SE 2.7)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		116.9 (SE 2.6)			<0.01								Not reported
		Step 1 diet	103/118		122.2 (SE 2.6)			<0.01								Not reported
17258	Men	Average American Diet	46/118		134.4 (SE 4.1)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	46/118		120.2 (SE 3.8)			<0.01								Not reported
		Step 1 diet	46/118		125.1 (SE 3.9)			<0.01								Not reported
17259	Women	Average American Diet	57/118		128.9 (SE 3.5)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	57/118		114.3 (SE 3.5)			<0.01								Not reported
		Step 1 diet	57/118		199.9 (SE 3.4)			<0.01								Not reported
17300	Black	Average American Diet	26/118		128.1 (SE 5.2)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	26/118		113.1 (SE 4.7)			<0.01								Not reported
		Step 1 diet	26/118		119.4 (SE 4.8)			<0.01								Not reported
17301	Non black	Average American Diet	77/118		132.4 (SE 3.1)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	77/118		118.2 (SE 3.1)			<0.01								Not reported
		Step 1 diet	77/118		123.1 (SE 3.0)			<0.01								Not reported
17316	Pre- menopausal	Average American Diet	39/118		116.73 (SE 3.0)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	39/118		102.1 (SE 2.8)			<0.01								Not reported
		Step 1 diet	39/118		108.0			<0.01								Not

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17317	Post-menopausal	Average American Diet	18/118		(SE 2.9) 155.4 (SE 5.2)							LDL-C	Fasting (mg/dL)	6 weeks	reported Not reported	No bias
		Low saturated fat diet	18/118		140.7 (SE 5.3)			<0.01							Not reported	
		Step 1 diet	18/118		145.4 (SE 4.9)			<0.01							Not reported	
17332	Men <40y	Average American Diet	30/118		123.7 (SE 4.5)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	30/118		109.5 (SE 4.2)			<0.01							Not reported	
		Step 1 diet	30/118		115.5 (SE 4.4)			<0.01							Not reported	
17333	Men >40y	Average American Diet	16/118		154.5 (SE 5.5)							LDL-C	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	16/118		140.3 (SE 4.7)			<0.01							Not reported	
		Step 1 diet	16/118		143.1 (SE 5.4)			<0.01							Not reported	
(Howard <i>et al.</i> , 2006) 16247		Control	approx n=1699 (5.8% sub-sample of 29294)	134.2 (SD 35.1)	127.0 (SD 34.0)	-6.2 (SD 29.1)						LDL-C	Fasting (mg/dL)	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub-sample of 19541)	133.3 (SD 35.3)	123.2 (SD 33.1)	-9.7 (SD 29.3)		<0.05							Decrease	
*17613		Low fat minus control	As above							-3.55 (CI - 6.58, -0.52)	<0.05	LDL-C	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Johnston		High	7/10	3.24 (SE		-12.4%	NS					LDL-C	Whole	6 weeks	Decrease	unclear

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<i>et al., 2004</i> *14861		carbohydrate, low fat		0.18)		(SE 6.5%)							blood (mmol/L)			
		High protein, low fat	9/10	3.09 (SE 0.25)		-8.2% (SE 4.4%)	NS	0.481							Decrease	
(Johnston <i>et al., 2006</i>) 17517		Low carbohydrate diet	10/10	3.38 (SE 0.29)								LDL-C	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low-carbohydrate diet	9/9	3.71 (SE 0.27)				NS							Decrease	
(Keogh <i>et al., 2007</i>) 15622		High carbohydrate diet	12/12	3.8 (SE 1.54)	3.01 (SE 1.34)		0.01					LDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	3.49 (SE 0.96)	2.86 (SE 0.76)		0.01	NS							Decrease	
*15623		High carbohydrate diet	12/12	3.8 (SE 1.54)	2.98 (SE 0.99)		0.01					LDL-C	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	3.49 (SE 0.96)	2.88 (SE 0.8)		0.01	NS							Decrease	
15624		High carbohydrate diet	completers not reported/12	4.16 (SE 0.49)	3.01 (SE 0.42)		0.01					LDL-C	Fasting plasma (mmol/L)	1 year	Decrease	unclear
		Low carbohydrate diet	completers not reported/13	3.48 (SE 0.21)	2.69 (SE 0.29)		NS	NS							Decrease	
(Keogh <i>et al., 2008</i>) *16722		High carbohydrate, low SFA	47/50	3.2 (SD 0.8)	2.9 (SD 0.6)		<0.001					LDL-C	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low carbohydrate, high SFA	52/57	3.2 (SD 1.0)	3.1 (SD 1.1)		<0.001	<0.05							Decrease	
(Kirkwood <i>et al., 2007</i>) 15668		Group 1: No advice	18/allocated not reported				NS					LDL-C	Fasting (mmol/L)	12 weeks	No change	unclear
		Group 2:	16/allo				NS	NS							Decrease	

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		Conventional weight loss diet	cated not report ed													
15669		Group 3: Exercise	19/allo cated not report ed				NS	NS				LDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
		Group 4: Conventional weight loss diet + exercise	16/allo cated not report ed	3.09	2.56	-0.53	0.05								Decrease	
(Krauss <i>et al.</i> , 2006) *17474		26% CHO High saturated fat	40/52	127.8 (SD 32.0)		1.1 (SE 2.7)		NS				LDL-C	Fasting plasma (mg/dL)	12 weeks	Decrease	unclear
		26% CHO Low saturated fat	47/59	129.1 (SD 25.7)		4.3 (SE 2.7)		<0.01							Decrease	
		39% CHO Low saturated fat	42/56	125.5 (SD 23.1)		-1.2 (SE 2.5)		NS							Decrease	
		54% CHO Low saturated fat	49/57	130.1 (SD 30.2)		-8.9 (SE 2.5)									Decrease	
(Lasker <i>et al.</i> , 2008) 15908		High carbohydrate	25/33			-6.50%						LDL-C	Fasting plasma (mmol/L)	4 months	Decrease	unclear
		High protein	25/32			4.9%		0.046							Decrease	
(Layman <i>et al.</i> , 2005) *16175		High carbohydrate diet	12/12	3.52 (SD 0.19)	3.07 (SD 0.15)		<0.05					LDL-C	Derived by calculation (mmol/L)	16 weeks	Decrease	unclear
		High protein diet	12/12	3.61 (SD 0.19)	3.54 (SD 0.22)		NS	<0.05							Decrease	
16176		High carbohydrate diet + exercise	12/12	3.24 (SD 0.15)	2.93 (SD 0.12)		<0.05					LDL-C	Derived by calculation (mmol/L)	16 weeks	Decrease	unclear
		High protein diet + exercise	12/12	3.2 (SD 0.22)	3.11 (SD 0.2)		NS	0.7							Decrease	
(Layman <i>et al.</i> , 2009) 14968		High carbohydrate, low protein diet	51/66		lower		<0.05					LDL-C	Fasting plasma	4 months	Decrease	unclear

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		Low carbohydrate, high protein diet	52/64		higher		<0.05	<0.05								Decrease	
14969		High carbohydrate, low protein diet	30/66				NS					LDL-C	Fasting plasma	1 year	Decrease	unclear	
		Low carbohydrate, high protein diet	41/64				NS	NS								Decrease	
(Leidy <i>et al.</i> , 2007) *16841		High protein, energy restricted	21/27	103 (SE 6)	85 (SE 4)	-18 (SE 5)		0.05				LDL-C	Derived by calculation Fasting, Serum (mg/dL)	12 weeks	Decrease	unclear	
		Moderate protein, energy restricted	25/27	118 (SE 4)	96 (SE 4)	-22 (SE 4)										Decrease	
(Ley <i>et al.</i> , 2004) *15934		Control	70/70			0.01 (SE 0.15)						LDL-C	Derived by calculation Fasting, Serum (mmol/L)	6 months	No change	unclear	
		Low fat	66/66			-0.26 (SE 0.09)		0.05								Decrease	
15935		Control	70/70			-0.02 (SE 0.15)						LDL-C	Derived by calculation Fasting serum (mmol/L)	1 year	No change	unclear	
		Low fat	66/66			-0.18 (SE 0.09)		NS								Decrease	
15936		Control	57/70			0.01 (SE 0.17)						LDL-C	Derived by calculation Fasting, Serum (mmol/L)	2 years	No change	unclear	
		Low fat	47/66			-0.07 (SE 0.09)		NS								Decrease	
15937		Control	51/70			-0.08 (SE 0.15)						LDL-C	Derived by calculation Fasting, Serum (mmol/L)	3 years	No change	unclear	

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15938		Low fat	48/66			-0.08 (SE 0.1)		NS								Decrease
		Control	52/70			-0.16 (SE 0.16)						LDL-C	Derived by calculation Fasting, Serum (mmol/L)	5 years	No change	unclear
		Low fat	51/66			-0.32 (SE 0.09)		NS								Decrease
(Lovejoy <i>et al.</i> , 2003) 14974		Control	13/15	2.85 (SE 0.16)		-0.09 (SE 0.13)		Unclear				LDL-C	Fasting (mmol/L)	3 months	Decrease	unclear
		Fat reduced	13/15	2.99 (SE 0.2)		-0.08 (SE 0.08)										Decrease
14975		Control	13/15	2.85 (SE 0.16)		0.09 (SE 0.14)						LDL-C	Fasting (mmol/L)	6 months	Decrease	unclear
		Fat reduced	13/15	2.99 (SE 0.2)		-0.03 (SE 0.12)										Decrease
*14976		Control	13/15	2.85 (SE 0.16)		0.1 (SE 0.1)						LDL-C	Fasting (mmol/L)	9 months	Decrease	unclear
		Fat reduced	13/15	2.99 (SE 0.2)		0.09 (SE 0.11)										Decrease
(Mahon <i>et al.</i> , 2007) *15071		Control	11/11	184 (SD 32)	174 (SD 30)	-10 (SD 46)	<0.05					LDL-C	Fasting serum (mg/dL)	9 weeks	No change	unclear
		Energy restriction + beef	14/14	157 (SD 49)	140 (SD 39)	-17 (SD 27)	<0.05	NS								Decrease
		Energy restriction + carbohydrate/fat	14/14	161 (SD 52)	141 (SD 33)	-20 (SD 50)	<0.05	NS								Decrease
		Energy restriction + chicken	15/15	141 (SD 40)	125 (SD 32)	-16 (SD 45)	<0.05	NS								Decrease
(Maki <i>et al.</i> , 2007) 17280		Ad libitum low GL diet	39/43	117.6 (SE 4.2)		-7 (SE 2.2)		NS				LDL-C	Derived by calculation Fasting (mg/dL)	12 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	123.4 (SE 5.7)		-3.6 (SE 2.9)										Decrease
*17281		Ad libitum low GL diet	39/43	117.6 (SE 4.2)		-2.8 (SE 3.2)		NS				LDL-C	Derived by calculation Fasting (mg/dL)	36 weeks	Decrease	unclear
		Low fat, energy	38/43	123.4 (SE		-1.9 (SE										Decrease

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(McMillan- Price <i>et al.</i> , 2006) *16222		restricted		5.7)		2.9)										
		High CHO, high GI diet	32/32	2.87 (SE 0.16)		0.04 (SE 0.10)						Change in LDL- C	Fasting (mmol/L)	12 weeks	Decrease	unclear
		High CHO, low GI diet	32/32	2.9 (SE 0.14)		-0.17 (SE 0.10)									Decrease	
		High protein, high GI diet	32/32	3.33 (SE 0.15)		0.26 (SE 0.10)		0.01 (compare d with high CHO, low GI diet)							Decrease	
		High protein, low GI diet	33/33	2.89 (SE 0.14)		-0.04 (SE 0.09)									Decrease	
(Meckling <i>et al.</i> , 2004) *14873		Low carbohydrate	15/20	169 (SE 11)	170 (SE 10)		NS	NS				LDL-C	Fasting (mg/dL)	10 weeks	Decrease	No bias
		Low fat	16/20	165 (SE 13)	113 (SE 9)		0.05								Decrease	
(Meckling and Sherfey, 2007) *16377 16378		Hypocaloric control diet	8/15	146 (SD 73)	190 (SD 221)		NS					LDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet	10/15	130 (SD 124)	76 (SD 37)		<0.05	NS							Decrease	
		Hypocaloric control diet + exercise	11/15	107 (SD 43)	93 (SD 45)		NS					LDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet + exercise	14/15	138 (SD 52)	130 (SD 31)		NS	NS							Decrease	
(Morgan <i>et al.</i> , 2009) 14707 *14708		Atkins	33/57	3.72 (SD 0.52)	3.59 (SD 0.73)		NS	Unclear						8 weeks	Decrease	
		Control	37/61	3.64 (SD 0.84)	3.79 (SD 0.78)		NS								No change	
		Slim Fast	44/59	3.55 (SE SD 0.81)	3.29 (SD 0.68)		0.01								Decrease	
		Weight Watchers	46/58	3.56 (SE SD 0.81)	3.12 (SD 0.71)		0.01								Decrease	
		Atkins	33/57	3.72 (SD	3.56		NS					LDL-C	Fasting	24 weeks	Decrease	unclear

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				0.52)	(SD 0.76)								whole blood (mmol/L)			
		Control	37/61	3.64 (SD 0.84)	3.55 (SD 0.73)		NS								No change	
		Slim Fast	44/59	3.55 (SE SD 0.81)	3.31 (SD 0.7)		0.01								Decrease	
		Weight Watchers	46/58	3.56 (SE SD 0.81)	3.13 (SD 0.58)		0.01								Decrease	
(Nelson <i>et al.</i> , 1995) *16940		High fat diet	11/11	112.8 (SD 26.8)	119.5 (SD 24.3)			NS				LDL-C	Fasting plasma (mg/dL)	50 days	Not reported	unclear
		Low fat diet	11/11	112.8 (SD 26.8)	114.5 (SD 21.3)										Not reported	
(Noakes <i>et al.</i> , 2005) 16992		High carbohydrate diet	48/48	3.90 (SE 0.12)	3.51 (SE 0.13)							LDL-C	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		High protein diet	52/52	3.79 (SE 0.14)	3.43 (SE 0.13)			NS							Decrease	
*16994		High carbohydrate diet	48/48	3.90 (SE 0.12)	3.71 (SE 0.13)	-0.19 (SE 0.08)						LDL-C	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	52/52	3.79 (SE 0.14)	3.53 (SE 0.13)	-0.26 (SE 0.09)		0.399							Decrease	
(Noakes <i>et al.</i> , 2006) 16576		High unsaturated fat	21/27	4.12 (SE 0.24)	3.54 (SE 0.25)			Unclear				LDL-C	Fasting plasma (mmol/L)	8 weeks	Decrease	unclear
		Very low carbohydrate	24/28	3.83 (SE 0.18)	3.89 (SE 0.28)										Decrease	
		Very low fat	22/28	3.65 (SE 0.22)	3.16 (SE 0.20)										Decrease	
*16577		High unsaturated fat	21/27	4.12 (SE 0.24)	3.78 (SE 0.22)	-0.34 (SE 0.14)						LDL-C	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear

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		Very low carbohydrate	24/28	3.83 (SE 0.18)	4.01 (SE 0.26)	0.18 (SE 0.18)										Decrease
		Very low fat	22/28	3.65 (SE 0.22)	3.25 (SE 0.22)	-0.4 (SE 0.11)										Decrease
(O'Brien <i>et al.</i> , 2005) 16954		Low carbohydrate	22/22					0.76				LDL-C	Fasting (mg/dL)	3 months	Decrease	unclear
		Moderate fat	19/19													Decrease
(Pereira <i>et al.</i> , 2004) *14581		Hypoenergetic low fat diet	11/23	124.3 (SE 9.86)	104.6 (SE 9.73)	-15% (SE 4.12%)						LDL-C	Derived by calculation Fasting, Serum (mg/dL)	67 days	Decrease	unclear
		Hypoenergetic low GL diet	14/23	138.7 (SE 9.75)	115.9 (SE 8.63)	-16.1% (SE 3.65%)		0.84								Decrease
(Pelkman <i>et al.</i> , 2004) 16879		Low fat, high carbohydrate diet	25/25	3.53 (SE 0.14)	2.89 (SE 0.14)		<0.05	Not reported/ unclear				LDL-C	Derived by calculation Fasting, (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	3.75 (SE 0.13)	3.24 (SE 0.13)		<0.05									Decrease
*16880		Low fat, high carbohydrate diet	25/25	3.53 (SE 0.14)	3.0 (SE 0.14)		<0.05					LDL-C	Derived by calculation Fasting, (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	3.75 (SE 0.13)	3.48 (SE 0.13)		<0.05									Decrease
16900	Weight stable during mainten ance	Low fat, high carbohydrate diet	12/25	3.57 (SE 0.20)	2.77 (SE 0.2)		<0.05					LDL-C	Derived by calculation Fasting, (mmol/L)	6 weeks	Decrease	unclear
	Weight stable during mainten ance	Moderate fat, lower carbohydrate diet	17/27	3.91 (SE 0.17)	3.35 (SE 0.17)		<0.05									Decrease
16901	Weight stable	Low fat, high carbohydrate diet	12/25	3.57 (SE 0.20)	3.05 (SE 0.2)		<0.05					LDL-C	Derived by calculation	10 weeks	Decrease	unclear

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
	during maintenance												Fasting, (mmol/L)			
	Weight stable during maintenance	Moderate fat, lower carbohydrate diet	17/27	3.91 (SE 0.17)	3.62 (SE 0.13)		NS								Decrease	
(Petersen <i>et al.</i> , 2006) 17202	Women	Hypoenergetic high carbohydrate, low fat diet	251/29 2	3.28 (SD 0.8)		-0.21 (SD 0.54)						LDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
	Women	Hypoenergetic low carbohydrate, high fat diet	235/28 7	3.24 (SD 0.82)		-0.11 (SD 0.49)									Decrease	
17203	Men	Hypoenergetic high carbohydrate, low fat diet	85/97	3.53 (SE (SD 0.78		-0.42 (SD 0.62)						LDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
	Men	Hypoenergetic low carbohydrate, high fat diet	77/95	3.41 (SD 0.75)		-0.24 (SD 0.51)									Decrease	
17204		Hyperenergetic low carbohydrate, high fat diet	312/38 2	3.28 (SD 0.81)		-0.14 (SD 0.5)						LDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
		Hypoenergetic high carbohydrate, low fat diet	336/38 9	3.34 (SD 0.8)		-0.26 (SD 0.57)									Decrease	
*17216		Hypoenergetic low carbohydrate, high fat diet minus hypoenergetic high carbohydrate, low fat diet	Low CHO: 312/38 3 High CHO: 336/38 9						0.11 (CI 0.03, 0.18)	0.01		LDL-C	Fasting plasma (mmol/L)	10 weeks	Decrease in both	bias
(Phillips <i>et al.</i> , 2008) *17421		Low carbohydrate diet	10/~14	82.4 (SE 14.2)	95.4 (SE 13.7)		NS	NS				LDL-C	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
		Low fat diet	8/~14	93.8 (SE 6.8)	84.4 (SE 9.2)		NS								Decrease	
(Sacks <i>et</i>		High-fat, average-	ITT:		123	-3.2%						LDL-C	Fasting	6 months	Decrease	No bias

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<i>al., 2009)</i> 15571		protein	/204		(SD 33)								serum (mg/dL)			
		High-fat, high-protein	ITT: /201		124 (SD 31)	-1.1%									Decrease	
		Low-fat, average-protein	ITT: /204		116 (SD 29)	-6.6%									Decrease	
		Low-fat, high-protein	ITT: /202		120 (SD 33)	-4.8%									Decrease	
*15572		High-fat, average-protein	ITT: /204	128 (SD 32)	127 (SD 33)	-0.2%						LDL-C	Fasting serum (mg/dL)	2 years	Decrease	No bias
		High-fat, high-protein	ITT: /201	126 (SD 31)	124 (SD 31)	-1.3%									Decrease	
		Low-fat, average-protein	ITT: /204	124 (SD 33)	117 (SD 31)	-5.9%		0.001 (compare d with high-fat diets)							Decrease	
		Low-fat, high-protein	ITT: /202	126 (SD 32)	121 (SD 33)	-3.9%									Decrease	
(Segal- Isaacson <i>et al.</i> , 2004) *14984		Low fat diet	4/4	138 (SD 19)	82 (SD 7)		<0.05					LDL-C	Fasting whole blood (mg/dL)	6 weeks	No change	unclear
		Very low carbohydrate	4/4	138 (SD 19)	92 (SD 23)		<0.05	0.333							Decrease	
(Sharman <i>et al.</i> , 2004) *14750		Low fat	15/15	3.25 (SD 0.73)	2.68 (SD 0.67)		0.05					LDL-C	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15	3.25 (SD 0.73)	3.05 (SD 0.8)		NS	0.05							Decrease	
(Stoernell <i>et al.</i> , 2008) *16522		Low carbohydrate diet	10/14	2.95 (SD 0.62)	2.83 (SD 0.45)			NS				LDL-C	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low fat diet	13/14	2.92 (SD 1.01)	2.88 (SD 1.10)										Decrease	
(Turley <i>et al.</i> , 1998) *15209		Low fat, high carbohydrate diet	36/38	3.61 (SD 0.82)	2.97 (SD 0.94)			0.001				LDL-C	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Western diet	36/38	3.61 (SD 0.82)	3.64 (SD 0.88)										Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
(Wolever and Mehling, 2002) 17010		High carbohydrate, high GI	11/11					NS				LDL-C	Fasting	16 weeks	Decrease	unclear
		High carbohydrate, low GI	13/13					NS							Decrease	
		Low carbohydrate, high MUFA	11/11					NS							Increase	
(Zambon <i>et al.</i> , 1999) 16265		High carbohydrate, energy restriction	11/11	3.35 (SD 0.54)	3.10 (SD 0.53)		<0.05					LDL-C	Fasting plasma (mmol/L)	3 months	Decrease	unclear
		Olive oil enriched energy restriction diet	9/9	3.50 (SE SD 0.7)	3.03 (SD 0.87)		NS	NS							Decrease	
*16266		High carbohydrate, energy restriction	5/11	3.35 (SD 0.54)	3.05 (SD 0.78)		NS					LDL-C	Fasting plasma (mmol/L)	6 months	Decrease	unclear
		Olive oil enriched energy restriction diet	7/9	3.2 (SD 0.7)	2.73 (SD 0.75)		<0.05	NS							Decrease	
Non-HDL-C																
Adolescent study																
(Sondike <i>et al.</i> , 2003) 15993		Low fat	14/19			-13.6 (SD 13.4)	<0.05					Non-HDL-C	Fasting serum (mg/dL)	12 weeks	Decrease	bias
		Very low carbohydrate	12/20			-26.0 (SD 22.3)	<0.05	0.036							Decrease	
Adult studies																
(Howard <i>et al.</i> , 2006) **16250		Control	approx n=1699 (5.8% sub- sample of 29294)	165.8 (SD 41.1)	158.4 (SD 37.0)	-6.6 (SD 32.6)						Non-HDL-C	Fasting (mg/dL)	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub- sample)	163.9 (SD 39.5)	154.3 (SD 36.5)	-9.7 (SD 32.0)		NS							Decrease	

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			of 19541)													
17616		Low fat minus control	As above							-3.08 (CI - 6.37, 0.22)	NS	Non- HDL-C	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Gardner <i>et al.</i> , 2007) **15117		Atkins: low carbohydrate	70/77			-8 (SD 26.3)		NS				Non- HDL-C	Fasting plasma (mg/dL)	2 months	Decrease	No bias
		Ornish: high carbohydrate	64/76			-7.8 (SD 17.8)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			-10.2 (SD 21.7)									Decrease	
15118		Atkins: low carbohydrate	70/77			-4.7 (SD 23.1)		NS				Non- HDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
		Ornish: high carbohydrate	64/76			-4.7 (SD 22.1)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			-3.7 (SD 18.8)									Decrease	
15119		Atkins: low carbohydrate	70/77			-5.1 (SD 22.5)		NS				Non- HDL-C	Fasting plasma (mg/dL)	1 year	Decrease	No bias
		Ornish: high carbohydrate	64/76			-6.8 (SD 20.3)		NS							Decrease	
		Zone: moderate carbohydrate	65/79			-0.5 (SD 20)									Decrease	
(Pelkman <i>et al.</i> , 2004) 16881		Low fat, high carbohydrate diet	25/25	4.36 (SE 0.15)	3.52 (SE 0.15)		<0.05	Not reported/ unclear				Non- HDL-C	Fasting (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	4.5 (SE 0.14)	3.79 (SE 0.14)		<0.05								Decrease	
**16882		Low fat, high carbohydrate diet	25/25	4.36 (SE 0.15)	3.73 (SE 0.15)		<0.05					Non- HDL-C	Fasting (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	4.5 (SE 0.14)	4.03 (SE 0.15)		<0.05								Decrease	

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
16902	Weight stable during mainte- nance	Low fat, high carbohydrate diet	12/25	4.35 (SE 0.21)	3.41 (SE 0.21)		<0.05					Non- HDL-C	Fasting (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	17/27	4.67 (SE 0.18)	3.90 (SE 0.18)		<0.05								Decrease	
16903	Weight stable during mainte- nance	Low fat, high carbohydrate diet	12/25	4.35 (SE 0.21)	3.88 (SE 0.21)		<0.05					Non- HDL-C	Fasting (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	17/27	4.67 (SE 0.18)	4.19 (SE 0.18)		<0.05								Decrease	

*This result has been used in the meta-analysis for high carbohydrate diets and LDL cholesterol

**This result has been used in the meta-analysis for high carbohydrate diets and non-HDL cholesterol

Triacylglycerol, total carbohydrate and high carbohydrate diets

Summary of cohort results

Data were extracted from two publications, reporting results from two studies (Ludwig *et al.*, 1999) (Schroeder *et al.*, 2007). The CARDIA study (Ludwig *et al.*, 1999) reported total carbohydrate intake as a percentage of total energy whereas total carbohydrates were presented as grams per day in the Middle-aged Runners Study (Schroeder *et al.*, 2007) as measured by a food diary. No significant association was seen between total carbohydrate intake and TAG levels in either of these studies.

The CARDIA study (Ludwig *et al.*, 1999) adjusted for an appropriate number of variables including age, gender, alcohol intake and smoking status while the Middle-aged Runners Study (Schroeder *et al.*, 2007) adjusted only for age.

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

Summary of RCT data

Sixty six studies, presented in seventy papers, explored the effects of dietary variation in carbohydrate - replacing carbohydrate with fat, protein or both - on TAG. Nine studies also presented results for non-esterified fatty acids (McMillan-Price *et al.*, 2006; Helge, 2002; Cornier *et al.*, 2005; Due *et al.*, 2004; Claessens *et al.*, 2009; Lofgren *et al.*, 2005; Wolever and Mehling, 2002; Noakes *et al.*, 2005; Kirk *et al.*, 2009; Wolever and Mehling, 2003), two of which did not provide extractable data on TAG – narrative descriptions only were provided (Kirk *et al.*, 2009; Wolever and Mehling, 2003).

Of the sixty six studies, nine employed a crossover design (Furtado *et al.*, 2008; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Dreon *et al.*, 1994; Ginsberg *et al.*, 1998; Nelson *et al.*, 1995; Segal-Isaacson *et al.*, 2004; Turley *et al.*, 1998), one implemented a factorial design (Dale *et al.*, 2009) and the remaining used a parallel group design.

Trials were conducted in a range of countries, which included: Australia (8), Canada (4), Denmark (3), New Zealand (3), the UK (2), Switzerland (2), Spain (1), Israel (1), France (1), Germany (1), Sweden (1), Italy (1), the Netherlands (1), Scotland (1) and one was a European trial. The majority of studies, however, were carried out in the USA (35).

Two trials studied adolescents (Demol *et al.*, 2009; Sondike *et al.*, 2003) and the remainder were studies of adults. Fifteen studies included women only (Brehm *et al.*, 2005; O'Brien *et al.*, 2005; Brehm *et al.*, 2003; Layman *et al.*, 2005; Leidy *et al.*, 2007; Mahon *et al.*, 2007; Meckling and Sherfey, 2007; Clifton *et al.*, 2004; Cornier *et al.*, 2005; Dale *et al.*, 2009; Kirkwood *et al.*, 2007; Lofgren *et al.*, 2005; Segal-Isaacson *et al.*, 2004; Zambon *et al.*, 1999; Howard *et al.*, 2006; Gardner *et al.*, 2007; Clifton *et al.*, 2008; Noakes *et al.*, 2005) and eleven studied males (Krauss *et al.*, 2006; Sharman *et al.*, 2004; Campos *et al.*, 1995; Clevidence *et al.*, 1992; Couture *et al.*, 2003; Dreon *et al.*, 1994; Lovejoy *et al.*, 2003; Nelson *et al.*, 1995; Turley *et al.*, 1998; Helge, 2002; Landry *et al.*, 2003).

Most of the studies that reported TAG recruited participants with a BMI greater than 25kg/m². In fact, only in two studies was the mean study BMI within the non-overweight range (Ginsberg *et al.*, 1998; Nelson *et al.*, 1995).

Final numbers of participants ranged from four to 811 (Segal-Isaacson *et al.*, 2004; Sacks *et al.*, 2009), other than the Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006), which had an extremely large sample size of 48,835 (only 5.8% provided a blood sample).

Six studies had four groups (Mahon *et al.*, 2007; Sacks *et al.*, 2009; Dansinger *et al.*, 2005; McMillan-Price *et al.*, 2006; Krauss *et al.*, 2006; Morgan *et al.*, 2009). Four studies compared lowest and highest carbohydrate intakes (Mahon *et al.*, 2007; Dansinger *et al.*, 2005; Krauss *et al.*, 2006; Morgan *et al.*, 2009). One study compared high and low carbohydrate with medium and high protein levels (Sacks *et al.*, 2009) and one study compared higher and lower carbohydrate on high and low GI diets (McMillan-Price *et al.*, 2006). Six studies had three groups and compared the lowest and highest carbohydrate intakes (Due *et al.*, 2008; Noakes *et al.*, 2006; Ginsberg *et al.*, 1998; Appel *et al.*, 2005; Gardner *et al.*, 2007; Raatz *et al.*, 2005).

Two studies were not included in the meta-analysis as the participants used were adolescents aged 12-18 years (Demol *et al.*, 2009; Sondike *et al.*, 2003). The study reported by Demol *et al.* compared the effects of a high carbohydrate low fat diet, with lower carbohydrate diets that varied in the proportion of energy derived from fat or protein using obese adolescents (Demol *et al.*, 2009). Triacylglycerol, measured at 12 weeks and one year, did not statistically significantly differ between diet groups. Likewise, Sondike *et al.* (Sondike *et al.*, 2003) explored the effects of a low carbohydrate diet and a low fat diet on serum lipids in obese adolescents. After 12 weeks, TAG had decreased from baseline in the low carbohydrate group ($p < 0.05$) but not in the low fat group. This outcome also marginally differed between conditions as the low carbohydrate group experienced a decrease in LDL cholesterol compared with the low fat group, albeit a not statistically significant difference ($p = 0.07$).

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 lipids. However, it was included in the meta-analysis as the carbohydrate differences between the groups met our inclusion criteria of >5% of energy.

Trials were separated into three main types on the basis of the proportion of energy derived from the macronutrients. For inclusion in a meta-analysis a 5% difference in energy from carbohydrate was taken as meaningful. Actual consumption was used rather than the intended diet unless otherwise stated – see the Trial Characteristics table.

If a trial tested the effects of diets which differed by 5% or more of energy from carbohydrate it was then further categorised into one of three categories. Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat also differed by 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein differed by 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more, but protein intakes were also different by more than 2%.

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

Papers from Campos *et al.* (Campos *et al.*, 1995) and Dreon *et al.* (Dreon *et al.*, 1994) are from same study. The results from Campos *et al.* (Campos *et al.*, 1995) are included in the meta-analysis.

Four studies did not report data that could be incorporated into the meta-analysis (Kirkwood *et al.*, 2007; Wolever and Mehling, 2002; Peterson and Jovanovic-Peterson, 1995): one of which provided baseline data only (Johnston *et al.*, 2006). Studies by Johnston *et al.* (Johnston *et al.*, 2006), Kirkwood *et al.* (Kirkwood *et al.*, 2007) and Wolever *et al.* (Wolever and Mehling, 2002) showed no differences in TAG in the diet groups at follow-up.

In a 12-week study, Kirkwood *et al.* randomised individuals to a low-fat, high-carbohydrate (including sucrose) energy-reduced diet or a 'no dietary change' diet or the same diets with the addition of an exercise regimen. The carbohydrate difference between the non-exercise groups was very small, but was 52 versus 44% energy in the high carbohydrate and 'no dietary advice' exercise groups respectively. No data were provided in the paper, but the authors reported that there were no differences between groups in TAG after 12 weeks (Kirkwood *et al.*, 2007).

(Wolever and Mehling, 2002) compared 4-month high carbohydrate (55%) diets that were high or low GI with a lower carbohydrate (45%), high monounsaturated fat diet. Data were not provided, however the authors did not report a statistically significant difference in TAG between diets.

Johnston *et al.* compared a ketogenic low-carbohydrate (5% carbohydrate) diet with a nonketogenic low-carbohydrate (40% carbohydrate) diet in 20 obese adults for six weeks. Both diets were equally effective in terms of weight loss, but the authors reported that there was no difference between the diets in terms of TAG reduction (follow up data not provided in the paper) (Johnston *et al.*, 2006).

Peterson *et al.* (Peterson and Jovanovic-Peterson, 1995) conducted a 12-week crossover trial to explore the effects of a 40% carbohydrate calorically restricted diet compared with a 55% carbohydrate calorically restricted diet in obese women. Overall, fasting TAG levels appeared to increase during the 55% carbohydrate diet and decrease during the 40% carbohydrate diet (Peterson and Jovanovic-Peterson, 1995). Women with previous gestational diabetes who consumed the 40% carbohydrate bar first then moved on to the 55% carbohydrate bar second experienced a statistically significant increase in TAG levels from one diet to the next ($p < 0.05$). Likewise, the obese group who consumed the 55% carbohydrate bars first and the 40% carbohydrate bars second witnessed a statistically significant decrease from baseline ($p < 0.05$). However, the significance of the between-group differences in TAG is not clear since the authors presented the data only by order of each trial arm. The authors concluded that there was a beneficial decrease in TAG on the lower carbohydrate phase and that this approach may assist in decreasing TAG levels in obese women.

One study could not be included in the meta-analysis as it had a difference in carbohydrate of less than 5% between groups (Dale *et al.*, 2009). Both high carbohydrate and low carbohydrate, high MUFA diet groups experienced a decrease in TAG at one and two years. However, the extent of change was not different in each diet group.

Twenty-six studies were included in the meta-analysis comparing different carbohydrate and fat intakes and changes in TAG reported as mmol/L.

Eleven studies had an energy restriction goal (Clifton *et al.*, 2004; Colette *et al.*, 2003; Cornier *et al.*, 2005; Foster *et al.*, 2003; Frisch *et al.*, 2009; Golay *et al.*, 2000; Lofgren *et al.*, 2005; Pelkman *et al.*, 2004; Petersen *et al.*, 2006; Raatz *et al.*, 2005; Segal-Isaacson *et al.*, 2004). Accordingly, 14 studies reported a decrease in weight across all study groups, although the magnitude of weight loss may have been greater in one group rather than another. Two studies reported no change in body weights (Campos *et al.*, 1995; Turley *et al.*, 1998), and one study reported a consistent increase (Landry *et al.*, 2003). In some studies the direction of weight change was different between dietary groups (Segal-Isaacson *et al.*, 2004; Pelkman *et al.*, 2004; Nelson *et al.*, 1995; Colette *et al.*, 2003; Frisch *et al.*, 2009). In all cases this entailed a reduction in weight in the high carbohydrate group and no change in the low carbohydrate group. The high carbohydrate groups consumed, on average, 57% energy from carbohydrate (range 47-65) and the low carbohydrate groups consumed 39% (range 5-52).

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. Heterogeneity was more than 75% (97%). This indicates substantial heterogeneity between the trials, so a pooled estimate would have little meaning. Accordingly, no pooled estimate is provided on the Forest plot. However, the majority of the trials report higher TAG levels with consumption of a higher carbohydrate diet. The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias.

Figure 2.37 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and TAG (mmol/L)

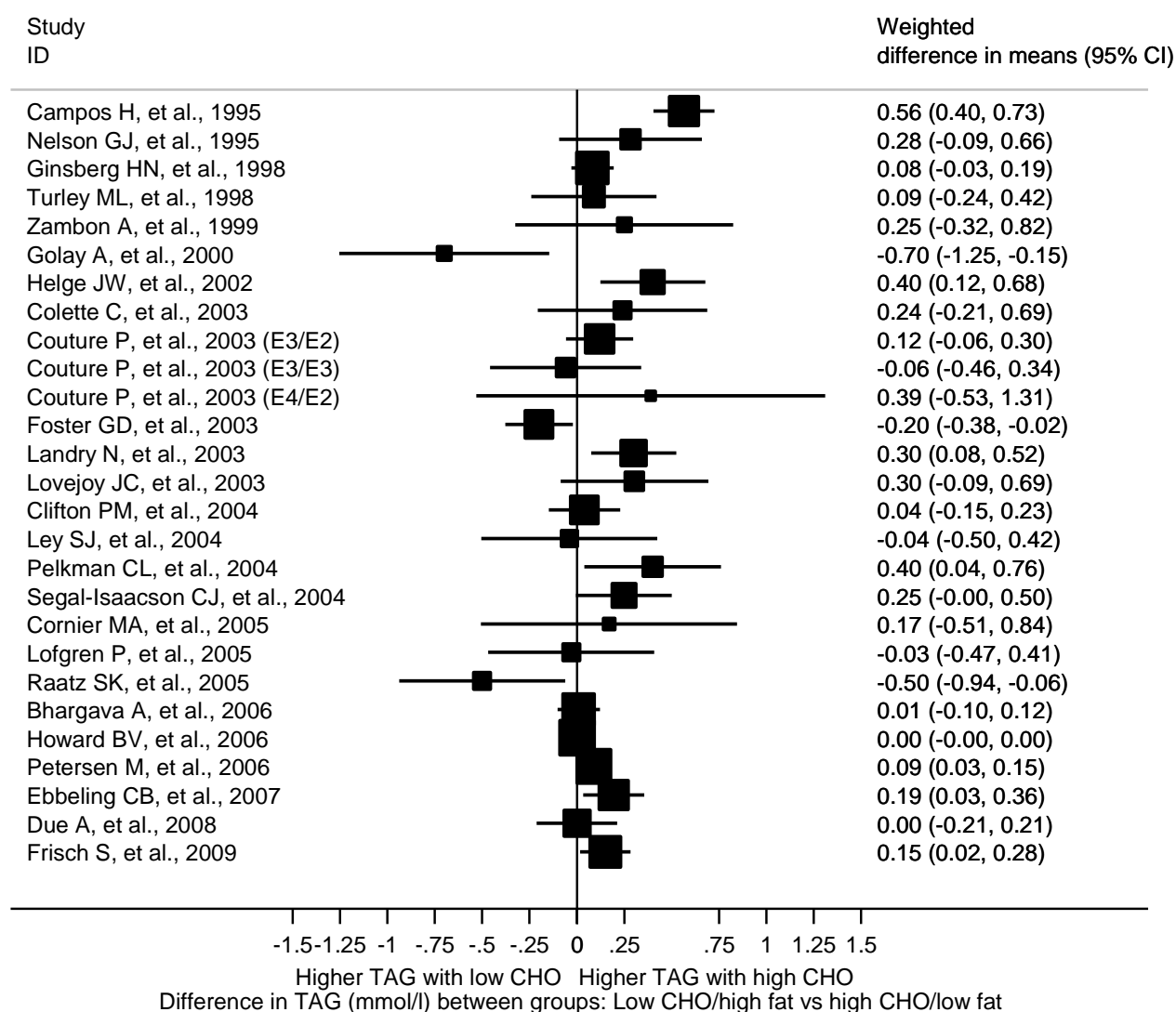
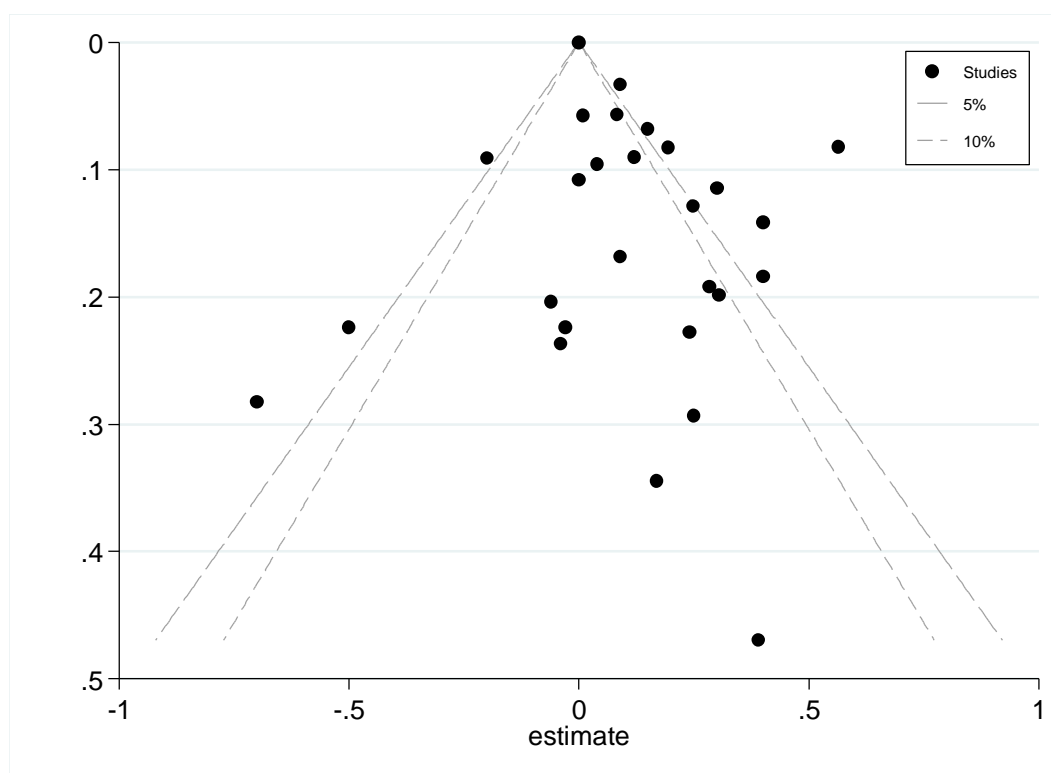


Figure 2.38 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat versus lower carbohydrate, higher fat diets and TAG



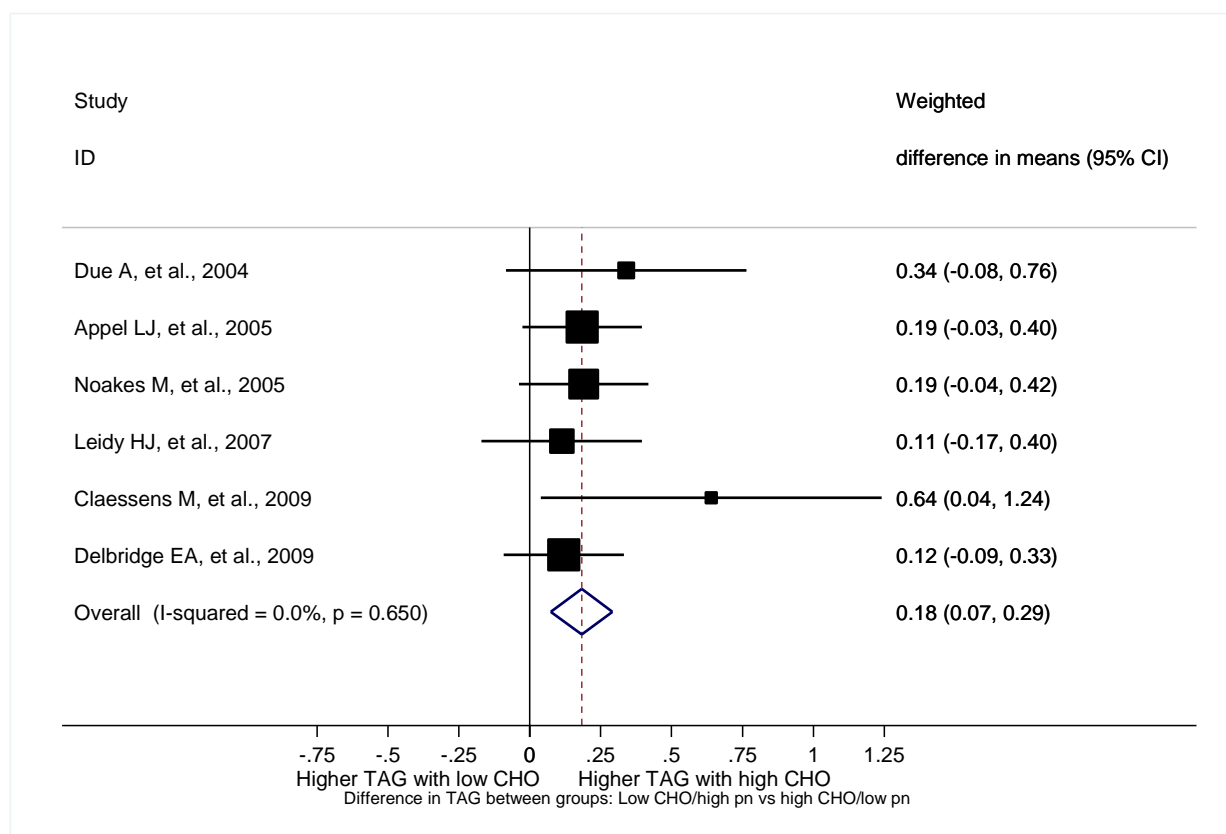
Comparison of higher carbohydrate, lower protein diets with lower carbohydrate, higher protein diets

Six studies were included in the meta-analysis comparing different carbohydrate and protein intakes and changes in TAG reported as mmol/L.

There was no change in body weights in the OMNI-Heart Study (no energy restriction protocol) (Furtado *et al.*, 2008; Appel *et al.*, 2005), an overall decrease in three studies (Due *et al.*, 2004; Leidy *et al.*, 2007; Clifton *et al.*, 2008) and an increase in one study (Delbridge *et al.*, 2009). In one study there was a decrease in the low carbohydrate group and an increase in the high carbohydrate group (Claessens *et al.*, 2009). The high carbohydrate groups consumed, on average, 57% energy from carbohydrate (range 51-63) and 16% protein, and the low carbohydrate groups consumed 45% energy from carbohydrate (range 40-49) and 27% protein.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to one year. The pooled estimate indicated that TAG levels were 0.18mmol/L (95% CI 0.07 to 0.29) higher with consumption of a higher carbohydrate, low protein diet. This was significantly different from zero ($p=0.001$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 62%). Statistically, there was evidence that high carbohydrate, low protein diets are associated with higher TAG levels.

Figure 2.39 Forest plot for higher carbohydrate, lower protein diets and lower carbohydrate, higher protein diets and TAG (mmol/L)



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

Two studies did not report data that could be incorporated into a meta-analysis (Lasker *et al.*, 2008; Layman *et al.*, 2009). In the 12-month randomised parallel group trial by Layman *et al.* (Layman *et al.*, 2009), overweight and obese men and women received a high carbohydrate, low protein diet or a low carbohydrate, high protein diet. At four months, TAG in the high carbohydrate, low protein group and the low carbohydrate, high protein group had statistically significantly increased and decreased, respectively ($p < 0.05$). In addition, TAG differed between conditions as the high carbohydrate, low protein group had lower TAG compared with the low carbohydrate, high protein group at four months ($p < 0.01$). These differences were also apparent at the 12-month follow-up ($p = 0.049$).

Lasker *et al.* (Lasker *et al.*, 2008), using a comparable parallel group design, found that TAG had reduced by 26.8% in the low carbohydrate, high protein diet group, but decreased by 7% in the high carbohydrate group ($p = 0.01$). This effect was also evident after controlling for changes in fat mass and body weight (Lasker *et al.*, 2008). Differences over time were not reported.

One study could not be included in the meta-analysis as no measure of variation was available (Dyson *et al.*, 2007). Changes in TAG were not statistically significant between or within groups.

Two genetic studies that used the same data were not included in the meta-analysis (de Luis *et al.*, 2008; de Luis *et al.*, 2009a) as an existing study (de Luis *et al.*, 2009b). De Luis *et al.* (de Luis *et al.*, 2008; de Luis *et al.*, 2009a) compared a low carbohydrate diet (1507kcal/day, 38% carbohydrates, 26% proteins, 36% fats) and a low fat diet (1500kcal/day, 52% carbohydrates, 20% proteins, 27% fats) in 118 participants (de Luis *et al.*, 2009b).

In the latter paper, changes in TAG were reported to be similar in both diet groups overall (de Luis *et al.*, 2009b). In individuals with different polymorphisms of the fatty acid binding protein 2 (FABP2) gene (de Luis *et al.*, 2008) some differences were apparent. In the wild type group, TAG decreased with both high and low carbohydrate diets, but no significant changes occurred in the mutant-type group.

In individuals with different polymorphisms of the uncoupling protein-3 gene (a gene with influence on energy expenditure and fat storage) (de Luis *et al.*, 2009a), separating participants according to genetic subgroups also showed differences in TAG response. A significant improvement in TAG – that is, a decrease from baseline in probands with the wild type allele of the UCP-3 gene treated with the low carbohydrate diet ($p < 0.05$) was reported. In carriers of the T variant TAG were unaffected by either diet.

Twenty seven studies were included in the meta-analysis comparing different carbohydrate, fat and protein intakes and changes in TAG reported as mmol/L.

The high carbohydrate groups consumed, on average, 55% energy from carbohydrate (range 43 to 67%) and the low carbohydrate groups consumed 33% (range 5 to 47%). Just three studies did not have an energy restriction goal for the participants (Maki *et al.*, 2007; Johnston *et al.*, 2004; Clevidence *et al.*, 1992). Accordingly, the studies almost uniformly reported decreased body weights in all dietary groups, although the magnitude of the change may have differed between groups.

All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years. Overall heterogeneity denoted by I^2 was high at 82% and therefore the pooled estimate which has little meaning was not reported. There is a suggestion of asymmetry in the funnel plot, but this could be the result of chance. A roughly symmetrical funnel plot would indicate an absence of publication bias. Generally, while there was considerable variation between studies, higher carbohydrate diets were associated with higher TAG levels.

Figure 2.40 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and TAG (mmol/L)

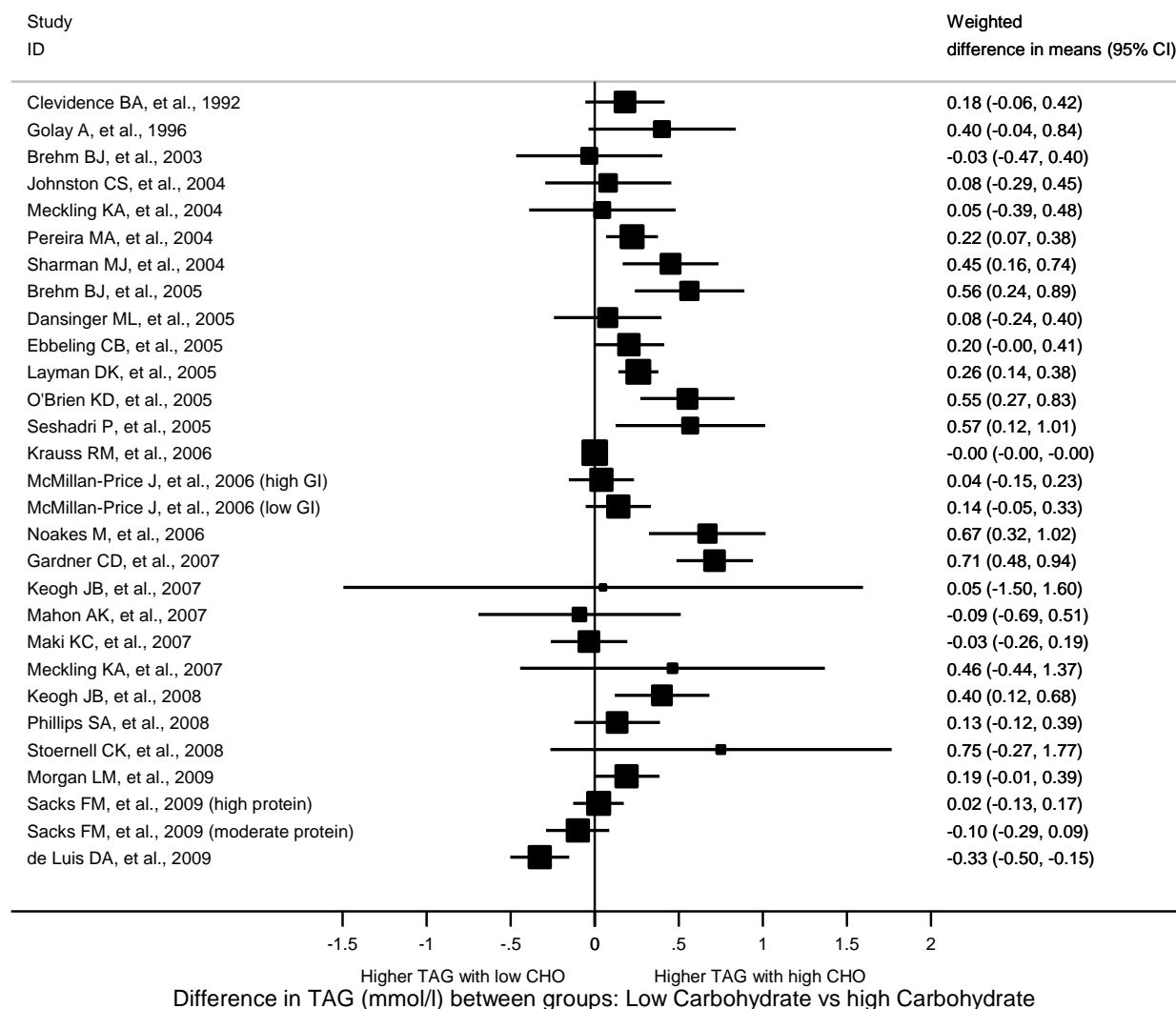


Figure 2.41 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower fat and protein versus lower carbohydrate, higher fat and protein diets and TAG

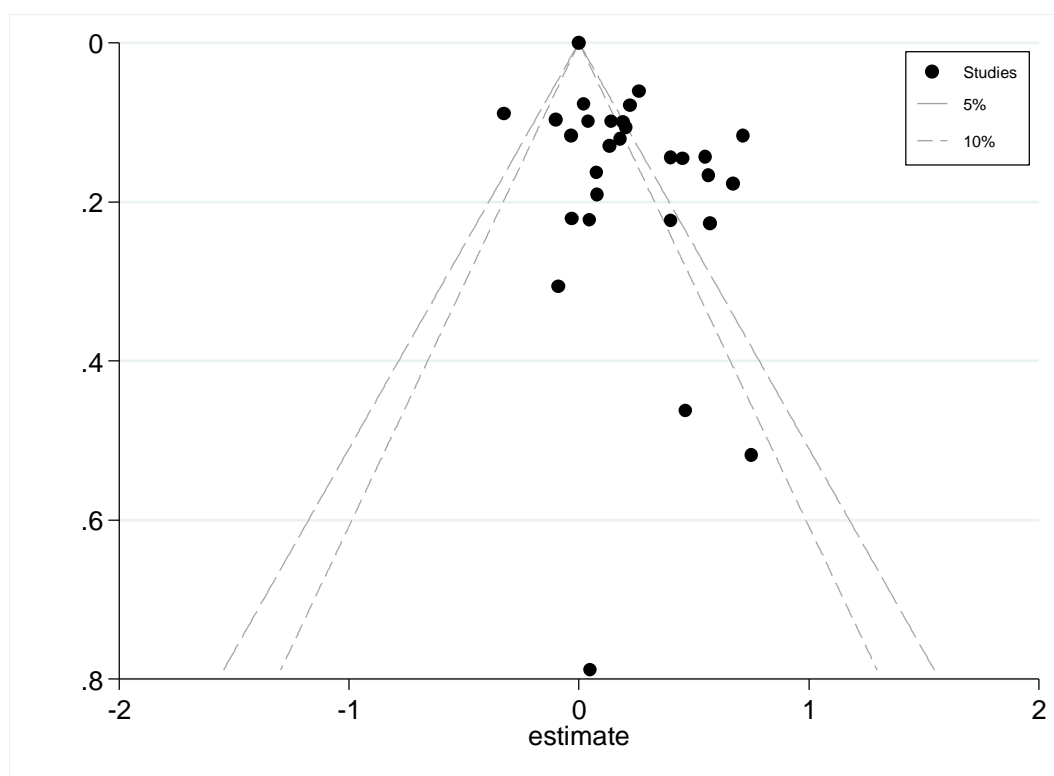


Table 2.53 Triacylglycerol and total carbohydrate: cohort studies in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub- group Details	Contrast	Exposure Units	Mean Outcome	Beta coefficient (SE)/(CI)	P trend	Adjustments
(Ludwig <i>et al.</i> , 1999) 13692 The CARDIA Study	USA, Multi- ethnic, Generally healthy, No hypertension, No T2DM	18-30 %M 45.9	5115	10 years	FFQ (700)	Carbohydrate, total (% energy)	TAG Fasting, mg/dL	Race - White	(51.9) vs (33.5)	% Energy	81.6 vs.81.4		0.82	age, alcohol, centre, education, energy intake, physical activity, gender, smoking, vitamin intake
13693 The CARDIA Study								Race - Black	(51.9) vs (33.5)	% Energy	68.9 vs. 68.1		0.50	As above
(Schroeder <i>et al.</i> , 2007) 14178 Middle-aged Runners Study	USA, Active people only, No CHD, No hypertension	(51) %M 62	91	10 years	Food diary	Carbohydrate, total (grams/day)	TAG Fasting			1 g/day		No effect on regression direction		Age

Table 2.54 Triacylglycerol and high carbohydrate diets: RCT data

Author / Result ID	Sub group detail	Intervention groups	Completers / Allocated	Baseline	Follow- up	Within group Δ from base- line	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
Adolescent studies																
(Demol <i>et al.</i> , 2009) 15407		High carbohydrate, low fat	20/20	106.4 (SD 11.6)	89.6 (SD 12.5)							TAG	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Low carbohydrate, high fat	17/17	126.3 (SD 12.6)	105.0 (SD 13.9)			NS							Decrease	
		Low carbohydrate, high protein	18/18	119.3 (SD 12.2)	78.8 (SD 12.8)			NS							Decrease	
15408		High carbohydrate, low fat	20/20	106.4 (SD 11.6)	78.7 (SD 14.6)							TAG	Fasting (mg/dL)	1 year	Decrease	unclear
		Low carbohydrate, high fat	17/17	126.3 (SD 12.6)	102.7 (SD 16.3)			NS							Decrease	

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
		Low carbohydrate, high protein	18/18	119.3 (SD 12.2)	121.1 (SD 13.9)			NS								Decrease
(Sondik <i>et al.</i> , 2003) 15992		Low fat	14/19			-5.9 (SD 70.0)	NS					TAG	Fasting serum (mg/dL)	12 weeks	Decrease	bias
		Very low carbohydrate	12/20			-48.3 (SD 29.0)	<0.05	0.07								Decrease
Adult studies																
(Brehm <i>et al.</i> , 2003) 15725		Moderate fat	20/20	109.25 (SE 9.49)	101.80 (SE 6.71)		<0.01					TAG	Fasting (μg/dL)	3 months	Decrease	unclear
		Low carbohydrate	22/22	148.73 (SE 13.41)	92.41 (SE 8.74)		<0.01	NS								Decrease
*15726		Moderate fat	20/20	109.25 (SE 9.49)	111.00 (SE 12.37)							TAG	Fasting (μg/dL)	6 months	Decrease	unclear
		Low carbohydrate	22/22	148.73 (SE 13.41)	113.86 (SE 15.25)			NS								Decrease
(Brehm <i>et al.</i> , 2005) 16383		Low carbohydrate	20/25	128.85 (SE 13.44)	78.8 (SE 4.82)			NS				TAG	Fasting plasma (mg/dL)	2 months	Decrease	No bias
		Moderate fat	20/25	145.63 (SE 19.95)	129.45 (SE 10.3)											Decrease
*16384		Low carbohydrate	20/25	128.85 (SE 13.44)	80.75 (SE 6.11)			NS				TAG	Fasting plasma (mg/dL)	4 months	Decrease	No bias
		Moderate fat	20/25	145.63 (SE 19.95)	130.65 (SE 13.41)											Decrease
(Campo <i>s et al.</i> , 1995) *16207		High-fat minus low-fat higher CHO	Crossover: 43/allocated not reported						-50 (SD 53)		0.0001	TAG	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17093		High-fat	43/allocated not reported	135 (SD 73)	104 (SD 59)			0.0001				TAG	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
		Low-fat higher CHO	43/allocated not reported	135 (SD 73)	154 (SD 85)											Not reported
(Claessens <i>et al.</i> , 2009) *16824		High carbohydrate supplement	16/allocated not reported	1.43 (SE 0.18)	1.98 (SE 0.42)	0.56 (SE 0.29)	NS					TAG	Fasting (mmol/L)	12 weeks	Increase	unclear
		High protein supplement - casein	14/allocated not reported	1.13 (SE 0.11)	1.08 (SE 0.13)	-0.05 (SE 0.08)	NS	<0.05								Decrease
		High protein supplement - whey	18/allocated not reported	1.23 (SE 0.17)	1.22 (SE 0.2)	-0.08 (SE 0.1)	NS									Decrease
(Clevidence <i>et al.</i> , 1992) *16604		High fat diet	42/46	1.24 (SE 0.09)	1.14 (SE 0.08)	-0.1	NS	0.008				TAG	Fasting plasma (mmol/L)	10 weeks	No change	unclear
		Low fat diet	42/46	1.24 (SE 0.09)	1.32 (SE 0.09)	0.08	NS									No change
(Clifton <i>et al.</i> , 2004) 16744		High MUFA	31/35	1.49 (SD 0.66)	1.21 (SD 0.42)			Unclear				TAG	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Very low fat	31/35	1.45 (SD 0.37)	1.27 (SD 0.27)											Decrease
*16745		High MUFA	31/35	1.49 (SD 0.66)	1.16 (SD 0.44)		<0.01					TAG	Fasting (mmol/L)	12 weeks	Decrease	unclear
		Very low fat	31/35	1.45 (SD 0.37)	1.20 (SD 0.3)		<0.01									Decrease
(Clifton <i>et al.</i> , 2008) *16007		High carbohydrate diet	38/38			-0.21 (SD 0.89)		NS				TAG	Fasting (mmol/L)	1.25 years	Decrease	unclear
		High protein diet	40/41			-0.19 (SD 0.52)										Decrease
(Colette <i>et al.</i> , 2003) *17415		High carbohydrate diet	15/15	1.62 (SE 0.22)	1.42 (SE 0.18)		NS					TAG	Fasting serum (mmol/L)	8 weeks	Decrease	unclear
		High MUFA diet	17/17	1.51 (SE 0.19)	1.18 (SE 0.14)		0.042	NS								Decrease

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Cornier <i>et al.</i> , 2005) *16712	Insulin sensitive	High carbohydrate, low fat	6/10	136 (SE 42)	118 (SE 26)			NS				TAG	Fasting (mg/dL)	16 weeks	Decrease	unclear
		Low carbohydrate, high fat	6/11	132 (SE 20)	103 (SE 16)		<0.05								Decrease	
(Couture <i>et al.</i> , 2003) *15882	Genetics - Apo E genotype E3/E2	High carbohydrate diet	3/3	1.19 (SD 0.44)	0.96 (SD 0.1)		0.43	Not reported/unclear				TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	5/5	1.12 (SD 0.27)	0.84 (SD 0.12)										Decrease	
*15883	Genetics - Apo E genotype E3/E3	High carbohydrate diet	22/22	1.27 (SD 0.68)	1.29 (SD 0.67)		0.74					TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	21/21	1.61 (SD 0.74)	1.35 (SD 0.68)		0.02								Decrease	
*15884	Genetics - Apo E genotype E3/E4	High carbohydrate diet	8/8	1.41 (SD 0.98)	1.44 (SD 1.26)		0.75					TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		High MUFA diet	6/6	1.42 (SD 0.44)	1.05 (SD 0.42)		<0.01								Decrease	
(Dale <i>et al.</i> , 2009) 15985		High MUFA diet minus high carbohydrate diet	High MUFA: 85/100 High CHO: 89/100						0 (CI -0.09, 0.09)		NS	TAG	Fasting (mmol/L)	2 years	Decrease in both	unclear
17401		High carbohydrate diet	89/100	1.2 (SD 0.6)	1.10 (SD 0.58)							TAG	Fasting (mmol/L)	1 year	Decrease	unclear
		High MUFA diet	85/100	1.2 (SD 0.6)	1.11 (SD 0.59)										Decrease	
17372		High carbohydrate diet	89/100	1.2 (SD 0.6)	1.11 (SD 0.62)							TAG	Fasting (mmol/L)	2 years	Decrease	unclear
		High MUFA diet	85/100	1.2 (SD 0.6)	1.11 (SD 0.61)										Decrease	

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Dansinger <i>et al.</i> , 2005) 15813		Atkins	40/40			-32.3 (SD 66)	0.01	Unclear				TAG	Fasting serum (mg/dL)	2 months	Decrease	No bias
		Ornish	40/40			-0.4 (SD 77)	NS								Decrease	
		Weight watchers	40/40			-9.2 (SD 39)	NS								Decrease	
		Zone	40/40			-54.1 (SD 105)	0.01								Decrease	
15814		Atkins	40/40			-10.6 (SD 40)	NS					TAG	Fasting serum (mg/dL)	6 months	Decrease	No bias
		Ornish	40/40			-2.3 (SD 71)	NS								Decrease	
		Weight watchers	40/40			-1.5 (SD 55)	NS								Decrease	
		Zone	40/40			-14.8 (SD 57)	NS								Decrease	
*15815		Atkins	40/40			-1.2 (SD 84)	NS					TAG	Fasting serum (mg/dL)	1 year	Decrease	No bias
		Ornish	40/40			5.6 (SD 36)	NS								Decrease	
		Weight watchers	40/40			-12.7 (SD 61)	NS								Decrease	
		Zone	40/40			2.5 (SD 147)	NS								Decrease	
(Delbridge <i>et al.</i> , 2009) *15325		Low fat, high carbohydrate weight maintenance diet	70/70			0.21 (SE 0.06)						TAG	Fasting (mmol/L)	1 year	Increase	unclear
		Low fat, high protein weight maintenance diet	68/71			0.09 (SE 0.09)		0.241							Increase	
(de Luis <i>et al.</i> ,	Genetics - wild-	Low carbohydrate	55/105	131.2 (SD 41.8)	118.3 (SD			NS				TAG	Fasting (mg/dL)	2 months	Decrease	unclear

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Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
2008) 16147	type Ala54/Ala54				44.3)											
		Low fat	55/99	112.7 (SD 59)	99.9 (SD 34)										Decrease	
16164	Genetics - mutant-type Ala54/Thr54 or Thr54/Thr54	Low carbohydrate	50/105	134.8 (SD 62.4)	123.3 (SD 86)			NS				TAG	Fasting (mg/dL)	2 months	Decrease	unclear
		Low fat	44/99	124.8 (SD 62.4)	106.3 (SD 86)										Decrease	
(de Luis <i>et al.</i> , 2009b) *16085		Low carbohydrate	52/52	149 (SD 87)	126 (SD 48)			NS				TAG	Fasting (mg/dL)	3 months	Decrease	unclear
		Low fat	66/66	104 (SD 47)	97 (SD 31)										Decrease	
(de Luis <i>et al.</i> , 2009a) 16701	Genetics - UCP3 Gene - 55CC polymorphism	Low carbohydrate	54/67	129.2 (SD 41.8)	117.3 (SD 44.3)		<0.05	Unclear				TAG	Serum (mg/dL)	2 months	Decrease	unclear
		Low fat	40/64	135.4 (SD 45.0)	128.8 (SD 39.0)		NS								Decrease	
16702	Genetics - UCP3 Gene - 55CT/TT polymorphism	Low fat	24/64	124.8 (SD 62.4)	123.3 (SD 44)		NS					TAG	Serum (mg/dL)	2 months	Decrease	unclear
		Low carbohydrate	13/67	122.88 (SD 62.4)	124.3 (SD 86.1)		NS								Decrease	
(Dreon <i>et al.</i> , 1994) 15635	Larger LDL particles	High-fat low CHO	87/105	121.2 (SD 61.1)	86.3 (SD 3.9)		<0.0001					TAG	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	87/105	121.2 (SD 61.1)	122.8 (SD 6.9)		<0.0001								Not reported	
15645	Smaller and denser LDL	High-fat low CHO	18/105	121.2 (SD 61.1)	166.1 (SD 12.4)		<0.01					TAG	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	particles															
		Low-fat higher CHO	18/105	121.2 (SD 61.1)	225.4 (SD 19.5)		<0.01								Not reported	
17045	Larger LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 87/105						36.5 (SD 6)		<0.0001	TAG	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17052	Smaller and denser LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 18/105						59.3 (SD 17)		<0.01	TAG	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17058	LDL particles remained large during study	High-fat low CHO	51/105	121.2 (SD 61.1)	76.6 (SD 4.7)		<0.0001					TAG	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	51/105	121.2 (SD 61.1)	96.8 (SD 4.6)		<0.0001								Not reported	
17062	LDL particles changed from large to small and dense during study	High-fat low CHO	36/105	121.2 (SD 61.1)	99.9 (SD 5.4)		<0.0001					TAG	Fasting plasma (mg/dL)	6 weeks	Not reported	unclear
		Low-fat higher CHO	36/105	121.2 (SD 61.1)	159.5 (SD 13.3)		<0.0001								Not reported	
17066	LDL particles remained large during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 51/105						20.2 (SD 5)		<0.0001	TAG	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17072	LDL particles changed from large to small and	Low-fat higher CHO minus high-fat low CHO	Crossover: 36/105						59.5 (SD 12)		<0.0001	TAG	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	dense during study															
(Due <i>et al.</i> , 2004) 17540		High protein	23/23	1.34 (CI 1.1, 1.7)	1.19 (CI 0.7, 1.5)			NS				TAG	Fasting (mmol/L)	6 months	Decrease	unclear
		Moderate protein	23/18	1.3 (CI 1.0, 1.7)	1.41 (CI 1.0, 2.1)										Decrease	
*17541		High protein	23/23	1.34 (CI 1.1, 1.7)	1.29 (CI 0.9, 1.5)			NS				TAG	Fasting (mmol/L)	1 year	Decrease	unclear
		Moderate protein	18/18	1.3 (CI 1.0, 1.7)	1.63 (CI 1.3, 1.9)										Decrease	
(Due <i>et al.</i> , 2008) *15302		Control	24/25	1.12 (CI 0.9, 1.3)	1.01 (CI 0.8, 1.2)	-0.11 (CI -0.2, 0)						TAG	Fasting plasma (mmol/L)	6 months	Increase	unclear
		High MUFA	39/52	1.02 (CI 0.9, 1.2)	0.88 (CI 0.8, 1.0)	-0.15 (CI -0.3, 0)		NS							Increase	
		Low fat	43/48	1.15 (CI 0.9, 1.3)	1.0 (CI 0.9, 1.1)	-0.15 (CI -0.3, 0)		NS							Increase	
(Dyson <i>et al.</i> , 2007) 16347		Healthy eating diet	4/~6	1.4	1.3	-0.2						TAG	(mmol/L)	3 months	Decrease	bias
		Low carbohydrate diet	6/~6	1.6	1.5	-0.1		NS							Decrease	
(Ebbeling <i>et al.</i> , 2005) 15509		Low fat diet	12/17	109 (SE 15)		-7.1% (CI -19.8, 7.6)						TAG	Fasting (mg/dL)	6 months	Decrease	unclear
		Low GI diet	11/17	133 (SE 17)		-35.4% (CI -44.6, -24.7)									Decrease	
*15510		Low fat diet	12/17	109 (SE 15)		-19.1% (CI -32.2, -3.6)		0.005				TAG	Fasting (mg/dL)	1 year	Decrease	unclear

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
		Low GI diet	11/17	133 (SE 17)		-37.2% (CI -47.7, -24.5)										Decrease
(Ebbeling <i>et al.</i> , 2007) *15453		Low fat diet	37/37			-4.0 (SE 5.6)						TAG	Fasting plasma (%)	6 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-21.2 (SE 4.7)		0.2								Decrease
15454		Low fat diet	37/37			2.0 (SE 6.0)						TAG	Fasting plasma (%)	18 months	Decrease	No bias
		Low GL diet	ITT: 36/36			-9.0 (SE 5.4)		0.18								Decrease
(Foster <i>et al.</i> , 2003) 15204		Conventional diet plan	30/30			1.1 (SD 34.6)	NS					TAG	Fasting serum (%)	3 months	Decrease	unclear
		Low carbohydrate diet	33/33			-18.7 (SD 25.7)	<0.05	0.01								Decrease
15205		Conventional diet plan	30/30			-7.6 (SD 19.3)	<0.05					TAG	Fasting serum (%)	6 months	Decrease	unclear
		Low carbohydrate diet	33/33			15.0 (SD 29.4)	<0.05	0.13								Decrease
*15206		Conventional diet plan	30/30			0.7 (SD 37.7)	NS					TAG	Fasting serum (%)	1 year	Decrease	unclear
		Low carbohydrate diet	33/33			-17.0 (SD 23.0)	<0.05	0.04								Decrease
(Frisch <i>et al.</i> , 2009) *15164		High carbohydrate diet	100/100			-0.03 (SD 0.55)	NS					TAG	Fasting serum (mmol/L)	6 months	Decrease	unclear
		Moderate carbohydrate diet	100/100			-0.18 (SD 0.4)	0.05	0.005								Decrease
15165		High carbohydrate diet	100/100			-0.04 (SD 0.5)	NS					TAG	Fasting serum (mmol/L)	1 year	Decrease	unclear

Author / Result ID	Sub group detail	Intervention groups	Completers / Allocated	Baseline	Follow-up	Within group Δ from base-line	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias	
		Moderate carbohydrate diet	100/100			-0.1 (SD 0.47)	0.05	0.164								Decrease	
(Furtado <i>et al.</i> , 2008) *16332		High carbohydrate	107/164	106 (SD 74)		-5.5 (SD 50)	0.3					TAG	Fasting serum (mg/dL)	6 weeks	No change	No bias	
		High protein	107/164	106 (SD 74)		-15 (SD 51)	0.01	0.02							No change		
		High PUFA	107/164	106 (SD 74)		-7.9 (SD 46)	0.08	0.6							No change		
16333		High protein minus high carbohydrate	Crossover: 107/164							-9.5 (SD 42)	0.02	TAG	Fasting serum (mg/dL)	6 weeks	No change in both	No bias	
16334		High PUFA minus high carbohydrate	Crossover: 107/164							-2.4 (SD 45)	0.6	TAG	Fasting serum (mg/dL)	6 weeks	No change in both	No bias	
(Gardner <i>et al.</i> , 2007) *15114		Atkins: low carbohydrate	70/77			-52.3 (SD 66.8)		NS				TAG	Fasting plasma (mg/dL)	2 months	Decrease	No bias	
		Ornish: high carbohydrate	64/76			10.9 (SD 55)		NS							Decrease		
		Zone: moderate carbohydrate	65/79			-24.8 (SD 53.1)									Decrease		
15115		Atkins: low carbohydrate	70/77			-35.6 (SD 64.4)		NS				TAG	Fasting plasma (mg/dL)	6 months	Decrease	No bias	
		Ornish: high carbohydrate	64/76			-7.6 (SD 54.4)		NS							Decrease		
		Zone: moderate carbohydrate	65/79			-21.3 (SD 58.9)									Decrease		
15116		Atkins: low carbohydrate	70/77			-29.3 (SD 59)		0.05				TAG	Fasting plasma (mg/dL)	1 year	Decrease	No bias	
		Ornish: high carbohydrate	64/76			-14.9 (SD 46.2)		NS							Decrease		
		Zone: moderate	65/79			-4.2 (SD									Decrease		

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		carbohydrate				48.5)										
(Ginsberg et al., 1998) *17250		Average American Diet	103/118		85.1 (SE 3.4)							TAG	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		93.0 (SE 3.7)			NS							Not reported	
		Step 1 diet	103/118		92.4 (SE 3.7)			<0.01							Not reported	
17262	Men	Average American Diet	46/118		96.5 (SE 6)							TAG	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	46/118		107.8 (SE 6)			NS							Not reported	
		Step 1 diet	46/118		104.6 (SE 6)			NS							Not reported	
17263	Women	Average American Diet	57/118		76.7 (SE 5)							TAG	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	57/118		82.3 (SE 5)			NS							Not reported	
		Step 1 diet	57/118		81.3 (SE 5)			<0.01							Not reported	
17304	Black	Average American Diet	26/118		71.5 (SE 8)							TAG	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	26/118		75.9 (SE 9)			NS							Not reported	
		Step 1 diet	26/118		76.7 (SE 8)			NS							Not reported	
17305	Non black	Average American Diet	77/118		90.0 (SE 4)							TAG	Fasting (mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	77/118		99.5 (SE 4)			<0.01							Not reported	

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17320	Pre-menopausal	Step 1 diet	77/118		98.5 (SE 4)			<0.01								Not reported	
		Average American Diet	39/118		72.2 (SE 5)							TAG	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	39/118		78.3 (SE 6)			NS								Not reported	
		Step 1 diet	39/118		78.3 (SE 5)			NS								Not reported	
17321	Post-menopausal	Average American Diet	18/118		87.4 (SE 9)							TAG	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	18/118		92.8 (SE 11)			NS								Not reported	
		Step 1 diet	18/118		95.6 (SE 10)			NS								Not reported	
17336	Men <40y	Average American Diet	30/118		94.6 (SE 8)							TAG	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	30/118		105.6 (SE 8)			NS								Not reported	
		Step 1 diet	30/118		100.5 (SE 8)			NS								Not reported	
17337	Men >40y	Average American Diet	16/118		100.5 (SE 10)							TAG	Fasting (mg/dL)	6 weeks		Not reported	No bias
		Low saturated fat diet	16/118		112.2 (SE 9)			NS								Not reported	
		Step 1 diet	16/118		114.4 (SE 10)			NS								Not reported	
(Golay et al., 1996) *16627		Low carbohydrate diet	completers not reported/22	1.7 (SE 0.1)	1.4 (SE 0.1)		<0.01	Not reported/unclear				TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear	
		Moderate carbohydrate diet	completers not reported/21	2.2 (SE 0.2)	2.2 (SE 0.2)		NS									Decrease	

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(Golay <i>et al.</i> , 2000) *14854		Higher carbohydrate, macronutrients not eaten simultaneously	26/26	2.3 (SE 0.3)	1.3 (SE 0.2)		<0.01	NS				TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Lower carbohydrate, macronutrients eaten simultaneously	28/28	2.5 (SE 0.4)	2 (SE 0.2)		<0.05								Decrease	
(Helge, 2002) *15905		High carbohydrate + exercise	16/16	1.1 (SE 0.1)	1.1 (SE 0.1)							TAG	Fasting serum (mmol/L)	7 weeks	Decrease	unclear
		High fat + exercise	17/17	1.1 (SE 0.1)	0.7 (SE 0.1)		<0.05	<0.05							Decrease	
(Howard <i>et al.</i> , 2006) 16251		Control	approx n=1699 (5.8% sub-sample of 29294)	141.1 (SD 66.3)	144.6 (SD 63.7)	1.0 (SD 0.3)						TAG	Fasting (mg/dL)	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub-sample of 19541)	138.6 (SD 65.1)	142.3 (SD 67.5)	1.0 (SD 0.4)		NS							Decrease	
*17617		Low fat minus control	As above							0.00 (CI - 0.03, 0.04)	NS	TAG	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Johnston <i>et al.</i> , 2004) *14864		High carbohydrate, low fat	7/10	1.25 (SE 0.17)		-11.9% (SE 13.8%)	NS					TAG	Whole blood (mmol/L)	6 weeks	Decrease	unclear
		High protein, low fat	9/10	1.15 (SE 0.18)		-18.6% (SE 7.9%)	NS	0.124							Decrease	
(Johnston <i>et al.</i> ,)		Low carbohydrate diet	10/10	1.48 (SE 0.12)								TAG	Fasting serum (mmol/L)	6 weeks	Decrease	unclear

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2006) 17519		Very low-carbohydrate diet	9/9	1.82 (SE 0.19)				NS								Decrease
(Keogh <i>et al.</i> , 2007) 15625		High carbohydrate diet	12/12	1.39 (SE 0.59)	1.13 (SE 0.52)		0.01					TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	1.72 (SE 0.87)	1.2 (SE 0.57)		0.01	NS								Decrease
*15626		High carbohydrate diet	12/12	1.39 (SE 0.59)	1.11 (SE 0.61)		0.01					TAG	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Low carbohydrate diet	13/13	1.72 (SE 0.87)	1.06 (SE 0.5)		0.01	NS								Decrease
15627		High carbohydrate diet	completers not reported/12	1.38 (SE 0.18)	1.34 (SE 0.27)		NS					TAG	Fasting plasma (mmol/L)	1 year	Decrease	unclear
		Low carbohydrate diet	completers not reported/13	1.35 (SE 0.08)	1.07 (SE 0.08)		NS	NS								Decrease
(Keogh <i>et al.</i> , 2008) *16723		High carbohydrate, low SFA	47/50	1.8 (SD 1.0)	1.5 (SD 0.9)		<0.001					TAG	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low carbohydrate, high SFA	52/57	1.6 (SD 0.7)	1.1 (SD 0.4)		<0.001	<0.05								Decrease
(Kirkwood <i>et al.</i> , 2007) 15672		Group 1: No advice	18/allocated not reported				NS					TAG	Fasting (mmol/L)	12 weeks	No change	unclear
		Group 2: Conventional weight loss diet	16/allocated not reported				NS	NS								Decrease
15673		Group 3: Exercise	19/allocated not reported				NS	NS				TAG	Fasting (mmol/L)	12 weeks	Decrease	unclear
		Group 4: Conventional weight loss diet + exercise	16/allocated not reported				NS									Decrease
(Krauss <i>et al.</i> ,		26% CHO High	40/52	2.18 (SD 0.25)		-0.03 (SE		NS				Log TAG	Fasting plasma	12 weeks	Decrease	unclear

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2006) *17477		saturated fat				0.02)							(mg/dL)			
		26% CHO Low saturated fat	47/59	2.1 (SD 0.24)		0.01 (SE 0.02)		NS								Decrease
		39% CHO Low saturated fat	42/56	2.19 (SD 0.23)		-0.06 (SE 0.02)		NS								Decrease
		54% CHO Low saturated fat	49/57	2.16 (SD 0.2)		-0.07 (SE 0.02)										Decrease
(Landry <i>et al.</i> , 2003) *15997		High carbohydrate	19/19	1.12 (SD 0.49)		0 (SD 0.4)	NS	Not reported				TAG	Fasting plasma (mmol/L)	7 weeks	Decrease	unclear
		Low carbohydrate, high fat diet	18/18	1.25 (SD 0.32)		-0.3 (SD 0.3)	<0.01									Decrease
16850		High carbohydrate	19/19	876 (SD 396)		-38 (SD 267)	NS					TAG	8 hour AUC, Post test meal (mmol.min/l)	7 weeks	Decrease	unclear
		Low carbohydrate, high fat diet	18/18	889 (SD 242)		-159 (SD 216)	<0.01									Decrease
(Lasker <i>et al.</i> , 2008) 15917		High carbohydrate	25/33			-7						TAG	Fasting plasma (%)	4 months	Decrease	unclear
		High protein	25/32			-26.8		0.01								Decrease
(Layman <i>et al.</i> , 2005) *16179		High carbohydrate diet	12/12	1.4 (SD 0.14)	1.38 (SD 0.18)		NS					TAG	Fasting serum (mmol/L)	16 weeks	Decrease	unclear
		High protein diet	12/12	1.42 (SD 0.15)	1.12 (SD 0.11)		<0.05	<0.05								Decrease
16180		High carbohydrate diet + exercise	12/12	1.08 (SD 0.13)	0.91 (SD 0.13)		NS					TAG	Fasting serum (mmol/L)	16 weeks	Decrease	unclear

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		High protein diet + exercise	12/12	1.31 (SD 0.21)	0.98 (SD 0.16)		<0.05	0.41								Decrease
(Layman <i>et al.</i> , 2009) 14960		High carbohydrate, low protein diet	51/66		Higher #		<0.05					TAG	Fasting plasma	4 months	Decrease	unclear
		Low carbohydrate, high protein diet	52/64		Lower#		<0.05	<0.01								Decrease
14965		High carbohydrate, low protein diet	30/66		Higher #		<0.05					TAG	Fasting plasma	1 year	Decrease	unclear
		Low carbohydrate, high protein diet	41/64		Lower#		<0.05	0.049								Decrease
(Leidy <i>et al.</i> , 2007) *16842		High protein, energy restricted	21/27	108 (SE 12)	85 (SE 11)	-22 (SE 10)		NS				TAG	Fasting serum (mg/dL)	12 weeks	Decrease	unclear
		Moderate protein, energy restricted	25/27	122 (SE 10)	110 (SE 9)	-12 (SE 8)										Decrease
(Ley <i>et al.</i> , 2004) 15958		Control	70/70			-0.01 (SE 0.23)						TAG	Fasting serum (mmol/L)	6 months	No change	unclear
		Low fat	66/66			-0.12 (SE 0.12)		NS								Decrease
*15959		Control	70/70			0.07 (SE 0.21)						TAG	Fasting serum (mmol/L)	1 year	No change	unclear
		Low fat	66/66			0.03 (SE 0.11)		NS								Decrease
15961		Control	57/70			0.01 (SE 0.22)						TAG	Fasting serum (mmol/L)	2 years	No change	unclear

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		Low fat	47/66			-0.11 (SE 0.15)		NS								Decrease
15962		Control	51/70			0.25 (SE 0.27)						TAG	Fasting serum (mmol/L)	3 years	No change	unclear
		Low fat	48/66			-0.02 (SE 0.14)		NS								Decrease
15963		Control	52/70			0.12 (SE 0.22)						TAG	Fasting serum (mmol/L)	5 years	No change	unclear
		Low fat	51/66			0.37 (SE 0.1)		NS								Decrease
(Lofgren <i>et al.</i> , 2005) *17273		High carbohydrate, low fat	20/20	1.30 (SE 0.2)	1.10 (SE 0.1)			NS				TAG	Fasting plasma (mmol/L)	10 weeks	Decrease	unclear
		High fat, moderate carbohydrate	20/20	1.22 (SE 0.1)	1.13 (SE 0.2)											Decrease
(Lovejoy <i>et al.</i> , 2003) 14981		Control	13/15	138.9 (SE 16.97)		5.61 (SE 11.66)		Unclear				TAG	Fasting (mg/dL)	3 months	Decrease	unclear
		Fat reduced	13/15	102.77 (SE 10.73)		27.68 (SE 8.64)										Decrease
14986		Control	13/15	138.9 (SE 16.97)		16.85 (SE 20.33)						TAG	Fasting (mg/dL)	6 months	Decrease	unclear
		Fat reduced	13/15	102.77 (SE 10.73)		18.31 (SE 9.09)										Decrease
*14987		Control	13/15	138.9 (SE 16.97)		7.42 (SE 15.82)						TAG	Fasting (mg/dL)	9 months	Decrease	unclear
		Fat reduced	13/15	102.77 (SE 10.73)		34.35 (SE 7.69)										Decrease
(Mahon <i>et al.</i> , 2007)		Control	11/11	156 (SD 46)	154 (SD 55)	-2 (SD 58)	NS					TAG	Fasting serum (mg/dL)	9 weeks	No change	unclear

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*15073		Energy restriction + beef	14/14	127 (SD 57)	104 (SD 45)	-23 (SD 50)	NS	NS								Decrease	
		Energy restriction + carbohydrate /fat	14/14	183 (SD 95)	173 (SD 87)	-10 (SD 69)	NS	NS								Decrease	
		Energy restriction + chicken	15/15	139 (SD 57)	114 (SD 61)	-25 (SD 45)	NS	NS								Decrease	
(Maki et al., 2007) 17286		Ad libitum low GL diet	39/43	127.1 (SE 8.3)		-24.8 (SE 5.3)		NS				TAG	Fasting (mg/dL)	12 weeks	Decrease	unclear	
		Low fat, energy restricted	38/43	134 (SE 10.6)		-11.5 (SE 6.5)										Decrease	
*17287		Ad libitum low GL diet	39/43	127.1 (SE 8.3)		-12.5 (SE 5.2)		NS				TAG	Fasting (mg/dL)	36 weeks	Decrease	unclear	
		Low fat, energy restricted	38/43	134 (SE 10.6)		-15.5 (SE 8.9)										Decrease	
(McMillan-Price et al., 2006) *16223		High CHO, high GI diet	32/32	1.37 (SE 0.15)		-0.14 (SE 0.07)		NS				TAG	Fasting (mmol/L)	12 weeks	Decrease	unclear	
		High CHO, low GI diet	32/32	1.39 (SE 0.13)		-0.05 (SE 0.07)		NS								Decrease	
		High protein, high GI diet	32/32	1.41 (SE 0.13)		-0.18 (SE 0.07)		NS								Decrease	
		High protein, low GI diet	33/33	1.25 (SE 0.12)		-0.19 (SE 0.07)		NS								Decrease	
(Meckling et al., 2004) *14875		Low carbohydrate	15/20	136 (SE 22)	96 (SE 17)		0.05	NS				TAG	Fasting (mg/dL)	10 weeks	Decrease	No bias	
		Low fat	16/20	134 (SE 24)	100 (SE 10)		0.05									Decrease	
(Meckling and Sherfey, 2007) *16381		Hypocaloric control diet	8/15	190 (SD 97)	182 (SD 105)		NS					TAG	Fasting (mg/dL)	12 weeks	Decrease	unclear	
		Hypocaloric protein rich	10/15	140 (SD 46)	141 (SD 49)		NS	NS								Decrease	

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		diet														
16382		Hypocaloric control diet + exercise	11/15	87 (SD 30)	87 (SD 29)		NS					TAG	Fasting (mg/dL)	12 weeks	Decrease	unclear
		Hypocaloric protein rich diet + exercise	14/15	154 (SD 69)	108 (SD 28)		<0.05	NS							Decrease	
(Morgan <i>et al.</i> , 2009) 14702		Atkins	33/57	1.65 (SD 0.7)	1.07 (SD 0.44)		0.01	Unclear				TAG	Fasting whole blood (mmol/L)	8 weeks	Decrease	unclear
		Control	37/61	1.4 (SD 0.65)	1.5 (SD 0.65)		NS								No change	
		Slim Fast	44/59	1.49 (SD 1)	1.47 (SD 1.04)		NS								Decrease	
		Weight Watchers	46/58	1.55 (SD 0.77)	1.25 (SD 0.47)		0.01								Decrease	
*14703		Atkins	33/57	1.65 (SD 0.7)	1.01 (SD 0.33)		NS					TAG	Fasting whole blood (mmol/L)	24 weeks	Decrease	unclear
		Control	37/61	1.4 (SD 0.65)	1.38 (SD 0.65)		NS								No change	
		Slim Fast	44/59	1.49 (SD 1)	1.29 (SD 0.87)		0.01								Decrease	
		Weight Watchers	46/58	1.55 (SD 0.77)	1.2 (SD 0.47)		0.01								Decrease	
(Nelson <i>et al.</i> , 1995) *16938		High fat diet	11/11	85.8 (SD 28.4)	66.4 (SD 41.7)			<0.002				TAG	Fasting plasma (mg/dL)	50 days	Not reported	unclear
		Low fat diet	11/11	85.8 (SD 28.4)	91.5 (SD 38.0)										Not reported	
(Noakes <i>et al.</i> , 2005)		High carbohydrate diet	48/48	1.47 (SE 0.11)	1.3 (SE 0.09)							TAG	Fasting serum (mmol/L)	8 weeks	Decrease	unclear

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16986		High protein diet	52/52	1.37 (SE 0.11)	1.10 (SE 0.06)			Unclear							Decrease	
*16987		High carbohydrate diet	48/48	1.47 (SE 0.11)	1.35 (SE 0.10)	-0.11 (SE 0.06)						TAG	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	52/52	1.37 (SE 0.11)	1.07 (SE 0.06)	-0.30 (SE 0.10)		0.007							Decrease	
17018	TAG < 1.5mmol /L	High carbohydrate diet	23/48									TAG	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	27/52					NS							Decrease	
17019	TAG > 1.5mmol /L	High carbohydrate diet	25/48			decrease						TAG	Fasting serum (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	25/52			decrease		0.023							Decrease	
(Noakes <i>et al.</i> , 2006)		High unsaturated fat	21/27	1.56 (SE 0.11)	1.29 (SE 0.11)			Unclear				TAG	Fasting plasma (mmol/L)	8 weeks	Decrease	unclear
16584		Very low carbohydrate	24/28	1.83 (SE 0.19)	1.16 (SE 0.1)										Decrease	
		Very low fat	22/28	1.51 (SE 0.13)	1.38 (SE 0.12)										Decrease	
*16585		High unsaturated fat	21/27	1.56 (SE 0.11)	1.42 (SE 0.12)	-0.15 (SE 0.07)						TAG	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		Very low carbohydrate	24/28	1.83 (SE 0.19)	1.11 (SE 0.1)	-0.73 (SE 0.12)									Decrease	
		Very low fat	22/28	1.51 (SE 0.13)	1.44 (SE 0.13)	-0.06 (SE 0.13)									Decrease	
(O'Brien <i>et al.</i> , 2005)		Low carbohydrate	22/22			-56.3 (SD 51.1)		<0.001				TAG	Fasting (mg/dL)	3 months	Decrease	unclear
*16956		Moderate fat	19/19			-7.5 (SD)									Decrease	

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30.3)																
(Pereira <i>et al.</i> , 2004) *14579		Hypoenergetic low fat diet	11/23	92.4 (SE 9.47)	102.3 (SE 8.11)	16.2% (SE 5.24%)						TAG	Fasting serum (mg/dL)	67 days	Decrease	unclear
		Hypoenergetic low GL diet	14/23	78.3 (SE 8.4)	72.4 (SE 7.19)	-3.5% (SE 4.63%)		0.01							Decrease	
(Pelkman <i>et al.</i> , 2004) 16889		Low fat, high carbohydrate diet	25/25	1.8 (SE 0.13)	1.38 (SE 0.13)		<0.05	Not reported/unclear				TAG	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	1.65 (SE 0.13)	1.19 (SE 0.13)		<0.05								Decrease	
*16890		Low fat, high carbohydrate diet	25/25	1.8 (SE 0.13)	1.61 (SE 0.13)		NS					TAG	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	1.65 (SE 0.13)	1.21 (SE 0.13)		<0.05								Decrease	
(Petersen <i>et al.</i> , 2006) 17199	Women	Hypoenergetic high carbohydrate, low fat diet	251/292	1.02 (SD 0.52)		-0.01 (SD 0.39)						TAG	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
	Women	Hypoenergetic low carbohydrate, high fat diet	235/287	1.04 (SD 0.83)		-0.12 (SD 0.67)									Decrease	
17200	Men	Hypoenergetic high carbohydrate, low fat diet	85/97	1.19 (SD 0.55)		-0.13 (SD 0.43)						TAG	Fasting plasma (mmol/L)	10 weeks	Decrease	bias
	Men	Hypoenergetic low carbohydrate, high fat diet	77/95	1.49 (SD 0.91)		-0.4 (SD 0.79)									Decrease	
17201		Hypoenergetic high carbohydrate, low fat diet	336/389	1.06 (SD 0.53)		-0.04 (SD 0.41)						TAG	Fasting plasma (mmol/L)	10 weeks	Decrease	bias

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
		Hypoenergetic low carbohydrate, high fat diet	312/382	1.15 (SD 0.87)		-0.19 (SD 0.71)										Decrease
*17215		Hypoenergetic low carbohydrate, high fat diet minus hypoenergetic high carbohydrate, low fat diet	Low CHO: 312/383 High CHO: 336/389							-0.09 (CI - 0.16, -0.03)	0.07	TAG	Fasting plasma (mmol/L)	10 weeks	Decrease in both	bias
(Peters on and Jovano vic-Peterso n, 1995) 17465	BMI - Obese (130-200% ideal BW)	40% CHO supplement bar 1st	4/13	119 (SD 36)	108 (SD 20)		NS	Not reported				TAG	Fasting serum (mg/dL)	6 weeks	Decrease	bias
		55% CHO supplement bar 1st	6/12	129 (SD 15)	125 (SD 13)		NS									Decrease
17467	BMI - Obese (130-200% ideal BW)	40% CHO supplement bar 2nd	6/12	129 (SD 15)	88 (SD 13)		0.05					TAG	Fasting serum (mg/dL)	6 weeks	No change	bias
		55% CHO supplement bar 2nd	4/13	119 (SD 36)	116 (SD 35)											No change
17468	Previous gestatio nal DM in last pregnan cy	40% CHO supplement bar 1st	5/13	111 (SD 34)	95 (SD 27)		NS					TAG	Fasting serum (mg/dL)	6 weeks	Decrease	bias
		55% CHO supplement bar 1st	4/12	142 (SD 75)	167 (SD 101)		NS									Decrease
17469	Previous gestatio nal DM	40% CHO supplement bar 2nd	2/12	111 (SD 34)	136 (SD 47)							TAG	Fasting serum (mg/dL)	6 weeks	No change	bias

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Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	in last pregnancy															
		55% CHO supplement bar 2nd	5/13	142 (SD 75)	143 (SD 46)		0.05								No change	
(Phillips <i>et al.</i> , 2008) *17423		Low carbohydrate diet	10/~14	77.9 (SE 14.1)	57.5 (SE 4.6)		0.05	NS				TAG	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
		Low fat diet	8/~14	60.6 (SE 6.9)	69.3 (SE 10.5)			NS							Decrease	
(Raatz <i>et al.</i> , 2005) *17235		High fat diet	10/8	1.04 (SE 0.1)		0 (SE 0.1)		0.02				TAG	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
		High GI diet	9/8	2.04 (SE 0.3)		-0.5 (SE 0.2)									Decrease	
		Low GI diet	10/6	1.79 (SE 0.3)		-0.4 (SE 0.3)									Decrease	
(Sacks <i>et al.</i> , 2009) 15585		High-fat, average-protein	ITT: /204		120 (SD 88)	-18.1%		NS				TAG	Fasting serum (mg/dL)	6 months	Decrease	No bias
		High-fat, high-protein	ITT: /201		114 (SD 71)	-19.5%		NS							Decrease	
		Low-fat, average-protein	ITT: /204		116 (SD 73)	-14.2%		NS							Decrease	
		Low-fat, high-protein	ITT: /202		114 (SD 63)	-20.4%		NS							Decrease	
*15586		High-fat, average-protein	ITT: /204	147 (SD 93)	129 (SD 89)	-12.4%		NS				TAG	Fasting serum (mg/dL)	2 years	Decrease	No bias
		High-fat, high-protein	ITT: /201	141 (SD 85)	118 (SD 71)	-16.7%		NS							Decrease	
		Low-fat, average-protein	ITT: /204	135 (SD 82)	120 (SD 83)	-11.5%		NS							Decrease	
		Low-fat, high-protein	ITT: /202	144 (SD 79)	120 (SD 67)	-16.6%		NS							Decrease	

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Segal-Isaacson <i>et al.</i> , 2004) *14983		Low fat diet	4/4	97 (SD 20)	75 (SD 20)		NS					TAG	Fasting whole blood (mg/dL)	6 weeks	Decrease	unclear
		Very low carbohydrate	4/4	97 (SD 20)	53 (SD 11)		<0.05	0.065							Decrease	
(Seshadri <i>et al.</i> , 2005) 16115		Low carbohydrate diet	40/allocated unclear			-58 (SD 94)	0.001	0.041				TAG	Fasting (mg/dL)	6 months	Decrease	unclear
		Standard diet, energy restricted	35/allocated unclear			-12 (SD 75)	NS								Decrease	
*16117	No diabetes	Low carbohydrate diet	23/allocated unclear			-46.17 (SD 86.38)	0.01	0.019				TAG	Fasting (mg/dL)	6 months	Decrease	unclear
		Standard diet, energy restricted	22/allocated unclear			4.22 (SD 42.58)	NS								Decrease	
(Sharm an <i>et al.</i> , 2004) 16942		Low fat	15/15	22.1 (SD 5.4)	17.8 (SD 6)		0.05					TAG 8hr AUC post meal response	Serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15	22.1 (SD 5.4)	13.8 (SD 3.6)		0.05	0.05							Decrease	
16943		Low fat	15/15			-23%	0.05					TAG 8hr peak AUC post meal response	Serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15			-34%	0.05								Decrease	
*14754		Low fat	15/15	1.55 (SD 0.49)	1.32 (SD 0.51)		NS					TAG	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15	1.55 (SD 0.49)	0.87 (SD 0.24)		0.05	0.05							Decrease	
(Stoernell <i>et al.</i> , 2008) *16513		Low carbohydrate diet	10/14	1.62 (SD 0.64)	1.33 (SD 0.61)			NS				TAG	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low fat diet	13/14	2.00 (SD 1.03)	2.08 (SD)										Decrease	

Author / Result ID	Sub group detail	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
					1.52)											
(Turley <i>et al.</i> , 1998)		Low fat, high carbohydrate diet	36/38	1.41 (SD 0.68)	1.57 (SD 0.69)			NS				TAG	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
*15220		Western diet	36/38	1.41 (SD 0.68)	1.48 (SD 0.74)										Decrease	
(Wolever and Mehlin <i>g.</i> , 2002)		High carbohydrate, high GI	11/11					NS				TAG	Fasting (mmol/L)	16 weeks	Decrease	unclear
17011		High carbohydrate, low GI	13/13					NS							Decrease	
		Low carbohydrate, high MUFA	11/11					NS							Increase	
(Zamboni <i>et al.</i> , 1999)		High carbohydrate, energy restriction	11/11	1.14 (SD 0.53)	1.06 (SD 0.57)		NS					TAG	Fasting plasma (mmol/L)	3 months	Decrease	unclear
16261		Olive oil enriched energy restriction diet	9/9	0.79 (SD 0.32)	1.06 (SD 0.58)		<0.05	NS							Decrease	
*16262		High carbohydrate, energy restriction	5/11	1.14 (SD 0.53)	1.06 (SD 0.61)		NS					TAG	Fasting plasma (mmol/L)	6 months	Decrease	unclear
		Olive oil enriched energy restriction diet	7/9	0.79 (SD 0.32)	0.81 (SD 0.24)		NS	NS							Decrease	

*This result has been used in the meta-analysis for high carbohydrate diets and TAG

data provided in a figure only

One paper (Appel *et al.*, 2005) presented results for TAG; however these have not been extracted as they are reported here in another paper (Furtado *et al.*, 2008).

Non-esterified fatty acids, total carbohydrate and high carbohydrate diets

Summary of cohort results

No cohort studies reported results concerning total carbohydrate and non-esterified fatty acids.

Summary of RCT data

Nine studies, reported in ten papers, explored the effects of dietary variation in carbohydrate on non-esterified fatty acids (McMillan-Price *et al.*, 2006; Cornier *et al.*, 2005; Helge, 2002; Kirk *et al.*, 2009; Lofgren *et al.*, 2005; Wolever and Mehling, 2002; Claessens *et al.*, 2009; Due *et al.*, 2004; Noakes *et al.*, 2005; Wolever and Mehling, 2003). Details of these included studies can be found in the Trial Characteristics table.

All included trials studied parallel groups. One third of studies were conducted in the USA (Cornier *et al.*, 2005; Kirk *et al.*, 2009; Wolever and Mehling, 2002; Wolever and Mehling, 2003) and the remaining were carried out in Australia (McMillan-Price *et al.*, 2006; Noakes *et al.*, 2005), Denmark (Due *et al.*, 2004; Helge, 2002), Sweden (Lofgren *et al.*, 2005) and the Netherlands (Claessens *et al.*, 2009).

Participants in the nine studies were adults (mean age ranged from 27 to 57 years), who were on average overweight or obese. Three studies recruited females only (Noakes *et al.*, 2005; Lofgren *et al.*, 2005; Cornier *et al.*, 2005) and one studied males only (Helge, 2002). The other five were mixed gender.

Final sample sizes ranged from 21 to 129 participants, with the mean number being 56, but the median being 41.

Since there are a small number of trials these have not been separated into the three main types on the basis of the replacement of carbohydrate with either fat or protein as previously.

Nine studies were included in the meta-analysis comparing diets with different carbohydrate intakes (diets differed by >5% energy from carbohydrates) and changes in non-esterified fatty acids reported as mmol/L (equal to mEq/L). One study reported results in mg/dL (Kirk *et al.*, 2009). This was transformed using the molecular weight of the most common fatty acid which is 86mol/g. One study had four groups and was divided into low GI and high GI (McMillan-Price *et al.*, 2006) – highest and lowest carbohydrate diets were used. Two studies had two groups (Claessens *et al.*, 2009) and (Wolever and Mehling, 2002). All studies included adults as participants. The first follow up reported at the end of the intervention was used. The pooled estimate indicated that non-esterified fatty acid levels were 0mmol/L (95% CI -0.04 to 0.05) higher with consumption of a higher carbohydrate diet. This was not significantly different from zero ($p=0.82$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 58%). A funnel plot was not prepared since less than 10 studies were included in the meta-analysis. Statistically, there was no evidence to suggest that high carbohydrate diets are associated with differences in fasting non-esterified fatty acid levels.

Figure 2.42 Forest plot for high carbohydrate diets and non-esterified fatty acids

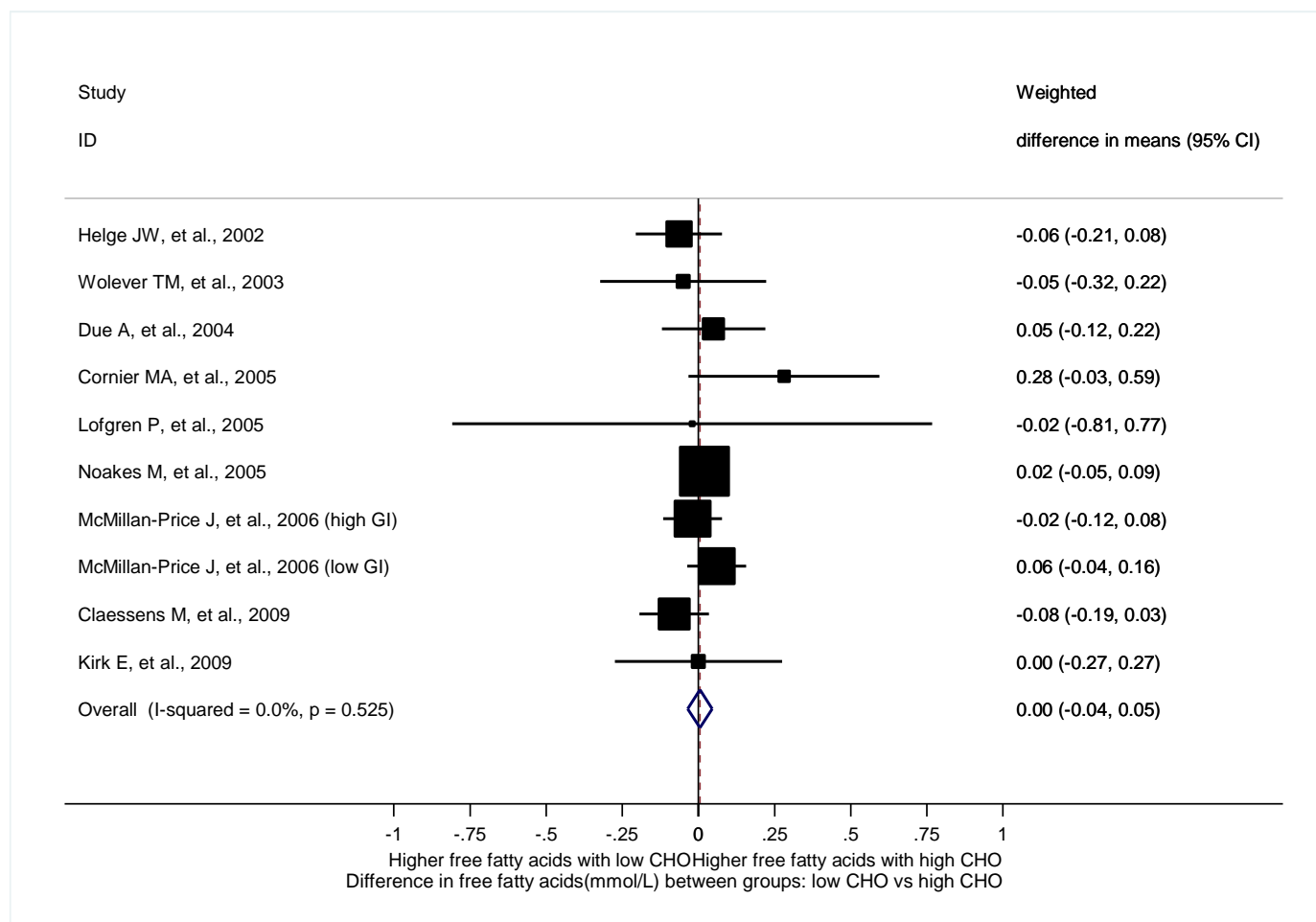


Table 2.55 Non-esterified fatty acids and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Claessen <i>et al.</i> , 2009) *16825		High carbohydrate supplement	16/allocated not reported	0.48 (SE 0.03)	0.39 (SE 0.03)	-0.07 (SE 0.03)	<0.05	NS	Free fatty acid	Fasting (mmol/L)	12 weeks	Increase	unclear
		High protein supplement - casein	14/allocated not reported	0.5 (SE 0.05)	0.45 (SE 0.06)	-0.06 (SE 0.08)	NS	NS				Decrease	
		High protein supplement - whey	18/allocated not reported	0.48 (SE 0.05)	0.48 (SE 0.04)	0.01 (SE 0.05)	NS	NS				Decrease	
(Cornier <i>et al.</i> , 2005) *16727	Insulin sensitive	High carbohydrate, low fat	6/10	858 (SE 110)	813 (SE 113)			NS	Free fatty acid	Fasting (μEq/l)	16 weeks	Decrease	unclear
		Low carbohyd rate, high fat	6/11	659 (SE 34)	532 (SE 113)							Decrease	
(Due <i>et al.</i> , 2004) 17542		High protein	23/23	500 (CI 324, 658)	294 (CI 232, 457)			0.01	Free fatty acid	Fasting (μmol/L)	6 months	Decrease	unclear
		Moderate protein	23/18	435 (CI 296, 626)	434 (CI 311, 561)							Decrease	
*17543		High protein	23/23	500 (CI 324, 658)	384 (CI 232, 493)			NS	Free fatty acid	Fasting (μmol/L)	1 year	Decrease	unclear
		Moderate protein	18/18	435 (CI 296, 626)	434 (CI 315, 533)							Decrease	
(Helge, 2002) *15912		High carbohydrate + exercise	16/16	459 (SE 49)	463 (SE 53)				Fatty acid	Fasting plasma (μmol/L)	7 weeks	Decrease	unclear
		High fat + exercise	17/17	461 (SE 38)	527 (SE 49)			NS				Decrease	
(Kirk <i>et al.</i> , 2009) *17557		High carbohydrate	completers not reported/11			-1.5 (SE 9.9)	NS		Free fatty acid	Fasting (mg/dL)	11 week	Decrease	unclear
		Very low carbohydrate	completers not reported/11			-1.5 (SE 7.5)	NS	>0.05				Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Lofgren <i>et al.</i> , 2005) *17272		High carbohydrate, low fat	20/20	0.65 (SE 0.05)	0.63 (SE 0.04)			NS	Free fatty acid	Fasting plasma (mmol/L)	10 weeks	Decrease	unclear
		High fat, moderate carbohydrate	20/20	0.71 (SE 0.4)	0.65 (SE 0.4)							Decrease	
(McMillan -Price <i>et al.</i> , 2006) *16229		High CHO, high GI diet	32/32	510 (SE 33)		-63 (SE 35)		NS	Free fatty acid	Fasting (μmol/L)	12 weeks	Decrease	Unclear
		High CHO, low GI diet	32/32	436 (SE 32)		3 (SE 36)		NS				Decrease	
		High protein, high GI diet	32/32	545 (SE 42)		-44 (SE 35)		NS				Decrease	
		High protein, low GI diet	33/33	520 (SE 53)		-57 (SE 34)		NS				Decrease	
(Noakes <i>et al.</i> , 2005) 17005 *17006		High carbohydrate diet	48/48	0.41 (SE 0.02)	0.37 (SE 0.02)				Free fatty acid	Fasting (mmol/L)	8 weeks	Decrease	unclear
		High protein diet	52/52	0.46 (SE 0.03)	0.39 (SE 0.02)			NS				Decrease	
		High carbohydrate diet	48/48	0.41 (SE 0.02)	0.39 (SE 0.02)	-0.02 (SE 0.02)			Free fatty acid	Fasting (mmol/L)	12 weeks	Decrease	unclear
		High protein diet	52/52	0.46 (SE 0.03)	0.42 (SE 0.03)	-0.04 (SE 0.03)		0.765				Decrease	
(Wolever and Mehling, 2002) 17013		High carbohydrate, high GI	11/11			-0.037 (SE 0.046)		NS	Non-esterified fatty acids	Fasting (mEq/l)	16 weeks	Decrease	unclear
		High carbohydrate, low GI	13/13			0.188 (SE 0.116)		NS				Decrease	
		Low carbohydrate, high MUFA	11/11			-0.144 (SE 0.068)		NS				Increase	
(Wolever and Mehling, 2003) *17135		High carbohydrate, high GI	11/13			0.04 (SE 0.05)		NS	Free fatty acids	Fasting (mEq/l)	4 months	Decrease	unclear
		High carbohydrate, low GI	13/13			-0.19 (SE 0.12)		NS				Decrease	
		Low	11/12			-0.14 (SE		NS				Increase	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
		carbohydrate, high MUFA				0.07)							

*This result was used in the meta-analysis for high carbohydrate diets and non-esterified fatty acids

Total cholesterol:HDL ratio, total carbohydrate and high carbohydrate diets

Summary of cohort results

Data were extracted from two cohort studies in children. Both the Amsterdam Growth and Health Study (Twisk *et al.*, 1997) and the Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) presented evidence concerning the association between total carbohydrates and total cholesterol:HDL ratio (TC:HDL). Both studies reported total carbohydrate intake as a percentage of total energy as measured by a dietary history. The Northern Ireland Young Hearts Project reported no association between total carbohydrates intake and TC:HDL cholesterol ratio in either boys or girls. However, the Amsterdam Growth and Health Study provided evidence of an increasing ratio of TC:HDL cholesterol with increasing percentage energy derived from carbohydrate. A higher ratio would indicate a worsening cardiometabolic risk.

The Northern Ireland Young Hearts Project (Boreham *et al.*, 1999) adjusted for socio-economic status and sexual maturity. The Amsterdam Growth and Health Study (Twisk *et al.*, 1997) adjusted for age, gender, sum of skinfolds and VO₂ max.

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

Summary of RCT data

Fifteen studies explored the effects of dietary variation in the carbohydrate proportion of diets – replacing carbohydrate with fat, protein or both – on TC:HDL ratio. Of these, 11 were conducted in the USA and the others in Australia (McMillan-Price *et al.*, 2006), France (Colette *et al.*, 2003), Scotland (Kirkwood *et al.*, 2007) and New Zealand (Ley *et al.*, 2004).

The majority of trials employed a parallel group design, whilst three opted for a crossover approach (Sharman *et al.*, 2004; Clevidence *et al.*, 1992; Ginsberg *et al.*, 1998). Trials were either unclear regarding blinding (11), open (2), double blind (1) or single blind (1). All studied adults. Predominantly mixed gender trials were included within this review although three studies chose to recruit males only (Krauss *et al.*, 2006; Sharman *et al.*, 2004; Clevidence *et al.*, 1992) and three females only (Howard *et al.*, 2006; Kirkwood *et al.*, 2007; Mahon *et al.*, 2007). Mean BMI tended to fall into the overweight or obese category for most trials, although one study by Ginsberg *et al.* (Ginsberg *et al.*, 1998) used participants indicative of a healthy weight (mean BMI: 24 kg/m²).

Final numbers of participants typically ranged from 15 to 224, although an exception to this is the Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006), which had a large sample size of 48,835 (5.8% only provided a blood sample).

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

If a trial tested the effects of diets which differed by 5% or more of energy from carbohydrate it was then further categorised into one of three categories. Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat also differed by 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein differed by 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more, but protein intakes were also different by more than 2%.

Four studies had four groups (Dansinger *et al.*, 2005) (Krauss *et al.*, 2006) (Mahon *et al.*, 2007) (McMillan-Price *et al.*, 2006). Three studies compared lowest and highest carbohydrate intakes (Mahon *et al.*, 2007) (Dansinger *et al.*, 2005) (Krauss *et al.*, 2006). One study compared higher and lower carbohydrate on high and low GI diets (McMillan-Price *et al.*, 2006). One study had three groups (Due *et al.*, 2008) where highest and lowest carbohydrate intakes were included.

Follow up varied from six weeks to three years.

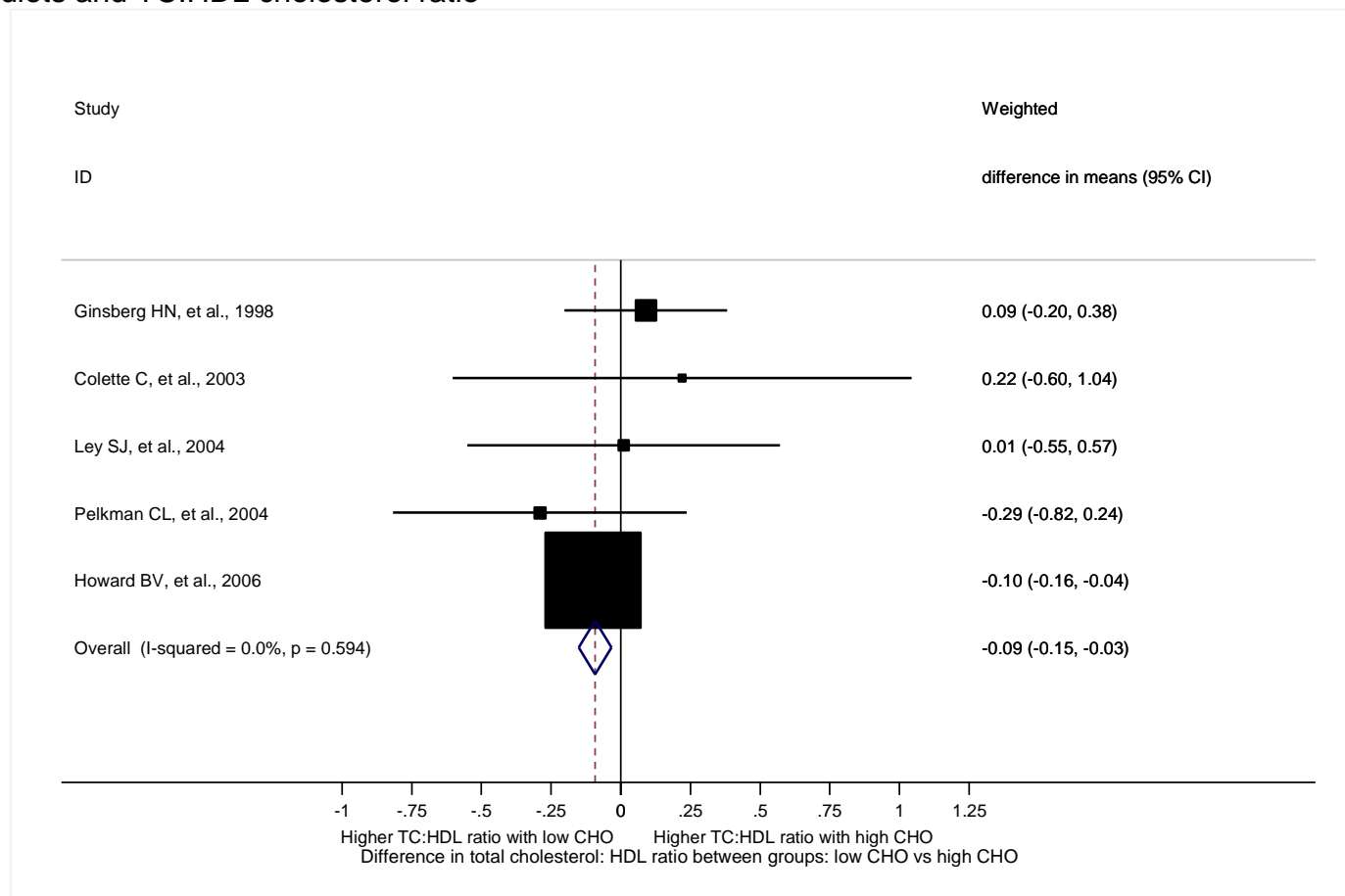
Comparison of higher carbohydrate, lower fat diets with lower carbohydrate, higher fat diets

One study did not provide the ratios at follow up, but reported in text that there was no differential impact of the diets on the ratio of total to HDL cholesterol (Kirkwood *et al.*, 2007). Five studies were included in the meta-analysis comparing different carbohydrate and fat intake and changes in TC:HDL ratio.

All studies included adults as participants. On average, the high carbohydrate diets provided 58% (range 52 to 64%) carbohydrate and the lower carbohydrate diets 46% (range 40 to 50.5%). The first follow up reported at the end of the intervention was used. This varied from six weeks to three years.

The pooled estimate indicated that the TC:HDL ratio was 0.09 (95% CI 0.03 to 0.15) lower with consumption of a higher carbohydrate, low fat diet. This was significantly different from zero ($p=0.001$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 77%). Statistically there was evidence that high carbohydrate low fat diets are associated with a lower TC:HDL ratio. However, Howard *et al.*, 2006 contributed 93% to the pooled estimate and therefore the results should be interpreted with caution.

Figure 2.43 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and TC:HDL cholesterol ratio



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

No studies were categorised as comparing changes in carbohydrate and protein only.

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

Nine studies were included in the meta-analysis comparing different carbohydrate, fat and protein intake and changes in the TC:HDL ratio. All studies included adults as participants. The first follow up reported at the end of the intervention was used. On average, the high carbohydrate diets provided 56% carbohydrate, and the low carbohydrate diets 35% of energy. The largest difference in carbohydrate between groups was in (Sharman *et al.*, 2004), which compared diets with 56% and 8% carbohydrate content.

The pooled estimate indicated that the TC:HDL ratio was 0.06 (95% CI -0.14 to 0.27) higher with consumption of a higher carbohydrate, low fat diet. This was not significantly different from zero ($p=0.42$). Overall heterogeneity denoted by I^2 was 73% (95% CI 51 to 86%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically there was no evidence that high carbohydrate lower fat, lower protein diets are associated with changes in levels of the TC:HDL ratio.

Figure 2.44 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and TC:HDL cholesterol ratio

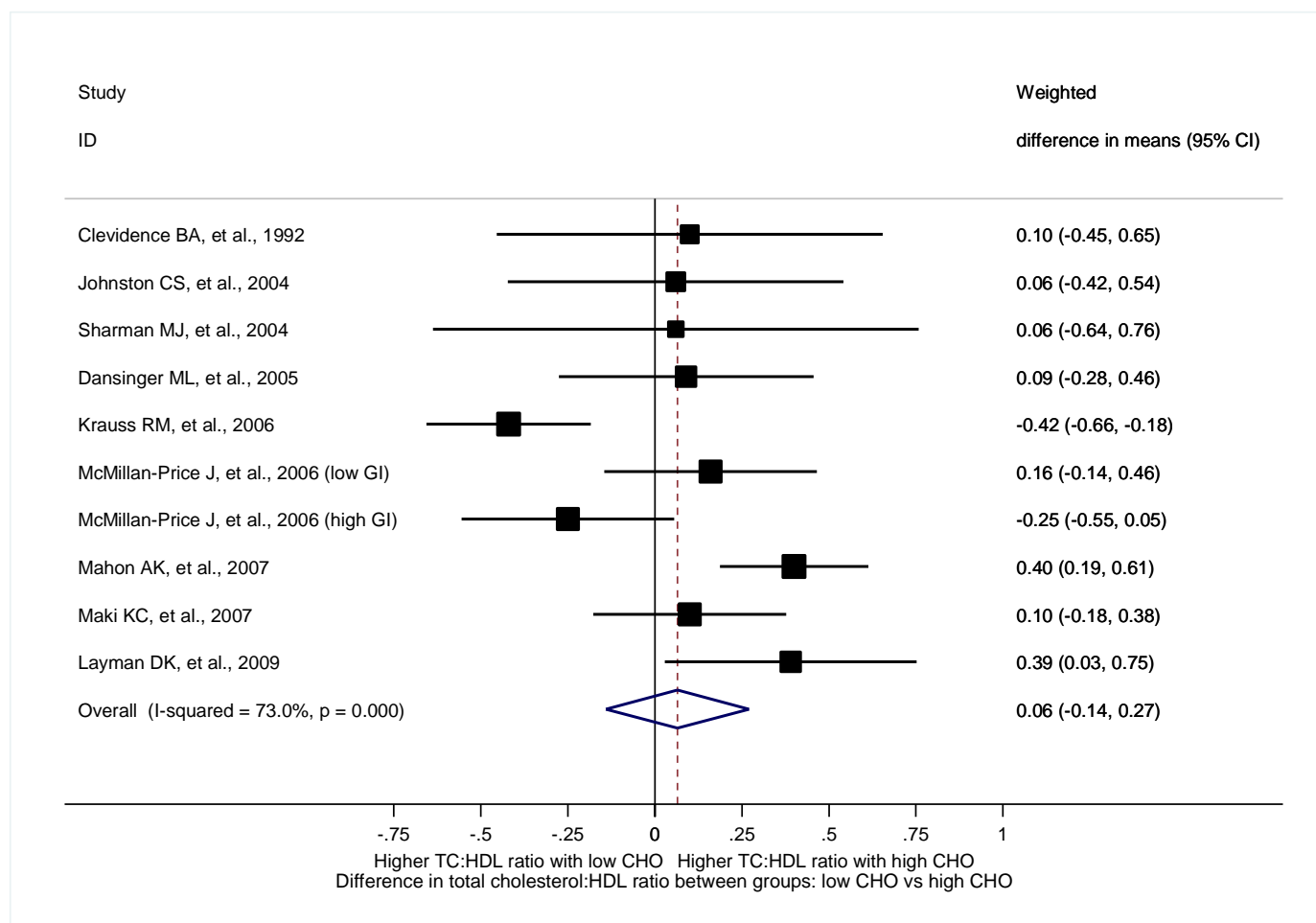


Figure 2.45 Contour-enhanced funnel plot for studies presenting data on effects of higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and TC:HDL cholesterol ratio

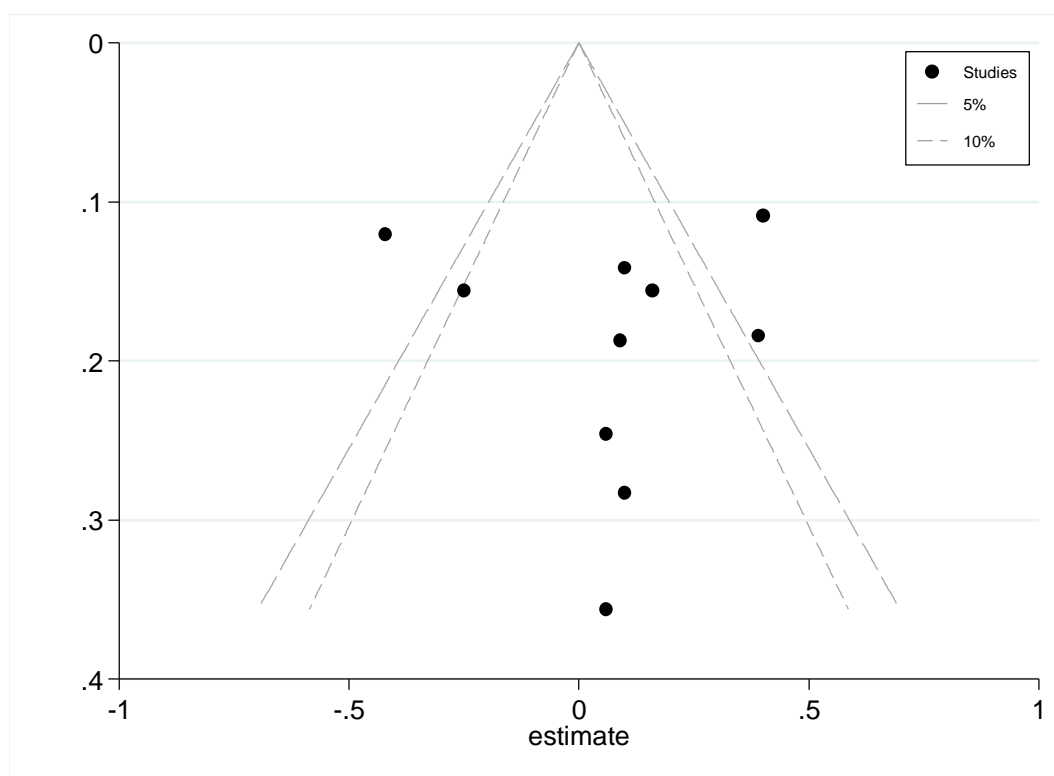


Table 2.56 Total cholesterol:HDL ratio and total carbohydrate: cohort studies in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub- group Details	Contrast	Exposure Units	RR (CI)	Beta coefficient (SE)/(CI)	p	P trend	Adjustments
(Boreham <i>et al.</i> , 1999) 14167 The Northern Ireland Young Hearts Project	Northern Ireland, Primarily White	12-15 %M 49.3	509	4 years (1.7)	Dietary history	Carbohydrate, total (% energy)	Total cholesterol :HDL ratio Serum	Male				Not reported		NS	SES/Class, sexual maturity
14193 The Northern Ireland Young Hearts Project								Female				Not reported		NS	SES/Class, sexual maturity
(Twisk <i>et al.</i> , 1997) 13271 Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 46	233	14 years	Dietary history	Carbohydrate, total (% energy)	Total cholesterol :HDL ratio* Serum Non-fasting		Continuous risk estimate	Per 1% CHO energy	1.3 (1.1, 1.6)		0.01		age, gender, sum of skinfolds, VO2 max

*Odds of a total cholesterol:HDL ratio of >4.0 (participants aged 13-16 years) and >5.5 (participants aged 21-27 years)

Table 2.57 Total cholesterol:HDL ratio and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
(Sharman <i>et al.</i> , 2004) 14753		Low fat	15/15	4.96 (SD 1.03)	4.59 (SD 1.17)		NS		Total cholesterol :HDL ratio	Fasting serum (mmol/L)	6 weeks	Decrease	unclear
		Very low carbohydrate	15/15	4.96 (SD 1.03)	4.53 (SD 0.73)		NS	NS				Decrease	
(Johnston <i>et al.</i> , 2004) 14863		High carbohydrate, low fat	7/10	4.2 (SE 0.3)		4.0% (SE 6.1%)	NS		Total cholesterol :HDL ratio	Whole blood	6 weeks	Decrease	unclear
		High protein, low fat	9/10	3.5 (SE 0.4)		2.2% (SE 3.5%)	NS	0.888				Decrease	
(Layman <i>et al.</i> , 2009) 14966		High carbohydrate, low protein diet	30/66			-0.5 (SE 0.12)			Change in Total cholesterol :HDL ratio	Fasting plasma	1 year	Decrease	unclear
		Low carbohydrate, high protein diet	41/64			-0.89 (SE 0.14)		0.044				Decrease	
(Mahon <i>et al.</i> , 2007) 15074		Energy restriction + beef	14/14	4.3 (SD 1.4)	3.8 (SD 0.7)	-0.5 (SD 0.1)	NS	NS	Total cholesterol :HDL ratio	Fasting serum	9 weeks	Decrease	unclear
		Energy restriction + carbohydrate/fat	14/14	4.0 (SD 1.0)	4.1 (SD 1.1)	0.1 (SD 0.3)	NS	NS				Decrease	
		Energy restriction + chicken	15/15	4.6 (SD 1.4)	4.2 (SD 1.0)	-0.3 (SD 0.3)	NS	NS				Decrease	
		Control	11/11	4.5 (SD 1.2)	4.2 (SD 1.0)	-0.3 (SD 0.2)	NS					No change	
(Krauss <i>et al.</i> , 2006) 17487		26% CHO High saturated fat	40/52	5.30 (SD 1.8)		-0.16 (SE 0.08)		NS	Total cholesterol :HDL ratio		12 weeks	Decrease	unclear
		26% CHO Low saturated fat	47/59	4.93 (SD 1.3)		-0.03 (SE 0.09)		<0.01				Decrease	
		39% CHO Low saturated fat	42/56	5.09 (SD 1.25)		0.29 (SE 0.11)		NS				Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
(McMillan- Price <i>et al.</i> , 2006) 16958		54% CHO Low saturated fat	49/57	5.03 (SD 1.17)		-0.45 (SE 0.08)						Decrease	
		High CHO, high GI diet	32/32	3.94 (SE 0.25)		-0.23 (SE 0.11)		NS	Total cholesterol :HDL ratio		12 weeks	Decrease	unclear
		High CHO, low GI diet	32/32	4.16 (SE 0.24)		-0.21 (SE 0.11)		NS				Decrease	
		High protein, high GI diet	32/32	4.75 (SE 0.32)		0.02 (SE 0.11)		NS				Decrease	
		High protein, low GI diet	33/33	3.83 (SE 0.26)		-0.37 (SE 0.11)		NS				Decrease	
(Kirkwood <i>et al.</i> , 2007) 15674		Group 1: No advice	18/allocated not reported				NS		Total cholesterol :HDL ratio	Fasting	12 weeks	No change	unclear
		Group 2: Conventional weight loss diet	16/allocated not reported				NS	NS				Decrease	
15675		Group 3: Exercise	19/allocated not reported				NS	NS	Total cholesterol :HDL ratio	Fasting	12 weeks	Decrease	unclear
		Group 4: Conventional weight loss diet + exercise	16/allocated not reported				NS					Decrease	
(Dansinger <i>et al.</i> , 2005)		Atkins	40/40			-0.36 (SD 0.66)	0.05	Unclear	Total cholesterol :HDL ratio	Fasting serum	2 months	Decrease	No bias

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
15704		Ornish	40/40			-0.18 (SD 1.01)	NS					Decrease	
		Weight watchers	40/40			-0.49 (SD 1.86)	NS					Decrease	
		Zone	40/40			-0.66 (SD 1.06)	0.05					Decrease	
15808		Atkins	40/40			-0.38 (SD 0.68)	0.05		Total cholesterol :HDL ratio	Fasting serum	6 months	Decrease	No bias
		Ornish	40/40			-0.25 (SD 1.07)	NS					Decrease	
		Weight watchers	40/40			-0.6 (SD 1.57)	0.01					Decrease	
		Zone	40/40			-0.46 (SD 0.93)	0.05					Decrease	
15809		Atkins	40/40			-0.39 (SD 0.69)	0.05		Total cholesterol :HDL ratio	Fasting serum	1 year	Decrease	No bias
		Ornish	40/40			-0.3 (SD 0.96)	NS					Decrease	
		Weight watchers	40/40			-0.7 (SD 1.67)	0.01					Decrease	
		Zone	40/40			-0.52 (SD 1.04)	0.05					Decrease	
(Ley <i>et al.</i> , 2004) 15953		Control	70/70			-0.01 (SE 0.26)			Total cholesterol :HDL ratio	Fasting serum	6 months	No change	unclear
		Low fat	66/66			-0.37 (SE 0.1)		0.05				Decrease	
15954		Control	70/70			-0.11 (SE 0.26)			Total cholesterol :HDL ratio	Fasting serum	1 year	No change	unclear
		Low fat	66/66			-0.1 (SE 0.14)		NS				Decrease	
15955		Control	57/70			-0.23 (SE 0.28)			Total cholesterol :HDL ratio	Fasting serum	2 years	No change	unclear
		Low fat	47/66			-0.44 (SE 0.13)		NS				Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
15956		Control	51/70			-0.12 (SE 0.27)			Total cholesterol :HDL ratio	Fasting serum	3 years	No change	unclear
		Low fat	48/66			-0.17 (SE 0.13)		NS				Decrease	
15957		Control	52/70			-0.53 (SE 0.24)			Total cholesterol :HDL ratio	Fasting serum	5 years	No change	unclear
		Low fat	51/66			-0.34 (SE 0.14)		NS				Decrease	
(Clevence <i>et al.</i> , 1992) 16610		High fat diet	42/46	4.1 (SE 0.2)	4.0 (SE 0.2)	-0.1	NS	NS	Total cholesterol :HDL ratio		10 weeks	No change	unclear
		Low fat diet	42/46	4.1 (SE 0.2)	4.1 (SE 0.2)	0	NS					No change	
(Pelkman <i>et al.</i> , 2004) 16883		Low fat, high carbohydrate diet	25/25	4.66 (SE 0.19)	4.3 (SE 0.19)		<0.05	Not reported	Total cholesterol :HDL ratio		6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	5.14 (SE 0.18)	4.58 (SE 0.18)		<0.05					Decrease	
16884		Low fat, high carbohydrate diet	25/25	4.66 (SE 0.19)	4.4 (SE 0.19)		NS		Total cholesterol :HDL ratio		10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	5.14 (SE 0.18)	4.69 (SE 0.19)		<0.05					Decrease	
16904	Weight stable during maintenance	Low fat, high carbohydrate diet	12/25	4.69 (SE 0.27)	4.25 (SE 0.27)		NS		Total cholesterol :HDL ratio		6 weeks	Decrease	unclear
	Weight stable during	Moderate fat, lower carbohydrate	17/27	5.17 (SE 0.23)	4.57 (SE 0.23)		<0.05					Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
	maintenance	diet											
16905	Weight stable during maintenance	Low fat, high carbohydrate diet	12/25	4.69 (SE 0.27)	4.59 (SE 0.27)		NS		Total cholesterol :HDL ratio		10 weeks	Decrease	unclear
	Weight stable during maintenance	Moderate fat, lower carbohydrate diet	17/27	5.17 (SE 0.23)	4.71 (SE 0.23)		<0.05					Decrease	
(Ginsberg <i>et al.</i> , 1998) 17254		Average American Diet	103/118		4.07 (SE 0.1)				Total cholesterol :HDL ratio	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		4.21 (SE 0.11)		NS					Not reported	
		Step 1 diet	103/118		4.16 (SE 0.11)		NS					Not reported	
17276	Men	Average American Diet	46/118		4.52 (SE 0.17)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Men	Low saturated fat diet	46/118		4.71 (SE 0.18)		NS					Not reported	
	Men	Step 1 diet	46/118		4.65 (SE 0.17)		NS					Not reported	
17277	Women	Average American Diet	57/118		3.71 (SE 0.11)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Women	Low saturated fat diet	57/118		3.8 (SE 0.13)		NS					Not reported	
	Women	Step 1 diet	57/118		3.77 (SE 0.12)		NS					Not reported	
17312	Black	Average American Diet	26/118		3.93 (SE 0.18)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
17313	Black	Low saturated fat diet	26/118		3.97 (SE 0.19)			NS				Not reported	
	Black	Step 1 diet	26/118		3.99 (SE 0.19)			NS				Not reported	
	Non black	Average American Diet	77/118		4.12 (SE 0.12)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Non black	Low saturated fat diet	77/118		4.28 (SE 0.14)			NS				Not reported	
	Non black	Step 1 diet	77/118		4.22 (SE 0.13)			NS				Not reported	
17328	Pre- menopausal	Average American Diet	39/118		3.47 (SE 0.12)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Pre- menopausal	Low saturated fat diet	39/118		3.53 (SE 0.14)			NS				Not reported	
	Pre- menopausal	Step 1 diet	39/118		3.50 (SE 0.13)			NS				Not reported	
17329	Post- menopausal	Average American Diet	18/118		4.23 (SE 0.17)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Post- menopausal	Low saturated fat diet	18/118		4.37 (SE 0.21)			NS				Not reported	
	Post- menopausal	Step 1 diet	18/118		4.35 (SE 0.21)			NS				Not reported	
17344	Men <40y	Average American Diet	30/118		4.15 (SE 0.18)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Men <40y	Low saturated fat diet	30/118		4.32 (SE 0.18)			NS			6 weeks	Not reported	No bias
	Men <40y	Step 1 diet	30/118		4.26 (SE 0.17)			NS			6 weeks	Not reported	No bias

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight Change	Outcome Assessment Bias
17345	Men >40y	Average American Diet	16/118		5.22 (SE 0.27)				Total cholesterol :HDL ratio		6 weeks	Not reported	No bias
	Men >40y	Low saturated fat diet	16/118		5.46 (SE 0.30)			NS				Not reported	
	Men >40y	Step 1 diet	16/118		5.38 (SE 0.32)			NS				Not reported	
(Colette <i>et al.</i> , 2003) 17412		High carbohydrate diet	15/15	4.80 (SE 0.32)	4.42 (SE 0.33)			NS	Total cholesterol :HDL ratio	Fasting serum	8 weeks	Decrease	unclear
		High MUFA diet	17/17	4.38 (SE 0.35)	4.20 (SE 0.26)			NS				Decrease	
(Howard <i>et al.</i> , 2006) 16249		Control	approx n=1699 (5.8% sub-sample of 29294)	4.1 (SD 1.3)	4.0 (SD 1.2)	-0.1 (SD 1.0)			Total cholesterol :HDL ratio	Fasting	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub-sample of 19541)	4.0 (SD 1.2)	3.8 (SD 1.1)	-0.2 (SD 0.8)		NS				Decrease	
(Maki <i>et al.</i> , 2007) 17284		Ad libitum low GL diet	39/43	3.7 (SE 0.1)		-0.2 (SE 0.1)		NS	Total cholesterol :HDL ratio	Fasting	12 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	3.8 (SE 0.2)		0 (SE 0.1)		NS				Decrease	
17285		Ad libitum low GL diet	39/43	3.7 (SE 0.1)		-0.3 (SE 0.1)		NS	Total cholesterol :HDL ratio	Fasting	36 weeks	Decrease	unclear
		Low fat, energy restricted	38/43	3.8 (SE 0.2)		-0.2 (SE 0.1)		NS				Decrease	

LDL:HDL cholesterol ratio, total carbohydrate and high carbohydrate diets

Summary of cohort results

Data were extracted from the Amsterdam Growth and Health Study, a cohort study of children (Twisk *et al.*, 1997). Baseline total carbohydrate intake as a percentage of total energy measured by a dietary history was reported.

The Amsterdam Growth and Health Study provided evidence of an increasing ratio of LDL:HDL cholesterol with increasing baseline percentage energy derived from carbohydrate. A higher ratio would indicate a worsening cardiometabolic risk.

The Amsterdam Growth and Health Study (Twisk *et al.*, 1997) adjusted for age, gender, sum of skinfolds and VO₂ max.

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

Summary of RCT data

Eleven studies explored the effects of dietary variation in carbohydrate on LDL:HDL cholesterol ratio. Details of these studies can be found in the Trial Characteristics table.

Of the included studies, nine used a parallel group design and two a crossover design (Turley *et al.*, 1998; Clevidence *et al.*, 1992). The majority did not state the extent of blinding of participants and/or researchers, but two studies were classified as open with regard to blinding (Petersen *et al.*, 2006; Due *et al.*, 2008). Overall, study durations ranged from six weeks to one year.

Studies were conducted in a range of countries such as the USA (4), Canada (1), Italy (1), France (1), Denmark (1), New Zealand (1), the Netherlands (1) and Europe as a whole (1).

All participants studied were adults. Two trials recruited males only (Turley *et al.*, 1998; Clevidence *et al.*, 1992), two studied females only (Zambon *et al.*, 1999; Layman *et al.*, 2005) and the remaining were mixed gender. Most studies that reported LDL:HDL cholesterol ratio used participants with a BMI > 25 kg/m², although studies by Meckling *et al.*, Clevidence *et al.* and Pelkman *et al.* failed to report this data (Meckling *et al.*, 2004; Clevidence *et al.*, 1992; Pelkman *et al.*, 2004).

Final sample sizes ranged from 20 to 771 participants, with three studies being particularly large with more than 100 participants in each (Petersen *et al.*, 2006; Due *et al.*, 2008; Dansinger *et al.*, 2005). The mean sample size was 131 and the median number was 52. Follow up varied from six weeks to 12 months.

Trials were separated into three main types on the basis of the proportion of energy derived from the macronutrients. For inclusion in a meta-analysis a 5% difference in energy from carbohydrate was taken as meaningful. Actual consumption was used rather than the intended diet unless otherwise stated – see the Trial Characteristics table.

If a trial tested the effects of diets which differed by 5% or more of energy from carbohydrate it was then further categorised into one of three categories. Higher carbohydrate, lower fat diets were differentiated from lower carbohydrate, higher fat diets where percentage of energy from fat also differed by 2% or more. Higher carbohydrate, lower protein diets were differentiated from lower carbohydrate, higher protein diets where percentage of energy from protein differed by 2% or more and higher carbohydrate, lower protein and fat diets were differentiated from lower carbohydrate, higher protein and fat diets where percentage of energy from fat was 2% or more, but protein intakes were also different by more than 2%.

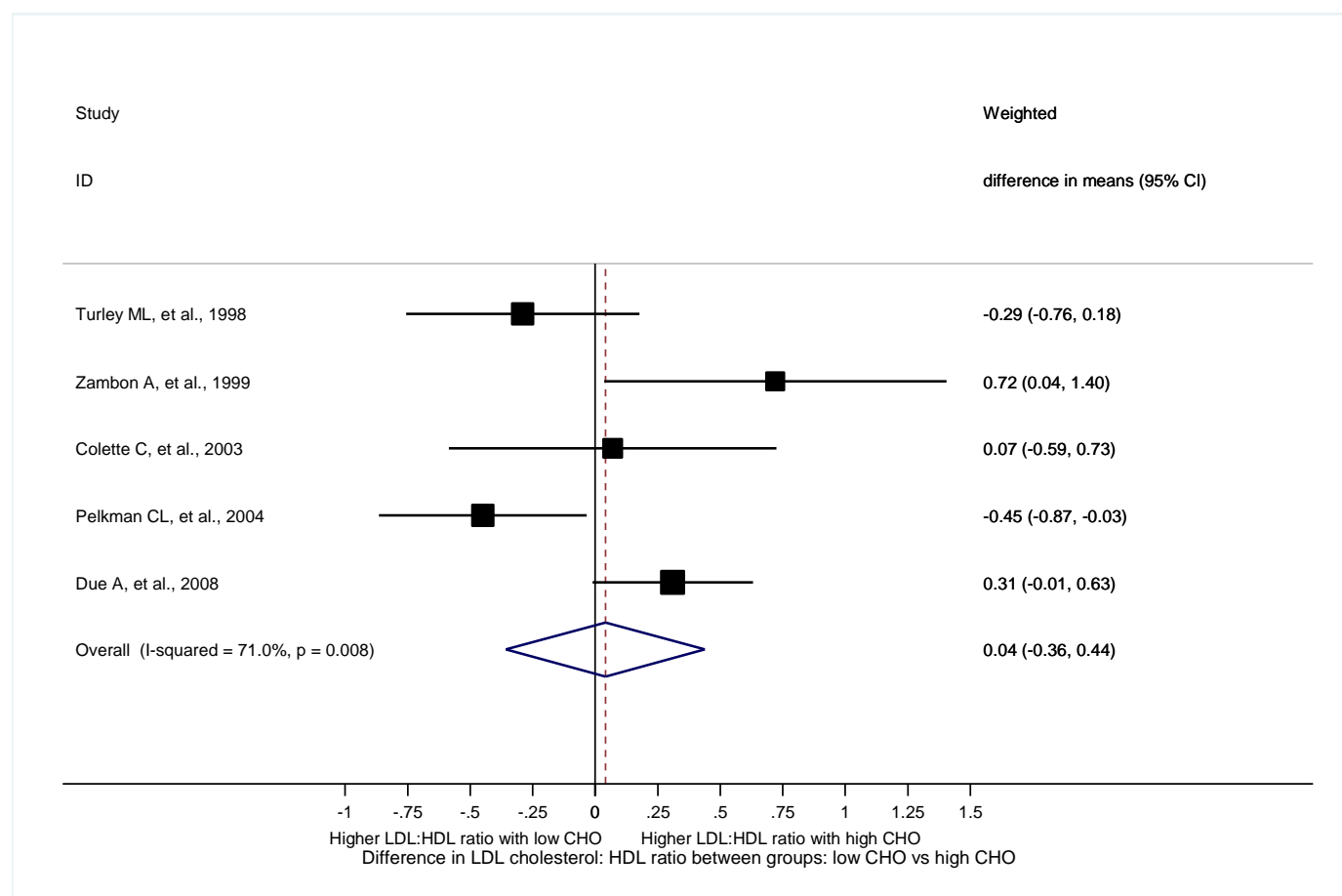
Comparison of higher carbohydrate, lower fat diets with lower carbohydrate, higher fat diets

Petersen *et al.* (Petersen *et al.*, 2006) observed no differential effect of hypoenergetic high and low carbohydrate diets over 10 weeks. The ratios were not provided in the paper, and so this study could not be included in a meta-analysis.

Five studies were included in the meta-analysis comparing different carbohydrate and fat intake and changes in LDL:HDL cholesterol ratio. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to three years.

The pooled estimate indicated that LDL:HDL cholesterol ratio was 0.04 (95% CI -0.36 to 0.44) higher with consumption of a higher carbohydrate, low fat diet. This was not significantly different from zero ($p=0.84$). Overall heterogeneity denoted by I^2 was 71% (95% CI 26 to 89%). Statistically, there was no evidence that high carbohydrate low fat diets are associated with changes in levels of LDL:HDL cholesterol ratio.

Figure 2.46 Forest plot for higher carbohydrate, lower fat diets and lower carbohydrate, higher fat diets and LDL:HDL cholesterol ratio



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

One study that compared an *ad libitum* higher carbohydrate, lower protein diet with a lower carbohydrate, higher protein diet provided data (Claessens *et al.*, 2009). The primary aim was to determine whether these diets would impact on weight maintenance and metabolic parameters over 12 weeks after a period of weight loss generated by a ketogenic very low carbohydrate diet. Overall, there was no differential improvement in the LDL:HDL cholesterol ratio comparing high with low carbohydrate diets.

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

Meckling *et al.* (Meckling *et al.*, 2004) studied the effects of high carbohydrate (62%) low fat diet and a low carbohydrate (15% carbohydrate) diet in 31 overweight and obese subjects. The ratio decreased to a greater extent on the high carbohydrate diet ($p < 0.05$). However, measures of variance around the mean ratios were not provided and so this study could not be included in the meta-analysis.

Three studies were included in the meta-analysis comparing different carbohydrate, fat and protein intake and changes in LDL:HDL cholesterol ratio. All studies included adults as participants. The first follow up reported at the end of the intervention was used.

The pooled estimate indicated that LDL:HDL cholesterol ratio was 0.10 (95% CI -0.14 to 0.34) lower with consumption of a higher carbohydrate, low fat, low protein diet. This was not significantly different from zero ($p=0.43$). Overall heterogeneity denoted by I^2 was 64% (95% CI 0 to 90%). Statistically, there was no evidence that high carbohydrate low fat, low protein diets are associated with changes in the LDL:HDL cholesterol ratio.

Figure 2.47 Forest plot for higher carbohydrate, lower protein and fat diets and lower carbohydrate, higher fat and protein diets and LDL:HDL cholesterol ratio

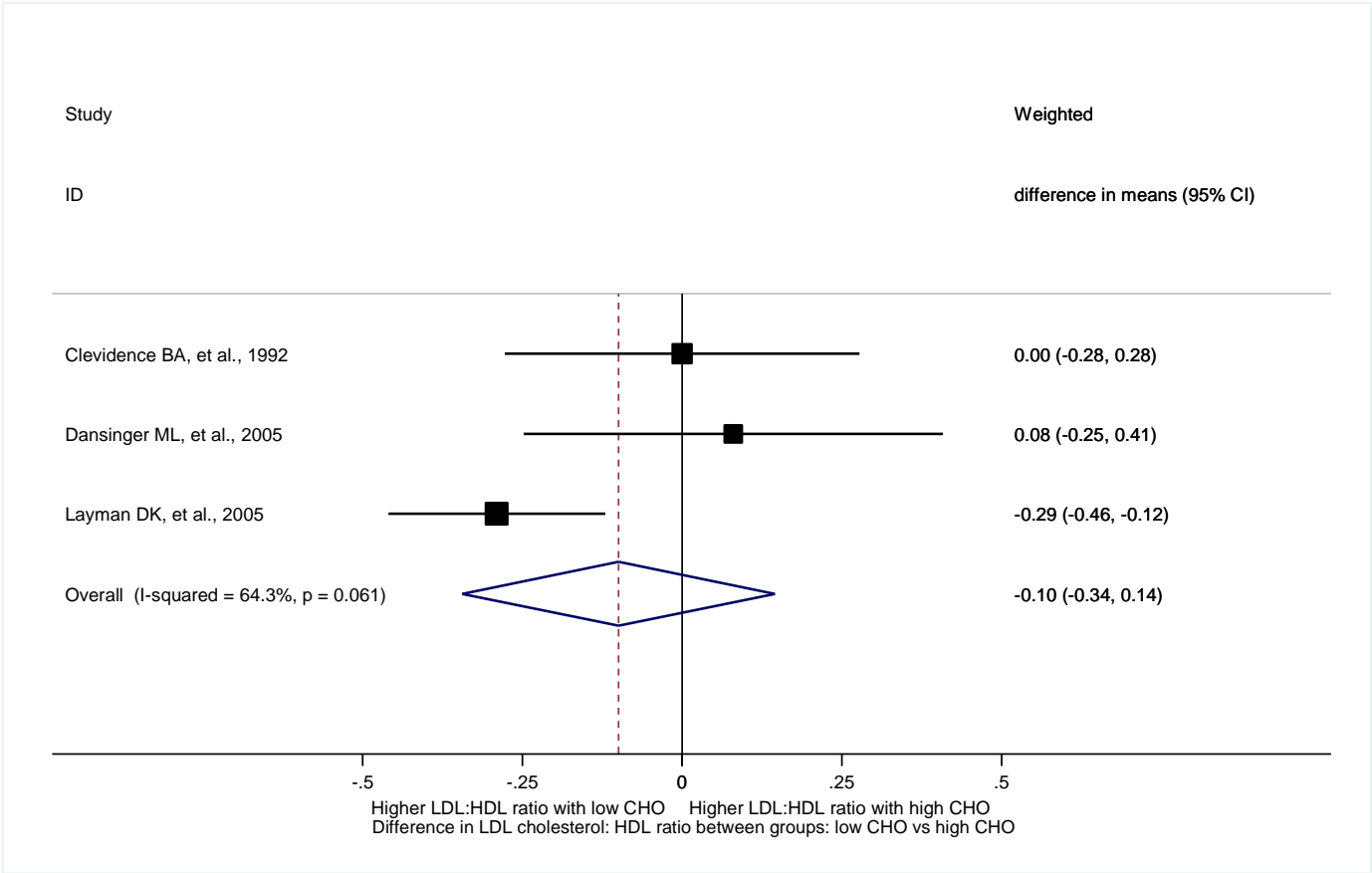


Table 2.58 LDL:HDL cholesterol ratio and total carbohydrate: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub- group Details	Contrast	Exposure Units	RR (CI)	Beta coefficient (SE)/(CI)	p	P trend	Adjustments
(Twisk <i>et al.</i> , 1997) 13271 Amsterdam Growth and Health Study	The Netherlands	12-15 (13) %M 46	233	14 years	Dietary history	Carbohydrate, total (% energy)	LDL:HDL ratio* Serum Non-fasting		Continuous risk estimate	Per 1% CHOI energy	1.2 (1, 1.5)		0.02		age, gender, sum of skinfolds, VO2 max

*Odds of HDL cholesterol of <1.1mmol/L (participants aged 13-16 years) and <0.9mmol/L (participants aged 21-27 years)

Table 2.59 LDL:HDL cholesterol ratio and high carbohydrate diets: 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	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Meckling <i>et al.</i> , 2004) 14876		Low carbohydrate	15/10	3.4	3.1		0.05	0.05	LDL:HDL cholesterol ratio	Fasting	10 weeks	Decrease	No bias
		Low fat	16/10	3.2	2.6		0.05					Decrease	
(Layman <i>et al.</i> , 2005) 16181		High carbohydrate diet	12/12	2.79 (SD 0.20)	2.59 (SD 0.13)		NS		LDL:HDL cholesterol ratio	(mmol/L)	16 weeks	Decrease	unclear
		High protein diet	12/12	2.84 (SD 0.23)	2.88 (SD 0.27)		NS	0.35				Decrease	
16182		High carbohydrate diet + exercise	12/12	2.48 (SD 0.19)	2.35 (SD 0.14)		NS		LDL:HDL cholesterol ratio		16 weeks	Decrease	unclear
		High protein diet + exercise	12/12	2.78 (SD 0.26)	2.64 (SD 0.26)		NS	0.64				Decrease	
(Turley <i>et al.</i> , 1998) 15219		Western diet	36/38	3.03 (SD 0.96)	3.17 (SD 1.05)				LDL:HDL cholesterol ratio	Fasting serum	6 weeks	Decrease	unclear
		Low fat, high carbohydrate diet	36/38	3.03 (SD 0.96)	2.88 (SD 0.97)			0.004				Decrease	
(Due <i>et al.</i> , 2008) 15301		Control	24/25	2.51 (CI 2.1, 2.9)	2.59 (CI 2.0, 3.1)	0.05 (CI -0.2, 0.3)			LDL:HDL cholesterol ratio	Fasting plasma	6 months	Increase	unclear
		High MUFA	39/52	2.49 (CI 2.1, 2.9)	2.16 (CI 1.9, 2.4)	-0.33 (CI -0.6, -0.1)		0.036				Increase	
		Low fat	43/48	2.42 (CI 2.1, 2.7)	2.4 (CI 2.0, 2.8)	-0.02 (CI -0.2, 0.2)		NS				Increase	
(Dansinger <i>et al.</i> , 2005) 15810		Atkins	40/40			-0.18 (SD 0.57)	0.05	Unclear	LDL:HDL cholesterol ratio	Fasting serum	2 months	Decrease	No bias
		Ornish	40/40			-0.21 (SD 0.67)	NS					Decrease	
		Weight watchers	40/40			-0.42 (SD 1.55)	NS					Decrease	
		Zone	40/40			-0.33 (SD 0.79)	0.01					Decrease	
15811		Atkins	40/40			-0.3 (SD 0.55)	0.01		LDL:HDL cholesterol ratio	Fasting serum	6 months	Decrease	No bias
		Ornish	40/40			-0.22 (SD 0.7)	NS					Decrease	
		Weight watchers	40/40			-0.47 (SD 1.37)	0.05					Decrease	
		Zone	40/40			-0.3 (SD 0.74)	0.01					Decrease	

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	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
15812		Atkins	40/40			-0.39 (SD 0.81)	0.01		LDL:HDL cholesterol ratio	Fasting serum	1 year	Decrease	No bias
		Ornish	40/40			-0.31 (SD 0.68)	0.01					Decrease	
		Weight watchers	40/40			-0.55 (SD 1.39)	0.05					Decrease	
		Zone	40/40			-0.4 (SD 0.81)	0.01					Decrease	
(Zambon <i>et al.</i> , 1999) 16267		High carbohydrate, energy restriction	11/11	2.47 (SD 0.8)	2.24 (SD 0.5)		NS		LDL:HDL cholesterol ratio	Fasting plasma (mmol/L)	3 months		unclear
		Olive oil enriched energy restriction diet	9/9	2.32 (SD 0.7)	2.08 (SD 0.4)		NS	NS					
16268		High carbohydrate, energy restriction	5/11	2.47 (SD 0.8)	2.31 (SD 0.6)		NS		LDL:HDL cholesterol ratio	Fasting plasma (mmol/L)	6 months		unclear
		Olive oil enriched energy restriction diet	7/9	2.32 (SD 0.7)	1.59 (SD 0.5)		<0.05	<0.05					
(Clevidence <i>et al.</i> , 1992) 16611		High fat diet	42/46	2.7 (SE 0.1)	2.6 (SE 0.1)	-0.1	NS	NS	LDL:HDL cholesterol ratio		10 weeks		unclear
		Low fat diet	42/46	2.7 (SE 0.1)	2.6 (SE 0.1)	-0.1	NS						
(Pelkman <i>et al.</i> , 2004) 16885		Low fat, high carbohydrate diet	25/25	2.97 (SE 0.15)	2.71 (SE 0.15)		NS	Not reported	LDL:HDL cholesterol ratio		6 weeks		unclear
		Moderate fat, lower carbohydrate diet	27/27	3.4 (SE 0.15)	3.06 (SE 0.15)		<0.05						
16886		Low fat, high carbohydrate diet	25/25	2.97 (SE 0.15)	2.73 (SE 0.15)		NS		LDL:HDL cholesterol ratio		10 weeks		unclear

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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	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		Moderate fat, lower carbohydrate diet	27/27	3.4 (SE 0.15)	3.18 (SE 0.15)		NS						
16906	Weight stable during maintenance	Low fat, high carbohydrate diet	12/25	3.04 (SE 0.23)	2.64 (SE 0.23)		NS		LDL:HDL cholesterol ratio		6 weeks		unclear
	Weight stable during maintenance	Moderate fat, lower carbohydrate diet	17/27	3.46 (SE 0.19)	3.05 (SE 0.19)		<0.05						
16907	Weight stable during maintenance	Low fat, high carbohydrate diet	12/25	3.04 (SE 0.23)	2.83 (SE 0.23)		NS		LDL:HDL cholesterol ratio		10 weeks		unclear
	Weight stable during maintenance	Moderate fat, lower carbohydrate diet	17/27	3.46 (SE 0.19)	3.2 (SE 0.19)		<0.05						
(Petersen <i>et al.</i> , 2006) 17225		Hypoenergetic high carbohydrate, low fat diet	336/389						Change in LDL:HDL cholesterol ratio		10 weeks	Decrease	bias
		Hypoenergetic low carbohydrate, high fat diet	312/382					NS				Decrease	
(Colette <i>et al.</i> , 2003) 17414		High carbohydrate diet	15/15	3.07 (SE 0.20)	2.82 (SE 0.26)		NS		LDL:HDL cholesterol ratio	Fasting serum	8 weeks	Decrease	unclear
		High MUFA diet	17/17	2.82 (SE 0.28)	2.75 (SE 0.21)		NS	NS				Decrease	
(Claessens <i>et al.</i> , 2009) 16826		High carbohydrate supplement	16/allocated not reported	2.56 (SE 0.19)	2.73 (SE 0.16)	0.17 (SE 0.14)	NS	NS	LDL:HDL cholesterol ratio		12 weeks		unclear

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	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
		High protein supplement - casein	14/allocated not reported	2.79 (SE 0.25)	2.5 (SE 0.23)	-0.29 (SE 0.15)	<0.05	NS					
		High protein supplement - whey	18/allocated not reported	2.68 (SE 0.21)	2.77 (SE 0.25)	0.09 (SE 0.12)	NS	NS					

Triacylglycerol:HDL cholesterol ratio, total carbohydrate and high carbohydrate diets

No cohort studies reported results concerning total carbohydrate and TAG:HDL ratio.

Summary of RCT data

One randomised controlled trial of overweight and obese participants (n=130) provided data on the TAG:HDL cholesterol ratio (TAG:HDL) (Layman *et al.*, 2009). In their study, Layman *et al.* (Layman *et al.*, 2009) compared a high carbohydrate, low protein diet (55% carbohydrate, 15% protein) to a low carbohydrate, high protein diet (~40% carbohydrate, 30% protein) over a 12-month period. Food was consumed as part of a free living diet plan. At four and 12 months, both groups experienced a reduction in TAG:HDL ratio with the difference between these dietary groups being statistically significant at both time-points ($p<0.01$ and $p=0.016$, respectively). It should also be noted that participants in both the two groups experienced weight loss; therefore any changes may not necessarily be attributable to the dietary intervention alone.

Table 2.60 Triacylglycerol:HDL ratio and high carbohydrate diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Layman <i>et al.</i> , 2009) 14961	High carbohydrate, low protein diet	51/66	-0.18 (SE 0.09)	<0.01	Change in TAG:HDL ratio	Fasting plasma	4 months	Decrease	unclear
	Low carbohydrate, high protein diet	52/64	-0.3 (SE 0.1)					Decrease	
14967	High carbohydrate, low protein diet	30/66	-0.38 (SE 0.13)	0.016	Change in TAG:HDL ratio	Fasting plasma	1 year	Decrease	unclear
	Low carbohydrate, high protein diet	41/64	-0.94 (SE 0.22)					Decrease	

Apolipoproteins, total carbohydrate and high carbohydrate diets

No cohort studies reported results concerning total carbohydrate and apolipoproteins.

Summary of RCT data

Fifteen studies, reported in sixteen papers, explored the effects of dietary variation in carbohydrate diets - replacing carbohydrate with fat, protein or both - on apolipoproteins. Details of these studies can be found in the Trial Characteristics table.

Four studies employed a crossover design (Ginsberg *et al.*, 1998; Campos *et al.*, 1995; Dreon *et al.*, 1994; Furtado *et al.*, 2008) and the remaining studied parallel groups. The majority of trials were conducted in the USA (10), although studies were also carried out in Australia (2), Spain (1), Canada (1) and Italy (1).

All participants were adults (mean age ranged from 30 to 62 years), who were typically overweight or obese. In fact, only (Ginsberg *et al.*, 1998) recruited subjects with a BMI indicative of a healthy weight (mean BMI: 24kg/m²). Five trials recruited males only (Couture *et al.*, 2003; Dreon *et al.*, 1994; Lovejoy *et al.*, 2003; Krauss *et al.*, 2006; Campos *et al.*, 1995), and two used females (Zambon *et al.*, 1999; Howard *et al.*, 2006). The other eight trials were mixed gender.

Final sample sizes generally ranged from 20 to 224 (mean=108; median=117), although an exception to this is The Women's Health Initiative Dietary Modification Trial (Howard *et al.*, 2006), which had an extremely large sample size of 48835 of which 5.8% provided blood samples for lipid analysis (approx. n=2832).

The majority of these intervention trials studied the effects of interventions in which dietary carbohydrate was manipulated to compare high and low carbohydrate diets with energy replacement from both fat and protein (fat and protein difference between high and low carbohydrate diets each of 2% or more).

De Luis *et al.* (de Luis *et al.*, 2009a; de Luis *et al.*, 2008) compared the effects of energy restricted high or low carbohydrate diets (52 vs. 38% carbohydrate) that were respectively low or moderate in fat (27 vs. 36% fat) in obese men and women.

Keogh *et al.* (Keogh *et al.*, 2008) compared the effects of rather extreme manipulations in dietary carbohydrate, comparing 4% and 46% carbohydrate diets in which the carbohydrate was replaced with both fat and protein (59 and 35% respectively) in the low carbohydrate diet. Similarly, Noakes *et al.* (Noakes *et al.*, 2006) employed a three group design, comparing a very low carbohydrate

(12%), a high carbohydrate (66%), and a moderate carbohydrate (49%) diet with high unsaturated fat content for 12 weeks.

Krauss *et al.* (Krauss *et al.*, 2006) using a four group design, studied diets which varied in carbohydrate content from 54 to 26%, and in saturated fat content from 7-15%.

Lasker *et al.* (Lasker *et al.*, 2008) compared the effects of energy restricted, high carbohydrate (215g/d) and low carbohydrate (153g/d) diets in which fat and protein levels were also different (67 vs. 121g/d protein).

Layman *et al.* (Layman *et al.*, 2009) manipulated carbohydrate and fat intakes in a two group trial comparing diets in which the carbohydrate to protein ratio was either more than 3.2 or less than 1.5. Dietary fat also differed by 6%, but fibre and energy intakes were similar in both groups. The first four months of this study explored the effects of weight loss and the latter eight months the effects of weight maintenance.

Seven studies devised interventions in which diets were manipulated to compare high and low carbohydrate diets with energy replacement from only fat (with the fat difference between high and low carbohydrate diets each of 2% or more, but protein similar).

The study by Pelkman *et al.* (Pelkman *et al.*, 2004) compared diets with 64% carbohydrate or 51% carbohydrate (18 vs. 33% fat respectively).

The study by Zambon *et al.* (Zambon *et al.*, 1999) compared isoenergetic high and low carbohydrate diets (60 vs. 40%) in which the low carbohydrate diet was higher in monounsaturated fat (7 vs. 27% energy).

The primary aim of the study by Lovejoy *et al.* (Lovejoy *et al.*, 2003) was to explore the effects of Olestra, a fat substitute. However, a comparison of two of the three study groups that did not include Olestra is possible in that they compare the effects of 52 vs. 58% carbohydrate diets (33 vs. 25% energy from fat) over nine months in healthy males.

The Women's Health Initiative Study, reported by Howard *et al.* (Howard *et al.*, 2006) was a large, parallel group design study of women, in which the low fat, high carbohydrate (29% fat, 54% carbohydrate) diet group were also encouraged to consume more fruit and vegetables and more grains compared with a control (no intervention) group (37% fat, 46% carbohydrate).

In the study by Ginsberg *et al.* (Ginsberg *et al.*, 1998), the three test diets varied in carbohydrate content from 59 to 48%, fat content varied from 26 to 37%, and the saturated fat content from 16

to 5%. Thus, the comparison was between an 'average American diet', the Step 1 diet and a low saturated fat diet.

Couture *et al.* (Couture *et al.*, 2003) provided high or low carbohydrate (58 vs. 45 % carbohydrate) diets to 65 healthy overweight males, in which fat was respectively low or high (26 vs. 40%) and in the higher fat diet was higher in monounsaturated fat.

In the study reported by Campos *et al.* (Campos *et al.*, 1995) and Dreon *et al.* (Dreon *et al.*, 1994) a crossover design that compared a low-fat, high carbohydrate diet (24% fat, 60% carbohydrate) with a high-fat diet (46% fat, 38% carbohydrate) was tested in healthy males for six weeks. Results were further explored in individuals with different LDL subclass patterns.

The Omni-Heart study reported by Furtado *et al.* (Furtado *et al.*, 2008) compared three diets that differed in carbohydrate (58 vs. 48%) and in which the carbohydrate was replaced with either fat (37 vs. 27%), or protein (25 vs. 15%), in 191 overweight and obese men and women for six weeks. Thus, an exploration of carbohydrate replacement with either fat or protein was possible in this study.

Apolipoprotein A-1 and high carbohydrate diets

Six studies provided data on the effects of high vs. low carbohydrate diets on apolipoprotein A-1. A meta-analysis was not conducted since in two studies conversion of units from mmol/L to mg/dL produced values that were not consistent with the other studies reported in mg/dL. It is unclear if this represents errors in reporting or is a reflection of variation in measurement technique. Two studies found significantly higher apolipoprotein A-1 levels after a lower carbohydrate diet had been consumed (Dreon *et al.*, 1994; Ginsberg *et al.*, 1998), but three others found no differential effect of dietary carbohydrate intake (Krauss *et al.*, 2006; Lovejoy *et al.*, 2003; Pelkman *et al.*, 2004; Zambon *et al.*, 1999).

Table 2.61 Apolipoprotein A-1 and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow- up	Weight change	Outcome Assess- ment Bias
(Dreon <i>et al.</i> , 1994) 17050	LDL particles remained large during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 87/105						-11.6 (SD 2)	<0.0001	Apolipoprotein A-1	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17056	Smaller and denser LDL particles	Low-fat higher CHO minus high-fat low CHO	Crossover: 18/105						-8.8 (SD 2)	<0.01	Apolipoprotein A-1	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17070	LDL particles remained large during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 51/105						-10.8 (SD 2)	<0.001	Apolipoprotein A-1	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
17075	LDL particles changed from large to small and dense during study	Low-fat higher CHO minus high-fat low CHO	Crossover: 36/105						-12.6 (SD 2)	<0.0001	Apolipoprotein A-1	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Ginsberg <i>et al.</i> , 1998) 17252		Average American Diet	103/118		142.2 (SE 2.0)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		130.4 (SE 1.9)		<0.01							Not reported	
		Step 1 diet	103/118		135.4 (SE 2.0)		<0.01							Not reported	
17266	Men	Average American Diet	46/118		132.0 (SE 2.4)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Men	Low saturated fat diet	46/118		120.6 (SE 1.9)		<0.01							Not reported	
	Men	Step 1 diet	46/118		124.1 (SE 2.1)		<0.01							Not reported	
17267	Women	Average American Diet	57/118		150.4 (SE 2.6)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Women	Low	57/118		138.4		<0.01							Not	

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Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow- up	Weight change	Outcome Assess- ment Bias
		saturated fat diet			(SE 2.6)									reported	
	Women	Step 1 diet	57/118		114.6 (SE 2.6)			<0.01						Not reported	
17308	Black	Average American Diet	26/118		140.3 (SE 4.1)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Black	Low saturated fat diet	26/118		130.2 (SE 4.2)			<0.01						Not reported	
	Black	Step 1 diet	26/118		135.1 (SE 4.2)			NS						Not reported	
17309	Non black	Average American Diet	77/118		142.8 (SE 2.3)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Non black	Low saturated fat diet	77/118		130.5 (SE 2.1)			<0.01						Not reported	
	Non black	Step 1 diet	77/118		135.5 (SE 2.3)			<0.01						Not reported	
17324	Pre- menopausal	Average American Diet	39/118		148.3 (SE 3.0)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Pre- menopausal	Low saturated fat diet	39/118		135.3 (SE 2.8)			<0.01						Not reported	
	Pre- menopausal	Step 1 diet	39/118		142.5 (SE 3.0)			<0.01						Not reported	
17325	Post- menopausal	Average American Diet	18/118		155.1 (SE 4.9)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Post- menopausal	Low saturated fat diet	18/118		145.3 (SE 5.4)			<0.01						Not reported	
	Post- menopausal	Step 1 diet	18/118		149.1 (SE 5.0)			NS						Not reported	
17340	Men <40y	Average American Diet	30/118		134.0 (SE 3.3)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Men <40y	Low saturated fat diet	30/118		121.9 (SE 2.5)			<0.01						Not reported	

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow- up	Weight change	Outcome Assess- ment Bias
17341	Men <40y	Step 1 diet	30/118		124.5 (SE 2.6)			<0.01						Not reported	
	Men >40y	Average American Diet	16/118		128.2 (SE 3.4)						Apolipoprotein A-1	(mg/dL)	6 weeks	Not reported	No bias
	Men >40y	Low saturated fat diet	16/118		118.2 (SE 2.9)			<0.01						Not reporter	
	Men >40y	Step 1 diet	16/118		123.2 (SE 3.8)			NS						Not reported	
(Krauss <i>et al.</i> , 2006) 17484		26% CHO High saturated fat	40/52	111.2 (SD 14.3)		0.8 (SE 1.7)		NS			Apolipoprotein A-1	Fasting plasma (mg/dL)	12 weeks	Decrease	unclear
		26% CHO Low saturated fat	47/59	111.0 (SD 16.4)		2.9 (SE 1.9)		NS						Decrease	
		39% CHO Low saturated fat	42/56	114.0 (SD 15.5)		-0.8 (SE 1.5)		NS						Decrease	
		54% CHO Low saturated fat	49/57	113.8 (SD 15.8)		-0.9 (SE 1.5)								Decrease	
(Lovejoy <i>et al.</i> , 2003) 14988		Control	13/15	1.08 (SE 0.03)		0.05 (SE 0.03)					Apolipoprotein A-1	Fasting (g/L)	3 months	Decrease	unclear
		Fat reduced	13/15	1.12 (SE 0.05)		0.06 (SE 0.02)		NS						Decrease	
14990		Control	13/15	1.08 (SE 0.03)		0.09 (SE 0.02)					Apolipoprotein A-1	Fasting (g/L)	6 months	Decrease	unclear
		Fat reduced	13/15	1.12 (SE 0.05)		0.07 (SE 0.04)		NS						Decrease	
14992		Control	13/15	1.08 (SE 0.03)		0.02 (SE 0.02)					Apolipoprotein A-1	Fasting (g/L)	9 months	Decrease	unclear
		Fat reduced	13/15	1.12		0.02 (SE		NS						Decrease	

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Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow- up	Weight change	Outcome Assess- ment Bias
				(SE 0.05)		0.03)									
(Pelkman <i>et al.</i> , 2004) 16891		Low fat, high carbohydrate diet	25/25	5.42 (SE 0.18)	4.88 (SE 0.18)		<0.05	Not reported/ unclear			Apolipoprotein A-1	(mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	5.14 (SE 0.17)	4.76 (SE 0.17)		<0.05							Decrease	
16892		Low fat, high carbohydrate diet	25/25	5.42 (SE 0.18)	5.13 (SE 0.18)		<0.05				Apolipoprotein A-1	(mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	27/27	5.14 (SE 0.17)	4.99 (SE 0.17)		NS							Decrease	
16910	Weight stable during maintenance	Low fat, high carbohydrate diet	12/25	5.35 (SE 0.21)	4.98 (SE 0.21)		NS				Apolipoprotein A-1	(mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	17/27	5.34 (SE 0.17)	4.92 (SE 0.17)		<0.05							Decrease	
16911	Weight stable during maintenance	Low fat, high carbohydrate diet	12/25	5.35 (SE 0.21)	5.29 (SE 0.21)		NS				Apolipoprotein A-1	(mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carbohydrate diet	17/27	5.34 (SE 0.17)	5.17 (SE 0.17)		NS							Decrease	
(Zambon <i>et al.</i> , 1999) 16271		High carbohydrate, energy restriction	11/11	1.62 (SD 0.22)	1.49 (SD 0.23)		NS				Apolipoprotein A-1	Fasting plasma (mmol/L)	3 months	Decrease	unclear
		Olive oil enriched energy restriction diet	9/9	1.55 (SD 0.16)	1.6 (SD 0.27)		NS	NS						Decrease	

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Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow- up	Weight change	Outcome Assess- ment Bias
16272	High carbohydrate, energy restriction	5/11	1.62 (SD 0.22)	1.47 (SD 0.27)			NS				Apolipoprotein A-1	Fasting plasma (mmol/L)	6 months	Decrease	unclear
	Olive oil enriched energy restriction diet	7/9	1.55 (SD 0.16)	1.64 (SD 0.21)			NS	NS						Decrease	

Apolipoprotein B and high carbohydrate diets

Thirteen papers from 12 studies were identified that reported information concerning the effects of high carbohydrate diets on apolipoprotein B levels.

Studies reported results in g/L, mg/dL or mmol/L. Five studies reported results in mmol/L (Zambon *et al.*, 1999; Pelkman *et al.*, 2004; Noakes *et al.*, 2006; Keogh *et al.*, 2008; Lasker *et al.*, 2008). On transformation to mg/dL the results from these studies were not compatible with those reported as mg/dL. Accordingly, a meta-analysis was deemed to be inappropriate.

The majority of these intervention trials studied the effects of interventions in which dietary carbohydrate was manipulated to compare high and low carbohydrate diets with energy replacement from both fat and protein (fat and protein difference between high and low carbohydrate diets each of 2% or more). All studies included adults as participants. The duration of the studies varied from six weeks to nine months.

Most of these studies found no significant differential effect of high compared with low carbohydrate diets (Keogh *et al.*, 2008; Noakes *et al.*, 2006; Lasker *et al.*, 2008; Layman *et al.*, 2009). However, Krauss *et al.* (Krauss *et al.*, 2006) using a four group design, found that the 26% carbohydrate, low saturated fat diet significantly reduced apolipoprotein B compared with the 54% carbohydrate, low saturated fat diet. This was also the case after post-hoc adjustment for differences in weight loss.

The study by Dreon *et al.* (Dreon *et al.*, 1994) found a differential response to high carbohydrate, low fat and low carbohydrate, high fat diets in individuals with different LDL subclass patterns. In those with smaller and denser LDL particles, a significant reduction in apolipoprotein B levels was observed with the high carbohydrate diet. The individuals with the alternative LDL subclass pattern characterised by large LDL particles were less responsive. Papers from Campos *et al.* (Campos *et al.*, 1995) and Dreon *et al.* (Dreon *et al.*, 1994) are from same study, but both are included in the table of results.

Five studies devised interventions in which diets were manipulated to compare high and low carbohydrate diets with energy replacement from only fat (fat difference between high and low carbohydrate diets each of 2% or more, but protein similar).

The study by Pelkman *et al.* (Pelkman *et al.*, 2004) compared diets with 64% carbohydrate or 51% carbohydrate (18 vs. 33% fat respectively). Changes in apolipoprotein B were similar to those of LDL cholesterol: both diet groups experienced a decrease in levels, but there was no difference between the diets. The studies by Lovejoy *et al.* (Lovejoy *et al.*, 2003) and by Couture *et al.* (Couture *et al.*, 2003) similarly found no effect of differences in carbohydrate.

The study by Zambon *et al.* (Zambon *et al.*, 1999) compared isoenergetic high and low carbohydrate diets (60 vs. 40%) in which the low carbohydrate diet was higher in monounsaturated fat (7 vs. 27% energy). At six months, apolipoprotein B levels were lower on the lower carbohydrate diet than the high carbohydrate diet ($p < 0.05$).

In the study by Ginsberg *et al.* (Ginsberg *et al.*, 1998), apolipoprotein B levels decreased as the subjects went from the 'average American diet' to the Step 1 diet, and they were further reduced on the low saturated fat diet (carbohydrate percentages 48, 55 and 59% respectively).

The Omni-Heart study reported by Furtado *et al.* (Furtado *et al.*, 2008) compared three diets that differed in carbohydrate (58 vs. 48%) and in which the carbohydrate was replaced with either fat (37 vs. 27%), or protein (25 vs. 15%), in 191 overweight and obese men and women for six weeks. Thus, an exploration of carbohydrate replacement with either fat or protein was possible in this study. Apolipoprotein B levels decreased to the greatest extent on the lower carbohydrate, high protein diet although differences between diet groups were not statistically significant.

Overall, most studies do not report a differential effect of higher carbohydrate and lower carbohydrate diets. However, there is some variation in outcome and this is likely to be due to the nature of the comparison diet and the extent of weight change within each intervention.

Table 2.62 Apolipoprotein B and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias
(Campos <i>et al.</i> , 1995) 17092		High-fat minus low- fat higher CHO	Crossover: 43/ allocated not reported						4 (SD 14)		0.06	Apolipoprotein B	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Couture <i>et al.</i> , 2003) 15885	Genetics - Apo E genotype E3/E2	High carb diet	3/3	0.69 (SD 0.2)	0.52 (SD 0.03)		0.3	Not reported/ unclear				Apolipoprotein B	Fasting plasma (g/L)	6 weeks	Decrease	No bias
		High MUFA diet	4/4	0.65 (SD 0.12)	0.56 (SD 0.1)		0.07								Decrease	
15886	Genetics - Apo E genotype E3/E3	High carb diet	22/22	0.82 (SD 0.25)	0.72 (SD 0.22)		<0.01					Apolipoprotein B	Fasting plasma (g/L)	6 weeks	Decrease	No bias
		High MUFA diet	21/21	0.96 (SD 0.19)	0.82 (SD 0.16)		<0.01								Decrease	
15887	Genetics - Apo E genotype E3/E4	High carb diet	8/8	0.96 (SD 0.26)	0.75 (SD 0.17)		<0.01					Apolipoprotein B	Fasting plasma (g/L)	6 weeks	Decrease	No bias
		High MUFA diet	6/6	0.94 (SD 0.27)	0.79 (SD 0.23)		<0.01								Decrease	
(Dreon <i>et al.</i> , 1994) 17051	LDL particles remained large during study	Low-fat higher CARB minus high- fat low CARB	Crossover: 87/105						1.1 (SD 1)			Apolipoprotein B	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Dreon <i>et al.</i> , 1994)1705 7	Smaller and denser LDL particles	Low-fat higher carb minus high- fat low carb	Crossover: 18/105						-11.6 (SD 3)		<0.001	Apolipoprotein B	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Dreon <i>et al.</i> , 1994)1707 1	LDL particles remained large during study	Low-fat higher carb minus high- fat low carb	Crossover: 51/105						0.1 (SD 1.8)			Apolipoprotein B	Fasting plasma (mg/dL)	6 weeks	Both not reported	unclear
(Dreon <i>et al.</i> , 1707)	LDL particles changed from	Low-fat higher carb	Crossover: 36/105						2.5 (SD 2)			Apolipoprotein B	Fasting plasma	6 weeks	Both not reported	unclear

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1994)1707 7	large to small and dense during study	minus high- fat low carb											(mg/dL)			
(Furtado <i>et al.</i> , 2008) 16336		High carb	88/164	83 (SD 28)		-4.9 (SD 23)	0.05					Apolipoprotein B	Fasting serum (mg/dL)	6 weeks	No change	No bias
		High protein	88/164	83 (SD 28)		-8.2 (SD 22)	0.01	0.1							No change	
		High PUFA	88/164	83 (SD 28)		-6.1 (SD 21)	0.01	0.6							No change	
16337		High protein minus high carb	Crossover: 88/164							-3.3 (SD 21)	0.1	Apolipoprotein B	Fasting serum (mg/dL)	6 weeks	No change in both	No bias
16338		High PUFA minus high carb	Crossover: 88/164							-1.2 (SD 21)	0.6	Apolipoprotein B	Fasting serum (mg/dL)	6 weeks	No change in both	No bias
(Ginsberg <i>et al.</i> , 1998) 17251		Average American Diet	103/118		116.8 (SE 2.4)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		111.6 (SE 2.6)			<0.01							Not reported	
		Step 1 diet	103/118		113.6 (SE 2.6)			NS							Not reported	
(Ginsberg <i>et al.</i> , 1998) 17264	Men	Average American Diet	46/118		121.4 (SE 3.8)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
	Men	Low saturated fat diet	46/118		116.7 (SE 4.0)			<0.01							Not reported	
	Men	Step 1 diet	46/118		117.6 (SE 4.0)			NS							Not reported	
17265	Women	Average American Diet	57/118		113.1 (SE 3.0)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
	Women	Low saturated fat diet	57/118		107.5 (SE 3.4)			<0.01							Not reported	
	Women	Step 1 diet	57/118		110.3 (SE 3.4)			NS							Not reported	
(Ginsberg	Black	Average	26/118		112.9							Apolipoprotein	(mg/dL)	6	Not	No bias

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<i>et al.</i> , 1998) 17306		American Diet			(SE 4.4)							B		weeks	reported	
	Black	Low saturated fat diet	26/118		106.4 (SE 5.3)			<0.01								Not reported
	Black	Step 1 diet	26/118		109.9 (SE 4.9)			NS								Not reported
17307	Non black	Average American Diet	77/118		118.1 (SD 2.8)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
	Non black	Low saturated fat diet	77/118		113.4 (SD 3.0)			<0.01								Not reported
	Non black	Step 1 diet	77/118		114.8 (SD 3.1)			NS								Not reported
(Ginsberg <i>et al.</i> , 1998) 17322	Pre- menopausal	Average American Diet	39/118		104.7 (SE 2.8)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
	Pre- menopausal	Low saturated fat diet	39/118		107.5 (SE 3.4)			<0.01								Not reported
	Pre- menopausal	Step 1 diet	39/118		101.0 (SE 3.0)			NS								Not reported
17323	Post- menopausal	Average American Diet	18/118		131.3 (SE 5)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
	Post- menopausal	Low saturated fat diet	18/118		129.2 (SE 6.6)			NS								Not reported
	Post- menopausal	Step 1 diet	18/118		130.6 (SE 6.5)			NS								Not reported
(Ginsberg <i>et al.</i> , 1998) 17338	Men <40y	Average American Diet	30/118		111.6 (SE 4.0)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias
	Men <40y	Low saturated fat diet	30/118		106.1 (SE 4.3)			<0.01								Not reported
	Men <40y	Step 1 diet	30/118		107.1 (SE 4.1)			NS								Not reported
17339	Men >40y	Average American	16/118		139.8 (SE 5.5)							Apolipoprotein B	(mg/dL)	6 weeks	Not reported	No bias

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias
		Diet														
	Men >40y	Low saturated fat diet	16/118		136.7 (SE 5.6)			NS								Not reported
	Men >40y	Step 1 diet	16/118		137.4 (SE 5.9)			NS								Not reported
(Keogh <i>et al.</i> , 2008) 16724		High carb, low SFA	47/50	0.99 (SD 0.19)	0.89 (SD 0.2)		Main effect of time <0.001	NS				Apolipoprotein B	Fasting (mmol/L)	8 weeks	Decrease	unclear
		Low carb, high SFA	52/57	0.97 (SD 0.26)	0.90 (SD 0.28)											Decrease
(Krauss <i>et al.</i> , 2006) 17482		26% carb High saturated fat	40/52	104.2 (SD 24.7)		-12.5 (SE 2.1)		NS				Apolipoprotein B	Fasting plasma (mg/dL)	12 weeks	Decrease	unclear
		26% carb Low saturated fat	47/59	100.0 (SD 21.2)		-15.8 (SE 1.9)		<0.01		0.04 within low SAFA groups						Decrease
		39% carb Low saturated fat	42/56	102.6 (SD 18.4)		-9.5 (SE 1.8)		NS								Decrease
		54% carb Low saturated fat	49/57	102.3 (SD 21.7)		-4.9 (SE 2.0)										Decrease
(Lasker <i>et al.</i> , 2008) 15909		High carb	25/33			-0.33 (SE 0.1)		NS				Apolipoprotein B	Fasting plasma (mmol/L)	4 months	Decrease	unclear
		High protein	25/32			-0.41 (SE 0.12)		NS	0.61							Decrease
(Layman <i>et al.</i> , 2009) 14962		High carb, low protein diet	51/66									Apolipoprotein B	Fasting plasma	4 months	Decrease	unclear
		Low carb, high protein diet	52/64					NS								Decrease
14963		High carb, low protein diet	30/66									Apolipoprotein B	Fasting plasma	1 year	Decrease	unclear

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias	
		Low carb, high protein diet	41/64					NS								Decrease	
(Lovejoy <i>et al.</i> , 2003) 14993	Control		13/15	0.93 (SE 0.05)		-0.02 (SE 0.03)						Apolipoprotein B	Fasting (g/L)	3 months		Decrease	unclear
	Fat reduced		13/15	0.89 (SE 0.06)		0.04 (SE 0.03)		NS								Decrease	
(Lovejoy <i>et al.</i> , 2003) 14994	Control		13/15	0.93 (SE 0.05)		0.03 (SE 0.04)						Apolipoprotein B	Fasting (g/L)	6 months		Decrease	unclear
	Fat reduced		13/15	0.89 (SE 0.06)		0.07 (SE 0.03)		NS								Decrease	
14995	Control		13/15	0.93 (SE 0.05)		0.11 (SE 0.04)						Apolipoprotein B	Fasting (g/L)	9 months		Decrease	unclear
	Fat reduced		13/15	0.89 (SE 0.06)		0.13 (SE 0.02)		NS								Decrease	
(Noakes <i>et al.</i> , 2006) 16578	High unsaturated fat		21/27	1.05 (SE 0.06)	0.93 (SE 0.06)							Apolipoprotein B	Fasting plasma (mmol/L)	8 weeks		Decrease	unclear
	Very low carb		24/28	1.01 (SE 0.05)	0.94 (SE 0.05)											Decrease	
	Very low fat		22/28	0.97 (SE 0.05)	0.85 (SE 0.05)											Decrease	
16579	High unsaturated fat		21/27	1.05 (SE 0.06)	0.99 (SE 0.05)	-0.06 (SE 0.02)				0.42		Apolipoprotein B	Fasting plasma (mmol/L)	12 weeks		Decrease	unclear
	Very low carb		24/28	1.01 (SE 0.05)	1.00 (SE 0.05)	-0.02 (SE 0.05)										Decrease	
	Very low fat		22/28	0.97 (SE 0.05)	0.89 (SE 0.06)	-0.07 (SE 0.02)										Decrease	
(Pelkman <i>et al.</i> , 2004)	Low fat, high carb diet		25/25	1.95 (SE 0.08)	1.58 (SE 0.08)		<0.05					Apolipoprotein B	(mmol/L)	6 weeks		Decrease	unclear

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16893		Moderate fat, lower carb diet	27/27	2.0 (SE 0.08)	1.67 (SE 0.08)		<0.05									Decrease	
(Pelkman <i>et al.</i> , 2004)		Low fat, high carb diet	25/25	1.95 (SE 0.08)	1.65 (SE 0.08)		<0.05					Apolipoprotein B	(mmol/L)	10 weeks		Decrease	unclear
16894		Moderate fat, lower carb diet	27/27	2.0 (SE 0.08)	1.74 (SE 0.08)		<0.05									Decrease	
(Pelkman <i>et al.</i> , 2004)	Weight stable during maintenance	Low fat, high carb diet	12/25	2.03 (SE 0.12)	1.59 (SE 0.12)		<0.05					Apolipoprotein B	(mmol/L)	6 weeks		Decrease	unclear
16912		Moderate fat, lower carb diet	17/27	2.06 (SE 0.10)	1.72 (SE 0.10)		<0.05									Decrease	
(Pelkman <i>et al.</i> , 2004)	Weight stable during maintenance	Low fat, high carb diet	12/25	2.03 (SE 0.12)	1.76 (SE 0.12)		<0.05					Apolipoprotein B	(mmol/L)	10 weeks		Decrease	unclear
16913		Moderate fat, lower carb diet	17/27	2.06 (SE 0.10)	1.77 (SE 0.10)		<0.05									Decrease	
(Zambon <i>et al.</i> , 1999)		High carb, energy restriction	11/11	1.17 (SD 0.21)	1.08 (SD 0.22)		NS					Apolipoprotein B	Fasting plasma (mmol/L)	3 months		Decrease	unclear
16269		Olive oil enriched energy restriction diet	9/9	1.10 (SD 0.19)	1.08 (SD 0.39)		NS	NS								Decrease	
(Zambon <i>et al.</i> , 1999)		High carb, energy restriction	5/11	1.17 (SD 0.21)	1.03 (SD 0.14)		NS					Apolipoprotein B	Fasting plasma (mmol/L)	6 months		Decrease	unclear
16270		Olive oil enriched energy restriction diet	7/9	1.10 (SD 0.19)	0.85 (SD 0.17)		<0.05	<0.05								Decrease	

Lipoprotein (a) and high carbohydrate diets

Five studies reported data on the effects of variation in dietary carbohydrate on lipoprotein (a). A meta-analysis was not conducted since there were concerns about pooling data for an outcome in which there appeared to be inconsistencies in response when comparing data reported as mmol/L or mg/dL. It is unclear if this represents errors in reporting or is a reflection of variation in the measurement approach.

All studies included adults as participants. The duration of the trials varied from six weeks to three years. A description of the trials is included in the Trials Characteristics table (Table 2.43), and also at the beginning of this section on lipoproteins. In the Women's Health Initiative Study (Howard *et al.*, 2006), both dietary groups experienced a decline in lipoprotein (a) levels at the three year follow up point. However, there was no differential effect of these diets on this outcome. Similarly, the study by Pelkman *et al.* (Pelkman *et al.*, 2004) found no differential impact of dietary carbohydrate on lipoprotein (a) in their 10 week study which compared diets with 64% carbohydrate or 51% carbohydrate (18 vs. 33% fat respectively).

In the study by Ginsberg *et al.* (Ginsberg *et al.*, 1998), the three test diets varied in carbohydrate content from 59 to 48%, fat content varied from 26 to 37%, and the saturated fat content from 16 to 5%. Thus, the comparison was between an 'average American diet', the Step 1 diet and a low saturated fat diet. Compared with the 'average American diet' group, the other groups, with a higher carbohydrate intake, both had significantly higher levels of lipoprotein (a) after six weeks.

Two papers (de Luis *et al.*, 2009a; de Luis *et al.*, 2008) explored the impact of high compared with low carbohydrate energy restricted diets on lipoprotein (a) in individuals with different genetic profiles. Changes in lipoprotein (a) were similar in both diet groups overall, in individuals with different polymorphisms of the fatty acid binding protein 2 (FABP2) gene (de Luis *et al.*, 2008), and in individuals with different polymorphisms of the uncoupling protein-3 gene (a gene with influence on energy expenditure and fat storage) (de Luis *et al.*, 2009a).

Overall, the studies are inconsistent in terms of the direction of effect of high carbohydrate diets.

Table 2.63 Lipoprotein (a) and high carbohydrate diets: RCT data

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias
(de Luis <i>et al.</i> , 2008) 16185	Genetics - mutant-type Ala54/Thr54 or Thr54/Thr54	Low carb	50/105	33.7 (SD 40.3)	32.8 (SD 35.4)			NS				Lipoprotein (a)	Fasting (mg/dL)	2 month s	Decrease	unclear
		Low fat	44/99	33.9 (SD 40.3)	52.1 (SD 45.4)										Decrease	
16186	Genetics - wild-type Ala54/Ala54	Low carb	50/105	28.1 (SD 32.1)	26.7 (SD 30.4)			NS				Lipoprotein (a)	Fasting (mg/dL)	2 month s	Decrease	unclear
		Low fat	44/99	34.1 (SD 38)	40.7 (SD 50.4)										Decrease	
(de Luis <i>et al.</i> , 2009a) 16703	Genetics - UCP3 Gene - 55CC polymorphis m	Low carb	54/67	44.3 (SD32. 1)	45.9 (SD 30.4)		NS	Unclear				Lipoprotein (a)	(mg/dL)	2 month s	Decrease	unclear
		Low fat	40/64	27.4 (SD 37.0)	23.3 (SD 33.0)		NS								Decrease	
16704	Genetics - UCP3 Gene - 55CT/TT polymorphis m	Low carb	13/67	47.6 (SD 40.3)	49.3 (SD 35.4)		NS					Lipoprotein (a)	(mg/dL)	2 month s	Decrease	unclear
		Low fat	24/64	37.9 (SD 40.3)	39.1 (SD 45.4)		NS								Decrease	
(Ginsberg <i>et al.</i> , 1998) 17253		Average American Diet	103/118		15.5 (SE 1.8)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	103/118		18.2 (SE 1.9)			<0.01							Not reported	
		Step 1 diet	103/118		17.0 (SE 1.8)			<0.01							Not reported	
17268	Men	Average American Diet	46/118		11.3 (SE 2.0)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	46/118		14.4 (SE 2.5)			<0.01							Not reported	
		Step 1 diet	46/118		12.8			<0.01							Not	

Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias
					(SE 2.3)											reported
17270	Women	Average American Diet	57/118		19.0 (SE 2.6)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	57/118		21.5 (SE 2.9)			<0.01								Not reported
		Step 1 diet	57/118		20.3 (SE 2.7)			<0.01								Not reported
17310	Black	Average American Diet	26/118		24.3 (SE 3.6)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	26/118		28.5 (SE 4.0)			<0.01								Not reported
		Step 1 diet	26/118		26.6 (SE 3.9)			<0.01								Not reported
17311	Non black	Average American Diet	77/118		12.7 (SE 1.8)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	77/118		14.4 (SE 2.5)			<0.01								Not reported
		Step 1 diet	77/118		13.9 (SE 1.9)			<0.01								Not reported
17326	Pre- menopausal	Average American Diet	39/118		20.2 (SE 3.5)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	39/118		22.9 (SE 3.9)			<0.01								Not reported
		Step 1 diet	39/118		21.6 (SE 3.7)			<0.01								Not reported
17327	Post- menopausal	Average American Diet	18/118		16.6 (SE 3.6)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	18/118		18.74 (SE 4.0)			<0.01								Not reported
		Step 1 diet	18/118		17.6 (SE 3.6)			NS								Not reported
17342	Men <40y	Average	30/118		7.9 (SE							Lipoprotein (a)	(mg/dL)	6	Not	No bias

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Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias
		American Diet			1.7)									weeks	reported	
		Low saturated fat diet	30/118		10.4 (SE 2.2)			<0.01								Not reported
		Step 1 diet	30/118		9.4 (SE 2.0)			<0.01								Not reported
17343	Men >40y	Average American Diet	16/118		19.3 (SE 5.2)							Lipoprotein (a)	(mg/dL)	6 weeks	Not reported	No bias
		Low saturated fat diet	16/118		23.3 (SE 5.9)			<0.01								Not reported
		Step 1 diet	16/118		20.4 (SE 5.6)			NS								Not reported
(Howard <i>et al.</i> , 2006) 16255		Control	approx n=1699 (5.8% sub-sample of 29294)	15.4 (SD 17.0)	13.8 (SD 15.5)	0.9 (SD 0.5)						Lipoprotein (a)	Fasting (mg/dL)	3 years	No change	No bias
		Low fat	approx n=1132 (5.8% sub-sample of 19541)	15.7 (SD 17.5)	13.2 (SD 15.1)	0.9 (SD 0.5)		NS								Decrease
17618		Low fat minus control	As above							-0.01 (CI - 0.07, 0.05)	NS	Lipoprotein (a)	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Pelkman <i>et al.</i> , 2004) 16895		Low fat, high carb diet	25/25	19.6 (SE 3.4)	16.4 (SE 3.4)			NS				Lipoprotein (a)	(g/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carb diet	27/27	21.6 (SE 3.6)	20.6 (SE 3.6)			NS								Decrease
16896		Low fat, high carb diet	25/25	19.6 (SE 3.4)	17.7 (SE 3.4)			NS				Lipoprotein (a)	(g/L)	10 weeks	Decrease	unclear
		Moderate	27/27	21.6	23.9			NS								Decrease

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Author/ Result ID	Subgroup detail	Trial groups	Completers / Allocated	Base- line	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value diff. between groups	Outcome/ Assessment method	Outcome details	Follow -up	Weight change	Outcome Assess- ment Bias
		fat, lower carb diet		(SE 3.6)	(SE 3.6)											
16914	Weight stable during maintenance	Low fat, high carb diet	12/25	23.2 (SE 5.4)	19.4 (SE 5.4)		NS					Lipoprotein (a)	(mmol/L)	6 weeks	Decrease	unclear
		Moderate fat, lower carb diet	17/27	18.3 (SE 4.9)	16.1 (SE 4.9)		NS								Decrease	
16915	Weight stable during maintenance	Low fat, high carb diet	12/25	23.2 (SE 5.4)	19.1 (SE 5.4)		NS					Lipoprotein (a)	(mmol/L)	10 weeks	Decrease	unclear
		Moderate fat, lower carb diet	17/27	18.3 (SE 4.9)	19.5 (SE 4.9)		NS								Decrease	

Results – Carbohydrate supplements

Total cholesterol and carbohydrate supplements

No cohort studies reported results concerning carbohydrate supplements and total cholesterol.

Summary of RCT data

One study provided data on the effects of carbohydrate supplementation on total cholesterol levels (Pasman *et al.*, 1997b; Peterson and Jovanovic-Peterson, 1995). Weight increased in both diet groups in the study by Pasman *et al.* (Pasman *et al.*, 1997b).

Pasman *et al.* (Pasman *et al.*, 1997b) randomly assigned obese female subjects (n=33) to three treatments designed to test the effects of a supplement containing carbohydrate, chromium, dietary fibre and caffeine, a supplement containing 50g plain carbohydrate (42% glucose and 58% maltodextrins) and a diet without supplementation. The latter two arms are the comparison groups of interest here. The 50g carbohydrate supplement was dissolved in water and consumed once daily in replacement of a habitual afternoon drink (the supplement may therefore be viewed as providing additional carbohydrate relative to habitual intake). Total cholesterol, measured at two, eight and 14 months, was not different at any time between the supplement group and control group in this study.

Table 2.64 Total cholesterol and carbohydrate supplements: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value diff. between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Pasman <i>et al.</i> , 1997b) 15475	Carbohydrate	11/11	4.3 (SD 1.0)	4.8 (SD 1.0)	NS	Total cholesterol	Fasting (mmol/L)	2 months	Increase	unclear
	Control	9/9	4.9 (SD 0.5)	5.3 (SD 0.5)					Increase	
15477	Carbohydrate	11/11	4.3 (SD 1.0)	4.9 (SD 1.0)	NS	Total cholesterol	Fasting (mmol/L)	8 months	Increase	unclear
	Control	9/9	4.9 (SD 0.5)	5.9 (SD 1.0)					Increase	
15478	Carbohydrate	11/11	4.3 (SD 1.0)	5.0 (SD 1.0)	NS	Total cholesterol	Fasting (mmol/L)	14 months	Increase	unclear
	Control	9/9	4.9 (SD 0.5)	6.0 (SD 0.8)					Increase	

HDL cholesterol and carbohydrate supplements

No cohort studies reported results concerning carbohydrate supplements and HDL cholesterol.

Summary of RCT data

One study provided data on the effects of carbohydrate supplementation on HDL cholesterol (Pasman *et al.*, 1997b). The results from this study can be found in Table 2.65.

HDL cholesterol, measured at two, eight and 14 months in the study reported by Pasman *et al.* (Pasman *et al.*, 1997b), was not found to statistically significantly differ between the supplement group and control group. Body weights were unchanged throughout the trial.

Table 2.65 HDL cholesterol and carbohydrate supplement: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Pasman <i>et al.</i> , 1997b) 15479	Carbohydrate	11/11	1.1 (SD 0.3)	1.4 (SD 0.2)	NS	HDL-C	(mmol/L)	2 months	Increase	unclear
	Control	9/9	1.4 (SD 0.3)	1.4 (SD 0.3)					Increase	
15480	Carbohydrate	11/11	1.1 (SD 0.3)	1.6 (SD 0.4)	NS	HDL-C	Fasting (mmol/L)	8 months	Increase	unclear
	Control	9/9	1.4 (SD 0.3)	1.6 (SD 0.3)					Increase	
15481	Carbohydrate	11/11	1.1 (SD 0.3)	1.4 (SD 0.2)	NS	HDL-C	Fasting (mmol/L)	14 months	Increase	unclear
	Control	9/9	1.4 (SD 0.3)	1.6 (SD 0.4)					Increase	

LDL cholesterol and carbohydrate supplements

No cohort studies reported results concerning carbohydrate supplements and LDL cholesterol.

Summary of RCT data

One randomised double blind trial, which explored the effects of a 50g carbohydrate supplement (42% glucose; 58% maltodextrins) compared with a control (no intervention) on maintenance of weight loss (Pasman *et al.*, 1997b), indicated a slight increase in body weight and LDL cholesterol levels in both the supplement and control groups from baseline. However the differences in LDL cholesterol were not statistically significant.

Table 2.66 LDL cholesterol and carbohydrate supplements: RCT data

Author/ Result ID	Intervention groups	Completers / Allocated	Baseline	Follow- up	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Pasman <i>et al.</i> , 1997b) 15482	Carbohydrate	11/11	2.8 (SD 0.8)	3.1 (SD 0.9)	NS	LDL-C	Fasting (mmol/L)	2 months	Increase	unclear
	Control	9/9	3.1 (SD 0.4)	3.5 (SD 0.6)		LDL-C			Increase	
15483	Carbohydrate	11/11	2.8 (SD 0.8)	3.1 (SD 0.9)	NS	LDL-C	Fasting (mmol/L)	8 months	Increase	unclear
	Control	9/9	3.1 (SD 0.4)	3.9 (SD 0.8)		LDL-C			Increase	
15484	Carbohydrate	11/11	2.8 (SD 0.8)	3.3 (SD 1.0)	NS	LDL-C	Fasting (mmol/L)	14 months	Increase	unclear
	Control	9/9	3.1 (SD 0.4)	3.8 (SD 0.6)		LDL-C			Increase	

Results – Dietary sugars and high sugars diets

Total cholesterol, dietary sugars and high sugars diets

Summary of cohort results

One publication from the National Heart, Lung and Blood Institute Growth and Health Study, which followed girls aged 9-10 years at baseline, explored the effect of sugar from breakfast cereals on total blood cholesterol (Albertson *et al.*, 2009). A small non-significant increase in total cholesterol with each percentage increase of sugar in consumed cereals was observed. This study presented unadjusted results.

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

Summary of RCT data

One study compared the effects of fructose and glucose on total cholesterol (Bantle *et al.*, 2000). In this study, subjects (n=24) were randomly assigned to two isoenergetic diets: a high fructose diet (17% energy) or a high glucose diet which was comprised of popular foods and the addition of crystalline fructose or crystalline glucose (3% of total energy from fructose), respectively. Overall, no differences between the high fructose diet group and the high glucose diet group were observed (p=0.169). This trial therefore suggests no change in total cholesterol with added fructose consumption in the context of an isoenergetic diet.

Six studies provided data from studies that compared higher sugars diets with lower sugars intakes (Poppitt *et al.*, 2002; Saris *et al.*, 2000; Surwit *et al.*, 1997; Drummond *et al.*, 2003; Ryle *et al.*, 1990; Black *et al.*, 2006). Quantitative data were not reported by Drummond *et al.* (Drummond *et al.*, 2003) and, as a result, data from this study could not be included in a meta-analysis. One further study was not included in the meta-analysis since it did not report any measures of variation in total cholesterol (Poppitt *et al.*, 2002).

Body weights were unchanged or not reported in the majority of trials but decreased in one group (low fat high complex carbohydrate group) in the study by Poppitt *et al.* (Poppitt *et al.*, 2002) and in two groups (low fat high simple and low fat high complex carbohydrate dietary groups) in the study by Saris *et al.* (Saris *et al.*, 2000). Both groups in the study by Surwit *et al.* experienced weight loss (Surwit *et al.*, 1997). Efforts were made to maintain stable body weights in the study by Black *et al.* (Black *et al.*, 2006) by careful manipulation of the energy content of the diets. Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

Thirteen subjects in the trial reported by Poppitt *et al.* (Poppitt *et al.*, 2002) (n=46) were participants in the CARMEN study reported by Saris *et al.* (Saris *et al.*, 2000) (n=398), so there is an overlap of subjects between these two trials. Four out of five studies employed a parallel group design: Drummond *et al.* (Drummond *et al.*, 2003) compared dietary advice to reduce fat or reduce both dietary fat and sugar whereas Surwit *et al.* (Surwit *et al.*, 1997) investigated the effects of a hypoenergetic low fat, high sucrose diet and a hypoenergetic low fat, low sucrose diet. Studies by Poppitt *et al.* (Poppitt *et al.*, 2002) and Saris *et al.* (Saris *et al.*, 2000) (the CARMEN study) were similar in that they compared a control group (diet representative of the national average – 48% carbohydrate), a high “complex carbohydrate” (49% carbohydrate - polysaccharides), low fat diet and a high simple carbohydrate, low fat diet. Black *et al.* (Black *et al.*, 2006), on the other hand, used a crossover design to compare a high and low sucrose diet, comparable to Surwit *et al.* (Surwit *et al.*, 1997). Trial durations ranged from six weeks to six months. The trials by Drummond *et al.* (Drummond *et al.*, 2003) and Black *et al.* (Black *et al.*, 2006) included males only, whereas Surwit *et al.* (Surwit *et al.*, 1997) recruited only females. The remaining two studies were mixed gender (Poppitt *et al.*, 2002; Saris *et al.*, 2000). All participants were adults and tended to be overweight or obese, with the exception of the study by (Drummond *et al.*, 2003) in which BMI was not reported. Participants in this latter study however had elevated blood cholesterol levels.

Drummond *et al.* (Drummond *et al.*, 2003) explored the effects of dietary advice to reduce dietary fat or reduce both dietary fat and sugar over 12 weeks in 25 men. Subjects were alternatively assigned to receive advice to reduce foods high in fat and replace with high carbohydrate foods or receive advice to reduce fat and sugar in their diet. Quantitative data were not reported, but the authors concluded that total cholesterol in both groups had not statistically significantly altered from baseline. However, compliance to both types of dietary advice was poor.

In Black *et al.* (Black *et al.*, 2006) 13 healthy male subjects were assigned to a eucaloric high (25% of total energy intake) or low (10% of total energy intake) sucrose diet. The intervention was achieved through the provision of all appropriate foodstuffs, with each diet being followed for six weeks with a four week washout period between phases. After six weeks, the authors reported a statistically significant difference in total cholesterol comparing the high sucrose diet with the low sucrose diet ($p<0.01$). In the low sucrose group, there was a small decrease, but in the high sucrose group it increased slightly.

Surwit *et al.* (Surwit *et al.*, 1997) conducted a six week weight loss trial designed to compare a hypoenergetic low fat, high sucrose diet (43% of total daily energy intake from sucrose) with a hypoenergetic low fat, low sucrose diet (4% of total daily energy intake from sucrose) in 42 normal weight women. Whilst there was no difference in total cholesterol between groups at the end of the intervention period, there was a statistically significant time-by-group effect for total cholesterol ($p=0.009$), with the low sucrose diet group experiencing a larger reduction in total cholesterol compared with the high sucrose diet group. There was some degree of baseline imbalance between the groups in total cholesterol, which makes interpretation of this result less clear. Body weights, however, decreased in both groups.

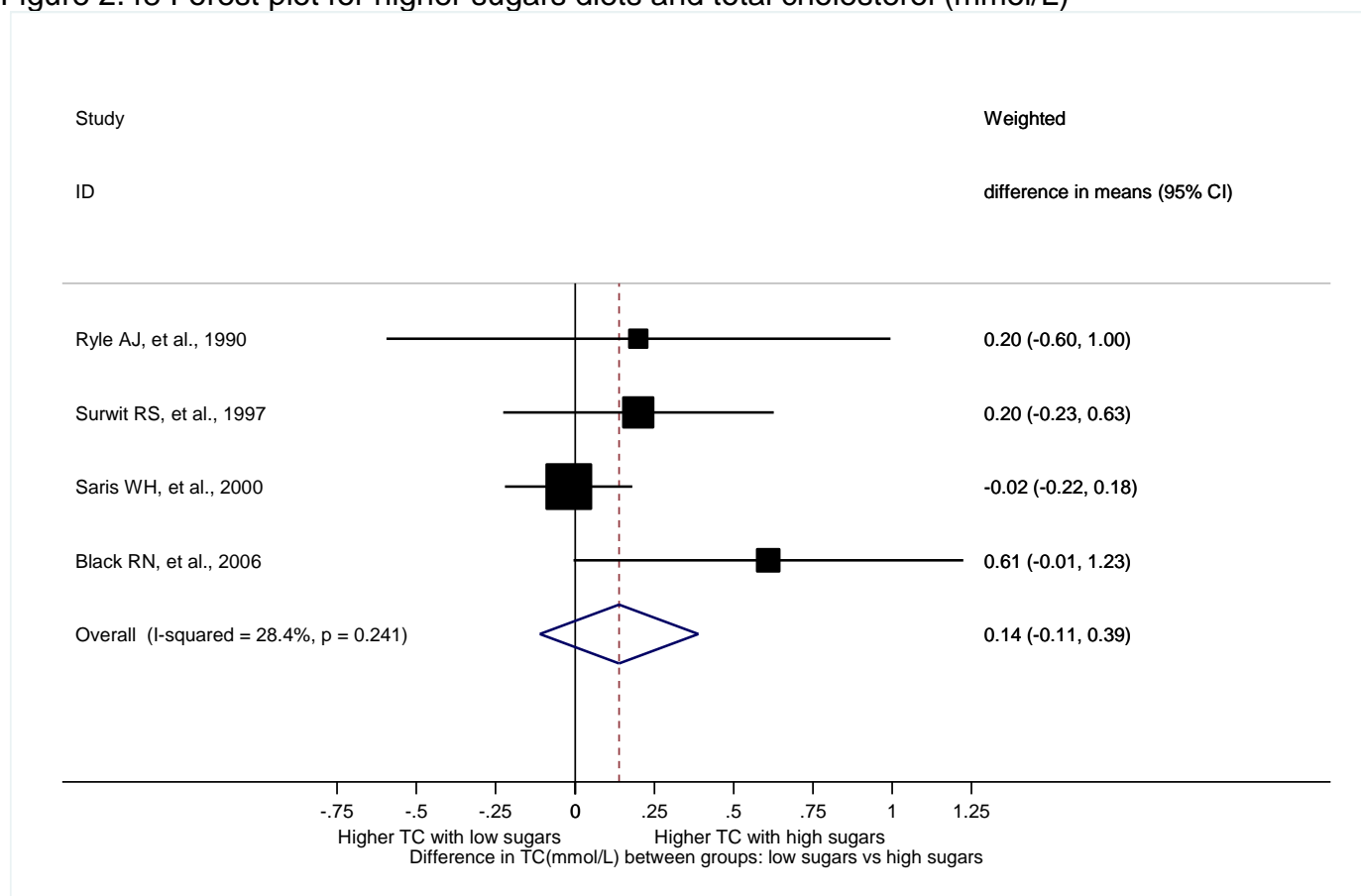
The study by (Poppitt *et al.*, 2002) was designed to test the effects of substituting a quarter of daily fat intake with either simple or “complex” carbohydrates on intermediary metabolism (Poppitt *et al.*, 2002). After six months, total cholesterol had changed by -0.33mmol/L, 0.63mmol/L and -0.06mmol/L in the control, low fat high complex carbohydrate and low fat high simple carbohydrate groups, respectively. A small but statistically significant difference between total cholesterol in the low fat high complex carbohydrate group and low fat high simple carbohydrate group only was also observed ($p<0.05$). However, it is noteworthy to highlight that participants in the former diet group lost weight, whilst the latter group did not. Changes in total cholesterol over time however were not statistically significant.

In the CARMEN study conducted by Saris *et al.* (Saris *et al.*, 2000) 398 moderately obese males and females were randomly allocated to a seasonal control group or one of three experimental groups: low fat high simple carbohydrate group, low fat high complex carbohydrate group or a control diet group. After six months, the authors reported no statistically significant diet-induced differences in total cholesterol between the control group, the low fat high simple carbohydrate group and the low fat high complex carbohydrate group, which are the comparison groups of interest here.

The study by Ryle *et al.* (Ryle *et al.*, 1990) compared the effects of dietary glucose and soluble fibre supplementation on total cholesterol levels in 11 non-obese men and women over six weeks. Study participants consumed a high fibre (15g guar gum per day), low glucose diet, or a low fibre, high glucose (500ml glucose drink providing 100g glucose per day) diet in a crossover design. Total cholesterol was not altered by either diet. With variation in both fibre and sugars intake, it is not possible to determine the independent effect of either dietary manipulation in this study.

Four studies were included in the meta-analysis comparing different sugars intakes and changes in total cholesterol reported as mmol/L (Saris *et al.*, 2000; Surwit *et al.*, 1997; Ryle *et al.*, 1990; Black *et al.*, 2006). All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months. The pooled estimate indicated that total cholesterol was 0.14mmol/L (95% CI -0.11 to 0.39) higher with consumption of a diet higher in sugars. This was not significantly different from zero ($p=0.28$). Overall heterogeneity denoted by I^2 was 28% (95% CI 0 to 74%). Statistically, there was no evidence that a higher sugars consumption influenced levels of total cholesterol.

Figure 2.48 Forest plot for higher sugars diets and total cholesterol (mmol/L)



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
(Albertson <i>et al.</i> , 2009) 13994 National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	7 years	Food diary	Sugar from breakfast cereals (Percent of cereal consumed that was sugar)	Total cholesterol Fasting, Whole blood	1 %	0.13 (0.08)	0.14	No adjustments made

Table 2.67 Total cholesterol and sugars: cohort study in children

Table 2.68 Total cholesterol and fructose vs. glucose: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Bantle <i>et al.</i> , 2000) *15269	High-fructose diet	12/12		4.3 (SE 0.05)		0.169	Total cholesterol	Plasma (mmol/L)	6 weeks	Decrease	unclear
	High-glucose diet	12/12		4.22 (SE 0.05)						Decrease	

Table 2.69 Total cholesterol and higher sugars diets: RCT data

Author/ Result ID	Intervention groups	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 at follow-up	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Black <i>et al.</i> , 2006) *16619	High sucrose diet	13/13	4.53 (SE 0.27)	4.62 (SE 0.8)			<0.01		Total cholesterol	Fasting (mmol/L)	6 weeks	No change	unclear
	Low sucrose diet	13/13	4.53 (SE 0.27)	4.01 (SE 0.80)								No change	
(Drummond <i>et al.</i> , 2003) 15105	Reduced fat	completers not reported/~22				NS	Not reported/ NS		Total cholesterol	Not reported (mmol/L)	12 weeks	Not reported	unclear
	Reduced fat and sugar	completers not reported/~22				NS						Not reported	
(Poppitt <i>et al.</i> , 2002) 15379	Control	7/15	6.2 (SD 1)		-0.33				Total cholesterol	Fasting serum (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	5.7 (SD 1)		-0.63		<0.05 diet effect (compared with the low-fat, high-simple carbohydrate diet)					Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	5.9 (SD 1.4)		-0.06							No change	
(Ryle <i>et al.</i> , 1990) *16204	High glucose low soluble fibre	11/11	5.2 (SD 0.7)	5.1 (SD 0.9)		NS			Total cholesterol	Fasting (mmol/L)	6 weeks	No change	unclear
	Low glucose high soluble fibre diet	11/11	5.2 (SD 0.7)	4.9 (SD 1.0)		NS	NS					No change	
(Saris <i>et al.</i> ,	Control diet	77/77	5.66 (SD 1.09)		-0.14 (SD 0.63)				Total cholesterol	Fasting serum (mmol/L)	6 months	No change	unclear

Author/ Result ID	Intervention groups	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 at follow-up	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
2000) *15094													
	Low-fat high-complex carbohydrate diet	83/83	5.66 (SD 1.09)		-0.22 (SD 0.65)		NS					Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	5.66 (SD 1.09)		-0.24 (SD 0.62)		NS					Decrease	
(Surwit <i>et al.</i> , 1997) *15051	High sucrose diet	20/28	4.63 (SD 0.77)	4.14 (SD 0.75)				0.009	Total cholesterol	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
	Low sucrose diet	22/24	4.92 (SD 0.84)	3.94 (SD 0.62)								Decrease	

HDL cholesterol, dietary sugars and high sugars diets

Summary of cohort results

Data were extracted from one study investigating the association between sucrose intake and change in HDL cholesterol (Archer *et al.*, 1998). The CARDIA study of young US adults (Archer *et al.*, 1998) reported sucrose intake as a percentage of total energy (measured using a 700 item FFQ). This study reported HDL cholesterol at follow up as well as the change in HDL cholesterol from baseline to follow up (year 7). For the whole cohort, there was a small but significant inverse association between baseline sucrose intake and follow up HDL cholesterol. HDL cholesterol decreased by 0.07mmol/L for each 10% increase in energy from sucrose.

Similarly, the longitudinal analysis of HDL cholesterol change from baseline to year 7 indicated an inverse association with dietary sucrose intakes for the cohort as a whole. The authors also reported that the inverse association was still apparent with the additional inclusion of starch (% energy) or dietary fat (% energy) as covariates in the model.

Both sucrose intake and change in HDL cholesterol, and sucrose intake and HDL cholesterol results at follow up were inconsistent in certain subgroups, with only some of these relationships achieving statistical significance. The CARDIA study (Archer *et al.*, 1998) adjusted for age, alcohol, BMI, smoking and physical activity.

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

Summary of RCT data

Four studies provided data on the effects of high sugars diets on HDL cholesterol (Black *et al.*, 2006; Surwit *et al.*, 1997; Poppitt *et al.*, 2002; Saris *et al.*, 2000). Further details of these studies can be found in the Trial Characteristics table.

Three trials used a parallel group approach (Poppitt *et al.*, 2002; Saris *et al.*, 2000; Surwit *et al.*, 1997), whereas the remaining trial employed a crossover design (Black *et al.*, 2006). All four studies were carried out in Europe, two of which were conducted in the UK (Surwit *et al.*, 1997; Black *et al.*, 2006). Samples varied in size from 14 to 398 participants. All were adults. Average BMI of trial participants was generally in the overweight or obese category for the four trials. One study recruited females only (Surwit *et al.*, 1997), one used males only (Black *et al.*, 2006) and the others were mixed gender.

The extent of blinding, if at all, in all studies was not reported.

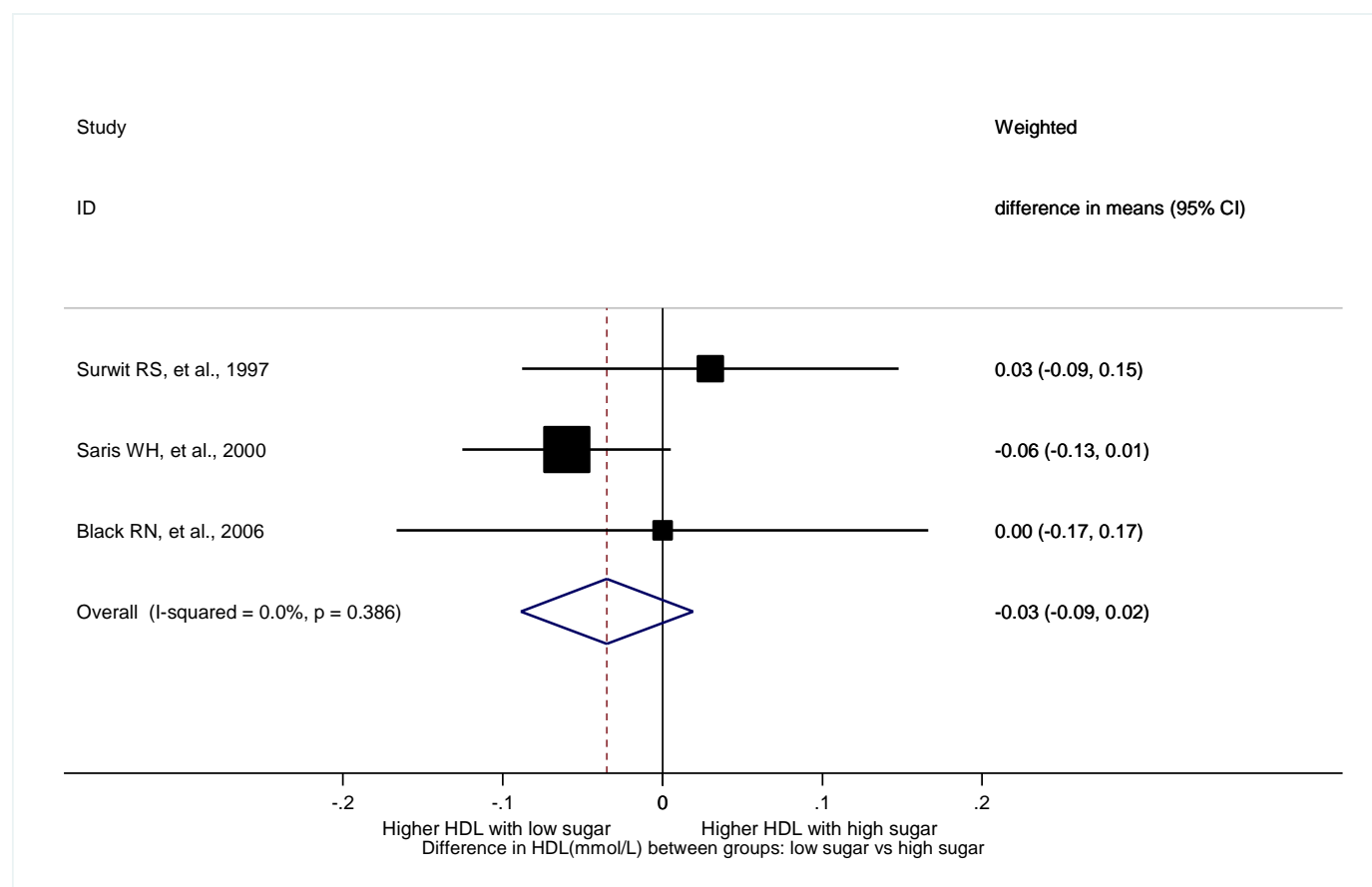
Of the four studies, body weights were unchanged in two. Poppitt *et al.* (Poppitt *et al.*, 2002) and Saris *et al.* (Saris *et al.*, 2000) reported a decrease in weight in the low fat high complex carbohydrate group and a weight loss in the two dietary intervention groups, respectively. Surwit *et al.*, on the other hand, reported weight loss in both dietary groups (Surwit *et al.*, 1997). Changes in HDL cholesterol therefore may not be solely attributable to the dietary intervention.

Three studies were included in the meta-analysis comparing different sugars intakes and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months.

Data from Poppitt *et al.* (Poppitt *et al.*, 2002) were not included in the meta-analysis as only baseline data were reported. Poppitt *et al.* (Poppitt *et al.*, 2002) explored the effects of a low fat high complex carbohydrate, a low fat high simple carbohydrate or a control diet in 46 subjects with three or more metabolic syndrome risk factors. This study was designed to test the effects of substituting a quarter of daily fat intake with either simple or “complex” carbohydrates on intermediary metabolism (Poppitt *et al.*, 2002). Diets were attained through the provision of food from a study grocery store located near the research clinic. No statistically significant differences in HDL cholesterol between the three treatment groups were observed (Poppitt *et al.*, 2002).

The pooled estimate indicated that HDL cholesterol was 0.03mmol/L (95% CI -0.02 to 0.09) lower with consumption of a diet higher in sugars. This was not significantly different from zero ($p=0.21$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 89%). Statistically, there was no evidence that higher sugar consumption is associated with variation in levels of HDL cholesterol.

Figure 2.49 Forest plot for high sugars diets and HDL cholesterol (mmol/L)



Data were provided from one trial comparing fructose and glucose intake and HDL cholesterol (Bantle *et al.*, 2000). The results of this study can be seen in Table 2.70.

Bantle *et al.* (Bantle *et al.*, 2000) conducted a crossover trial to test the effects of dietary fructose on plasma lipids in healthy volunteers (n=24). The authors reported a decrease in body weight in both groups during the intervention. Overall, no differences between the high fructose diet group and the high glucose diet group were observed for HDL cholesterol (p=0.965). This trial therefore suggests no change in HDL cholesterol with added fructose consumption in the context of an isoenergetic diet.

Table 2.70 HDL cholesterol and sucrose: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
(Archer <i>et al.</i> , 1998) 13715 The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, No T2DM	18-30 %M 45.9	5115	7 years	FFQ (700)	Sucrose	Change in HDL-C Plasma,mmol/L		10 % Energy	-0.04 (0.01)	<0.001	age, alcohol, BMI, smoking, ethnicity, physical activity, gender
13711 The CARDIA Study								Race - Black Men	10 % Energy	-0.03 (0.02)		age, alcohol, BMI, smoking, physical activity
13712 The CARDIA Study								Race - White Men	10 % Energy	-0.04 (0.01)	<0.01	As above
13713 The CARDIA Study								Race - Black Women	10 % Energy	-0.03 (0.01)	<0.05	As above
13714 The CARDIA Study								Race - White Women	10 % Energy	-0.04 (0.01)	<0.01	As above
13710 The CARDIA Study	USA, Multi-ethnic, Generally healthy, No hypertension, No T2DM	18-30 %M 45.9	5115	7 years	FFQ (700)	Sucrose	HDL-C Plasma,mmol/L		10 % Energy	-0.07 (0.01)	<0.001	age, alcohol, BMI, smoking, ethnicity, physical activity, gender
13706 The CARDIA Study								Race - Black Men	10 % Energy	-0.06 (0.04)		age, alcohol, BMI, smoking, physical activity
13707 The CARDIA Study								Race - White Men	10 % Energy	-0.08 (0.02)	<0.01	As above
13708 The CARDIA Study								Race - Black Women	10 % Energy	-0.09 (0.03)	<0.01	As above
13709 The CARDIA Study								Race - White Women	10 % Energy	-0.05 (0.03)		As above

Table 2.71 HDL cholesterol and fructose vs. glucose: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Bantle <i>et al.</i> , 2000) 15282	High-fructose diet	12/12		1.3 (SE 0.03)	0.965	HDL-C	Plasma (mmol/L)	6 weeks	Decrease	unclear
	High-glucose diet	12/12		1.3 (SE 0.03)					Decrease	

Table 2.72 HDL cholesterol and high sugars diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Black <i>et al.</i> , 2006) *16621	High sucrose diet	13/13	1.26 (SE 0.05)	1.2 (SE 0.06)			NS	HDL-C	Fasting (mmol/L)	6 weeks	No change	unclear
	Low sucrose diet	13/13	1.26 (SE 0.05)	1.2 (SE 0.06)							No change	
(Poppitt <i>et al.</i> , 2002) 15381	Control	7/15	1.4 (SD 0.3)				NS	HDL-C	Fasting serum (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	1.3 (SD 0.2)				NS				Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	1.1 (SD 0.3)				NS				No change	
(Ryle <i>et al.</i> , 1990) *16205	High glucose low soluble fibre	11/11	1.1 (SD 0.3)	1.1 (SD 0.3)		NS		HDL-C	Fasting (mmol/L)	6 weeks	No change	unclear
	Low glucose high soluble fibre diet	11/11	1.1 (SD 0.3)	1.0 (SD 0.3)		NS	NS				No change	
(Saris <i>et al.</i> , 2000) *15095	Control diet	77/77	1.28 (SD 0.34)		-0.07 (SD 0.23)			HDL-C	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	1.28 (SD 0.34)		-0.08 (SD 0.22)		NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	1.28 (SD 0.34)		-0.13 (SD 0.18)		NS				Decrease	
(Surwit <i>et al.</i> , 1997) *15053	High sucrose diet	20/28	1.35 (SD 0.34)	1.06 (SD 0.19)			NS	HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
	Low sucrose diet	22/24	1.29 (SD 0.22)	1.03 (SD 0.19)							Decrease	

*This result was used in the meta-analysis of high sugar diets and HDL cholesterol

LDL cholesterol, dietary sugars and high sugars diets

No cohort studies reported results concerning sugars and LDL cholesterol.

Summary of RCT data

Only one trial provided data on fructose and glucose intake and LDL cholesterol (Bantle *et al.*, 2000). No differences between the high fructose diet group and the high glucose diet group were observed ($p=0.658$) in this study. This trial therefore suggests no change in LDL cholesterol with added fructose consumption in the context of an isoenergetic diet.

Four studies provided data on the effects of high sugars diets on LDL cholesterol (Black *et al.*, 2006; Surwit *et al.*, 1997; Poppitt *et al.*, 2002; Saris *et al.*, 2000). These trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol, dietary sugars and high sugars trials.

Of the four studies, body weights were unchanged in one (Black *et al.*, 2006). Only (Poppitt *et al.*, 2002) and (Saris *et al.*, 2000) reported a decrease in weight in the low fat high complex carbohydrate group and a weight loss in the two dietary intervention groups, respectively. Surwit *et al.*, on the other hand, reported weight loss in both dietary groups (Surwit *et al.*, 1997). Changes in LDL cholesterol therefore may not be solely attributable to the dietary intervention.

Three studies were included in the meta-analysis comparing different sugars intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months.

Data from Poppitt *et al.* (Poppitt *et al.*, 2002) were not included in the meta-analysis as only baseline data were reported. Poppitt *et al.* (Poppitt *et al.*, 2002) explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in 46 subjects with three or more metabolic syndrome risk factors. This study was designed to test the effects of substituting a quarter of daily fat intake with either simple or “complex” carbohydrates on intermediary metabolism (Poppitt *et al.*, 2002). Diets were attained through the provision of food from a study grocery store located near the research clinic. No statistically significant differences in LDL cholesterol between the three treatment groups were observed (Poppitt *et al.*, 2002).

The pooled estimate indicated that LDL cholesterol was 0.10mmol/L (95% CI -0.18 to 0.38) higher with consumption of a diet higher in sugars. This was not significantly different from zero (p=0.49). Overall heterogeneity denoted by I^2 was 44% (95% CI 0 to 83%). Statistically, there was no evidence that high sugar consumption is associated with levels of LDL cholesterol.

Figure 2.50 Forest plot for high sugars diets and LDL cholesterol (mmol/L)

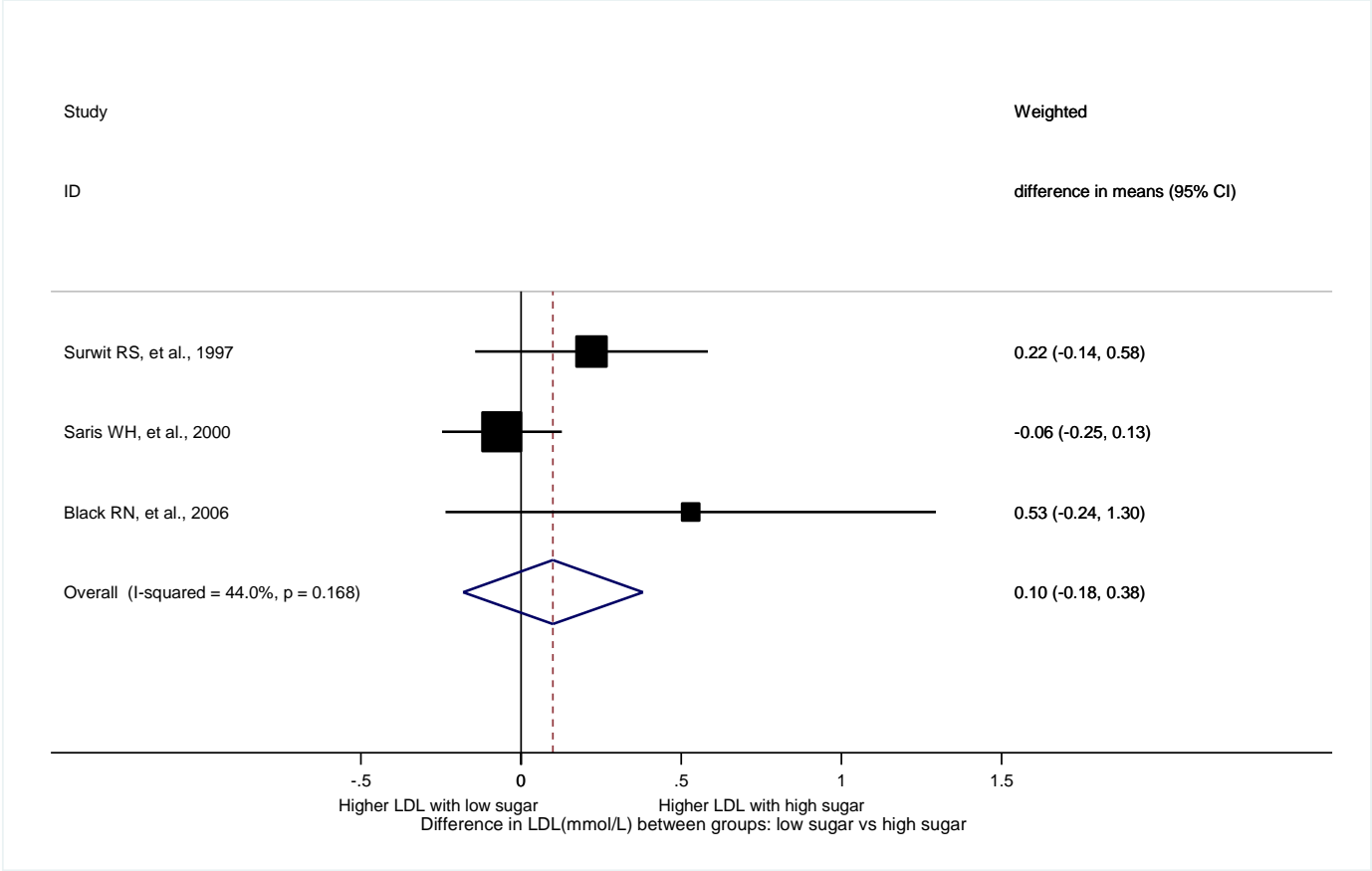


Table 2.73 LDL cholesterol and fructose vs. glucose: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Bantle <i>et al.</i> , 2000) 15275	High-fructose diet	12/12		2.54 (SE 0.05)	0.658	LDL-C	Plasma (mmol/L)	6 weeks	Decrease	unclear
	High-glucose diet	12/12		2.56 (SE 0.05)					Decrease	

Table 2.74 LDL cholesterol and high sugars diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Black <i>et al.</i> , 2006) *16620	High sucrose diet	13/13	2.78 (SE 0.27)	2.78 (SE 0.30)		<0.01		LDL-C	Fasting (mmol/L)	6 weeks	No change	unclear
	Low sucrose diet	13/13	2.78 (SE 0.27)	2.25 (SE 0.25)							No change	
(Poppitt <i>et al.</i> , 2002) 15380	Control	7/15	4.1 (SD 0.9)			NS		LDL-C	Fasting serum (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	3.7 (SD 0.7)			NS					Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	3.8 (SD 0.8)			NS					No change	
(Saris <i>et al.</i> , 2000) *15096	Control diet	77/77	3.7 (SD 1.02)		-0.03 (SD 0.65)			LDL-C	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	3.7 (SD 1.02)		-0.02 (SD 0.56)	NS					Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	3.7 (SD 1.02)		-0.09 (SD 0.53)	NS					Decrease	
(Surwit <i>et al.</i> , 1997) *15052	High sucrose diet	20/28	2.7 (SD 0.5)	2.6 (SD 0.62)			0.01	LDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
	Low sucrose diet	22/24	3.04 (SD 0.74)	2.38 (SD 0.55)							Decrease	

*This result has been used in the meta-analysis for high sugars and LDL cholesterol

Triacylglycerol, dietary sugars and high sugars diets

No cohort studies reported results concerning sugar and triacylglycerol.

Summary of RCT data

Only one study provided data on sugars and TAG levels (Bantle *et al.*, 2000). In Bantle *et al.* (Bantle *et al.*, 2000), results concerning TAG levels and daylong TAG observation were reported at six weeks from baseline randomisation. Overall, plasma TAG levels for both diet groups were statistically significantly different at follow up among men and women (Bantle *et al.*, 2000). Males experienced statistically significantly higher TAG levels during the high fructose diet compared with the high glucose diet ($p < 0.001$), whilst triacylglycerol levels of women remained unchanged. The authors also reported that daylong values for TAG were 32% greater in the high fructose diet than the high glucose diet for men ($p < 0.001$). Women did not show any statistically significant differences between diets for triacylglycerol daylong observation. It is also of note that body weights decreased throughout the trial, which may have impacted on TAG.

Five trials explored the effects of sugar reduction diets on TAG levels (Poppitt *et al.*, 2002; Saris *et al.*, 2000; Black *et al.*, 2006; Sorensen *et al.*, 2005; Surwit *et al.*, 1997). Further details of these studies can be found in the Trial Characteristics table and in the section on total cholesterol and high sugars diets.

Four trials used a parallel group approach (Poppitt *et al.*, 2002; Saris *et al.*, 2000; Surwit *et al.*, 1997; Sorensen *et al.*, 2005), whereas the remaining trial employed a crossover design (Black *et al.*, 2006). All four studies were carried out in Europe, two of which were conducted in the UK (Surwit *et al.*, 1997; Black *et al.*, 2006) and one in Denmark (Sorensen *et al.*, 2005). Samples varied in size from 14 to 398 participants with three studies tending to have less than 52 participants (Poppitt *et al.*, 2002; Sorensen *et al.*, 2005; Surwit *et al.*, 1997). All were adults. Average BMI of trial participants was generally in the overweight or obese category for the five trials. One study recruited females only (Surwit *et al.*, 1997), one used males only (Black *et al.*, 2006) and the others were mixed gender.

The extent of blinding, if at all, in all studies was not reported.

Of the five studies, body weights were unchanged in one (Black *et al.*, 2006; Surwit *et al.*, 1997). One study by Surwit *et al.* reported body weight loss in both dietary groups (Surwit *et al.*, 1997). Poppitt *et al.* (Poppitt *et al.*, 2002), Saris *et al.* (Saris *et al.*, 2000) and Sorensen *et al.* (Sorensen *et al.*, 2005) reported a decrease in weight in their low fat high complex carbohydrate group, a weight loss in their two dietary intervention groups, and a weight decrease in their sweetener group respectively. An increase in weight was also noted in one group in the study by Sorensen *et al.* (Sorensen *et al.*, 2005). Changes in TAG therefore may not be solely attributable to the dietary intervention.

Due to the absence or form of measures of variation in two studies (Black *et al.*, 2006; Sorensen *et al.*, 2005) and only baseline data being reported in Poppitt *et al.* (Poppitt *et al.*, 2002), it was not possible to combine studies to perform a meta-analysis.

Two studies compared low fat, high simple carbohydrate diets against low fat complex carbohydrate diets, rather than investigating high sucrose vs. low sucrose diets. In one study, Poppitt *et al.* (Poppitt *et al.*, 2002) explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in 46 subjects with three or more metabolic syndrome risk factors. This study was designed to test the effects of substituting a quarter of daily fat intake with either simple or “complex” carbohydrates on intermediary metabolism (Poppitt *et al.*, 2002). Diets were attained through the provision of food from a study grocery store located near the research clinic. At follow up (six months), TAG levels were statistically significantly higher in the low fat high simple carbohydrate group compared with the low fat high complex carbohydrate group and control group ($p < 0.05$).

In the CARMEN study conducted by Saris *et al.* (Saris *et al.*, 2000) 398 moderately obese males and females were randomly allocated to a low fat high simple carbohydrate group, low fat high complex carbohydrate group or a control diet group. The authors reported minor changes in TAG levels overall (by -0.13mmol/L, -0.16mmol/L and 0.01mmol/L in the control group, low fat high complex carbohydrate group and low fat high simple carbohydrate group, respectively). However no statistically significant differences between the groups were observed.

These trials show inconsistent findings concerning the impact of reduced sugars diets on TAG levels.

Three other studies explored the effects of a high sucrose diet compared with a low sucrose diet or sweeteners. Black *et al.* (Black *et al.*, 2006) conducted a randomised crossover study to test the effects of a eucaloric high (25% of total energy intake) or low (10% of total energy intake) sucrose diet in 13 healthy non-diabetic subjects. Fasting TAG measured after six weeks had decreased in both groups, but no statistically significant differences between groups were reported.

One study provided information concerning the effects of high and low sucrose diets on markers of cardiometabolic health in 41 overweight men and women (Sorensen *et al.*, 2005). The intervention was achieved through provision of food and drinks high in sucrose or sweetened with artificial sweeteners, with the majority of the additional sucrose being derived from sweetened beverages (70% of sucrose). At the 10-week follow up, no differences in plasma TAG between groups were apparent.

Finally, Surwit *et al.* (Surwit *et al.*, 1997) conducted a six week weight loss trial designed to compare a hypoenergetic low fat, high sucrose diet (43% of total daily energy intake from sucrose) with a hypoenergetic low fat, low sucrose diet (4% of total daily energy intake from sucrose) in 42 normal weight women. Whilst there were small reductions in fasting TAG levels at 10 weeks, these changes did not statistically significantly differ between groups.

These three trials jointly indicate that TAG levels are unaffected by sugar reduction diets in a variety of subjects.

Table 2.75 Triacylglycerol and fructose vs. glucose: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Follow-up	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Bantle <i>et al.</i> , 2000) 15288	Women	High-fructose diet	12/12	0.93 (SE 0.06)	0.631	TAG	Plasma (mmol/L)	6 weeks	Decrease	unclear
		High-glucose diet	12/12	0.97 (SE 0.06)					Decrease	
15294	Men	High-fructose diet	12/12	1.25 (SE 0.06)	<0.001	TAG	Plasma (mmol/L)	6 weeks	Decrease	unclear
		High-glucose diet	12/12	0.95 (SE 0.06)					Decrease	
15223	Women	High-fructose diet	12/12	30.8	0.722	TAG daylong observation	Plasma (mmol/hour/L)	6 weeks	Decrease	unclear
		High-glucose diet	12/12	29.9					Decrease	
15260	Men	High-fructose diet	12/12	46.1	<0.001	TAG daylong observation	Plasma (mmol/hour/L)	6 weeks	Decrease	unclear
		High-glucose diet	12/12	35					Decrease	

Table 2.76 Triacylglycerol and high sugars diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Black <i>et al.</i> , 2006) 16622	High sucrose diet	13/13	1.03	0.95			NS	TAG	Fasting (mmol/L)	6 weeks	No change	unclear
	Low sucrose diet	13/13	1.03	0.92							No change	
(Poppitt <i>et al.</i> , 2002) 15383	Control	7/15	2.1 (SD 1.1)	lower				TAG	Fasting serum (mmol/L)	6 months	No change	bias
	Low-fat, high- complex carbohydrate diet	12/16	1.9 (SD 1.3)	lower							Decrease	
	Low-fat, high- simple carbohydrate diet	13/15	2.3 (SD 1.3)	higher							No change	

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Ryle <i>et al.</i> , 1990) 16206	High glucose low soluble fibre	11/11	0.77 (SD 0.33)	0.89 (SD 0.39)		NS		TAG	Fasting (mmol/L)	6 weeks	No change	unclear
	Low glucose high soluble fibre diet	11/11	0.77 (SD 0.33)	0.7 (SD 0.29)		NS	NS				No change	
(Saris <i>et al.</i> , 2000) 15098	Control diet	77/77	1.45 (SD 0.8)		-0.13 (SD 0.57)			TAG	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high- complex carbohydrate diet	83/83	1.45 (SD 0.8)		-0.16 (SD 0.61)		NS				Decrease	
	Low-fat, high- simple carbohydrate diet	76/76	1.45 (SD 0.8)		0.01 (SD 0.53)		NS				Decrease	
(Sorensen <i>et al.</i> , 2005) 17446	Sucrose	19/21	1.1	1.2			NS	TAG	Plasma (mmol/L)	10 weeks	Increase	unclear
	Sweetener	18/20	1.1	0.9							Decrease	
(Surwit <i>et al.</i> , 1997) 15054	High sucrose diet	20/28	1.19 (SD 0.94)	1.08 (SD 0.59)			NS	TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
	Low sucrose diet	22/24	1.29 (SD 0.71)	1.05 (SD 0.45)							Decrease	

Total cholesterol:HDL ratio and high sugars diets

No cohort studies reported results concerning sugar and the TC:HDL ratio.

Summary of RCT data

One study explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in 46 subjects with three or more metabolic syndrome risk factors (Poppitt *et al.*, 2002). Body weights were unchanged in the control and low fat, high simple carbohydrate groups but decreased in the low fat, high complex carbohydrate group. At follow up (six months), the authors reported an increase in the TC:HDL ratio across all treatment groups ($p < 0.01$ for all); however no statistically significant changes between groups were detected.

Table 2.77 Total cholesterol:HDL ratio and high sugars diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Poppitt <i>et al.</i> , 2002) 15382	Control	7/15	4.6 (SD 0.9)	increase	<0.01	NS	Change in Total cholesterol :HDL ratio	Fasting serum (mmol/L)	6 months	No change	bias
	Low fat, high complex carb	12/16	4.6 (SD 1)	increase	<0.01	NS				Decrease	
	Low fat, high simple carb	13/15	5.3 (SD 1.9)	increase	<0.01	NS				No change	

LDL:HDL cholesterol ratio and high sugars diets

No cohort studies reported results concerning sugars and LDL:HDL cholesterol ratio.

Summary of RCT data

In the CARMEN study, Saris *et al.* (Saris *et al.*, 2000) randomly allocated participants to a low fat high simple carbohydrate diet, a low fat high complex carbohydrate diet or a control diet. Body weights were unchanged in the control group, although decreased in the two low fat, carbohydrate groups. At six months, minor changes in LDL:HDL cholesterol ratio were noted on all three diets; however the differences between groups were not statistically significant.

Table 2.78 LDL:HDL cholesterol ratio and high sugars diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Saris <i>et al.</i> , 2000) 15097	Control diet	77/77	0.39 (SD 0.19)	-0.04 (SD 0.15)		Change in HDL/LDL	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	0.39 (SD 0.19)	-0.03 (SD 0.11)	NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	0.39 (SD 0.19)	-0.03 (SD 0.08)	NS				Decrease	

Results – Sweetened beverages

Incident hyperlipidaemias and sweetened beverages

Summary of cohort results

Data were extracted from one study (Dhingra *et al.*, 2007) which explored the association between consumption of mixed sugar and artificially sweetened beverages and both incident hypertriglyceridaemia and incident low HDL cholesterol (for definitions see table below). The Framingham Heart Study (Dhingra *et al.*, 2007) reported mixed sugar and artificial sweetener beverages using a general questionnaire. A serving was described as a 12oz can of fizzy drink.

The study reported a 25% increased risk of incident hypertriglyceridaemia comparing more than one serving per day of mixed sugar and artificial sweetener beverages to no servings per day (Dhingra *et al.*, 2007). The risk of incident low HDL cholesterol was also significantly increased by 32% with more than one serving per day compared with no servings per day (Dhingra *et al.*, 2007). The results were adjusted for an appropriate range of potential confounders, including age, gender, smoking and physical activity, but not BMI.

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

Summary of RCT data

No RCTs reported outcomes concerning sweetened beverages and incident lipidaemia.

Table 2.79 Incident lipidaemias and sweetened beverages: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follo w Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Contrast	Exposure Units	RR (CI)	Adjustments
(Dhingra <i>et al.</i> , 2007) 14264 The Framingham Heart Study	USA, No CHD, Without metabolic syndrome	(53) %M 43	(1093) /8997	4 years	Questionnaire (general)	Mixed sugar and artificial sweetener beverages (soft drink - number of 12oz cans of fizzy drinks sugar or sweetener)	Incident hypertriglyceridemia (1.7mmol/L or receiving treatment) Experimenter/ clinic assessed	≥1 vs 0	servings/ day	1.25 (1.04, 1.51)	age, smoking, SAFA, energy intake, dietary fibre, GI, Mg intake, physical activity, gender, trans fatty acid intake
14265 The Framingham Heart Study			(739) /8997	4 years	Questionnaire (general)	Mixed sugar and artificial sweetener beverages (soft drink - number of 12oz cans of fizzy drinks sugar or sweetener)	Incident low HDL-C <40mg/dL (1.03mmol/L) men or <50mg/dL (1.3mmol/L) women Experimenter/ clinic assessed	≥1 vs 0	servings/ day	1.32 (1.06, 1.64)	As above

Results – “Complex” carbohydrates

Definitions of “complex” carbohydrates were not provided by the authors of the included studies, although the prevailing definition at that time stated that “complex” carbohydrates are composed of complex sugar 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 Organization (WHO), and as stated in Farchi *et al.* (Farchi *et al.*, 1995), intakes of “complex” carbohydrates should make up 50-70% of total carbohydrate intake. Trials that reported “complex” carbohydrate intake compared a low fat, high complex carbohydrate diet to a low fat, high simple carbohydrate diet and control diet (Poppitt *et al.*, 2002; Saris *et al.*, 2000).

Total cholesterol and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and total cholesterol.

Summary of RCT data

Two studies provided data on the effects of “complex” carbohydrate intake on total cholesterol (Poppitt *et al.*, 2002; Saris *et al.*, 2000). As there was an insufficient number of studies, it was not possible to perform a meta-analysis.

Body weights were unchanged in the study conducted by Saris *et al.* (Saris *et al.*, 2000), but in Poppitt *et al.* the authors reported that there was a weight decrease in the low fat complex carbohydrate group (Poppitt *et al.*, 2002).

Poppitt *et al.* (Poppitt *et al.*, 2002) explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in 46 subjects with three or more metabolic syndrome risk factors. This study was designed to test the effects of substituting a quarter of daily fat intake with either simple or “complex” carbohydrates on intermediary metabolism (Poppitt *et al.*, 2002). Diets were attained through the provision of food from a study grocery store located near the research clinic. After six months, total cholesterol had changed by -0.33mmol/L, 0.63mmol/L and -0.06mmol/L in the control, low fat high complex carbohydrate and low fat high simple carbohydrate groups, respectively. A small but statistically significant difference between total cholesterol in the low fat high complex carbohydrate group and the low fat high simple carbohydrate group only was also observed ($p < 0.05$). However, it is noteworthy to highlight that participants in the former diet group lost weight, whilst the latter group did not; changes in total cholesterol therefore may not be solely attributable to “complex” carbohydrate intake. Changes in total cholesterol over time were not statistically significant.

In the CARMEN study conducted by Saris *et al.* (Saris *et al.*, 2000) 398 moderately obese males and females were randomly allocated to a seasonal control group or one of three experimental groups: low fat high simple carbohydrate group, low fat high complex carbohydrate group or a control diet group. Diets for the low fat high simple carbohydrate group and low fat high complex carbohydrate group were achieved using both a purpose-built shop with a recorded choice of food items and conventional supermarkets. After six months, the authors reported no statistically significant diet-induced changes between the control group, the low fat high simple carbohydrate group and the low fat high complex carbohydrate group, which are the comparison groups of interest.

These two trials provide inconsistent findings concerning the effect of “complex” carbohydrate intake on total cholesterol levels.

Table 2.80 Total cholesterol and “complex” carbohydrates: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Poppitt <i>et al.</i> , 2002) 15379	Control	7/15	6.2 (SD 1)		-0.33		Total cholesterol	Fasting Serum, (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	5.7 (SD 1)		-0.63	<0.05 diet effect (compared with the low-fat, high-simple carbohydrate diet)				Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	5.9 (SD 1.4)		-0.06					No change	
(Saris <i>et al.</i> , 2000) 15094	Control diet	77/77	5.66 (SD 1.09)		-0.14 (SD 0.63)		Total cholesterol	Fasting Serum, (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	5.66 (SD 1.09)		-0.22 (SD 0.65)	NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	5.66 (SD 1.09)		-0.24 (SD 0.62)	NS				Decrease	

HDL cholesterol and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and HDL cholesterol.

Summary of RCT data

Two trials tested the effects of “complex” carbohydrate intake on HDL cholesterol (Poppitt *et al.*, 2002; Saris *et al.*, 2000). As there was an insufficient number of studies, it was not possible to perform a meta-analysis.

Body weights decreased in both treatment groups in the study by Saris *et al.* (Saris *et al.*, 2000) whilst the low fat high complex carbohydrate group experienced weight loss in Poppitt *et al.* (Poppitt *et al.*, 2002). Changes in blood lipids therefore may not be solely attributable to “complex” carbohydrate intake.

In the study by (Poppitt *et al.*, 2002), consumption of a low fat, high complex carbohydrate diet or a low fat, high simple carbohydrate diet did not produce statistically significant differences in HDL cholesterol compared with a control diet. No statistically significant differences over time for the three groups were observed.

Additionally, in the CARMEN study conducted by Saris *et al.* (Saris *et al.*, 2000) 398 moderately obese males and females were randomly allocated to a low fat high simple carbohydrate group, low fat high complex carbohydrate group or a control diet group. After six months, all groups experienced a decrease in HDL cholesterol levels (by 0.07mmol/L, 0.08mmol/L and 0.13mmol/L in the control, low fat high complex carbohydrate and low fat high simple carbohydrate groups, respectively), although the difference between these means was not statistically significant.

Overall, neither study provides evidence of an impact of “complex” carbohydrate intake on HDL cholesterol.

Table 2.81 HDL cholesterol and “complex” carbohydrates: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Poppitt <i>et al.</i> , 2002) 15381	Control	7/15	1.4 (SD 0.3)		NS	HDL-C	Fasting serum (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	1.3 (SD 0.2)		NS				Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	1.1 (SD 0.3)		NS				No change	
(Saris <i>et al.</i> , 2000) 15095	Control diet	77/77	1.28 (SD 0.34)	-0.07 (SD 0.23)		HDL-C	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	1.28 (SD 0.34)	-0.08 (SD 0.22)	NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	1.28 (SD 0.34)	-0.13 (SD 0.18)	NS				Decrease	

LDL cholesterol and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and LDL cholesterol.

Summary of RCT data

Two studies investigated the effects of “complex” carbohydrate intake on LDL cholesterol (Poppitt *et al.*, 2002; Saris *et al.*, 2000). As there was an insufficient number of studies, it was not possible to combine studies in a meta-analysis.

Body weights were unchanged in all trials other than in (Poppitt *et al.*, 2002), in which the authors reported that there was a decrease in the low fat, high complex carbohydrate group (Poppitt *et al.*, 2002).

Poppitt *et al.* (Poppitt *et al.*, 2002) explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in subjects with three or more metabolic syndrome risk factors. At follow up (six months), consumption of either low fat diet did not statistically significantly alter LDL cholesterol levels compared with the control diet. Similarly, no statistically significant changes in LDL cholesterol over time were observed.

Similarly in the CARMEN study conducted by Saris *et al.* (Saris *et al.*, 2000) 398 moderately obese males and females were randomly allocated to a seasonal control group or one of three experimental groups, which are the comparison groups of interest: low fat high simple carbohydrate group, low fat high complex carbohydrate group or a control diet group. Diets for the low fat high simple carbohydrate group and low fat high complex carbohydrate group were achieved using both a purpose-built shop with a recorded choice of food items and conventional supermarkets. After six months, there was a small decrease in LDL cholesterol of 0.03mmol/L, 0.02mmol/L and 0.09mmol/L in the control diet group, low fat high complex carbohydrate diet group and low fat high simple carbohydrate diet group respectively, although the differences between groups were not statistically significant.

Overall, these two trials provide evidence that a low-fat diet rich in “complex” carbohydrate is not associated with a difference in LDL cholesterol compared with a low fat, high simple carbohydrate diet.

Table 2.82 LDL cholesterol and “complex” carbohydrates: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Poppitt <i>et al.</i> , 2002) 15380	Control	7/15	4.1 (SD 0.9)		NS	LDL-C	Fasting Serum, (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	3.7 (SD 0.7)		NS				Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	3.8 (SD 0.8)		NS				No change	
(Saris <i>et al.</i> , 2000) 15096	Control diet	77/77	3.7 (SD 1.02)	-0.03 (SD 0.65)		LDL-C	Fasting Serum, (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	3.7 (SD 1.02)	-0.02 (SD 0.56)	NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	3.7 (SD 1.02)	-0.09 (SD 0.53)	NS				Decrease	

Triacylglycerol and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and TAG.

Summary of RCT data

Data from two trials concerning “complex” carbohydrate intake and TAG were extracted (Poppitt *et al.*, 2002; Saris *et al.*, 2000). As there was an insufficient number of studies, it was not possible to combine studies in a meta-analysis. The results from these studies are shown in Table 2.83.

Body weights were unchanged in all trials other than in Poppitt *et al.* (Poppitt *et al.*, 2002) in which the authors reported that there was a decrease in the low fat, high complex carbohydrate group.

In one study, Poppitt *et al.* (Poppitt *et al.*, 2002) explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in 46 subjects with three or more metabolic syndrome risk factors. This study was designed to test the effects of substituting a quarter of daily fat intake with either simple or “complex” carbohydrates on intermediary metabolism (Poppitt *et al.*, 2002). Diets were attained through the provision of food from a study grocery store located near the research clinic. At follow up (six months), TAG levels were statistically significantly higher in the low fat, high simple carbohydrate group than the low fat high complex carbohydrate and control group ($p=0.05$).

In the CARMEN study conducted by Saris *et al.* (Saris *et al.*, 2000) 398 moderately obese males and females were randomly allocated to a seasonal control group or one of three experimental groups, which are the comparison groups of interest: low fat high simple carbohydrate group, low fat high complex carbohydrate group or a control diet group. Diets for the low fat high simple carbohydrate group and low fat high complex carbohydrate group were achieved using both a purpose-built shop with a recorded choice of food items and conventional supermarkets. The authors reported minor changes in TAG levels overall (by -0.13mmol/L, -0.16mmol/L and 0.01mmol/L in the control group, low fat high complex carbohydrate group and low fat high simple carbohydrate group, respectively). However no statistically significant differences between the groups were observed.

These trials show inconsistent findings concerning the effect of “complex” carbohydrate intake on TAG levels.

Table 2.83 Triacylglycerol and “complex” carbohydrates: RCT data

Results Number	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Poppitt <i>et al.</i> , 2002) 15383	Control	7/15	2.1 (SD 1.1)	lower			TAG	Fasting serum (mmol/L)	6 months	No change	bias
	Low-fat, high-complex carbohydrate diet	12/16	1.9 (SD 1.3)	lower						Decrease	
	Low-fat, high-simple carbohydrate diet	13/15	2.3 (SD 1.3)	higher		<0.05 diet effect (compared with control and low-fat, high-complex carbohydrate diets)				No change	
(Saris <i>et al.</i> , 2000) 15098	Control diet	77/77	1.45 (SD 0.8)		-0.13 (SD 0.57)		TAG	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	1.45 (SD 0.8)		-0.16 (SD 0.61)	NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	1.45 (SD 0.8)		0.01 (SD 0.53)	NS				Decrease	

Total cholesterol:HDL ratio and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and total cholesterol:HDL ratio.

Summary of RCT data

One study explored the effects of a low fat, high complex carbohydrate diet, a low fat, high simple carbohydrate diet or a control diet in 46 subjects with three or more metabolic syndrome risk factors (Poppitt *et al.*, 2002). Body weights were unchanged in the control and low fat, high simple carbohydrate groups but decreased in the low fat, high complex carbohydrate group. At follow up (six months), the authors reported an increase in the TC:HDL ratio across all treatment groups ($p < 0.01$ for all); however no statistically significant changes between groups was detected.

Table 2.84 Total cholesterol:HDL ratio and “complex” carbohydrates: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Poppitt <i>et al.</i> , 2002) 15382	Control	7/15	4.6 (SD 0.9)	increase	<0.01		Change in Total cholesterol :HDL ratio	Fasting serum (mmol/L)	6 months	No change	bias
	Low fat, high complex carb	12/16	4.6 (SD 1)	increase	<0.01	NS				Decrease	
	Low fat, high simple carb	13/15	5.3 (SD 1.9)	increase	<0.01					No change	

LDL:HDL cholesterol ratio and “complex” carbohydrates

No cohort studies reported results concerning “complex” carbohydrates and LDL:HDL cholesterol ratio.

Summary of RCT data

In the CARMEN study, Saris *et al.* (Saris *et al.*, 2000) randomly allocated participants to a low-fat high-simple carbohydrate diet, a low fat high complex carbohydrate diet or a control diet. Body weights were unchanged in the control group, but decreased in the two low fat, high carbohydrate groups. At six months, minor changes in LDL:HDL cholesterol ratio were noted on all three diets but the differences between groups did not achieve statistical significance.

Table 2.85 LDL:HDL cholesterol ratio and “complex” carbohydrates: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Saris <i>et al.</i> , 2000) 15097	Control diet	77/77	0.39 (SD 0.19)	-0.04 (SD 0.15)		Change in HDL/LDL	Fasting serum (mmol/L)	6 months	No change	unclear
	Low-fat high-complex carbohydrate diet	83/83	0.39 (SD 0.19)	-0.03 (SD 0.11)	NS				Decrease	
	Low-fat, high-simple carbohydrate diet	76/76	0.39 (SD 0.19)	-0.03 (SD 0.08)	NS				Decrease	

Results – Dietary fibre and high fibre diets

Total cholesterol, dietary fibre and high fibre diets

The effects of high fibre diets composed of higher fibre food choices rather than through the use of dietary fibre isolates are included in this section. This generally means that the sources of fibre are variable both within and between studies. In all trials, the author definitions of 'high' and 'low' fibre were used to classify studies.

Summary of cohort results

Data were extracted from two publications, reporting results from two cohort studies (Albertson *et al.*, 2009; de Castro *et al.*, 2006). There was no association between dietary fibre derived from breakfast cereals and total cholesterol in the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) which followed girls aged 9-10 years for an average of seven years. Lower total cholesterol was associated with greater intake of fibre in the Japanese-Brazilian Diabetes Study, but there was no evidence of an association with LDL-cholesterol (de Castro *et al.*, 2006) (Table 2.86).

Dietary fibre intake was assessed using a food diary in the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009). This study provided unadjusted results only. Diet was assessed using a validated FFQ in the Japanese-Brazilian Diabetes Study (de Castro *et al.*, 2006)

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

Summary of RCT data

Six studies provided data on the effects of diets high or low in dietary fibre on total blood cholesterol (Andersson *et al.*, 2007; Kesaniemi *et al.*, 1990; Olendzki *et al.*, 2009; Thompson *et al.*, 2005; Aller *et al.*, 2004; Singh *et al.*, 1992). Data from one study were not included in the tables or the meta-analysis due to convincing evidence of poor study quality (Singh *et al.*, 1992).

All studies were open-blinded (or unclear). Two studies used a crossover design (Andersson *et al.*, 2007; Kesaniemi *et al.*, 1990), and the others used parallel groups. All were conducted on adults in Spain, Sweden, Finland and the USA (2). The fibre intakes in the high fibre groups ranged from 24-30.5g/day and from 6-17.4g/day in the low fibre groups, thus fibre intakes are 2-3 times greater in the high fibre groups. Mean BMI in each trial ranged between 26 and 36kg/m², and average age in each trial ranged from 41 to 59 years. The trial by Kesaniemi *et al.* (Kesaniemi *et al.*, 1990) included males only, but the other trials were mixed gender. Two trials imposed an energy intake restriction as part of each intervention diet (Thompson *et al.*, 2005; Olendzki *et al.*, 2009), and body weight decreased in each intervention group accordingly. In the other trials, body weights were unchanged or were slightly increased and these differences in weight change between trials may have impacted on blood lipid changes.

Five studies were included in the meta-analysis comparing different fibre intakes and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 48 weeks. The pooled estimate indicated that total cholesterol was 0.08mmol/L (95% CI -0.11 to 0.27) lower with consumption of a high fibre diet. This was not significantly different from zero (p=0.4). Overall heterogeneity denoted by I² was 0% (95% CI 0 to 36%). Statistically, there was no evidence that a diet higher in fibre is associated with differences in total cholesterol.

Figure 2.51 Forest plot for high fibre diets and total cholesterol (mmol/L)

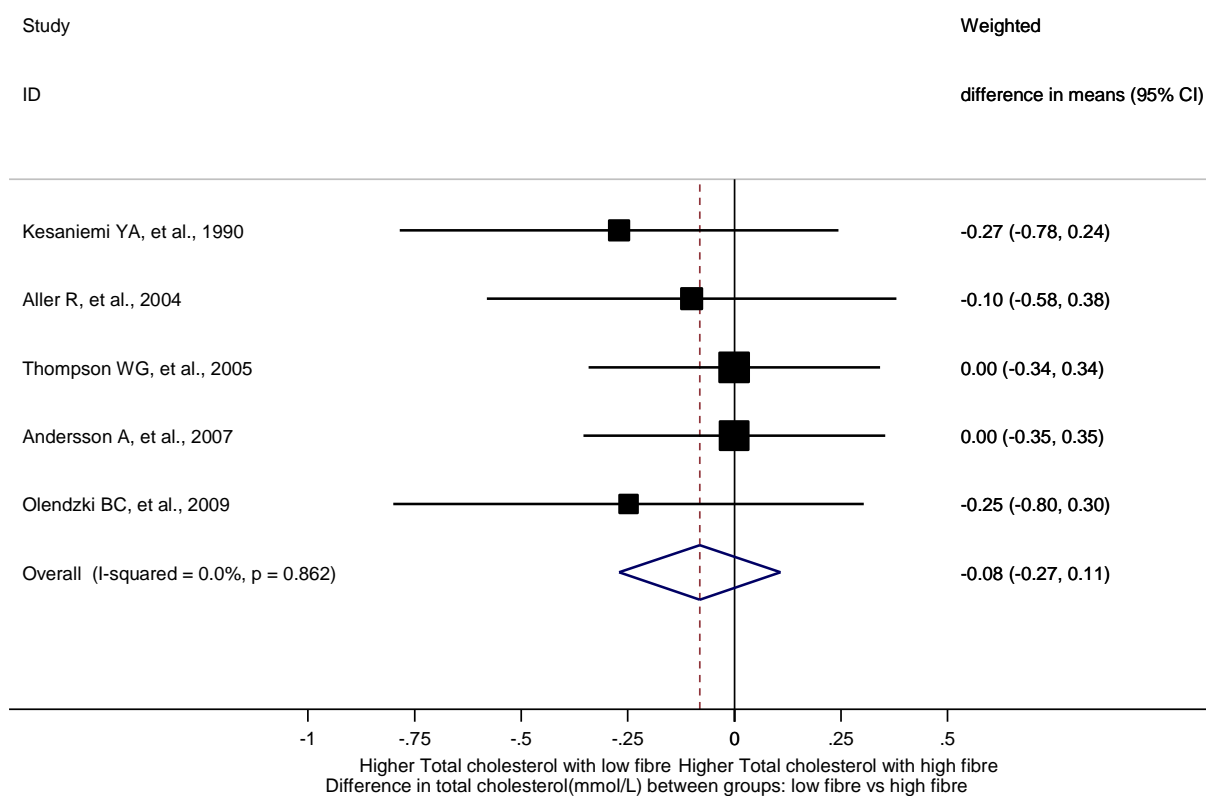


Table 2.86 Total cholesterol and dietary fibre: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
(Albertson <i>et al.</i> , 2009) 13984 National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	7 years	Food diary	Fibre from breakfast cereals (Percent of cereal consumed that was fibre)	Total cholesterol Fasting, Whole blood	1 %	-0.2 (0.18)	0.27	No adjustments made
(de Castro <i>et al.</i> , 2006) 14201/14196 Japanses-Brazilian Diabetes Study	Brazil, First and second generation Japanese	40-79 (57) %M48	647	7 years	FFQ	Fibre	Fasting total cholesterol Fasting LDL- cholesterol	1g/day	-1.250 (-2.061 to -0.437) -0.002 (-0.005 to 0.001)	<0.05 >0.05	BMI, waist, smoking, alcohol, morbidity (diabetes, hypertension, medication use)

Table 2.87 Total cholesterol and high fibre diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Aller <i>et al.</i> , 2004) *15573	High fibre	27/27	5.1 (SD 0.5)	4.9 (SD 0.9)		<0.05	Not reported	Total cholesterol	Fasting serum (mmol/L)	3 months	No change	unclear
	Low fibre	26/26	5.0 (SD 1.1)	5.0 (SD 0.9)		NS					No change	
(Andersson <i>et al.</i> , 2007) *16300	Refined grain products	30/30	5.5 (SD 0.8)	5.5 (SD 0.7)		NS		Total cholesterol	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	5.5 (SD 0.7)	5.5 (SD 0.7)		NS	0.76				Increase	
(Kesaniemi <i>et al.</i> , 1990) *14672	High fibre	34/34		5.68 (SE 0.17)			<0.05	Average of follow up assessments Total cholesterol	Serum (mmol/L)	8 weeks	No change	bias
	Low fibre	34/34		5.95 (SE 0.2)							No change	
(Olendzki <i>et al.</i> , 2009) *14590	Hypoenergetic high fibre	12/12	205.9 (SE 9.6)		-13.2 (SE 6.8)		NS	Total cholesterol	Serum (mg/dL)	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	207.0 (SE 11.1)		-18.0 (SE 7.9)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	200.2 (SE 10.5)		-8.4 (SE 7.5)		NS				Decrease	
14592	Hypoenergetic high fibre	12/12	205.9 (SE 9.6)		-11.8 (SE 7)		NS	Total cholesterol	Serum (mg/dL)	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	207.0 (SE 11.1)		-20.8 (SE 7.9)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	200.2 (SE 10.5)		-6.4 (SE 7.8)		NS				Decrease	
(Singh <i>et al.</i> , 1992) 16353	Control, normal diet	311/311	246 (SD 46.4)	240.6 (SD 40)	-2.10%			Total cholesterol	Fasting serum (mg/dL)	8 weeks	Decrease	unclear
	Low SFA, high fibre diet	310/310	243.8 (SD 44)	223.8 (SD 40)	-8.20%	0.02	0.02				Decrease	
(Thompson <i>et al.</i> , 2005) *17079	Energy restriction + dairy	21/30			-0.72 (SD 0.57)			Total cholesterol	Fasting (mM)	48 weeks	Decrease	bias
	Energy restriction + dairy + fibre	21/31			-0.72 (SD 0.56)		NS				Decrease	

*This result was used in the meta-analysis of high fibre diets and total cholesterol

HDL cholesterol, dietary fibre and high fibre diets

Summary of cohort results

One study reported data on the relationship between dietary fibre and HDL cholesterol, (Ludwig *et al.*, 1999). Dietary fibre (AOAC method) was measured by an FFQ and expressed as grams per unit energy in the CARDIA study (Ludwig *et al.*, 1999). This study showed that higher fibre density was statistically significantly associated with higher HDL cholesterol in white but not in black subjects.

The CARDIA study (Ludwig *et al.*, 1999) adjusted for an appropriate number of variables including age, gender, smoking and physical activity.

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

Summary of RCT data

Six studies provided data on the effects of diets high or low in dietary fibre on HDL cholesterol (Andersson *et al.*, 2007; Kesaniemi *et al.*, 1990; Olendzki *et al.*, 2009; Thompson *et al.*, 2005; Aller *et al.*, 2004; Singh *et al.*, 1992). Data from one study were not included in the tables or the meta-analysis due to convincing evidence of poor study quality (Singh *et al.*, 1992).

These five trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol, dietary fibre and high fibre diets.

Five studies were included in the meta-analysis comparing different fibre intakes and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 48 weeks.

The pooled estimate indicated that HDL cholesterol was 0.07mmol/L (95% CI -0.04 to 0.17) lower with consumption of a high fibre diet. This was not significantly different from zero ($p=0.2$). Overall heterogeneity denoted by I^2 was 67% (95% CI 15 to 87%). Statistically, there was no evidence that a diet higher in fibre is associated with differences in HDL cholesterol.

Figure 2.52 Forest plot for high fibre diets and HDL cholesterol (mmol/L)

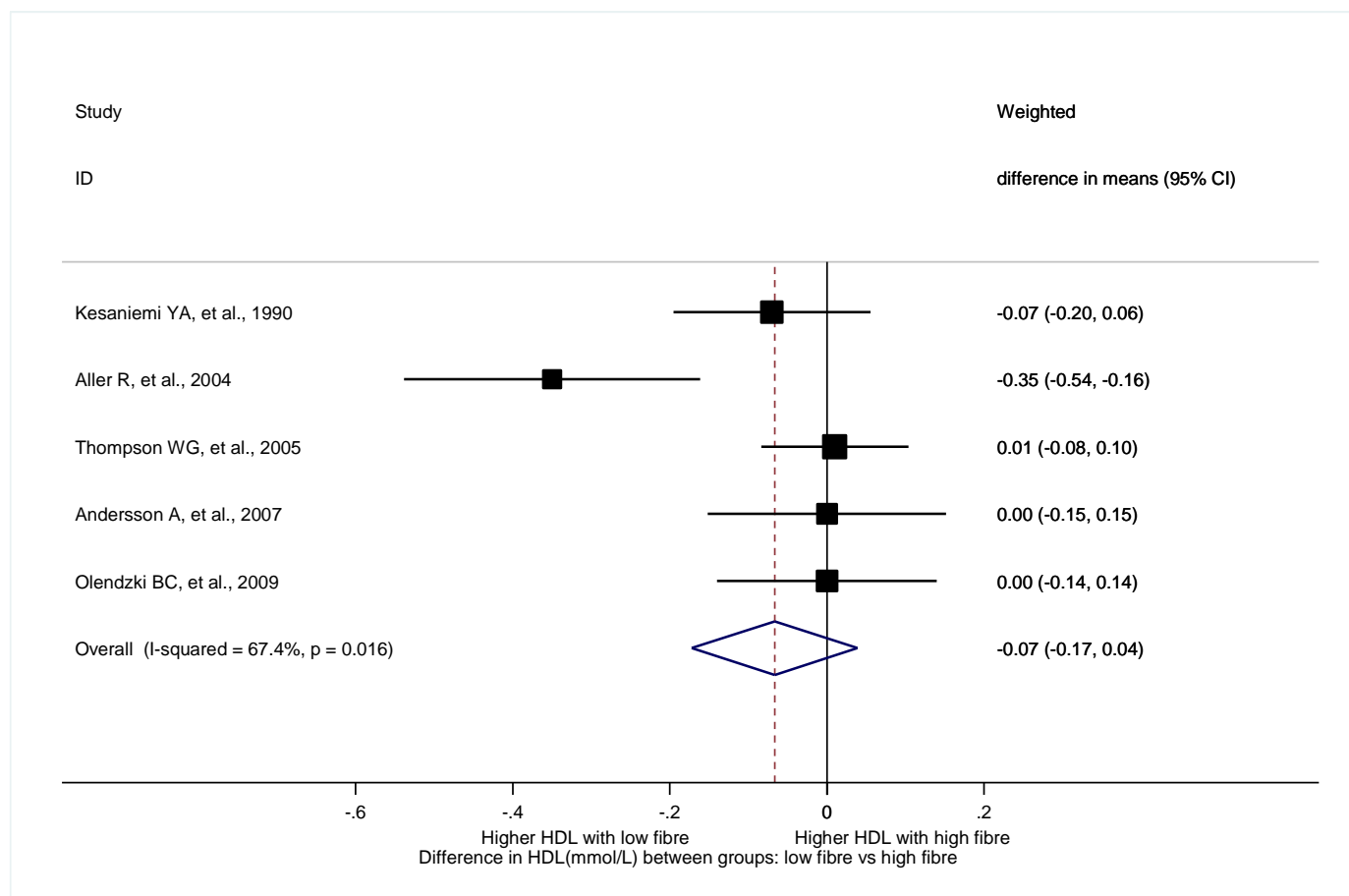


Table 2.88 HDL cholesterol and high fibre diets: cohort studies in adults

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 Details	Contrast	Exposure Units	Mean Outcome	P trend	Adjustments
(Ludwig <i>et al.</i> , 1999) 13694 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)	HDL-C Fasting, mg/dL	Race - White	(12.3) vs (5.2)	g/4184kJ/day	49.0 vs. 46.5	0.005	age, alcohol, centre, education, energy intake, HDL-C, physical activity, gender, smoking, vitamin intake
13695 The CARDIA Study								Race - Black	(12.3) vs (5.2)	g/4184kJ/day	51.4 vs. 51.5	0.28	age, alcohol, centre, education, energy intake, HDL-C, physical activity, gender, smoking, vitamin intake

Table 2.89 HDL cholesterol and high fibre diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Aller <i>et al.</i> , 2004) *15574	High fibre	27/27	1.57 (SD 0.5)	1.41 (SD 0.4)		NS	Not reported	HDL-C	Fasting serum (mmol/L)	3 months	No change	unclear
	Low fibre	26/26	1.72 (SD 0.4)	1.76 (SD 0.3)		NS					No change	
(Andersson <i>et al.</i> , 2007) *16301	Refined grain products	30/30	1.2 (SD 0.2)	1.2 (SD 0.3)		NS		HDL-C	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	1.3 (SD 0.3)	1.2 (SD 0.3)		NS	0.15				Increase	
(Kesaniemi <i>et al.</i> , 1990) *14675	High fibre	34/34		1.16 (SE 0.04)			<0.05	Average of follow up assessments HDL-C	Serum (mmol/L)	8 weeks	No change	bias
	Low fibre	34/34		1.23 (SE 0.05)							No change	
(Olendzki <i>et al.</i> , 2009) *14595	Hypoenergetic high fibre	12/12	55.3 (SE 3.7)		-2.3 (SE 1.7)		NS	HDL-C	Not reported (mg/dL)	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	53.0 (SE 4.3)		-3.9 (SE 2.0)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	49.0 (SE 4.0)		-3.9 (SE 1.9)		NS				Decrease	
14596	Hypoenergetic high fibre	12/12	55.3 (SE 3.7)		-1.3 (SE 1.8)		NS	HDL-C	Not reported (mg/dL)	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	53.0 (SE 4.3)		-2.6 (SE 2.0)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	49.0 (SE 4.0)		0.2 (SE 2.0)		NS				Decrease	
(Singh <i>et al.</i> , 1992) 16355	Control, normal diet	189/311	45.3 (SD 8.6)	43 (SD 9.4)	5%			HDL-C	Fasting serum (mg/dL)	8 weeks	Decrease	unclear
	Low SFA, high fibre diet	180/310	46.3 (SD 8)	44.3 (SD 7.4)	4.30%	NS	NS				Decrease	
(Thompson <i>et al.</i> , 2005) *17082	Energy restriction + dairy	21/30			0.07 (SD 0.16)			HDL-C	Fasting (mM)	48 weeks	Decrease	bias
	Energy restriction + dairy + fibre	21/31			0.08 (SD 0.15)		NS				Decrease	

*This result was used in the meta-analysis of high fibre diets and HDL cholesterol

LDL cholesterol, dietary fibre and high fibre diets

Summary of cohort results

Data were extracted from one publication, reporting results from one study (Ludwig *et al.*, 1999). The CARDIA study of young adults reported fibre intake as fibre density (grams/unit energy). Fibre was estimated using the AOAC method and diet was captured using a 700 item FFQ.

The CARDIA study (Ludwig *et al.*, 1999) provided evidence concerning the association between fibre density with continuous LDL cholesterol in black and white ethnic subgroups. In white participants, the mean difference in LDL cholesterol between the highest and lowest fibre density quintiles was -0.12mmol/L (4.8 mg/dL, $p=0.06$). In the black participants, however, there was no evidence of an association. This study adjusted for an appropriate number of variables including age, gender, alcohol intake and smoking status.

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

Summary of RCT data

Six studies provided data on the effects of diets high or low in dietary fibre on LDL cholesterol (Andersson *et al.*, 2007; Kesaniemi *et al.*, 1990; Olendzki *et al.*, 2009; Thompson *et al.*, 2005; Aller *et al.*, 2004; Singh *et al.*, 1992). Data from one study were not included in the tables or the meta-analysis due to convincing evidence of poor study quality (Singh *et al.*, 1992).

These five trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol, dietary fibre and high fibre diets.

Five studies were included in the meta-analysis comparing different fibre intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 48 weeks.

The pooled estimate indicated that LDL cholesterol was 0.02mmol/L (95% CI -0.15 to 0.20) lower with consumption of a high fibre diet. This was not significantly different from zero ($p=0.8$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 71%). Statistically, there was no evidence that a diet higher in fibre is associated with differences in LDL cholesterol.

Figure 2.53 Forest plot for high fibre diets and LDL cholesterol (mmol/L)

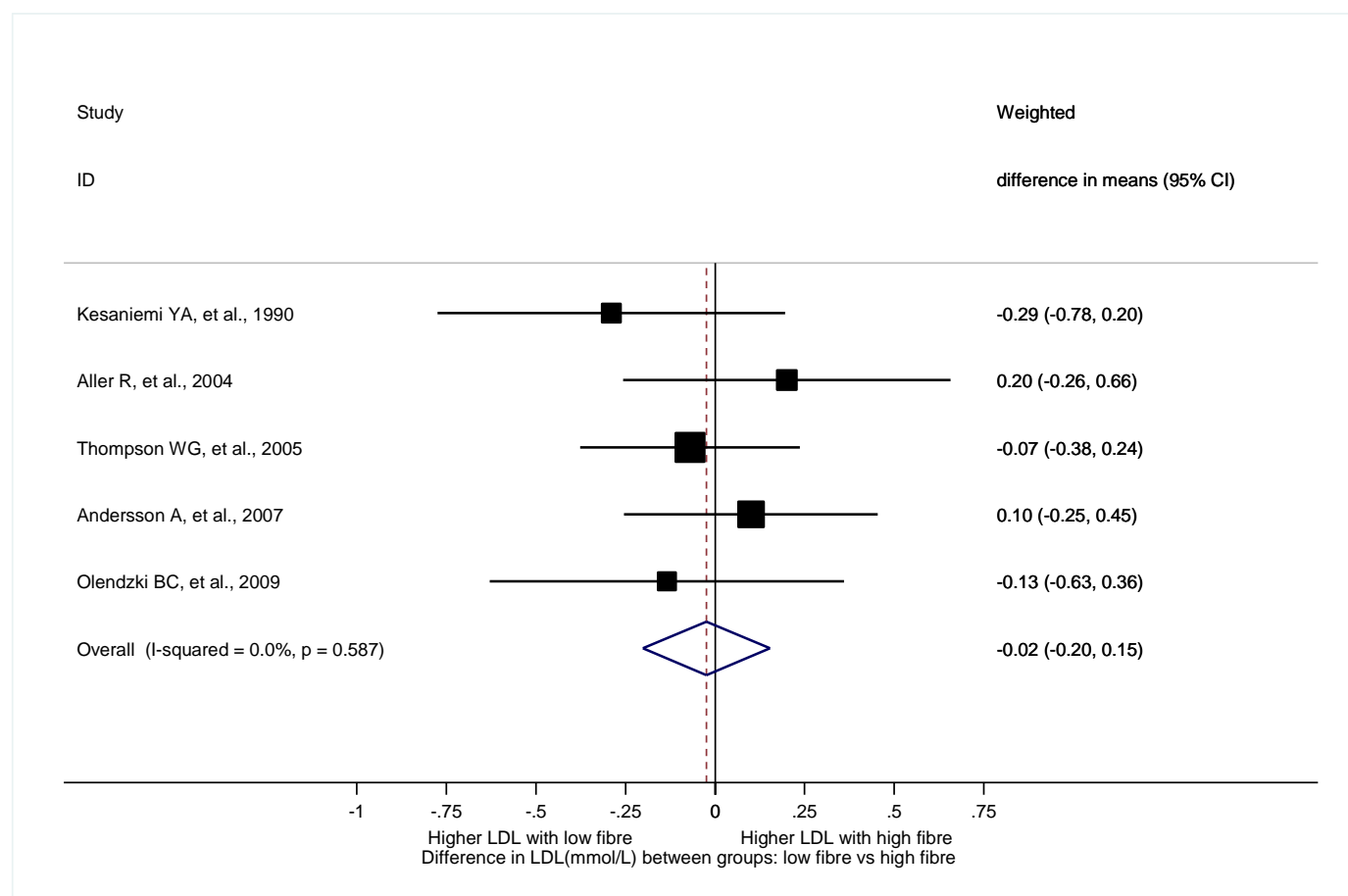


Table 2.90 LDL cholesterol and dietary fibre: cohort study in adults

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub- group Details	Contrast	Exposure Units	Mean outcome	P trend	Adjustments
(Ludwig <i>et al.</i> , 1999) 13698 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)	LDL-C Fasting, mg/dL	Race - White	(12.3) vs (5.2)	g/4184kJ/d ay	108.0 vs.112.8	0.06	age, alcohol, centre, education, energy intake, LDL-C, physical activity, gender, smoking, vitamin intake
13699 The CARDIA Study								Race - Black	(12.3) vs (5.2)	g/4184kJ/d ay	104.7 vs. 108.	0.20	age, alcohol, centre, education, energy intake, LDL-C, physical activity, gender, smoking, vitamin intake

Table 2.91 LDL cholesterol and high fibre diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Aller <i>et al.</i> , 2004) *15575	High fibre	27/27	3.5 (SD 0.9)	3.1 (SD 0.81)		<0.05	Not reported	Change in LDL-C	Fasting serum (mmol/L)	3 months	No change	unclear
	Low fibre	26/26	2.8 (SD 0.9)	2.9 (SD 0.9)		NS					No change	
(Andersson <i>et al.</i> , 2007) *16302	Refined grain products	30/30	3.7 (SD 0.8)	3.6 (SD 0.7)		NS		LDL-C	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	3.7 (SD 0.8)	3.7 (SD 0.7)		NS	0.4				Increase	
(Kesaniami <i>et al.</i> , 1990) *14674	High fibre	34/34		3.9 (SE 0.17)			<0.05	Average of follow up assessments LDL-C	Serum (mmol/L)	8 weeks	No change	bias
	Low fibre	34/34		4.19 (SE 0.18)							No change	
(Olendzki <i>et al.</i> , 2009) *14593	Hypoenergetic high fibre	12/12	129.9 (SE 8.1)		-8.1 (SE 6.2)		NS	LDL-C	Not reported (mg/dL)	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	119.8 (SE 9.4)		-6.0 (SE 7.1)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	116.2 (SE 8.9)		-0.8 (SE 6.7)		NS				Decrease	
14594	Hypoenergetic high fibre	12/12	129.9 (SE 8.1)		-7.2 (SE 6.3)		NS	LDL-C	Not reported (mg/dL)	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	119.8 (SE 9.4)		-4 (SE 7.1)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	116.2 (SE 8.9)		-1.5 (SE 7.0)		NS				Decrease	
(Singh <i>et al.</i> , 1992) 16354	Control, normal diet	311/311	166.33 (SD 82)		2.70%			LDL-C	Fasting serum (mg/dL)	8 weeks	Decrease	unclear
	Low SFA, high fibre diet	310/310	168.6 (SD 85.2)	152.4 (SD 67.4)	-9.80%	0.02	0.05				Decrease	
(Thompson <i>et al.</i> , 2005) *17083	Energy restriction + dairy	21/30			-0.63 (SD 0.47)			LDL-C	Fasting (mM)	48 weeks	Decrease	bias
	Energy restriction + dairy + fibre	21/31			-0.7 (SD 0.54)		NS				Decrease	

*This result was used in the meta-analysis for high fibre diets and LDL cholesterol

Triacylglycerol, dietary fibre and high fibre diets

Summary of cohort results

One study, the CARDIA study (Ludwig *et al.*, 1999), provided evidence concerning the association between fibre density (grams/unit energy) and fasting TAG in young adult black and white ethnic subgroups. Fibre was estimated using the AOAC method and diet was captured using a 700 item FFQ. This study showed a borderline statistically significant association between fibre density and TAG in white but not black subgroups. In the white sub-group higher fibre density corresponded with lower TAG levels. The CARDIA study (Ludwig *et al.*, 1999) adjusted for an appropriate number of variables including age, gender, alcohol intake and smoking status, but not BMI.

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

Summary of RCT data

Six studies provided data on the effects of diets high or low in dietary fibre on fasting TAG levels (Andersson *et al.*, 2007; Kesaniemi *et al.*, 1990; Olendzki *et al.*, 2009; Thompson *et al.*, 2005; Aller *et al.*, 2004; Singh *et al.*, 1992). Data from one study were not included in the tables or the meta-analysis due to convincing evidence of poor study quality (Singh *et al.*, 1992).

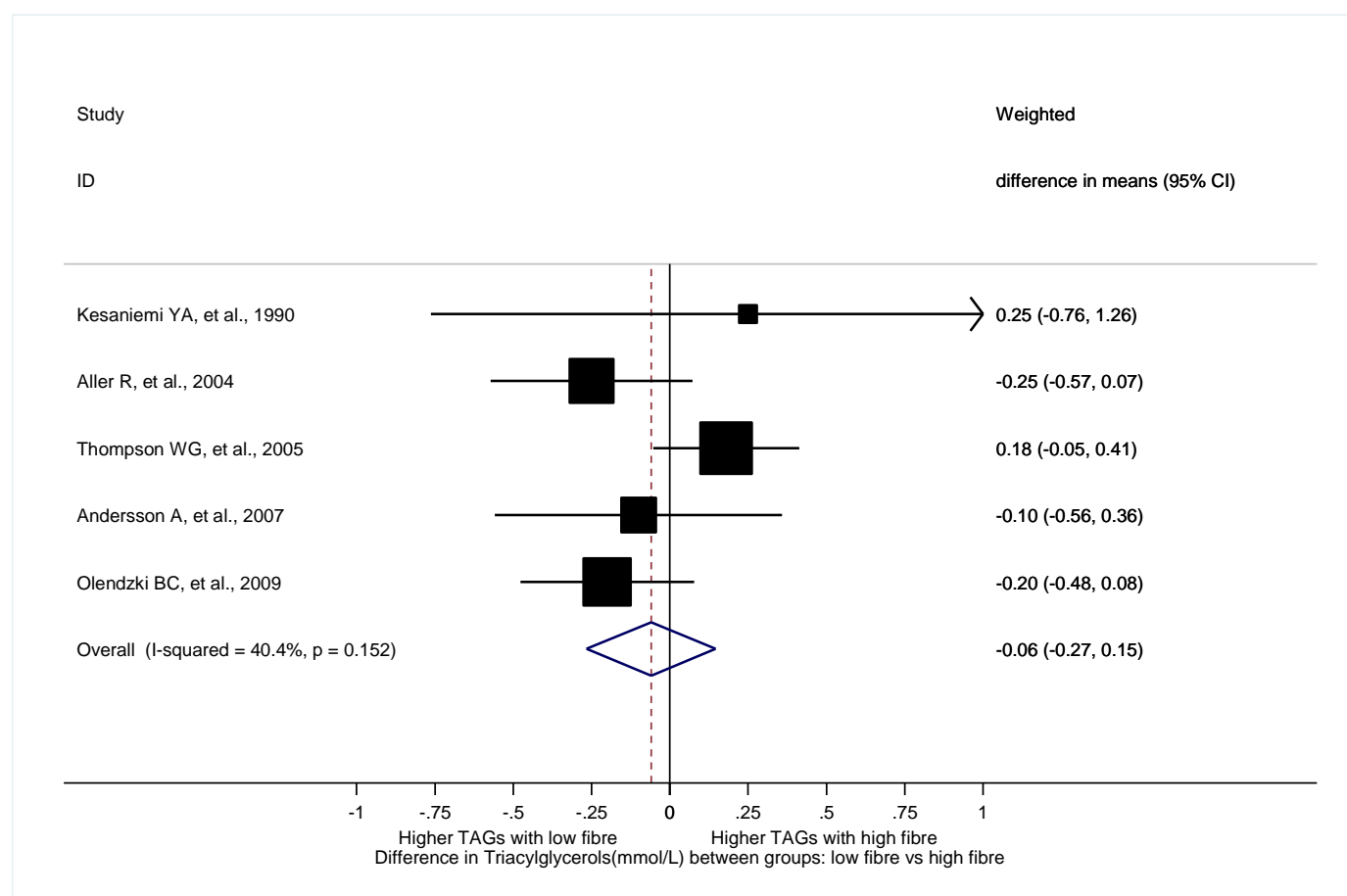
One study also presented results on non-esterified fatty acids and high fibre diets (Andersson *et al.*, 2007).

These five trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol, dietary fibre and high fibre diets.

Five studies were included in the meta-analysis comparing different fibre intakes and changes in TAG reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 48 weeks.

The pooled estimate indicated that TAG were 0.06mmol/L (95% CI -0.15 to 0.27) lower with consumption of a high fibre diet. This was not significantly different from zero ($p=0.57$). Overall heterogeneity denoted by I^2 was 40% (95% CI 0 to 78%). Statistically, there was no evidence that a diet higher in fibre is associated with changes in TAG.

Figure 2.54 Forest plot for high fibre diets and TAG (mmol/L)



One study also explored the effects of a diet rich in wholegrains or a diet containing refined grains on non-esterified fatty acids using 34 overweight and obese participants (Andersson *et al.*, 2007). Participants were requested to consume the intervention food products as part of a free living diet plan. After six weeks, the authors concluded that the dietary intervention had not affected non-esterified fatty acids within or between groups.

Table 2.92 Triacylglycerol 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 Details	Contrast	Exposure Units	Mean Outcome	P trend	Adjustments
(Ludwig <i>et al.</i> , 1999) 13690 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)	TAG Fasting, mg/dL	Race - White	(12.3) vs (5.2)	g/4184kJ/day	80.5 vs. 88.5	0.05	age, alcohol, centre, education, energy intake, physical activity, gender, smoking, blood TAG, vitamin intake
13691 The CARDIA Study								Race - Black	(12.3) vs (5.2)	g/4184kJ/day	65.8 vs. 70.1	0.11	As above

Table 2.93 Triacylglycerol, fatty acids and high fibre diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Aller <i>et al.</i> , 2004) *15576	High fibre	27/27	1 (SD 0.4)	1.02 (SD 0.3)		NS	Not reported	TAG	Fasting serum (mmol/L)	3 months	No change	unclear
	Low fibre	26/26	1.07 (SD 0.6)	1.27 (SD 0.8)		NS					No change	
(Andersson <i>et al.</i> , 2007) *16303	Refined grain products	30/30	1.3 (SD 0.6)	1.6 (SD 1.0)		<0.05		TAG	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	1.4 (SD 0.8)	1.5 (SD 0.8)		NS	0.19				Increase	
(Kesanani <i>et al.</i> , 1990) 14676	High fibre	34/34		2.11 (SE 0.38)			NS	Average of follow up assessments TAG	Serum (mmol/L)	8 weeks	No change	bias
	Low fibre	34/34		1.86 (SE 0.35)							No change	
(Olendzki <i>et al.</i> , 2009) *14597	Hypoenergetic high fibre	12/12	4.6 (SE 0.1)		-0.2 (SE 0.1)		NS	TAG	Not reported	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	5.1 (SE 0.1)		-0.3 (SE 0.1)		NS				Decrease	
	Hypoenergetic low saturated fat	10/10	5.1 (SE 0.1)		-0.1 (SE 0.1)		NS				Decrease	
14601	Hypoenergetic high fibre	12/12	4.6 (SE 0.1)		-0.2 (SE 0.1)		NS	TAG	Not reported	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	5.1 (SE 0.1)		-0.6 (SE 0.1)		NS				Decrease	

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
	Hypoenergetic low saturated fat	10/10	5.1 (SE 0.1)		-0.2 (SE 0.1)		NS				Decrease	
(Singh <i>et al.</i> , 1992) 16356	Control, normal diet	189/311	160.4 (SD 20)	154.6 (SD 16.8)	-3.60%			TAG	Fasting serum (mg/dL)	8 weeks	Decrease	unclear
	Low SFA, high fibre diet	180/310	158.5 (SD 18.6)	140.6 (SD 15.3)	-11.20%	0.01	0.02				Decrease	
(Thompson <i>et al.</i> , 2005) *17081	Energy restriction + dairy	21/30			-0.36 (SD 0.40)			TAG	Fasting (mM)	48 weeks	Decrease	bias
	Energy restriction + dairy + fibre	21/31			-0.18 (SD 0.37)		NS				Decrease	
Fatty acid												
(Andersson <i>et al.</i> , 2007) 16304	Refined grain products	30/30	0.63 (SD 0.17)	0.62 (SD 0.18)		NS		Free fatty acid	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	0.56 (SD 0.19)	0.61 (SD 0.18)		NS	0.99				Increase	

*This result was used in the meta-analysis for high fibre diets and TAG

Total cholesterol:HDL ratio, dietary fibre and high fibre diets

No cohort studies reported results concerning dietary fibre and total cholesterol:HDL ratio.

Summary of RCT data

Two studies provided data on the effects of high fibre diets on the TC:HDL ratio in generally healthy adults. Body weights were unchanged in one trial (Aller *et al.*, 2004) but decreased in another (Olendzki *et al.*, 2009). Aller *et al.* (Aller *et al.*, 2004) reported that over three months the TC:HDL ratio did not change in either low (10.4g/day) or high (30.5g/day) fibre diet groups. Similarly, the six month trial conducted by Olendzki *et al.* (Olendzki *et al.*, 2009) which investigated the effects of a high fibre diet, a high fibre and low saturated fat diet and a low saturated fat diet reported no differences between diet groups. In this study, all diets were hypoenergetic.

These two trials therefore suggest no effect of high fibre diets on the TC:HDL ratio.

Table 2.94 Total cholesterol:HDL ratio and high fibre diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Olendzki <i>et al.</i> , 2009) 14602	Hypoenergetic high fibre	12/12	3.9 (SE 0.3)		-0.1 (SE 0.1)		NS	Total cholesterol :HDL ratio	3 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	4.1 (SE 0.3)		-0.1 (SE 0.1)		NS			Decrease	
	Hypoenergetic low saturated fat	10/10	4.1 (SE 0.3)		0.2 (SE 0.1)		NS			Decrease	
14603	Hypoenergetic high fibre	12/12	3.9 (SE 0.3)		-0.2 (SE 0.1)		NS	Total cholesterol :HDL ratio	6 months	Decrease	unclear
	Hypoenergetic high fibre and low saturated fat	9/9	4.1 (SE 0.3)		-0.1 (SE 0.1)		NS			Decrease	
	Hypoenergetic low saturated fat	10/10	4.1 (SE 0.3)		-0.3 (SE 0.1)		NS			Decrease	
(Aller <i>et al.</i> , 2004) 15578	High fibre	27/27	3.73 (SD 0.9)	3.65 (SD 1.1)		NS	Not reported	Total cholesterol :HDL ratio	3 months	No change	unclear
	Low fibre	26/26	3.35 (SD 0.8)	3.25 (SD 1.2)		NS				No change	

Total cholesterol:LDL ratio, dietary fibre and high fibre diets

No cohort studies reported results concerning dietary fibre and TC:LDL ratio.

Summary of RCT data

One trial of healthy subjects provided data on TC:LDL ratio and high fibre diets (Aller *et al.*, 2004). Body weights were unchanged in this trial.

The study reported by Aller *et al.* (Aller *et al.*, 2004) explored the effects of fibre on blood glucose and lipids over a three month period. Total cholesterol:LDL ratio did not change in either low (10.4g/day) or high (30.5g/day) fibre diet groups.

Table 2.95 Total cholesterol:LDL ratio and high fibre diets: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Aller <i>et al.</i> , 2004) 15577	High fibre	27/27	1.61 (SD 0.2)	1.65 (SD 0.2)	NS	Not reported	TC : LDL ratio	3 months	No change	unclear
	Low fibre	26/26	1.88 (SD 0.3)	1.87 (SD 0.4)	NS				No change	

LDL:HDL cholesterol ratio and high fibre diets

No cohort studies reported results concerning dietary fibre and LDL:HDL cholesterol ratio.

Summary of RCT data

One study explored the effects of fibre on the ratio of LDL to HDL cholesterol over a three month period (Aller *et al.*, 2004). Fifty three healthy eligible subjects were randomised to receive a diet with 10.4g fibre (low fibre diet) or a diet with 30.5g fibre (high fibre diet). Body weights were unchanged in this trial. The LDL:HDL cholesterol ratio did not change in either diet group.

Table 2.96 LDL:HDL cholesterol ratio and high fibre diets: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Aller <i>et al.</i> , 2004) 15579	High fibre	27/27	2.21 (SD 0.9)	2.17 (SD 0.8)	NS	Not reported	Change in LDL:HDL cholesterol ratio	3 months	No change	unclear
	Low fibre	26/26	1.65 (SD 0.7)	1.66 (SD 1.1)	NS				No change	

Results – Fibre isolates, fermentable oligosaccharides

Intakes of fermentable oligosaccharides in Western populations have been estimated to range between 2 to 12g per day (Roberfroid, 1993), certain plants being rich sources such as artichokes, onions, asparagus and chicory. Additionally, certain fermentable oligosaccharides are used as a food additive, either for gelling and/or thickening effects or as a prebiotic. Various fructan preparations have been explored in studies with an intervention duration ranging from two weeks to six months. The range of different fermentable oligosaccharides here included mixed inulin-type fructans which are a mixture of low, medium and high degree of polymerisation fructans, such as Synergy 1 or Synergy HP (Forcheron and Beylot, 2007), Yacon root syrup (Genta *et al.*, 2009), or inulin (Raftiline) with an average degree of polymerisation of 10 to 25 (Davidson *et al.*, 1998; Jackson *et al.*, 1999; Letexier *et al.*, 2003). These were administered in doses ranging from 10 to 18g/day, and compared with placebo or control products such as maltodextrin. For a review of the chemistry, nomenclature and functional food properties of the inulin-type fructans, see (Roberfroid, 2007).

Various methods of administration were employed to incorporate the fermentable oligosaccharide products into the diet. The majority of studies asked the participants to add the powdered product to either food or drinks, generally in two or three doses across the day (Forcheron and Beylot, 2007; Letexier *et al.*, 2003; Jackson *et al.*, 1999). Alternatively, the fermentable oligosaccharides were incorporated into food products such as spreads (Davidson *et al.*, 1998), or consumed as a naturally rich source e.g. yacon root syrup (Genta *et al.*, 2009).

Total cholesterol and fibre isolates, fermentable oligosaccharides

No cohort studies reported results concerning fibre isolates, fermentable oligosaccharides and total cholesterol.

Summary of RCT data

Five studies provided data on the effects of high fermentable oligosaccharide diets on total blood cholesterol (Davidson *et al.*, 1998; Forcheron and Beylot, 2007; Genta *et al.*, 2009; Jackson *et al.*, 1999; Letexier *et al.*, 2003).

Two studies used a crossover design (Letexier *et al.*, 2003; Davidson *et al.*, 1998), and the others used parallel groups. All were conducted on adults in France (2), Argentina, the UK and the USA. The studies were small with a median number of participants within the trials of 28. All were double blind. The study by Letexier *et al.* (Letexier *et al.*, 2003) included only participants with a BMI of less than 25kg/m², but the other studies included lean and overweight, or mainly overweight or obese participants. The study by Genta *et al.* (Genta *et al.*, 2009) included only women, but the other studies were mixed gender. The study durations ranged from six weeks to six months.

Three studies compared 10g/day of inulin with a similar amount of maltodextrin (Jackson *et al.*, 1999; Letexier *et al.*, 2003; Forcheron and Beylot, 2007). The study by Genta *et al.* administered fermentable oligosaccharides in the form of yacon syrup, a naturally rich source (Genta *et al.*, 2009), and this was compared with a similar dose of placebo syrup. In the study by Davidson *et al.* (Davidson *et al.*, 1998), 18g/d of inulin was incorporated into chocolate, spreads and sweeteners and compared with un-supplemented products.

Body weights were unchanged in all trials other than in Genta *et al.* in which the authors reported that there was a decrease in the low dose yacon syrup group (Genta *et al.*, 2009).

All five studies were included in the meta-analysis comparing different oligosaccharide intakes and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months. It should be noted that in the study by Jackson *et al.* (Jackson *et al.*, 1999) the TC values were significantly higher in the placebo group at baseline due to incomplete stratified randomisation, and this differential remained throughout the study. The 8-week follow up data only were provided, rather than changes from baseline, and it is these values that have been included in the meta-analysis.

The pooled estimate indicated that total cholesterol was 0.13mmol/L (95% CI -0.30 to 0.55) lower with consumption of a diet higher in oligosaccharides. This was not significantly different from zero ($p=0.57$). Overall heterogeneity denoted by I^2 was 69% (95% CI 22 to 88%). Statistically, there was no evidence that diets higher in fermentable oligosaccharides are associated with changes in total cholesterol.

Figure 2.55 Forest plot for fibre isolates, fermentable oligosaccharides and total cholesterol (mmol/L)

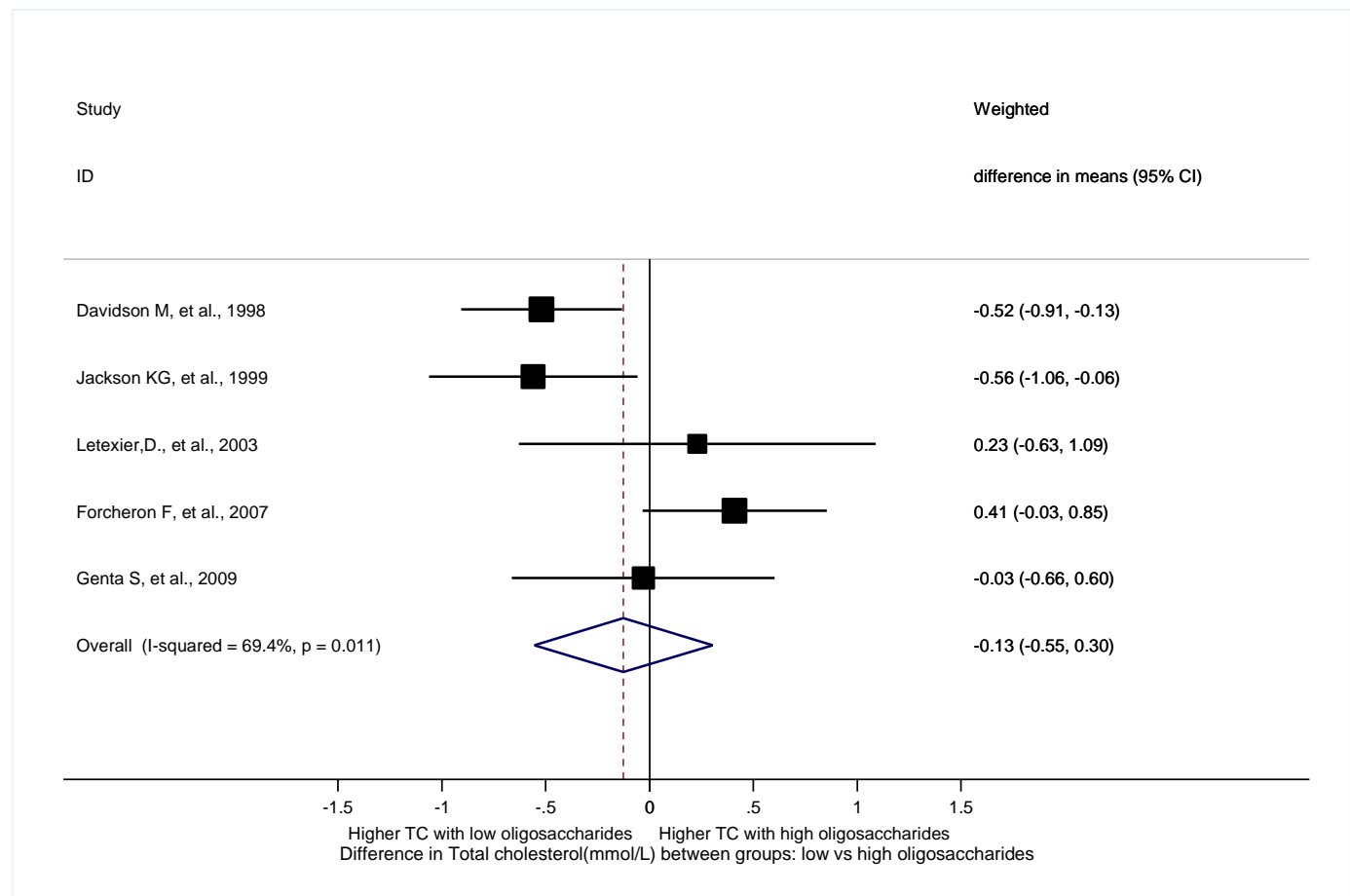


Table 2.97 Total cholesterol and fibre isolates, fermentable oligosaccharides: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Davidson <i>et al.</i> , 1998) *17577	Control	21/25	5.88 (SE 0.12)	6.28 (SE 0.12)	<0.05	<0.05	Total cholesterol	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Inulin	21/25	6.16 (SE 0.13)	6.07 (SE 0.17)	NS					No change	
(Forcheron and Beylot, 2007) *14827	Fructans	9/10	4.48 (SE 0.16)	4.14 (SE 0.16)	<0.05	NS	Total cholesterol	Fasting plasma (mmol/L)	6 months	No change	No bias
	Placebo	8/10	3.91 (SE 0.33)	3.73 (SE 0.16)						No change	
(Genta <i>et al.</i> , 2009) *14553	Low dose fructooligosaccharide syrup	completers not reported/20	5.28 (SD 0.8)	5.17 (SD 0.97)	NS	Not reported	Total cholesterol	Fasting serum (mmol/L)	120 days	Decrease	No bias
	Placebo syrup	15/15	5.33 (SD 0.97)	5.2 (SD 1.07)	NS					No change	
(Jackson <i>et al.</i> , 1999) *14795	Inulin	27/27	5.86 (SD 1)	5.9 (SD 0.97)	NS	NS	Total cholesterol	Fasting plasma (mmol/L)	8 weeks	No change	No bias
	Placebo	27/27	6.43 (SD 0.79)	6.46 (SD 0.91)	NS					No change	
14796	Inulin	27/27	5.86 (SD 1)	5.87 (SD 0.9)	NS	NS	Total cholesterol	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Placebo	27/27	6.43 (SD 0.79)	6.23 (SD 0.75)	NS					No change	
(Letexier <i>et al.</i> , 2003) *14841	Inulin	8/8		4.35 (SE 0.3)		NS	Total cholesterol	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Placebo	8/8		4.12 (SE 0.32)						No change	

*This result was used in the meta-analysis of fermentable oligosaccharides and total cholesterol

HDL Cholesterol and fibre isolates, fermentable oligosaccharides

No cohort studies reported results concerning fibre isolates, fermentable oligosaccharides and HDL cholesterol.

Summary of RCT data

Five studies provided data on the effects of high fermentable oligosaccharide diets on HDL cholesterol, and all were included in a meta-analysis (Davidson *et al.*, 1998; Forcheron and Beylot, 2007; Genta *et al.*, 2009; Jackson *et al.*, 1999; Letexier *et al.*, 2003). Details of these studies are provided in the section on total cholesterol.

The pooled estimate indicated that HDL cholesterol was 0.04mmol/L (95% CI -0.12 to 0.20) higher with consumption of a high oligosaccharide diet. This was not significantly different from zero ($p=0.60$). Overall heterogeneity denoted by I^2 was 69% (95% CI 20 to 88%). Statistically, there was no evidence that a diet higher in oligosaccharides is associated with changes in HDL cholesterol.

Figure 2.56 Forest plot for fibre isolates, fermentable oligosaccharides and HDL cholesterol (mmol/L)

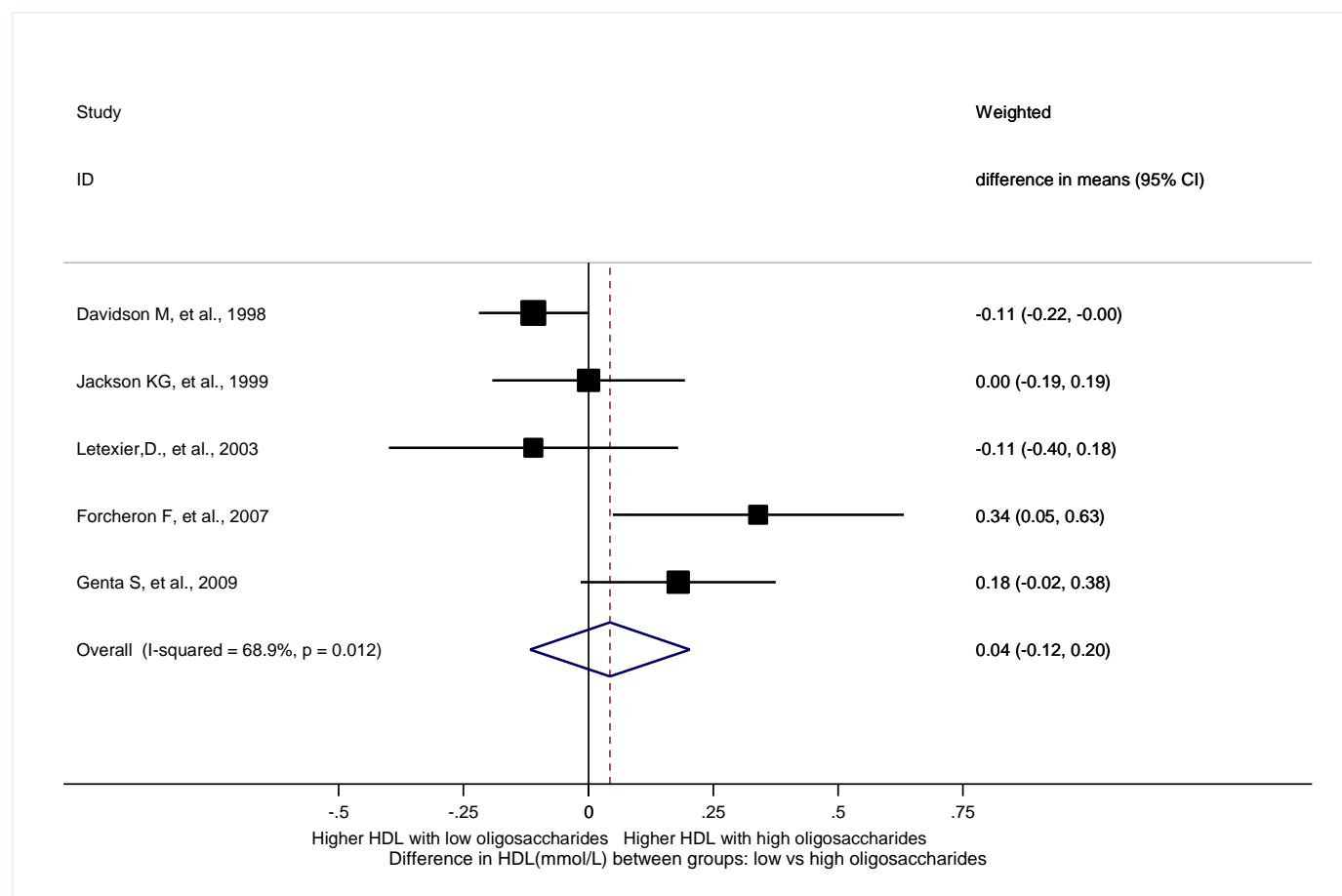


Table 2.98 HDL cholesterol and fibre isolates, fermentable oligosaccharides: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Davidson <i>et al.</i> , 1998) *17578	Control	21/25	1.29 (SE 0.06)	1.39 (SE 0.07)			HDL-C	Serum Fasting (mmol/L)	6 weeks	No change	No bias
	Inulin	21/25	1.34 (SE 0.07)	1.35 (SE 0.07)		NS				No change	
(Letexier <i>et al.</i> , 2003) *14840	Inulin	8/8		1.31 (SE 0.10)		NS	HDL-C	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Placebo	8/8		1.2 (SE 0.11)						No change	
(Forcheron and Beylot, 2007) *14829	Fructans	9/10	1.29 (SE 0.09)	1.47 (SE 0.11)	<0.05	NS	HDL-C	Fasting Plasma, (mmol/L)	6 months	No change	No bias
	Placebo	8/10	1.03 (SE 0.09)	1.13 (SE 0.1)						No change	
(Genta <i>et al.</i> , 2009) *14555	Low dose fructooligosaccharide syrup	completers not reported/20	1.2 (SD 0.16)	1.48 (SD 0.33)	NS	Not reported	HDL-C	Fasting Serum, (mmol/L)	120 days	Decrease	No bias
	Placebo syrup	15/15	1.14 (SD 0.35)	1.3 (SD 0.3)	NS					No change	
(Jackson <i>et al.</i> , 1999) *14801	Inulin	27/27	1.24 (SD 0.28)	1.31 (SD 0.33)	NS	NS	HDL-C	Fasting Plasma, (mmol/L)	8 weeks	No change	No bias
	Placebo	27/27	1.256 (SD 0.28)	1.31 (SD 0.39)	NS					No change	
14802	Inulin	27/27	1.24 (SD 0.28)	1.32 (SD 0.39)	NS	NS	HDL-C	Fasting Plasma, (mmol/L)	12 weeks	No change	No bias
	Placebo	27/27	1.26 (SD 0.28)	1.31 (SD 0.45)	NS					No change	

*This result was used in the meta-analysis of fermentable oligosaccharides and HDL cholesterol

LDL cholesterol and fibre isolates, fermentable oligosaccharides

No cohort studies reported results concerning fibre isolates, fermentable oligosaccharides and LDL cholesterol.

Summary of RCT data

Five studies provided data on the effects of high fermentable oligosaccharide diets on LDL cholesterol and all were included in a meta-analysis (Davidson *et al.*, 1998;Forcheron and Beylot, 2007;Genta *et al.*, 2009;Jackson *et al.*, 1999;Letexier *et al.*, 2003). Details of these studies are provided in the section on total cholesterol.

The pooled estimate indicated that LDL was 0.39mmol/L (95% CI 0.03 to 0.76) lower with consumption of a diet higher in oligosaccharides. This was significantly different from zero ($p=0.04$). Overall heterogeneity denoted by I^2 was 73% (95% CI 32 to 89%). Statistically, there was evidence that diets higher in fermentable oligosaccharides are associated with lower levels of LDL cholesterol.

Figure 2.57 Forest plot for fibre isolates, fermentable oligosaccharides and LDL cholesterol (mmol/L)

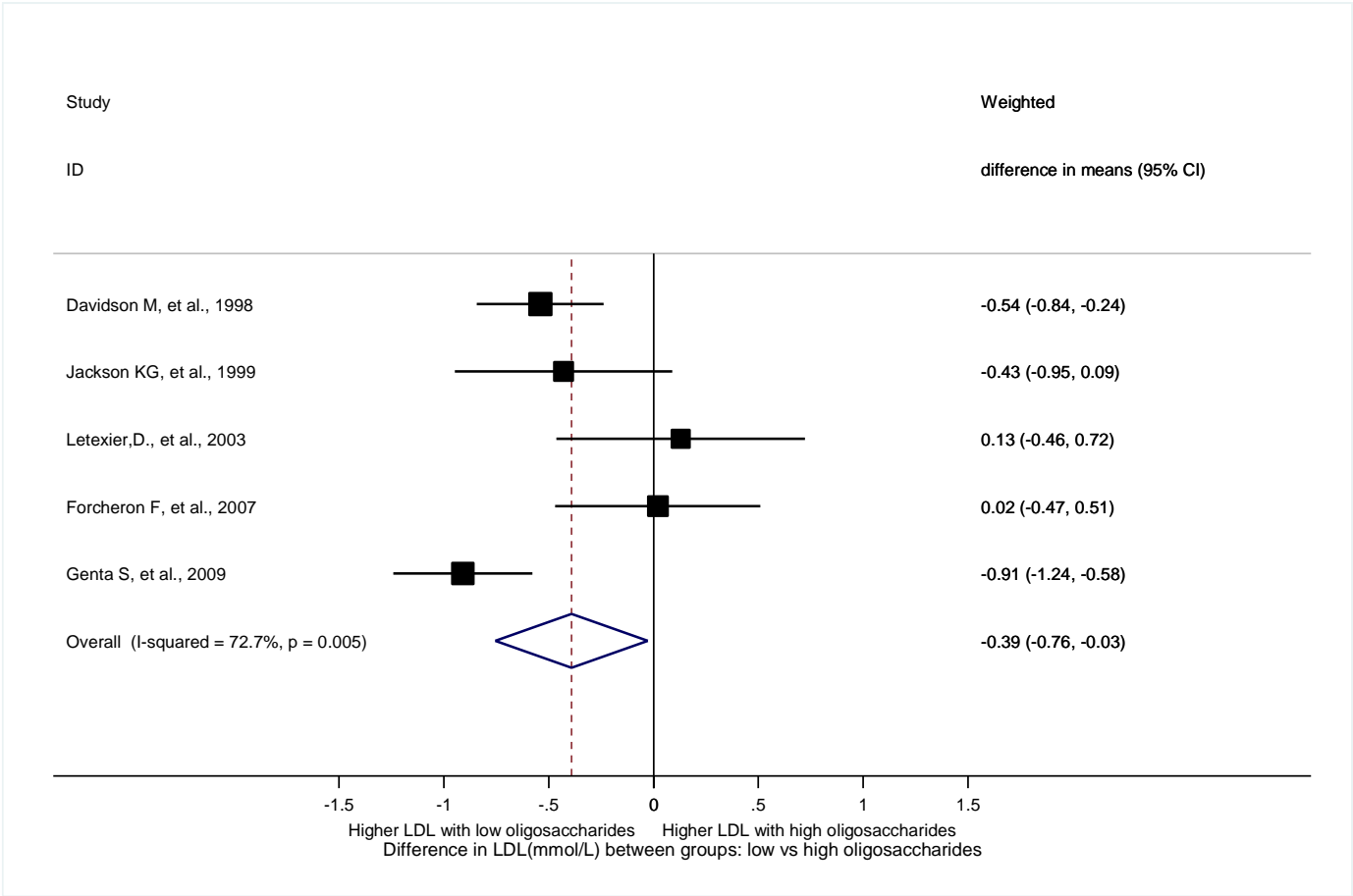


Table 2.99 LDL cholesterol and fibre isolates, fermentable oligosaccharides: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outco me	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Davidson <i>et al.</i> , 1998) *17576	Control	21/25	3.68 (SE 0.10)	4.10 (SE 0.10)			LDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Inulin	21/25	4.08 (SE 0.09)	3.98 (SE 0.12)		NS				No change	
(Letexier <i>et al.</i> , 2003) *14840	Inulin	8/8		2.90 (SE 0.22)		NS	LDL-C	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Placebo	8/8		2.77 (SE 0.21)						No change	
(Genta <i>et al.</i> , 2009) *14554	Low dose fructooligosaccharide syrup	completers not reported/20	3.54 (SD 0.71)	2.52 (SD 0.26)	0.05	Not reported	LDL-C	Fasting serum (mmol/L)	120 days	Decrease	No bias
	Placebo syrup	15/15	3.64 (SD 0.63)	3.43 (SD 0.71)	NS					No change	
(Forcheron and Beylot, 2007) *14830	Fructans	9/10	2.88 (SE 0.13)	2.33 (SE 0.2)	<0.05	NS	LDL-C	Fasting plasma (mmol/L)	6 months	No change	No bias
	Placebo	8/10	2.55 (SE 0.33)	2.31 (SE 0.15)						No change	
(Jackson <i>et al.</i> , 1999) *14804	Inulin	27/27	3.97 (SD 0.86)	4 (SD 0.85)	NS	NS	LDL-C	Fasting plasma (mmol/L)	8 weeks	No change	No bias
	Placebo	27/27	4.55 (SD 0.92)	4.43 (SD 1.08)	NS					No change	
14805	Inulin	27/27	3.97 (SD 0.86)	3.85 (SD 0.76)	NS	NS	LDL-C	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Placebo	27/27	4.55 (SD 0.92)	4.24 (SD 0.93)	NS					No change	

*This result was used in the meta-analysis for fermentable oligosaccharides and LDL cholesterol

Triacylglycerol and fibre isolates, fermentable oligosaccharides

No cohort studies reported results concerning fermentable oligosaccharides and TAG.

Summary of RCT data

Five studies provided data on the effects of high fermentable oligosaccharide diets on TAG and all were included in a meta-analysis (Davidson *et al.*, 1998;Forcheron and Beylot, 2007;Genta *et al.*, 2009;Jackson *et al.*, 1999;Letexier *et al.*, 2003). The five trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in total cholesterol.

The pooled estimate indicated that TAG were 0.13mmol/L (95% CI 0 to 0.27) lower with consumption of a high fermentable oligosaccharide diet. This was not significantly different from zero (p=0.06). Overall heterogeneity denoted by I² was 0% (95% CI 0 to 79%). Statistically, there was no evidence that a diet higher in fermentable oligosaccharides is associated with differences in TAG levels.

Figure 2.58 Forest plot for fibre isolates, fermentable oligosaccharides and TAG (mmol/L)

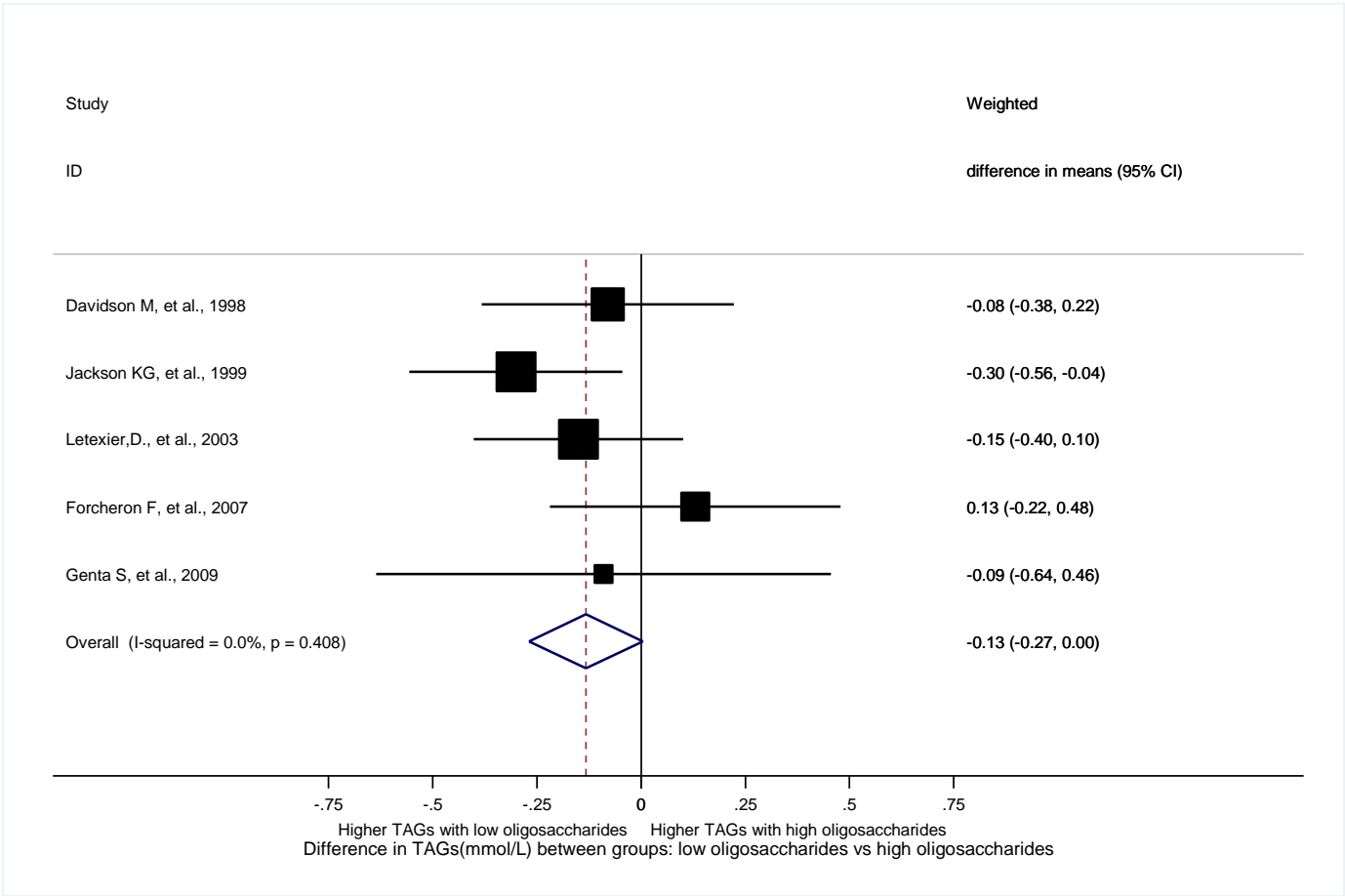


Table 2.100 Triacylglycerol and fibre isolates, fermentable oligosaccharides: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Davidson <i>et al.</i> , 1998) *17580	Control	21/25	1.65 (SE 0.11)	1.70 (SE 0.17)			TAG	Serum fasting (mmol/L)	6 weeks	No change	No bias
	Inulin	21/25	1.66 (SE 0.14)	1.61 (SE 0.08)		NS				No change	
(Genta <i>et al.</i> , 2009) *14556	Low dose fructooligosaccharide syrup	completers not reported/20	2.02 (SD 1.1)	2.1 (SD 0.97)	NS	Not reported	TAG	Fasting serum (mmol/L)	120 days	Decrease	No bias
	Placebo syrup	15/15	2.28 (SD 0.97)	2.19 (SD 0.78)	NS					No change	
(Forcheron and Beylot, 2007) *14826	Fructans	9/10	0.71 (SE 0.07)	0.77 (SE 0.14)		NS	TAG	Fasting plasma (mmol/L)	6 months	No change	No bias
	Placebo	8/10	0.78 (SE 0.16)	0.64 (SE 0.11)						No change	
(Jackson <i>et al.</i> , 1999) *14798	Inulin	27/27	1.46 (SD 0.55)	1.29 (SD 0.35)	NS	<0.05	TAG	Fasting plasma (mmol/L)	8 weeks	No change	No bias
	Placebo	27/27	1.4 (SD 0.4)	1.59 (SD 0.58)	NS						
14799	Inulin	27/27	1.46 (SD 0.55)	1.45 (SD 0.61)	NS	NS	TAG	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Placebo	27/27	1.4 (SD 0.4)	1.51 (SD 0.54)	NS					No change	
(Letexier <i>et al.</i> , 2003) *14840	Inulin	8/8		0.77 (SE 0.08)		<0.05	TAG	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Placebo	8/8		0.92 (SE 0.1)						No change	

*This result was used in the meta-analysis for fermentable oligosaccharides and TAG

LDL:HDL cholesterol ratio and fibre isolates, fermentable oligosaccharides

No cohort studies reported results concerning fermentable oligosaccharides and LDL:HDL cholesterol ratio.

Summary of RCT data

Two studies explored the effects of fermentable oligosaccharide intake on the ratio of LDL:HDL cholesterol in male and female adults (Jackson *et al.*, 1999; Davidson *et al.*, 1998). Body weights were unchanged throughout the two trials. In the study by Jackson *et al.* (Jackson *et al.*, 1999) 54 British participants were randomised to receive inulin administered as two 5g sachets per day or a comparable placebo administered in the same manner. LDL:HDL cholesterol ratio, measured at the end of the intervention, at eight weeks and at 12 weeks, was not altered by consumption of fermentable oligosaccharides.

In the study by Davidson *et al.* (Davidson *et al.*, 1998), participants with mild to moderate lipidaemias (n=25) were instructed to consume food products containing inulin – as a substitute to the sugar content of study foods – or comparable products containing maltodextrin for six weeks. When LDL:HDL cholesterol ratio values were compared at the end of the intervention, no statistically significant differences were reported.

The two studies presented here provide consistent evidence that fermentable oligosaccharide intake in the form of inulin does not differentially affect LDL:HDL cholesterol ratio.

Table 2.101 LDL:HDL cholesterol ratio and fibre isolates, fermentable oligosaccharides: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Jackson <i>et al.</i> , 1999) 14807	Inulin	27/27	3.32 (SD .9)	3.26 (SD 1.13)	NS	NS	LDL:HDL cholesterol ratio	8 weeks	No change	No bias
	Placebo	27/27	3.88 (SD 1.26)	3.77 (SD 1.55)	NS				No change	
14808	Inulin	27/27	3.32 (SD .9)	3.04 (SD .95)	NS	NS	LDL:HDL cholesterol ratio	12 weeks	No change	No bias
	Placebo	27/27	3.88 (SD 1.26)	3.65 (SD 1.45)	NS				No change	
(Davidson <i>et al.</i> , 1998) 17579	Control	21/25	2.98 (SE 0.69)	3.13 (SE 0.87)			LDL:HDL cholesterol ratio	6 weeks	No change	No bias
	Inulin	21/25	3.19 (SE 0.83)	3.11 (SE 0.81)		NS			No change	

Apolipoproteins and fibre isolates, fermentable oligosaccharides

No cohort studies reported results concerning fibre isolates, fermentable oligosaccharides and apolipoproteins.

Summary of RCT data

One parallel group study provided data on the effects of high fermentable oligosaccharide diets on apolipoproteins (Jackson *et al.*, 1999). In this study, 10g/day of inulin was compared with a similar amount of maltodextrin. Body weights remained unchanged in each dietary group.

Neither apolipoprotein A-1 or B were differentially affected by consumption of inulin in this trial.

Table 2.102 Apolipoproteins and fibre isolates, fermentable oligosaccharides: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Jackson <i>et al.</i> , 1999) 14810	Inulin	27/27	887 (SD 180)	869 (SD 142)	NS	NS	Apolipoprotein B	Fasting plasma (mg/L)	8 weeks	No change	No bias
	Placebo	27/27	958 (SD 180)	959 (SD 179)	NS					No change	
14811	Inulin	27/27	887 (SD 180)	879 (SD 143)	NS	NS	Apolipoprotein B	Fasting plasma (mg/L)	12 weeks	No change	No bias
	Placebo	27/27	958 (SD 180)	951 (SD 166)	NS					No change	
14814	Inulin	27/27	1193 (SD 218)	1165 (SD 211)	NS	NS	Apolipoprotein A-1	Fasting plasma (mg/L)	8 weeks	No change	No bias
	Placebo	27/27	1242 (SD 271)	1222 (SD 236)	NS					No change	
14819	Inulin	27/27	1193 (SD 218)	1160 (SD 233)	NS	NS	Apolipoprotein A-1	Fasting plasma (mg/L)	12 weeks	No change	No bias
	Placebo	27/27	1242 (SD 271)	1202 (SD 262)	NS					No change	

Results – Fibre isolates, mixed soluble types

Total cholesterol and fibre isolates, mixed soluble types

No cohort studies reported results concerning fibre isolates, mixed soluble types and total cholesterol.

Summary of RCT data

Four trials reported data on mixed water-soluble types of fibre isolate and total cholesterol (Haskell *et al.*, 1992; Knopp *et al.*, 1999; Salas-Salvado *et al.*, 2008; Jensen *et al.*, 1997). All were included in a meta-analysis.

These four studies all employed a parallel group design to compare the effects of a soluble fibre supplement and a placebo. All were similar in that they administered soluble fibre as a powder, which was mixed with water or an alternative beverage.

Haskell *et al.* (Haskell *et al.*, 1992) compared a water-soluble dietary fibre mixture of acacia gum, psyllium husk and guar gum (17g fibre/day) to a placebo in 62 subjects for 12 weeks. The fibre supplement was prepared as a powder in a carbohydrate base (approximately 15g of fructose per serving), and the control was the carbohydrate base only.

This same research group later conducted a longer duration trial (six months) with a similar protocol, but comparing 15g/day of a water-soluble dietary fibre supplement (a mixture of psyllium, pectin, guar gum, and locust bean gum) with an inactive water-soluble dietary fibre control (acacia gum). The 58 trial participants who were mildly to moderately hypercholesterolaemic consumed a self-selected, low-fat and low-cholesterol diet comparable to the National Cholesterol Education Program (NCEP) Step 1 diet throughout the trial (Jensen *et al.*, 1997). It should be noted that there was some baseline imbalance in blood lipids between the groups, with higher initial LDL cholesterol levels in the control group.

Knopp *et al.* explored the effects of a low fat (NCEP Step 1) diet plus mixed water soluble fibre supplementation (15g/d of guar gum and pectin and 5g/d of a mixture of soy fibre, pea fibre and corn bran) with a Step 1 diet plus placebo (non-water soluble fibre from cellulose) for 15 weeks. Subjects with mild to moderate hypercholesterolaemia (LDL cholesterol, 3.37– 4.92mmol/L) were randomly allocated to either the fibre (n= 87) or placebo group (n= 82) (Knopp *et al.*, 1999).

The primary endpoint of the study by Salas-Salvado *et al.* (Salas-Salvado *et al.*, 2008) was to compare the effect of two doses of a mixed water-soluble fibre supplement (3g *Plantago ovata* husk and 1g glucomannan - consumed twice or three times per day) with a placebo product on weight change. The 200 overweight or obese patients recruited were randomised to one of three experimental groups whilst also following an energy-restricted diet for 16 weeks.

The high mixed soluble fibre groups consumed 8-20g of fibre supplement per day, and for comparison, the low fibre groups consumed similar amounts of 'inactive' supplements (as defined by the authors) which included non-water soluble cellulose, acacia gum, or a fructose carrier.

Three out of the four studies were double blind (Haskell *et al.*, 1992; Salas-Salvado *et al.*, 2008; Jensen *et al.*, 1997) and one was single blind (Knopp *et al.*, 1999).

Trials were conducted in the USA (Haskell *et al.*, 1992; Knopp *et al.*, 1999; Jensen *et al.*, 1997) and in Spain (Salas-Salvado *et al.*, 2008). Sample sizes ranged from 58 to 200, with an average number of 122 subjects per study (median= 116). All subjects tended to be aged 48 or over and all studies were mixed gender. Of the studies that reported BMI, subjects were, on average, overweight (Knopp *et al.*, 1999; Jensen *et al.*, 1997) or obese (Salas-Salvado *et al.*, 2008).

Body weights were unchanged in all trials other than one (Salas-Salvado *et al.*, 2008) in which the authors reported that there was a decrease in all intervention groups (Salas-Salvado *et al.*, 2008). As such, any differences in total cholesterol may not be solely attributable to the dietary treatment.

Four studies were included in the meta-analysis comparing different mixed soluble fibre intakes and changes in total cholesterol reported as mmol/L. The first follow up reported at the end of the intervention was used. This varied from 12 to 24 weeks. The pooled estimate indicated that total cholesterol was 0.36mmol/L (95% CI 0.23 to 0.50) lower with consumption of a diet higher in soluble fibre. This was significantly different from zero ($p < 0.001$). Overall heterogeneity denoted by I^2 was 6% (95% CI 0 to 86%). Statistically, there was evidence that supplements of mixed water-soluble dietary fibre in the range of 15 to 20g per day, when consumed for six or more weeks, are associated with lower levels of total cholesterol.

Figure 2.59 Forest plot for fibre isolates, mixed soluble types and total cholesterol (mmol/L)

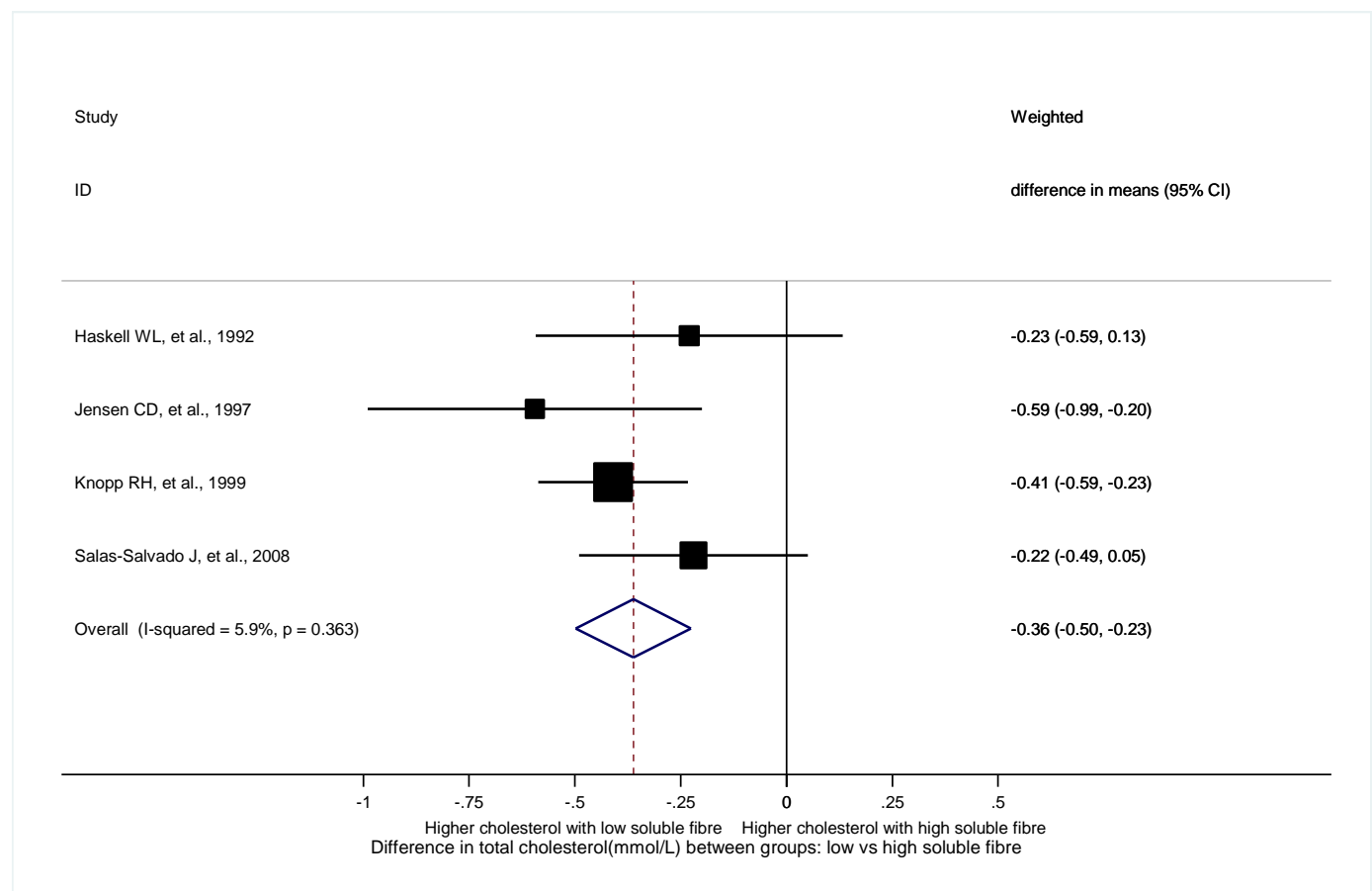


Table 2.103 Total cholesterol and fibre isolates, mixed soluble-type: RCT data

Author/ Result ID	Intervention groups	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	Outcom e details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Haskell <i>et al.</i> , 1992) 16088	Study1 Placebo	29/30	6.05 (SD 0.47)	6.05 (SD 0.65)		NS			Total cholesterol	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Study1 Soluble fibre	29/32	5.97 (SD 0.82)	5.86 (SD 0.91)		NS	NS					No change	
*16089	Study1 Placebo	29/30	6.05 (SD 0.47)	6.02 (SD 0.47)		NS			Total cholesterol	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Study1 Soluble fibre	29/32	5.97 (SD 0.82)	5.79 (SD 0.88)		NS	NS					No change	
(Jensen <i>et al.</i> , 1997) 15555	Control (Acacia gum)	27/27	232 (SD 22)	235 (SD 28)		NS			Total cholesterol	Fasting plasma (mg/dL)	8 weeks	No change	No bias
	Soluble fibre	24/24	235 (SD 19)	220 (SD 18)		<0.05	<0.05					No change	
15556	Control (Acacia gum)	27/27	232 (SD 22)	234 (SD 22)		NS			Total cholesterol	Fasting plasma (mg/dL)	16 weeks	No change	No bias
	Soluble fibre	24/24	235 (SD 19)	220 (SD 19)		<0.05	<0.05					No change	
*15557	Control (Acacia gum)	27/27	232 (SD 22)	242 (SD 31)		NS			Total cholesterol	Fasting plasma (mg/dL)	24 weeks	No change	No bias
	Soluble fibre	24/24	235 (SD 19)	219 (SD 26)		<0.05	<0.05					No change	
(Knopp <i>et al.</i> , 1999) 15838	Fibre supplementation	63/87	6.29 (SD 0.57)		-0.47 (SD 0.59)		<0.001		Total cholesterol	Plasma (mmol/L)	6 weeks	No change	unclear
	Placebo	56/82	6.17 (SD 0.5)		-0.07 (SD 0.5)							No change	
15839	Fibre supplementation	54/87	6.29 (SD 0.57)		-0.55 (SD 0.53)		<0.001		Total cholesterol	Plasma (mmol/L)	12 weeks	No change	unclear
	Placebo	58/82	6.17 (SD 0.5)		-0.16 (SD 0.41)							No change	
*15840	Fibre supplementation	52/87	6.29 (SD 0.57)		-0.44 (SD 0.45)		<0.001		Total cholesterol	Plasma (mmol/L)	15 weeks	No change	unclear
	Placebo	50/82	6.17 (SD		-0.03 (SD							No	

Author/ Result ID	Intervention groups	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	Outcom e details	Result- specific follow-up	Weight change	Outcome Assessment Bias
			0.5)		0.47)							change	
(Salas-Salvado <i>et al.</i> , 2008) 14506	Mixed soluble fibre 3 times a day	58/68			-0.33 (SD 0.11)		NS		Total cholesterol	Fasting (mmol/L)	16 weeks	Decrease	No bias
	Mixed soluble fibre twice a day	53/66			-0.43 (SD 0.12)							Decrease	
	Placebo	55/66			-0.11 (SD 0.11)							Decrease	
*14774	Mixed soluble fibre twice a day minus Placebo	Intervention: 53/66 Placebo: 55/66						-0.32 (CI -0.6, 0.04)	Total cholesterol	Fasting (mmol/L)	16 weeks	Decrease in both	No bias
*14775	Mixed soluble fibre 3 times a day minus placebo	Intervention: 58/58 Placebo: 55/66						-0.22 (CI -0.49, 0.05)	Total cholesterol	Fasting (mmol/L)	16 weeks	Decrease in both	No bias

*This result was used in the meta-analysis of soluble fibre and total cholesterol

HDL cholesterol and fibre isolates, mixed soluble types

No cohort studies reported results concerning fibre isolates, mixed soluble types and HDL cholesterol.

Summary of RCT data

Four trials reported data on mixed-soluble types of fibre and HDL cholesterol (Haskell *et al.*, 1992;Knopp *et al.*, 1999;Salas-Salvado *et al.*, 2008;Jensen *et al.*, 1997). All were included in a meta-analysis.

The four trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, mixed soluble types.

Four studies were included in the meta-analysis comparing different soluble fibre intakes and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 24 weeks. The pooled estimate indicated that HDL cholesterol was 0.04mmol/L (95% CI -0.04 to 0.11) lower with consumption of a diet higher in soluble fibre. This was not significantly different from zero ($p=0.36$). Overall heterogeneity denoted by I^2 was 45% (95% CI 0 to 82%). Statistically, there was no evidence that high soluble fibre consumption is associated with improved levels of HDL cholesterol.

Figure 2.60 Forest plot for fibre isolates, mixed soluble types and HDL cholesterol (mmol/L)

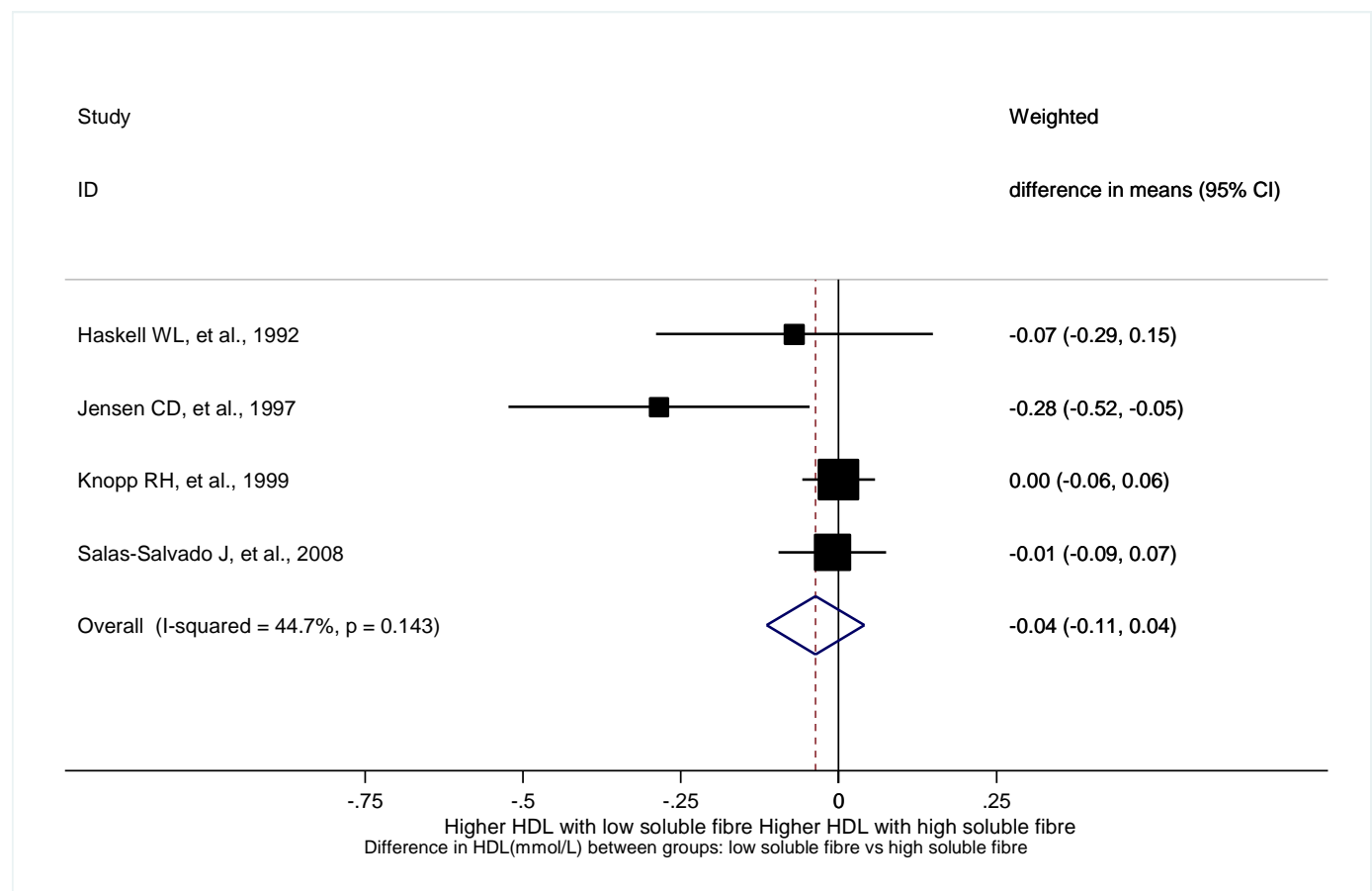


Table 2.104 HDL cholesterol and fibre isolates, mixed soluble types: RCT data

Author/ Result ID	Intervention groups	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	Outcom e details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Haskell <i>et al.</i> , 1992) 16092	Study1 Placebo	29/30	1.54 (SD 0.44)	1.57 (SD 0.44)		NS			HDL-C	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Study1 Soluble fibre	29/32	1.47 (SD 0.47)	1.41 (SD 0.31)		NS	NS					No change	
*16093	Study1 Placebo	29/30	1.54 (SD 0.44)	1.46 (SD 0.41)		NS			HDL-C	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Study1 Soluble fibre	29/32	1.47 (SD 0.47)	1.39 (SD 0.44)		NS	NS					No change	
(Jensen <i>et al.</i> , 1997) 15561	Control (Acacia gum)	27/27	60 (SD 21)	63 (SD 22)		NS			HDL-C	Fasting plasma (mg/dL)	8 weeks	No change	No bias
	Soluble fibre	24/24	54 (SD 15)	51 (SD 15)		NS	NS					No change	
15562	Control (Acacia gum)	27/27	60 (SD 21)	62 (SD 22)		NS			HDL-C	Fasting plasma (mg/dL)	16 weeks	No change	No bias
	Soluble fibre	24/24	54 (SD 15)	52 (SD 17)		NS	NS					No change	
*15563	Control (Acacia gum)	27/27	60 (SD 21)	62 (SD 20)		NS			HDL-C	Fasting plasma (mg/dL)	24 weeks	No change	No bias
	Soluble fibre	24/24	54 (SD 15)	51 (SD 14)		NS	NS					No change	
(Knopp <i>et al.</i> , 1999) 15841	Fibre supplementation	56/87	1.4 (SD 0.32)		-0.03 (SD 0.11)		NS		HDL-C	Plasma (mmol/L)	6 weeks	No change	unclear
	Placebo	63/82	1.35 (SD 0.34)		-0.01 (SD 0.16)							No change	
15842	Fibre supplementation	54/87	1.4 (SD 0.32)		-0.04 (SD 0.12)		NS		HDL-C	Plasma (mmol/L)	12 weeks	No change	unclear
	Placebo	58/82	1.35 (SD 0.34)		-0.06 (SD 0.14)							No change	
*15843	Fibre supplementation	52/87	1.4 (SD 0.32)		-0.02 (SD 0.14)		NS		HDL-C	Plasma (mmol/L)	15 weeks	No change	unclear
	Placebo	50/82	1.35 (SD		-0.02 (SD							No	

Author/ Result ID	Intervention groups	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	Outcom e details	Result- specific follow-up	Weight change	Outcome Assessment Bias
			0.34)		0.16)							change	
(Salas-Salvado <i>et al.</i> , 2008) *14508	Mixed soluble fibre 3 times a day	58/68			-0.06 (SD 0.03)		NS		HDL-C	Fasting (mmol/L)	16 weeks	Decrease	No bias
	Mixed soluble fibre twice a day	53/66			-0.01 (SD 0.04)							Decrease	
	Placebo	55/66			-0.05 (SD 0.03)							Decrease	
14770	Mixed soluble fibre twice a day minus Placebo	Intervention: 53/66 Placebo: 55/66						0.05 (CI -0.04, 0.14)	HDL-C	Fasting (mmol/L)	16 weeks	Decrease in both	No bias
14771	Mixed soluble fibre 3 times a day minus placebo	Intervention: 58/68 Placebo: 55/66						-0.01 (CI -0.09, 0.08)	HDL-C	Fasting (mmol/L)	16 weeks	Decrease in both	No bias

*This result was used in the meta-analysis of soluble fibre and HDL cholesterol

LDL cholesterol and fibre isolates, mixed soluble types

No cohort studies reported results concerning fibre isolates, soluble types and LDL cholesterol.

Summary of RCT data

Four trials reported data on mixed-soluble types of fibre and LDL cholesterol (Haskell *et al.*, 1992;Knopp *et al.*, 1999;Salas-Salvado *et al.*, 2008;Jensen *et al.*, 1997). All were included in a meta-analysis.

The four trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, mixed soluble types.

Four studies were included in the meta-analysis comparing different soluble fibre intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from 12 to 24 weeks. The pooled estimate indicated that LDL cholesterol was 0.29mmol/L (95% CI 0.12 to 0.45) lower with consumption of a diet higher in mixed soluble fibre isolates. This was significantly different from zero ($p=0.001$). Overall heterogeneity denoted by I^2 was 39% (95% CI 0 to 79%). There was evidence that high soluble fibre consumption is associated with lower levels of LDL cholesterol.

Figure 2.61 Forest plot for fibre isolates, mixed soluble types and LDL cholesterol (mmol/L)

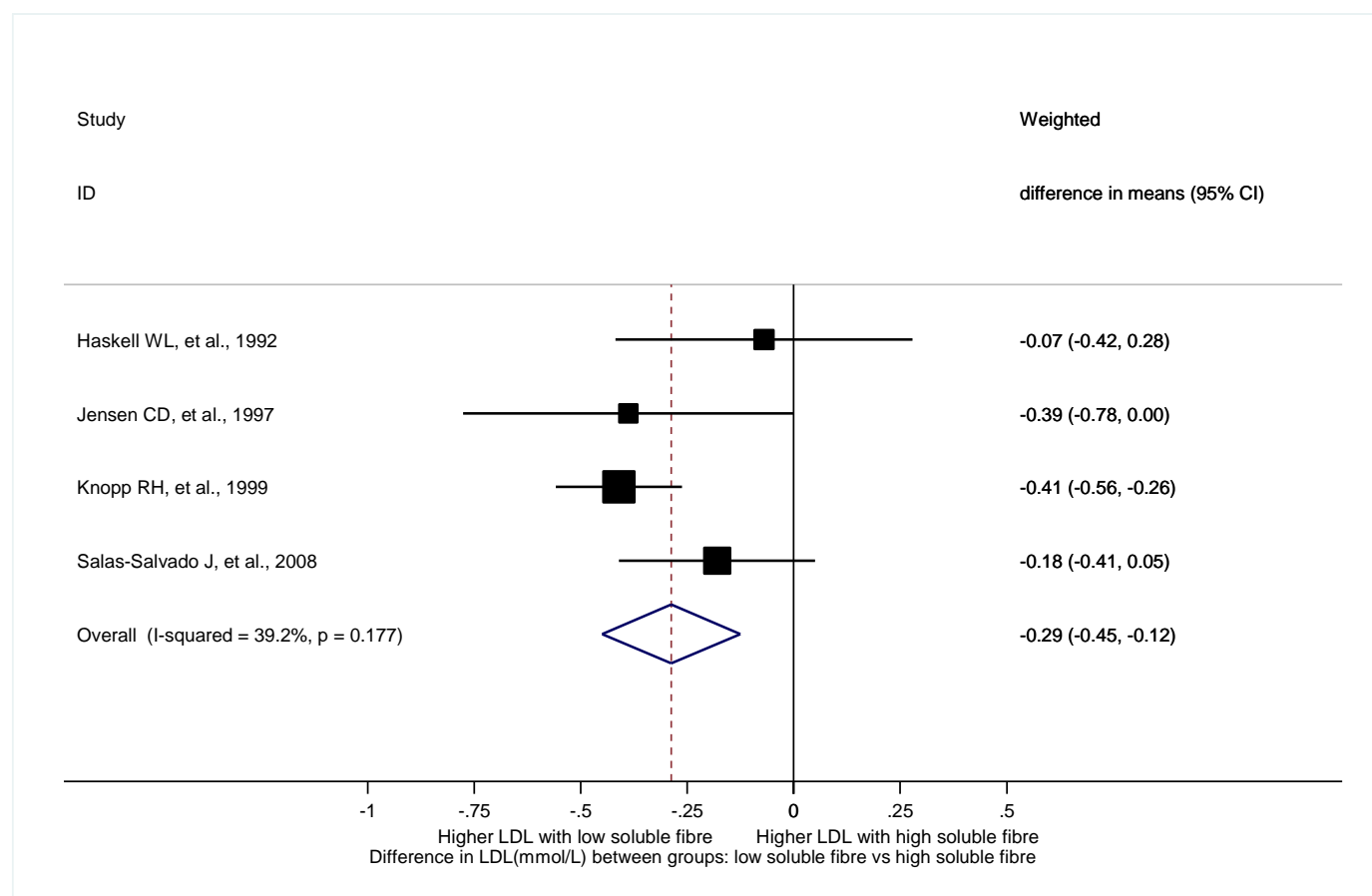


Table 2.105 LDL cholesterol and fibre isolates, mixed soluble types: RCT data

Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Haskell <i>et al.</i> , 1992) 16090	Study1 Placebo	29/30	3.86 (SD 0.57)	3.74 (SD 0.75)		NS			LDL-C	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Study1 Soluble fibre	29/32	4.01 (SD 0.67)	3.88 (SD 0.73)		NS	NS					No change	
*16091	Study1 Placebo	29/30	3.86 (SD 0.57)	3.71 (SD 0.62)		NS			LDL-C	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Study1 Soluble fibre	29/32	4.01 (SD 0.67)	3.78 (SD 0.73)		NS	NS					No change	
(Jensen <i>et al.</i> , 1997) 15558	Control (Acacia gum)	27/27	144 (SD 23)	142 (SD 27)		NS			LDL-C	Fasting plasma (mg/dL)	8 weeks	No change	No bias
	Soluble fibre	24/24	152 (SD 22)	136 (SD 20)		<0.05	<0.05					No change	
15559	Control (Acacia gum)	27/27	144 (SD 23)	145 (SD 27)		NS			LDL-C	Fasting plasma (mg/dL)	16 weeks	No change	No bias
	Soluble fibre	24/24	152 (SD 22)	142 (SD 21)		<0.05	<0.05					No change	
*15560	Control (Acacia gum)	27/27	144 (SD 23)	152 (SD 31)		NS			LDL-C	Fasting plasma (mg/dL)	24 weeks	No change	No bias
	Soluble fibre	24/24	152 (SD 22)	137 (SD 25)		<0.05	<0.05					No change	
(Knopp <i>et al.</i> , 1999) 15834	Fibre supplementation	56/87	4.22 (SD 0.44)		-0.47 (SD 0.49)		<0.001		LDL-C	Plasma (mmol/L)	6 weeks	No change	unclear
	Placebo	63/82	4.13 (SD 0.44)		0.01 (SD 0.42)								
15836	Fibre supplementation	54/87	4.22 (SD 0.44)		-0.56 (SD 0.49)		<0.001		LDL-C	Plasma (mmol/L)	12 weeks	No change	unclear
	Placebo	58/82	4.13 (SD 0.44)		-0.15 (SD 0.37)								
*15837	Fibre supplementation	52/87	4.22 (SD 0.44)		-0.41 (SD 0.36)		<0.001		LDL-C	Plasma (mmol/L)	15 weeks	No change	unclear
	Placebo	50/82	4.13 (SD 0.44)		-0.00 (SD 0.41)							No change	
(Salas- Salvado	Mixed soluble fibre 3 times a day	58/68			-0.24 (SD 0.09)		0.03		LDL-C	Fasting (mmol/L)	16 weeks	Decrease	No bias

Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
<i>et al.</i> , 2008) *14507	Mixed soluble fibre twice a day	53/66			-0.38 (SD 0.10)							Decrease	
	Placebo	55/66			-0.06 (SD 0.09)							Decrease	
14772	Mixed soluble fibre twice a day minus Placebo	Intervention: n: 53/66 Placebo: 55/66						-0.32 (CI -0.56, - 0.07)	LDL-C	Fasting (mmol/L)	16 weeks	Decrease in both	No bias
14773	Mixed soluble fibre 3 times a day minus Placebo	Intervention: n: 58/58 Placebo: 55/66						-0.18 (CI -0.41, 0.05)	LDL-C	Fasting (mmol/L)	16 weeks	Decrease in both	No bias

*This result was used in the meta-analysis for soluble fibre and LDL cholesterol

Triacylglycerol and fibre isolates, mixed soluble-type

No cohort studies reported results concerning fibre isolates, mixed soluble-type and TAG.

Summary of RCT data

Four trials reported data on mixed-soluble types of fibre and TAG (Haskell *et al.*, 1992;Knopp *et al.*, 1999;Salas-Salvado *et al.*, 2008;Jensen *et al.*, 1997). All were included in a meta-analysis. The four trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, mixed soluble types.

Four studies were included in the meta-analysis comparing different soluble fibre intakes and changes in TAG reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from 12 to 24 weeks.

The pooled estimate indicated that TAG was 0mmol/L (95% CI -0.18 to 0.18) lower with consumption of a diet higher in soluble fibre. This was not significantly different from zero ($p=1.00$). Overall heterogeneity denoted by I^2 was 16% (95% CI 0 to 87%). Statistically, there was no evidence that high soluble fibre consumption is associated with different levels of TAG.

Figure 2.62 Forest plot for fibre isolates, mixed soluble types and TAG (mmol/L)

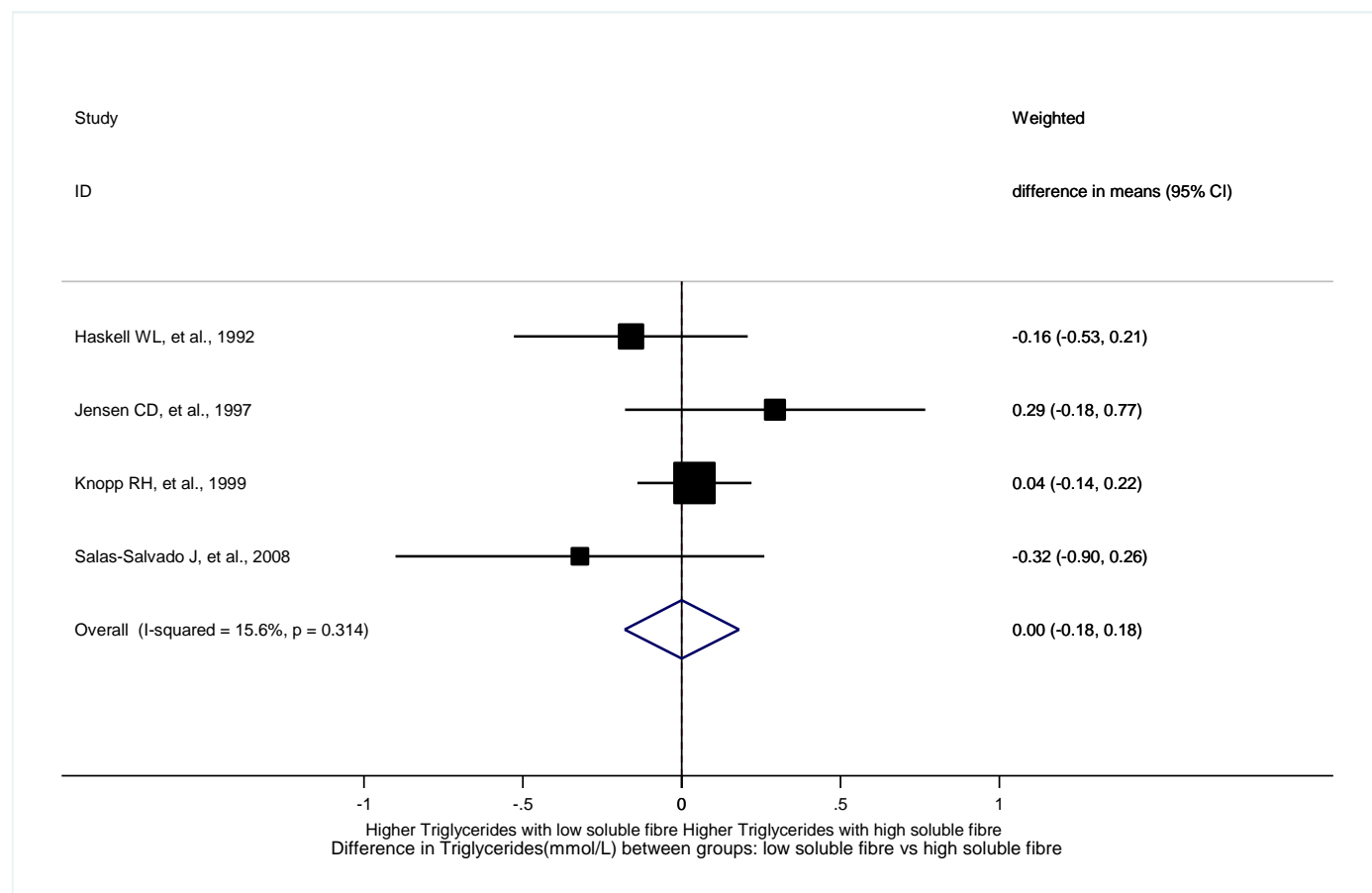


Table 2.106 Triacylglycerol and fibre isolates, mixed soluble-type: RCT data

Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Haskell <i>et al.</i> , 1992) 16096	Study1 Placebo	29/30	1.44 (SD 0.64)	1.57 (SD 0.7)		NS			TAG	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Study1 Soluble fibre	29/32	1.36 (SD 0.61)	1.53 (SD 0.73)		NS	NS					No change	
*16097	Study1 Placebo	29/30	1.44 (SD 0.64)	1.68 (SD 0.73)		NS			TAG	Fasting plasma (mmol/L)	12 weeks	No change	No bias
	Study1 Soluble fibre	29/32	1.36 (SD 0.61)	1.52 (SD 0.7)		NS	NS					No change	
(Jensen <i>et al.</i> , 1997) 15564	Control (Acacia gum)	27/27	137 (SD 70)	149 (SD 118)		NS			TAG	Fasting plasma (mg/dL)	8 weeks	No change	No bias
	Water soluble dietary fibre (WSDF)	24/24	142 (SD 64)	200 (SD 207)		NS	NS					No change	
15565	Control (Acacia gum)	27/27	137 (SD 70)	133 (SD 85)		NS			TAG	Fasting plasma (mg/dL)	16 weeks	No change	No bias
	Water soluble dietary fibre (WSDF)	24/24	142 (SD 64)	153 (SD 108)		NS	NS					No change	
*15566	Control (Acacia gum)	27/27	137 (SD 70)	140 (SD 70)		NS			TAG	Fasting plasma (mg/dL)	24 weeks	No change	No bias
	Water soluble dietary fibre (WSDF)	24/24	142 (SD 64)	166 (SD 86)		NS	NS					No change	
(Knopp <i>et al.</i> , 1999) 15847	Fibre supplementation	56/87	1.47 (SD 0.57)		0.07 (SD 0.43)		NS		TAG	Plasma (mmol/L)	6 weeks	No change	unclear
	Placebo	63/82	1.51 (SD 0.64)		0.17 (SD 0.65)							No change	
15848	Fibre supplementation	54/87	1.47 (SD 0.57)		0.1 (SD 0.51)		NS		TAG	Plasma (mmol/L)	12 weeks	No change	unclear
	Placebo	58/82	1.51 (SD)		0.09 (SD 0.51)							No change	

Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
*15849	Fibre supplementation	52/87	0.64) 1.47 (SD 0.57)		0.01 (SD 0.43)		NS		TAG	Plasma (mmol/L)	15 weeks	No change	unclear
	Placebo	50/82	1.51 (SD 0.64)		-0.03 (SD 0.5)							No change	
(Salas-Salvado <i>et al.</i> , 2008) *14511	Mixed soluble fibre 3 times a day	58/68			-0.01 (SD 0.23)		NS		TAG	Fasting (mmol/L)	16 weeks	Decrease	No bias
	Mixed soluble fibre twice a day	53/66			-0.03 (SD 0.26)							Decrease	
	Placebo	55/66			0.33 (SD 0.23)							Decrease	
14764	Mixed soluble fibre twice a day minus Placebo	Intervention: 53/66 Placebo: 55/66						-0.36 (CI -0.97, 0.25)	TAG	Fasting (mmol/L)	16 weeks	Decrease in both	No bias
14765	Mixed soluble fibre 3 times a day minus Placebo	Intervention: 58/58 Placebo: 55/66						-0.32 (CI -0.9, 0.26)	TAG	Fasting (mmol/L)	16 weeks	Decrease in both	No bias

*This result was used in the meta-analysis for soluble fibre and TAG

Total cholesterol:HDL ratio and fibre isolates, mixed soluble-type

No cohort studies reported results concerning fibre isolates, mixed soluble-type and TC:HDL ratio.

Summary of RCT data

Salas-Salvado *et al.* (Salas-Salvado *et al.*, 2008) reported the results of a parallel group trial with 200 adults who were randomly assigned to a mixed soluble fibre dose three times a day, a mixed soluble fibre dose twice a day or a placebo, consumed as part of an energy-restricted diet, for 16 weeks. Body weights decreased in both groups throughout this trial. At follow up, the TC:HDL ratio decreased in the two soluble fibre treatment groups relative to the placebo group ($p=0.03$).

Table 2.107 Total cholesterol:HDL ratio and fibre isolates, mixed soluble types: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Within group Δ from baseline	p-value difference between groups	Difference between groups in Δ from baseline	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Salas-Salvado <i>et al.</i> , 2008) 14509	Mixed soluble fibre 3 times a day	58/68	-0.13 (SD 0.12)	0.03		TC:HDL ratio	Fasting	16 weeks	Decrease	No bias
	Mixed soluble fibre twice a day	53/66	-0.36 (SD 0.14)						Decrease	
	Placebo	55/66	0.07 (SD 0.12)						Decrease	
14768	Mixed soluble fibre twice a day minus placebo	Intervention: 53/66 Placebo: 55/66			-0.44 (CI -0.76, -0.12)	TC:HDL ratio	Fasting	16 weeks	Decrease	No bias
14769	Mixed soluble fibre 3 times a day minus placebo	Intervention: 58/58 Placebo: 55/66			-0.21 (CI -0.51, 0.09)	TC:HDL ratio	Fasting	16 weeks	Decrease	No bias

LDL:HDL cholesterol ratio and fibre isolates, mixed soluble-type

No cohort studies reported results concerning fibre isolates, mixed soluble-type and LDL:HDL cholesterol ratio.

Summary of RCT data

Knopp *et al.* (Knopp *et al.*, 1999) conducted a 15-week parallel group trial to investigate the effects of a dietary supplement of soluble fibres (guar gum, pectin, soy fibre, pea fibre and corn bran) compared with a matching placebo (non water-soluble fibre from cellulose) using 169 generally healthy participants. Concurrently, participants in both groups were required to follow a Step 1 diet. Body weights were unchanged throughout the trial. There was evidence of a decrease in LDL:HDL cholesterol ratio for the fibre supplementation group compared with the placebo at six, 12 and 15 weeks ($p < 0.001$ for all).

Table 2.108 LDL:HDL cholesterol ratio and fibre isolates, mixed soluble type: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Knopp <i>et al.</i> , 1999) 15844	Fibre supplementation	56/87	3.17 (SD 0.82)	-0.29 (SD 0.41)	<0.001	LDL:HDL cholesterol ratio	Plasma	6 weeks	No change	unclear
	Placebo	63/82	3.23 (SD 0.83)	0.04 (SD 0.42)					No change	
15845	Fibre supplementation	54/87	3.17 (SD 0.82)	-0.34 (SD 0.39)	<0.001	LDL:HDL cholesterol ratio	Plasma	12 weeks	No change	unclear
	Placebo	58/82	3.23 (SD 0.83)	0.02 (SD 0.48)					No change	
15846	Fibre supplementation	52/87	3.17 (SD 0.82)	-0.25 (SD 0.37)	<0.001	LDL:HDL cholesterol ratio	Plasma	15 weeks	No change	unclear
	Placebo	50/82	3.23 (SD 0.83)	0.06 (SD 0.42)					No change	

Apolipoproteins and fibre isolates, mixed soluble type

No cohort studies reported results concerning fibre isolates, mixed soluble fibre and apolipoproteins.

Summary of RCT data

One study by Knopp *et al.* (Knopp *et al.*, 1999) tested the effects of a soluble fibre supplement on blood lipids in 169 healthy participants. Using a parallel group design, participants were randomised to receive a 20g/day fibre supplement (15g/day mixture of guar gum and pectin plus 5g/day mixture of soy fibre, corn bran and pea fibre) or a placebo product which was identical in taste and appearance (Knopp *et al.*, 1999). Body weights were unchanged in this trial.

Apolipoprotein B was measured at six and 15 weeks and had statistically significantly decreased with the 20g fibre supplement compared with the placebo ($p < 0.001$ for six and 15 weeks).

No statistically significant differences from baseline in apolipoprotein A-1 levels were found.

Table 2.109 Apolipoproteins and fibre isolates, mixed soluble-type: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Knopp <i>et al.</i> , 1999) 15850	Fibre supplementation	56/87	1.5 (SD 0.26)		-3.2 (SD 11)	NS	Apolipoprotein A- 1	Plasma (g/L)	6 weeks	No change	unclear
	Placebo	63/82	1.47 (SD 0.23)		1.7 (SD 16.5)					No change	
15852	Fibre supplementation	52/87	1.5 (SD 0.26)		-2.6 (SD 14.5)	NS	Apolipoprotein A- 1	Plasma (g/L)	15 weeks	No change	unclear
	Placebo	50/82	1.47 (SD 0.23)		-3.8 (SD 18.1)					No change	
15853	Fibre supplementation	56/87	1.42 (SD 0.26)		-13.7 (SD 20.8)	<0.001	Apolipoprotein B	Plasma (g/L)	6 weeks	No change	unclear
	Placebo	63/82	1.36 (SD 0.24)		1.6 (SD 19.0)					No change	
15855	Fibre supplementation	52/87	1.42 (SD 0.26)		-14.3 (SD 21.1)	<0.001	Apolipoprotein B	Plasma (g/L)	15 weeks	No change	unclear
	Placebo	50/82	1.36 (SD 0.24)		3.6 (SD 20.5)					No change	

Results – Fibre isolates, mixed soluble and insoluble type

Total cholesterol and fibre isolates, mixed soluble and insoluble type

No cohort studies reported results concerning fibre isolates, mixed type and total cholesterol.

Summary of RCT data

Three trials tested the effects of mixed-insoluble and soluble types of fibre isolate on total cholesterol levels (Cairella *et al.*, 1995;Hunninghake *et al.*, 1994;Birketvedt *et al.*, 2000). Quantitative data were not reported by Cairella *et al.* (Cairella *et al.*, 1995); therefore there was an insufficient number of studies to perform a meta-analysis.

All trials were comparable in that they employed a parallel group design. Different methods of fibre administration were implemented to incorporate the fibre into the diet. Cairella *et al.* (Cairella *et al.*, 1995) and Birketvedt *et al.* (Birketvedt *et al.*, 2000) instructed subjects to consume fibre tablets three times a day, whereas a fibre supplement, in a powdered form, was mixed and consumed with a beverage twice a day in the study by Hunninghake *et al.* (Hunninghake *et al.*, 1994).

The trials were carried out in Norway (Birketvedt *et al.*, 2000), Italy (Cairella *et al.*, 1995) and the USA (Hunninghake *et al.*, 1994).

Subjects in these three trials were typically either, on average, overweight (Birketvedt *et al.*, 2000;Cairella *et al.*, 1995) or obese (Hunninghake *et al.*, 1994). All used adults as participants and the average age ranged between 36 and 52 years. One study used females only who had mild to moderate hypercholesterolaemia (Birketvedt *et al.*, 2000), but the remaining two studies recruited both males and females (Cairella *et al.*, 1995;Hunninghake *et al.*, 1994).

The paper by Hunninghake *et al.* (Hunninghake *et al.*, 1994) reported no changes in body weight during the trial, but the other two trials reported a decrease in weight from baseline to follow up (Cairella *et al.*, 1995;Birketvedt *et al.*, 2000). Any differences in total cholesterol therefore may not be solely attributable to the dietary intervention.

In Cairella *et al.* (Cairella *et al.*, 1995), a dietary fibre supplement (fibre sourced from vegetables, citrus fruit and cereals – 6g fibre per day) was compared with a placebo in 30 obese subjects. An initial 15-day weight loss phase with a very low caloric diet was employed, after which subjects in both the fibre supplement group and placebo group were encouraged to follow a balanced diet (with 17-22g fibre content) for the remaining 60 days of the study. At follow up, total cholesterol normalised in both groups and no statistically significant differences were observed between groups.

In the trial recorded by Hunninghake *et al.* (Hunninghake *et al.*, 1994) 127 eligible subjects were randomly allocated to receive a 10g/day fibre supplement, a 20g/day fibre supplement or a matching placebo. The fibre supplement contained guar gum, pectin, soy, corn bran, and pea fibre. Overall, both fibre intervention groups showed a statistically significant decrease in total cholesterol relative to the placebo group ($p < 0.05$ for both).

Finally, Birketvedt *et al.* (Birketvedt *et al.*, 2000) used 53 subjects on a reduced energy diet (1200kcal/day) to test a fibre supplement compared with no supplement over a 24-week period. Fibre tablets (mixture of fibre from grain and citrus; 15% soluble fibre and 85% insoluble fibre) were prescribed three times a day for eight weeks (6g fibre/d). The dosage was then reduced to five tablets per day for the rest of the study. The authors reported a decrease of 0.5mmol/L from baseline in total cholesterol in both the mixed fibre supplement group and placebo group ($p < 0.05$) with no additional benefit from fibre supplementation.

These data therefore provide inconsistent evidence concerning the effects of mixed-insoluble fibre on total cholesterol.

Table 2.110 Total cholesterol and fibre isolates, mixed soluble and insoluble-type: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Birketvedt <i>et al.</i> , 2000) 14926	Energy restricted diet and mixed fibre tablets	28/28	5.7 (SE 0.2)	5.2 (SE 0.2)		<0.05	NS	Total cholesterol	Fasting serum (mmol/L)	24 weeks	Decrease	No bias
	Energy restricted diet and placebo tablets	25/25	6 (SE 0.3)	5.5 (SE 0.2)		<0.05					Decrease	
(Cairella <i>et al.</i> , 1995) 15687	Balanced diet and fibre tablets	completers not reported/15						Total cholesterol	Not reported	60 days	Decrease	No bias
	Balanced diet and placebo tablets	completers not reported/15					NS				Decrease	
(Hunninghake <i>et al.</i> , 1994) 15309	Fibre 10g	40/53	5.98 (SE 0.08)	5.79 (SE 0.09)	-6%		<0.05	Total cholesterol	Fasting plasma (mmol/L)	15 weeks	No change	No bias
	Fibre 20g	39/55	6.10 (SE 0.11)	5.81 (SE 0.1)	-5%		<0.05				No change	
	Placebo	48/53	6.08 (SE 0.08)	6.11 (SE 0.08)							No change	

HDL cholesterol and fibre isolates, mixed soluble and insoluble types

No cohort studies reported results concerning fibre isolates, mixed soluble and insoluble types and HDL cholesterol.

Summary of RCT data

One trial provided data on the effects of mixed soluble and insoluble types of fibre on HDL cholesterol (Hunninghake *et al.*, 1994). The results of this study are shown in Table 2.111

The trial recorded by Hunninghake *et al.* (Hunninghake *et al.*, 1994) recruited subjects who had mild to moderate hypercholesterolaemia. In this study (Hunninghake *et al.*, 1994), the authors tested the effects of fibre supplementation on cholesterol levels over a 15-week period. One hundred and twenty seven eligible subjects were randomly allocated to receive a 10g/day fibre supplement, a 20g/day fibre supplement or a matching placebo. The fibre supplement was administered in 296mL skimmed milk, juice or water for use in the morning or evening. Body weight changes were not reported. Changes in HDL cholesterol did not differ markedly from baseline and the authors concluded that such changes were not statistically significantly different among treatment groups over time.

Table 2.111 HDL cholesterol and fibre isolates, mixed soluble and insoluble types: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Hunninghake <i>et al.</i> , 1994) 15310	Fibre 10g	40/53	1.22 (SE 0.04)	1.22 (SE 0.04)	NS	HDL	Fasting plasma (mmol/L)	15 weeks	No change	No bias
	Fibre 20g	39/55	1.26 (SE 0.05)	1.3 (SE 0.05)	NS				No change	
	Placebo	48/53	1.34 (SE 0.05)	1.35 (SE 0.05)					No change	

LDL cholesterol and fibre isolates, mixed soluble and insoluble types

No cohort studies reported results concerning mixed fibre isolates and LDL cholesterol.

Summary of RCT data

One intervention tested the effects of mixed soluble and insoluble fibre supplementation on LDL cholesterol (Hunninghake *et al.*, 1994).

In this study (Hunninghake *et al.*, 1994), 127 eligible subjects were randomly allocated to receive a 10g/day fibre supplement, a 20g/day fibre supplement or a matching placebo. The authors found a statistically significant decrease in LDL cholesterol in the fibre supplement groups relative to the placebo group ($p < 0.05$ for both). Similarly, by the end of the treatment, changes of -8% and -7% in LDL cholesterol in the 10g/day fibre supplement group and 20g/day fibre supplement groups respectively were observed, although statistically significant values were not reported. This study therefore indicates a small improvement in LDL cholesterol levels with additional fibre supplementation.

Table 2.112 LDL cholesterol and fibre isolates, mixed soluble and insoluble type: RCT data

Author/ Result ID	Interven- tion groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Hunninghake <i>et al.</i> , 1994) 15311	Fibre 10g	40/53	4.11 (SE 0.07)	3.89 (SE 0.08)	-8%	<0.05	LDL-C	Derived by calculation Fasting, Plasma (mmol/L)	15 weeks	No change	No bias
	Fibre 20g	39/55	4.17 (SE 0.08)	3.86 (SE 0.08)	-7%	<0.05				No change	
	Placebo	48/53	4.08 (SE 0.06)	4.1 (SE 0.07)						No change	

Triacylglycerol and fibre isolates, mixed soluble and insoluble types

No cohort studies reported results concerning mixed soluble and insoluble fibre isolates and TAG.

Summary of RCT data

Three studies provided data on the effects of mixed soluble and insoluble-type fibre on TAG (Cairella *et al.*, 1995; Hunninghake *et al.*, 1994; Birketvedt *et al.*, 2000). Data from Cairella *et al.* (Cairella *et al.*, 1995) did not provide quantitative data and therefore it was not possible to combine studies to perform a meta-analysis.

The three trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, mixed insoluble types.

Bar Hunninghake *et al.* (Hunninghake *et al.*, 1994), in which no weight change occurred in any dietary group, two trials reported a decrease in weight from baseline to follow up (Cairella *et al.*, 1995; Birketvedt *et al.*, 2000). Any differences in TAG therefore may not be solely attributable to the dietary intervention.

Cairella *et al.* (Cairella *et al.*, 1995) conducted a randomised, double blind trial to explore the effects of a dietary fibre supplement compared with placebo on weight loss and blood lipids in 30 obese subjects (BMI range: 30.9 to 47.0 kg/m²). An initial 15-day weight loss phase with a very low caloric diet was employed, after which subjects in both the fibre supplement group and placebo group were encouraged to follow a balanced diet (with 17-22g fibre content) for the remaining 60 days of the study. Fibre was administered by tablets (fibre sourced from vegetables, citrus fruit and cereals), whereby three tablets were taken six times daily. This group was compared with an identical placebo tablet, which followed similar administration patterns and consumption as the intervention. After 60 days, total cholesterol normalised in both groups, however no statistically significant differences were observed between groups.

The trial recorded by Hunninghake *et al.* (Hunninghake *et al.*, 1994) recruited subjects who had mild to moderate hypercholesterolaemia. In this study, the authors tested the effects of fibre supplementation on TAG levels over a 15-week period. One hundred and twenty seven eligible subjects were randomly allocated to receive a 10g/day fibre supplement, a 20g/day fibre supplement or a matching placebo. The fibre supplement was administered in 296mL skimmed milk, juice or water for use in the morning or evening. Overall, supplementation with fibre at either dose did not produce changes in TAG levels over time or between treatment groups.

Finally, Birketvedt *et al.* (Birketvedt *et al.*, 2000) used 53 moderately overweight subjects on a reduced energy diet (1200kcal/day) to test a fibre supplement (mixture of fibre from grain and citrus; 15% soluble fibre and 85% insoluble fibre) compared with no supplement over a 24-week period. Fibre was initially administered in tablet form (6g) and prescribed three times a day for eight weeks. The dosage was then reduced to five tablets per day for the rest of the study. Overall, both groups experienced a statistically significant decrease in serum TAG levels from baseline ($p>0.05$) but no difference between dietary groups. These changes were also accompanied by weight loss in both groups; consequently a reduction in TAG levels cannot be solely attributed to this dietary fibre supplement.

These three trials generally show that fibre supplementation does not impact on TAG levels in overweight subjects and those with mild to moderate hypercholesterolaemia.

Table 2.113 Triacylglycerol and fibre isolates, mixed soluble and insoluble types: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Birketvedt <i>et al.</i> , 2000) 14927	Energy restricted diet and mixed fibre tablets	28/28	1.34 (SE 0.2)	0.92 (SE 0.1)		<0.05	NS	TAG	Fasting serum (mmol/L)	24 weeks	Decrease	No bias
	Energy restricted diet and placebo tablets	25/25	1.47 (SE 0.2)	0.92 (SE 0.1)		<0.05					Decrease	
(Cairella <i>et al.</i> , 1995) 15688	Balanced diet and fibre tablets	completers not reported/15						TAG	Not reported	60 days	Decrease	No bias
	Balanced diet and placebo tablets	completers not reported/15					NS				Decrease	
(Hunninghake <i>et al.</i> , 1994) 15313	Fibre 10g	40/53	1.44 (SE 0.1)	1.3 (SE 0.13)			NS	TAG	Fasting plasma (mmol/L)	15 weeks	No change	No bias
	Fibre 20g	39/55	1.48 (SE 0.08)	1.43 (SE 0.09)			NS				No change	
	Placebo	48/53	1.47 (SE 0.1)	1.46 (SE 0.1)							No change	

LDL:HDL cholesterol ratio and fibre isolates, mixed soluble and insoluble types

No cohort studies reported results concerning mixed soluble and insoluble fibre isolates and LDL:HDL cholesterol ratio.

Summary of RCT data

One trial reported by Hunninghake *et al.* (Hunninghake *et al.*, 1994) randomly allocated participants with mild to moderate hypercholesterolaemia (n=127) to a 10g/day fibre supplement, a 20g/day fibre supplement or a matching placebo. The fibre supplement was administered in 296mL skimmed milk, juice or water for use in the morning or evening. At 15 weeks, supplementation with 10g or 20g fibre statistically significantly reduced the ratio of LDL to HDL cholesterol relative to the placebo.

Table 2.114 LDL:HDL cholesterol ratio and fibre isolates, mixed soluble and insoluble types: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	Baseline	Follow-up	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Hunninghake <i>et al.</i> , 1994) 15312	Fibre 10g	40/53	3.49 (SE 0.12)	3.34 (SE 0.14)	<0.05	LDL:HDL cholesterol ratio	Fasting plasma	15 weeks	No change	No bias
	Fibre 20g	39/55	3.46 (SE 0.12)	3.12 (SE 0.12)	<0.05				No change	
	Placebo	48/53	3.25 (SE 0.12)	3.27 (SE 0.14)					No change	

Apolipoproteins and fibre isolates, mixed soluble and insoluble types

No cohort studies reported results concerning mixed soluble and insoluble fibre isolates and apolipoproteins.

Summary of RCT data

Data from one trial were extracted concerning insoluble fibre and apolipoproteins (Hunninghake *et al.*, 1994). In this study, 127 eligible subjects were randomly allocated to receive a 10g/day fibre supplement, a 20g/day fibre supplement or a matching placebo (Hunninghake *et al.*, 1994). Body weights did not change in this trial. In all three treatment groups, apolipoprotein A-1 levels showed a decrease from baseline, although these differences were not statistically significant. Furthermore, when compared with the placebo there was a statistically significant reduction in apolipoprotein B in the 20g fibre supplement group.

Table 2.115 Apolipoproteins and fibre isolates, mixed soluble and insoluble types: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Hunninghake <i>et al.</i> , 1994) 15314	Fibre 10g	40/53	1322 (SE 35)	1375 (SE 31)		NS	Apolipoprote in A-1	Fasting plasma (mg/L)	15 weeks	No change	No bias
	Fibre 20g	39/55	1385 (SE 43)	1407 (SE 42)		NS				No change	
	Placebo	48/53	1433 (SE 41)	1468 (SE 42)						No change	
15315	Fibre 10g	40/53	1325 (SE 25)	1318 (SE 30)		<0.05	Apolipoprote in B	Fasting plasma (mg/L)	15 weeks	No change	No bias
	Fibre 20g	39/55	1342 (SE 36)	1270 (SE 35)		<0.05				No change	
	Placebo	48/53	1279 (SE 25)	1284 (SE 30)						No change	

Results – Fibre isolates, psyllium

Total cholesterol and fibre isolates, psyllium

No cohort studies reported results concerning fibre isolates, psyllium and total cholesterol.

Summary of RCT data

Three trials provided data on changes in total cholesterol in response to the addition of psyllium (Williams *et al.*, 1995; Romero *et al.*, 1998; Bell *et al.*, 1990). One study by Williams *et al.* (Williams *et al.*, 1995) included children as participants leaving too few adult studies to perform a meta-analysis.

All studies used a parallel group approach. Trials were conducted in the USA (2) and in Mexico. One study by (Williams *et al.*, 1995) included children aged 2-11 years whereas the other two studies recruited adults as participants. Trials were single blind (Williams *et al.*, 1995), double blind (Bell *et al.*, 1990) or the blinding was not reported (Romero *et al.*, 1998).

Of the three trials, two studied males only (Romero *et al.*, 1998; Bell *et al.*, 1990). Williams *et al.* (Williams *et al.*, 1995) was mixed gender. All studies were small in size, and had fewer than 100 participants in each.

Body weights were unchanged in all trials.

Williams *et al.* (Williams *et al.*, 1995) conducted a parallel intervention trial to test the effects of psyllium in lowering total cholesterol in a sample of healthy children (n=50). In this study, children were randomly allocated to either a Step 1 diet of low dietary fat, saturated fat and cholesterol plus a psyllium-enriched cereal (3.2g of soluble fibre) or a Step 1 diet (as above) plus a placebo cereal (less than 0.5g soluble fibre). Overall, total cholesterol measured at 12 weeks was found to have statistically significantly decreased by 21mg/dL and 11.5mg/dL in the high soluble fibre cereal group and low soluble fibre cereal group, respectively. Between groups, there was also a statistically significant difference, as the high soluble fibre cereal group experienced a further 14.5mg/dL decrease in total cholesterol compared with the low soluble fibre cereal group ($p<0.05$).

In the study by Bell *et al.* (Bell *et al.*, 1990) the authors explored the effects of psyllium fibre on cholesterol levels of 58 males with mild to moderate hypercholesterolaemia. A Step 1 diet was employed during the first six 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 control (cornflakes) whilst continuing with the Step 1 diet over a second six-week period. Cereals were administered as 57g portions and were consumed as part of breakfast. The psyllium-added cereal provided in the region of 3g psyllium per day. A 5.9% decrease in serum total cholesterol was observed in the latter six-week phase of the study in the psyllium cereal group, compared with no change in the cornflake (placebo) group. The difference between placebo and psyllium groups was statistically significant ($P=0.005$). The decrease in total cholesterol was similar in the pectin and psyllium groups.

The study reported by Romero *et al.* (Romero *et al.*, 1998) explored the effects of psyllium and oat bran in lowering plasma cholesterol over an eight-week period. Sedentary normal subjects ($n=30$) were randomly assigned to one of three interventions: a control group consuming wheat bran cookies (100g), a group consuming psyllium cookies (100g) or a group consuming oat bran cookies (100g). One hundred grams of psyllium and oat bran cookies was equivalent to 1.3g and 2.6g/ per day of soluble fibre, respectively. An additional sample of hypercholesterolaemic men was also included in the study and similarly these subjects were randomised to one of three groups. These data, however, were not extracted. The authors reported statistically significant reductions in plasma total cholesterol levels following eight weeks of psyllium or oat bran cookies ($p<0.05$ for both) in subjects.

The change in total cholesterol was significantly greater in the oat and psyllium groups compared with the control (wheat) group ($p<0.001$).

Table 2.116 Total cholesterol and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention groups	Completers / Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Williams <i>et al.</i> , 1995) 15681	High soluble fibre cereal plus Step 1 diet	25/26	218.60 (SD 28.61)	197.60 (SD 26.72)	-21	<0.001	<0.05	Total cholesterol	Fasting serum (mg/dL)	12 weeks	No change	bias
	Low soluble fibre cereal plus Step 1 diet	24/24	208.21 (SD 25.21)	196.67 (SD 27.62)	-11.54	<0.01					No change	
Adult studies												
(Bell <i>et al.</i> , 1990) 17609	Pectin enriched cereal	20/20	5.69	5.56 (SE 0.16)	-0.12 (0.4%)	NS	NS vs. placebo	Total cholesterol	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Placebo (cornflakes)	19/20	5.60	5.62 (SE 0.19)	0.02 (2.1%)	NS					No change	
	Psyllium enriched cereal	19/20	5.63	5.29 (SE 0.16)	-0.34 (5.9%)	0.0011	0.005 vs placebo				No change	
(Romero <i>et al.</i> , 1998) 15424	Oat bran cookies	12/12	214 (SD 13)	184 (SD 22)	-30	<0.05	<0.001	Total cholesterol	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium cookies	10/10	214 (SD 19)	193 (SD 26)	-21	<0.05	<0.001				No change	
	Wheat bran cookies	14/14	180 (SD 33)	185 (SD 30)	5	NS					No change	

HDL cholesterol and fibre isolates, psyllium

No cohort studies reported results concerning fibre isolates, psyllium and HDL cholesterol.

Summary of RCT data

Three trials provided data on the impact of psyllium fibre on HDL cholesterol (Williams *et al.*, 1995; Bell *et al.*, 1990; Romero *et al.*, 1998). One study by Williams *et al.* (Williams *et al.*, 1995) included children as participants leaving too few adult studies to perform a meta-analysis.

The three trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, psyllium.

Body weights were unchanged in all three trials.

Williams *et al.* (Williams *et al.*, 1995) conducted a study using healthy children (n=50) in which a Step 1 diet of low dietary fat, saturated fat and cholesterol plus a psyllium-enriched cereal (3.2g of soluble fibre) was compared with a Step 1 diet (as above) plus a placebo cereal (less than 0.5g soluble fibre). HDL cholesterol at the end of the intervention (12 weeks) had increased in both groups; however reported differences were not statistically significant.

In the study conducted by Bell *et al.* (Bell *et al.*, 1990), psyllium fibre-enriched cereal consumption did not improve HDL levels compared with the control product (cornflakes).

Similarly, Romero *et al.* (Romero *et al.*, 1998) did not report an improvement in HDL levels in the group assigned to psyllium-added cookies compared to the wheat bran cookie group.

Collectively, these three studies indicate that HDL cholesterol levels are unaffected by the addition of psyllium fibre to either a Step 1 (low fat) or a habitual diet.

Table 2.117 HDL cholesterol and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Williams <i>et al.</i> , 1995) 15682	High soluble fibre cereal plus Step 1 diet	25/26	40.96 (SD 11.47)	45.04 (SD 13.57)	4.08	NS	NS	HDL-C	Fasting serum ($\mu\text{g/dL}$)	12 weeks	No change	bias
	Low soluble fibre cereal plus Step 1 diet	24/24	48.57 (SD 7.73)	49.58 (SD 8.79)	1.52	NS					No change	
Adult studies												
(Bell <i>et al.</i> , 1990) 17191	Pectin enriched cereal	20/20		1.21 (SE 0.06)	0.03 (2.5%)	NS	NS	HDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Placebo	19/20		1.19 (SE 0.07)	0.02 (1.6%)	NS					No change	
	Psyllium enriched cereal	19/20		1.19 (SE 0.08)	-0.02 (1.6%)	NS	NS				No change	
(Romero <i>et al.</i> , 1998) 15426	Oat bran cookies	12/12	27 (SD 7)	32 (SD 8)		NS	NS	HDL-C	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium cookies	10/10	37 (SD 8)	41 (SD 9)		NS	NS				No change	
	Wheat bran cookies	14/14	47 (SD 19)	50 (SD 17)		NS	NS				No change	

LDL cholesterol and fibre isolates, psyllium

No cohort studies reported results concerning the fibre isolate psyllium and LDL cholesterol.

Summary of RCT data

Three studies reported data on the effects of psyllium fibre on LDL cholesterol (Williams *et al.*, 1995; Bell *et al.*, 1990; Romero *et al.*, 1998). One study by Williams *et al.* (Williams *et al.*, 1995) included children as participants leaving too few adult studies to perform a meta-analysis.

The three trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, psyllium. Body weights were unchanged in all three trials.

One study of healthy children (n=50) (Williams *et al.*, 1995) compared a Step 1 diet of low dietary fat, saturated fat and cholesterol plus psyllium-enriched cereal (3.2g of soluble fibre) with a Step 1 diet (as above) plus a placebo cereal (less than 0.5g soluble fibre) over a 12-week period. At follow up, LDL cholesterol was found to have decreased by 23.67µg/dL and 8.53µg/dL in the high soluble fibre plus Step 1 diet group and low soluble fibre plus Step 1 diet groups, respectively. This difference from baseline was only statistically significant in the high soluble fibre group (p<0.01), however. There was also evidence of a statistically significant change between diet groups (p<0.01).

In the study conducted by Bell *et al.* (Bell *et al.*, 1990), the psyllium fibre-enriched cereal group experienced a 5.7% decrease in serum LDL cholesterol compared with minimal change in the cornflake (placebo) group. The difference between placebo and psyllium groups was statistically significant (P=0.03). The difference in serum LDL cholesterol changes in the pectin and psyllium groups was not statistically significant.

Similarly, Romero *et al.* (Romero *et al.*, 1998) reported a greater decrease in LDL cholesterol in the group consuming oat bran cookies compared with the other groups of interest (-37mg/dL, p<0.05), although subjects consuming psyllium cookies also experienced a statistically significant reduction in this outcome (p<0.0001). The change in total cholesterol was significantly greater in the oat and psyllium groups compared with the control (wheat) group (p<0.001).

Overall, these findings provide some evidence of a reduction in LDL cholesterol with the addition of psyllium fibre to a Step 1 and habitual diet.

Table 2.118 LDL cholesterol and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Williams <i>et al.</i> , 1995) 15683	High soluble fibre cereal plus Step 1 diet	25/26	150.43 (SD 31.63)	126.76 (SD 26.06)	-23.67	<0.01	<0.01	LDL-C	Fasting Serum, ($\mu\text{g/dL}$)	12 weeks	No change	bias
	Low soluble fibre cereal plus Step 1 diet	24/24	132.77 (SD 29.26)	124.25 (SD 25.02)	-8.53	NS					No change	
Adult studies												
(Bell <i>et al.</i> , 1990) 17170	Pectin enriched cereal	20/20	3.92	3.76 (SE 0.14)	-0.16 (3.9%)	NS	NS vs. placebo	LDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Placebo	19/20	3.82	3.81 (SE 0.18)	-0.02 (0.4%)	NS					No change	
	Psyllium enriched cereal	19/20	3.83	3.6 (SE 0.12)	-0.23 (5.7%)	0.0066	0.034 vs. placebo				No change	
(Romero <i>et al.</i> , 1998) 15425	Oat bran cookies	12/12	140 (SD 43)	103 (SD 33)	-37	<0.05	<0.001	LDL-C	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium cookies	10/10	146 (SD 16)	121 (SD 20)	-25	<0.0001	<0.001				No change	
	Wheat bran cookies	14/14	109 (SD 29)	109 (SD 22)	-0.1	NS					No change	

Triacylglycerol and fibre isolates, psyllium

No cohort studies reported results concerning fibre isolates, psyllium and TAG.

Summary of RCT data

Three trials provided data on the effects of psyllium fibre on TAG (Williams *et al.*, 1995; Bell *et al.*, 1990; Romero *et al.*, 1998). Williams *et al.* (Williams *et al.*, 1995) included children as participants; therefore there was an insufficient number of adult studies to perform a meta-analysis.

The three trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, psyllium. Body weights were unchanged in all three trials.

One study of healthy children (n=50) (Williams *et al.*, 1995) which compared a Step 1 diet of low dietary fat, saturated fat and cholesterol plus a psyllium-enriched cereal (3.2g of soluble fibre) to a Step 1 diet (as above) plus a placebo cereal (less than 0.5g soluble fibre) detected changes in TAG, particularly in the low soluble fibre cereal plus Step 1 diet group; however these changes did not statistically significantly differ from baseline or between groups.

In the study conducted by Bell *et al.* (Bell *et al.*, 1990), psyllium fibre-enriched cereal consumption did not improve TAG levels compared with the control product (cornflakes).

Similarly, Romero *et al.* (Romero *et al.*, 1998) did not report an improvement in TAG levels in the group assigned to psyllium-added cookies compared with the wheat bran cookie group.

Collectively, these three studies indicate that TAG levels are unaffected by the addition of psyllium fibre to either a Step 1 (low fat) or a habitual diet.

Table 2.119 Triacylglycerol and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Williams <i>et al.</i> , 1995) 15684	High soluble fibre cereal plus Step 1 diet	25/26	141.46 (SD 46.56)	128.88 (SD 55.05)	-12.58	NS	NS	Change in TAG	Fasting serum (mg/dL)	12 weeks	No change	bias
	Low soluble fibre cereal plus Step 1 diet	24/24	134.48 (SD 45.13)	113.92 (SD 47.42)	-20.56	NS					No change	
Adult studies												
(Bell <i>et al.</i> , 1990) 17192	Pectin enriched cereal	20/20		1.3 (SE 0.16)	0.05	NS	NS	TAG	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Placebo	19/20		1.38 (SE 0.14)	0.03	NS					No change	
	Psyllium enriched cereal	19/20		1.07 (SE 0.15)	-0.135	NS	NS				No change	
(Romero <i>et al.</i> , 1998) 15427	Oat bran cookies	12/12	234 (SD 187)	246 (SD 97)		NS	NS	Change in TAG	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium cookies	10/10	154 (SD 67)	154 (SD 73)		NS	NS				No change	
	Wheat bran cookies	14/14	127 (SD 83)	130 (SD 91)		NS	NS				No change	

Total cholesterol:HDL ratio and fibre isolates, psyllium

No cohort studies reported results concerning fibre isolates, psyllium and TC:HDL ratio.

Summary of RCT data

One study provided data concerning psyllium and the TC:HDL ratio. Williams *et al.* (Williams *et al.*, 1995) compared a Step 1 diet of low dietary fat, saturated fat and cholesterol plus a psyllium-enriched cereal (3.2g of soluble fibre) to a Step 1 diet (as above) plus a placebo cereal (less than 0.5g soluble fibre) using a sample of healthy children. Body weights were unchanged in this trial. After the 12 week intervention, the TC:HDL ratio had decreased in both dietary groups but only statistically significantly so in the high soluble fibre group ($p < 0.05$). When comparing groups, this difference in change was statistically significant ($p < 0.001$).

Table 2.120 Total cholesterol:HDL ratio and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Williams <i>et al.</i> , 1995) 15685	High soluble fibre cereal plus Step 1 diet	25/26	5.6 (SD 1.5)	4.6 (SD 1.2)	-1	<0.05	<0.001	Change in TCI :HDL ratio	Fasting serum ($\mu\text{g/dL}$)	12 weeks	No change	bias
	Low soluble fibre cereal plus Step 1 diet	24/24	4.3 (SD 0.7)	4.1 (SD 0.9)	-0.2	NS					No change	

LDL:HDL cholesterol ratio and fibre isolates, psyllium

No cohort studies reported results concerning fibre isolates, psyllium and LDL:HDL cholesterol ratio.

Summary of RCT data

Two studies reported data on the effects of psyllium fibre on LDL:HDL cholesterol ratio (Williams *et al.*, 1995; Romero *et al.*, 1998). Changes in body weight were not evident throughout the two trials. Williams *et al.* (Williams *et al.*, 1995), compared a psyllium-enriched cereal and a placebo cereal using children aged 2-11 years and concluded that the LDL:HDL cholesterol ratio had statistically significantly decreased in the high soluble fibre cereal group ($p < 0.05$), but not in the placebo group. Furthermore, the difference between the two groups was statistically significant ($p < 0.001$).

Romero *et al.* (Romero *et al.*, 1998) also tested the effects of psyllium and oat bran on lowering plasma cholesterol over an eight-week period. Sedentary adults ($n=30$) were randomly assigned to a control group consuming wheat bran cookies, a group consuming psyllium cookies or a group consuming oat bran cookies. Over a period of eight weeks, an improvement in the LDL:HDL cholesterol ratio was observed for both fibre groups compared with the control group ($p < 0.01$ for both). However whilst the decrease in the ratio was larger in the psyllium group compared with the oat group, these groups were not significantly different from each other.

These two studies reported a consistent improvement in the LDL:HDL cholesterol ratio with psyllium-supplemented diets.

Table 2.121 LDL:HDL cholesterol ratio and fibre isolates, psyllium: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value Within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Williams <i>et al.</i> , 1995) 15686	High soluble fibre cereal plus Step 1 diet	25/26	3.8 (SD 1.2)	3.0 (SD 0.9)	-0.8	<0.05	<0.001	Change in LDL:HDL	12 weeks	No change	bias
	Low soluble fibre cereal plus Step 1 diet	24/24	2.7 (SD 0.6)	2.5 (SD 0.7)	-0.2	NS				No change	
(Romero <i>et al.</i> , 1998) 15428	Oat bran cookies	12/12	5.2 (SD 1.6)	3.4 (SD 1.5)	-1.8		<0.01	Change in LDL:HDL	8 weeks	No change	unclear
	Psyllium cookies	10/10	4.2 (SD 1)	3 (SD 1)	-1.2		<0.01			No change	
	Wheat bran cookies	14/14	3 (SD 1.6)	2.4 (SD 0.8)	-0.6					No change	

Results – Fibre isolates, gums and extracts

Total cholesterol and fibre isolates, gums and extracts

No cohort studies reported results concerning fibre isolates, gums and extracts and total cholesterol.

Summary of RCT data

Twelve studies provided data on the effects of specific fibre isolates including gums and other extracts on total cholesterol. These included guar gum, arabinogalactan, arabinoxylan and pectin. These differ from the studies of mixed isolates in that each study administered a single defined fibre isolate, rather than a combination of different fibre isolates.

Of the 12 included trials, seven employed a parallel group design (Pasman *et al.*, 1997a; Wood *et al.*, 2007; Marett and Slavin, 2004; Vido *et al.*, 1993; Schwab *et al.*, 2006; Reppas *et al.*, 2009; Bell *et al.*, 1990) and five a crossover design (Ryle *et al.*, 1990; Garcia *et al.*, 2006; Mee and Gee, 1997; Lehtimäki *et al.*, 2005; Panlasigui *et al.*, 2003). More than half of the studies were double blind (Wood *et al.*, 2007; Marett and Slavin, 2004; Vido *et al.*, 1993; Schwab *et al.*, 2006; Lehtimäki *et al.*, 2005; Reppas *et al.*, 2009; Bell *et al.*, 1990) and one was single blind (Garcia *et al.*, 2006). The remaining four were either unclear with regard to blinding of participant/researchers (Ryle *et al.*, 1990; Mee and Gee, 1997; Panlasigui *et al.*, 2003), or open (Pasman *et al.*, 1997a). Overall, the study durations ranged from six weeks to 14 months.

Studies were primarily conducted in the USA (5), but were also carried out in Finland (2), the Philippines (1), the Netherlands (1), the UK (1), Germany (1) and Italy (1).

One single study used adolescents as participants (Vido *et al.*, 1993) whereas the remaining 10 studied adults. Average BMI tended to fall into the overweight (25-30kg/m²) and obese (>30kg/m²) ranges for most studies; however one study reported a mean BMI of 22kg/m² (Ryle *et al.*, 1990). Two studies did not report the average BMI of participants (Vido *et al.*, 1993; Mee and Gee, 1997). Predominantly mixed gender trials were included within this review although three studies chose to recruit males only (Bell *et al.*, 1990; Wood *et al.*, 2007; Mee and Gee, 1997) and one study women only (Pasman *et al.*, 1997a).

Body weights were unchanged or not reported in the majority of trials. Exceptions to this include Vido *et al.* (Vido *et al.*, 1993), Wood *et al.* (Wood *et al.*, 2007) and Schwab *et al.* (Schwab *et al.*, 2006) which reported weight loss throughout the intervention. The three intervention groups in the

study by Pasman *et al.* (Pasman *et al.*, 1997a), however, gained weight (this was a trial to determine whether fibre would improve weight maintenance after an initial weight loss intervention). Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

Three studies compared guar gum with a control or placebo (Ryle *et al.*, 1990; Landin *et al.*, 1992; Pasman *et al.*, 1997a). Two studies compared pectin with a placebo or control product (Bell *et al.*, 1990; Schwab *et al.*, 2006). Single studies explored the effects of gum arabic (Mee and Gee, 1997), carageenan (Panlasigui *et al.*, 2003), arabinogalactan (Marett and Slavin, 2004), chitosan (Lehtimäki *et al.*, 2005), arabinoxylan (Garcia *et al.*, 2006), konjac mannan (Wood *et al.*, 2007), and methylcellulose (Reppas *et al.*, 2009).

Two studies were not included in the meta-analysis as one was a study of children less than 15 years of age (Vido *et al.*, 1993), and one did not provide quantitative data (Pasman *et al.*, 1997a). Neither study reported statistically significant differences in total cholesterol with the addition of the fibre isolates glucomannan and guar gum respectively. In their study, Vido *et al.* (Vido *et al.*, 1993) randomised children to receive either glucomannan (2g/day) or a placebo product for two months whilst consuming their usual diet. It was concluded that the dietary intervention had not affected total cholesterol in either diet group.

Similarly, Pasman *et al.* (Pasman *et al.*, 1997a) provided data on the effects of guar gum supplements on blood lipids in weight-reduced subjects for maintenance of weight loss over 16 months. This study compared a 20g water soluble fibre (guar gum) supplement with a no treatment condition for 14 months, following a two month very low calorie diet. No statistically significant differences in total cholesterol between or within groups were observed.

Ten studies were included in the meta-analysis comparing different gums and fibre isolates and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months. Three studies had more than two groups. In the studies by Bell *et al.*, and Schwab *et al.* the results for a comparison of control versus pectin were used (Bell *et al.*, 1990; Schwab *et al.*, 2006). In the study by Reppas *et al.* the placebo vs. high dose hydroxypropylmethylcellulose result was used (Reppas *et al.*, 2009). One study explored the effects of two different galactans: larch and tamarack (Marett and Slavin, 2004); tamarack was used in the meta-analysis.

The pooled estimate indicated that total cholesterol was 0.15mmol/L (95% CI -0.01 to 0.32) lower with consumption of a diet higher in gums or fibre extracts. This was not significantly different from zero ($p=0.07$). Overall heterogeneity denoted by I^2 was 56% (95% CI 13 to 78%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was no evidence that a diet higher in dietary fibre in the form of gums or extracts is associated with changes in total cholesterol.

Figure 2.63 Forest plot for fibre isolates, gums and extract and total cholesterol (mmol/L)

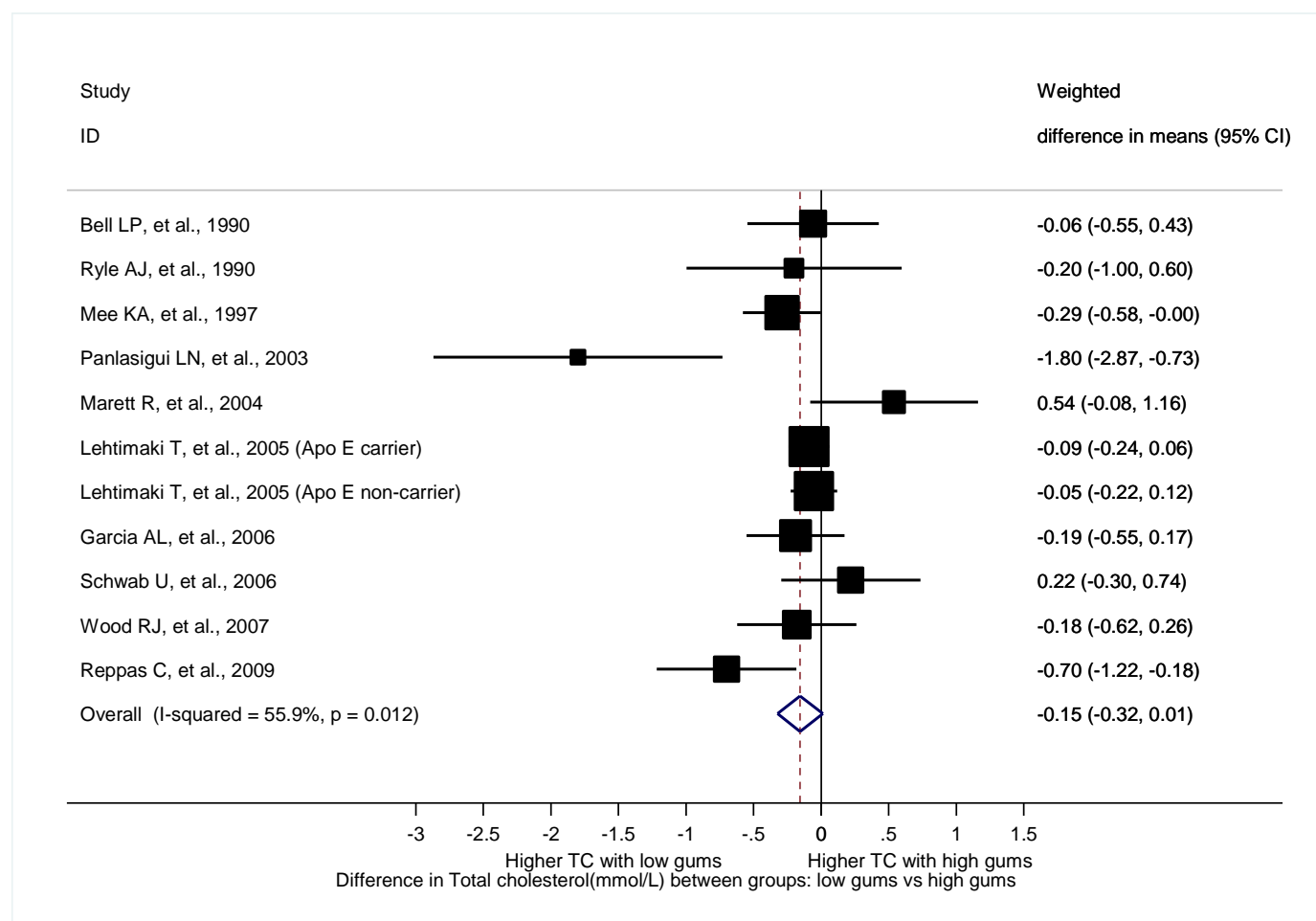


Figure 2.64 Contour-enhanced funnel plot for studies presenting data on dietary fibre isolates as gums and other extracts and total cholesterol

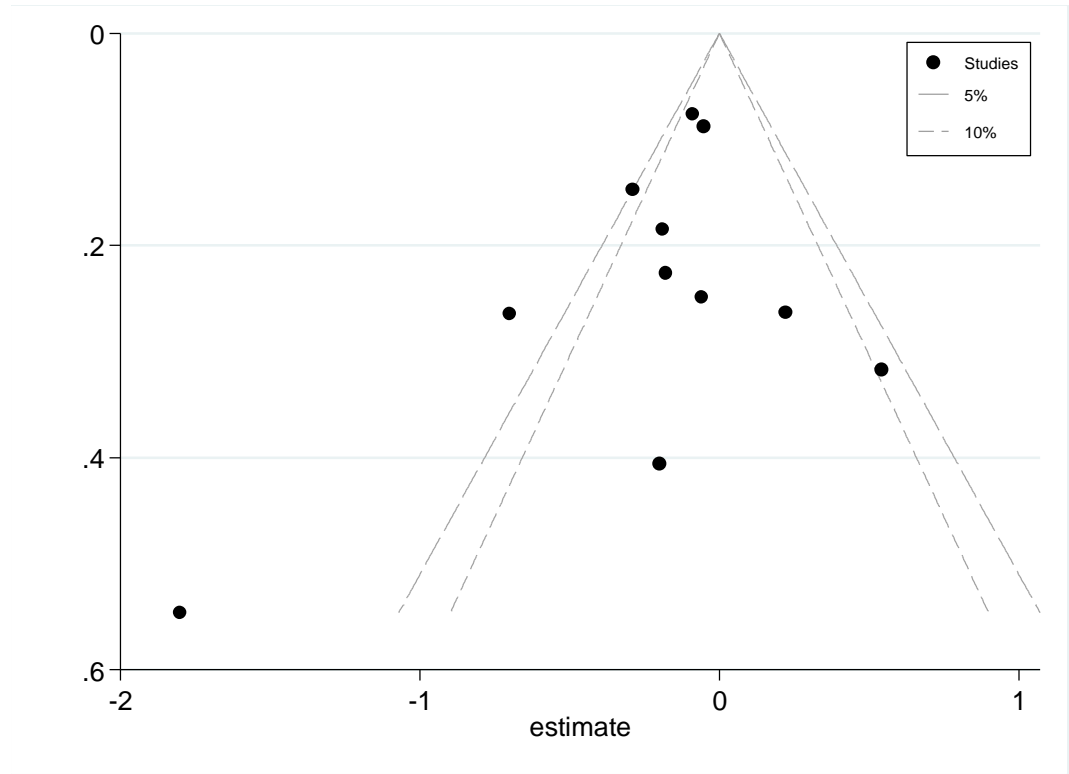


Table 2.122 Total cholesterol and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
Adolescent study													
(Vido <i>et al.</i> , 1993) 16936		Glucomannan supplement	30/30	194 (SD 49.3)	193 (SD 41.7)		NS	NS	Total cholesterol	Not reported (mg/dL)	2 months	Decrease	unclear
		Placebo	30/30	175 (SD 33.9)	181.8 (SD 40.6)		NS					Decrease	
Adult studies													
(Bell <i>et al.</i> , 1990) 17609		Pectin enriched cereal	20/20		5.56 (SE 0.16)	-0.12	NS	NS	Total cholesterol	Fasting serum (mmol/L)	6 weeks	No change	No bias
		Placebo	19/20		5.62 (SE 0.19)	0.02	NS					No change	
		Psyllium enriched cereal	19/20		5.29 (SE 0.16)	-0.34	0.0011	0.0052				No change	
(Garcia <i>et al.</i> , 2006) *17377		Arabinoxylan	11/11	5.37 (CI 5.15, 5.61)	5.05 (CI 4.74, 5.38)			0.656	Total cholesterol	Fasting Geometric mean, Serum (mmol/L)	6 weeks	No change	unclear
		Placebo	11/11	5.30 (CI 5.05, 5.57)	5.24 (CI 5.07, 5.41)							No change	
(Lehtima <i>ki et al.</i> , 2005) *17498	Apo E genotype E4 carrier	Encapsulated microcrystalline chitosan	86/96	6.01 (SD 0.5)	5.7 (SD 0.48)	-4.3 (SD 8.6)		NS	Total cholesterol	Fasting plasma (mmol/L)	3 months	Not reported	No bias
		Starch capsules	85/96	6.01 (SD 0.5)	5.79 (SD 0.51)	-2.8 (SD 8)						Not reported	
*17499	Apo E genotype E4 non-carrier	Encapsulated microcrystalline chitosan	86/96	5.94 (SD 0.69)	5.61 (SD 0.67)	-5.3 (SD 9.5)		NS	Total cholesterol	Fasting plasma (mmol/L)	3 months	Not reported	No bias
		Starch capsules	85/96	5.94 (SD 0.69)	5.66 (SD 0.66)	-4.4 (SD 9.8)						Not reported	
(Marett and Slavin, 2004) 16629		Larch arabinogalactan	18/18	4.64 (SD 1.00)	4.30 (SD 0.87)		NS	NS	Total cholesterol	Fasting plasma (mmol/L)	2 months	No change	No bias
		Placebo	17/17	4.64 (SD 1.04)	4.34 (SD 0.95)		NS	NS				No change	
		Tamarack arabinogalactan	19/19	4.68 (SD 0.99)	4.46 (SD 0.88)		NS	NS				No change	
16630		Larch arabinogalactan	18/18	4.64 (SD 1.00)	4.31 (SD 0.8)		NS	NS	Total cholesterol	Fasting plasma	3 months	No change	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
16631		Placebo	17/17	4.64 (SD 1.04)	4.53 (SD 1.07)		NS	NS	(mmol/L)				No change
		Tamarack arabinogalactan	19/19	4.68 (SD 0.99)	4.63 (SD 0.86)		NS	NS					No change
		Larch arabinogalactan	18/18	4.64 (SD 1.00)	4.27 (SD 0.75)		NS	NS	Total cholesterol	Fasting plasma (mmol/L)	4 months	No change	
		Placebo	17/17	4.64 (SD 1.04)	4.52 (SD 1.08)		NS	NS					No change
		Tamarack arabinogalactan	19/19	4.68 (SD 0.99)	4.61 (SD 0.97)		NS	NS					No change
16632		Larch arabinogalactan	18/18	4.64 (SD 1.00)	4.32 (SD 0.62)		NS	NS	Total cholesterol	Fasting plasma (mmol/L)	5 months	No change	
		Placebo	17/17	4.64 (SD 1.04)	4.42 (SD 1.04)		NS	NS					No change
		Tamarack arabinogalactan	19/19	4.68 (SD 0.99)	4.77 (SD 1.07)		NS	NS					No change
*16633		Larch arabinogalactan	18/18	4.64 (SD 1.00)	4.52 (SD 0.79)		NS	NS	Total cholesterol	Fasting plasma (mmol/L)	6 months	No change	
		Placebo	17/17	4.64 (SD 1.04)	4.32 (SD 0.92)		NS	NS					No change
		Tamarack arabinogalactan	19/19	4.68 (SD 0.99)	4.86 (SD 0.98)		NS	NS					No change
(Mee and Gee, 1997) *15629		Filtered apple juice	25/27	5.90 (SD 0.6)	5.59 (SD 0.49)		<0.05		Total cholesterol	Fasting serum (mmol/L)	6 weeks	No change	unclear
		Gum arabic- supplemented apple juice	25/27	5.90 (SD 0.6)	5.30 (SD 0.55)		<0.05	<0.05					No change
(Panlasi gui <i>et al.</i> , 2003) *15189		Carageenan- added test foods	20/20		3.64 (SD 1.43)				Total cholesterol	Fasting serum (mmol/L)	8 weeks	No change	unclear
		Usual diet	20/20		5.44 (SD 1.98)			0.0014					No change
(Pasman <i>et al.</i> , 1997a) 15522		Control	11/14				NS		Total cholesterol	Fasting plasma	14 months	Increase	unclear
		Guar gum - High compliance	10/10				NS	NS					Increase
		Guar Gum - Low	10/10				NS	NS					Increase

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
		compliance											
(Reppas <i>et al.</i> , 2009) 16782		High dose Hydroxypropyl methylcellulose	completers not reported/10	5.93 (SE 0.14)	5.09 (SE 0.17)		<0.05		Total cholesterol	Serum (mmol/L)	6 weeks	Not reported	No bias
		Low dose Hydroxypropyl methylcellulose	completers not reported/10	5.94 (SE 0.11)	5.51 (SE 0.15)		<0.05					Not reported	
		Placebo	completers not reported/10	6.03 (SE 0.15)	6.09 (SE 0.25)		NS					Not reported	
16783		High dose Hydroxypropyl methylcellulose	completers not reported/10	5.93 (SE 0.14)	5.1 (SE 0.15)		<0.05		Total cholesterol	Serum (mmol/L)	7 weeks	Not reported	No bias
		Low dose Hydroxypropyl methylcellulose	completers not reported/10	5.94 (SE 0.11)	5.52 (SE 0.15)		<0.05					Not reported	
		Placebo	completers not reported/10	6.03 (SE 0.15)	6.21 (SE 0.2)		NS					Not reported	
*16784		High dose Hydroxypropyl methylcellulose	completers not reported/ 10	5.93 (SE 0.14)	5.43 (SE 0.16)		<0.05	<0.05	Total cholesterol	Serum (mmol/L)	8 weeks	Not reported	No bias
		Low dose Hydroxypropyl methylcellulose	completers not reported/ 10	5.94 (SE 0.11)	5.54 (SE 0.13)		<0.05	<0.05				Not reported	
		Placebo	completers not reported/ 10	6.03 (SE 0.15)	6.13 (SE 0.21)		NS					Not reported	
(Ryle <i>et al.</i> , 1990) *16204		High glucose low soluble fibre	11/11	5.2 (SD 0.7)	5.1 (SD 0.9)		NS		Total cholesterol	Fasting (mmol/L)	6 weeks	No change	unclear
		Low glucose high soluble fibre diet (guar gum)	11/11	5.2 (SD 0.7)	4.9 (SD 1.0)		NS	NS				No change	
(Schwab <i>et al.</i> , 2006) 16485		Pectin	22/22	5.71 (SD 1.08)	5.68 (SD 0.85)			NS	Total cholesterol	Fasting serum (mmol/L)	8 weeks	Decrease	No bias
		Placebo	22/22	5.61 (SD 0.94)	5.54 (SD 1.10)							Decrease	
		Polydextrose	22/22	5.43 (SD 0.87)	5.36 (SD 0.97)			NS				Decrease	
*16486		Pectin	22/22	5.71 (SD 1.08)	5.54 (SD 0.68)		NS	NS	Total cholesterol	Fasting serum (mmol/L)	12 weeks	Decrease	No bias
		Placebo	22/22	5.61 (SD	5.32 (SD		<0.05					Decrease	

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Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
		Polydextrose	22/22	0.94) 5.43 (SD 0.87)	1.03) 5.27 (SD 0.82)		NS	NS				Decrease	
(Wood <i>et al.</i> , 2007) 17224		Low carbohydrate diet + placebo	15/15	4.58 (SD 1.18)	4.38 (SD 0.85)				Total cholesterol	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	4.64 (SD 0.75)	4.09 (SD 0.6)			NS				Decrease	
*17226		Low carbohydrate diet + placebo	15/15	4.58 (SD 1.18)	4.3 (SD 0.92)	-0.29 (SD 0.67)			Total cholesterol	Fasting plasma (mmol/L)	12 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	4.64 (SD 0.75)	4.18 (SD 0.63)	-0.47 (SD 0.56)		NS				Decrease	

*This result was used in the meta-analysis of gums and extracts and total cholesterol

HDL cholesterol and fibre isolates, gums and extracts

No cohort studies reported results concerning fibre isolates, gums and extracts and HDL cholesterol.

Summary of RCT data

Ten trials provided data on the relationship between soluble fibre isolates in the form of gums or fibre isolates and HDL cholesterol (Bell *et al.*, 1990; Pasman *et al.*, 1997a; Ryle *et al.*, 1990; Wood *et al.*, 2007; Marett and Slavin, 2004; Mee and Gee, 1997; Schwab *et al.*, 2006; Lehtimäki *et al.*, 2005; Panlasigui *et al.*, 2003; Reppas *et al.*, 2009). These studies also provided data on total cholesterol and so study details are provided in the Trial Characteristics table and in the text concerning total cholesterol.

Nine studies were included in the meta-analysis comparing different fibre intakes and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months.

Data from Pasman *et al.* (Pasman *et al.*, 1997a) were not included in the meta-analysis as quantitative data were not reported. In this study, the authors (Pasman *et al.*, 1997a) provided data on the effects of guar gum supplements on blood lipids in weight-reduced subjects for maintenance of weight loss over 16 months. This study compared a 20g water soluble fibre (guar gum) supplement with no treatment condition for 14 months, following a two month very low calorie diet. No differential effect of guar gum on HDL cholesterol was found.

The pooled estimate indicated that HDL cholesterol was 0.0mmol/L (95% CI -0.04 to 0.05) higher with consumption of a high gums diet. This was not significantly different from zero ($p=0.89$). Overall heterogeneity denoted by I^2 was 27% (95% CI 0 to 65%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was no evidence that a diet higher in dietary fibre in the form of gums or extracts is associated with changes in HDL cholesterol.

Figure 2.65 Forest plot for fibre isolates, gums and extract and HDL cholesterol (mmol/L)

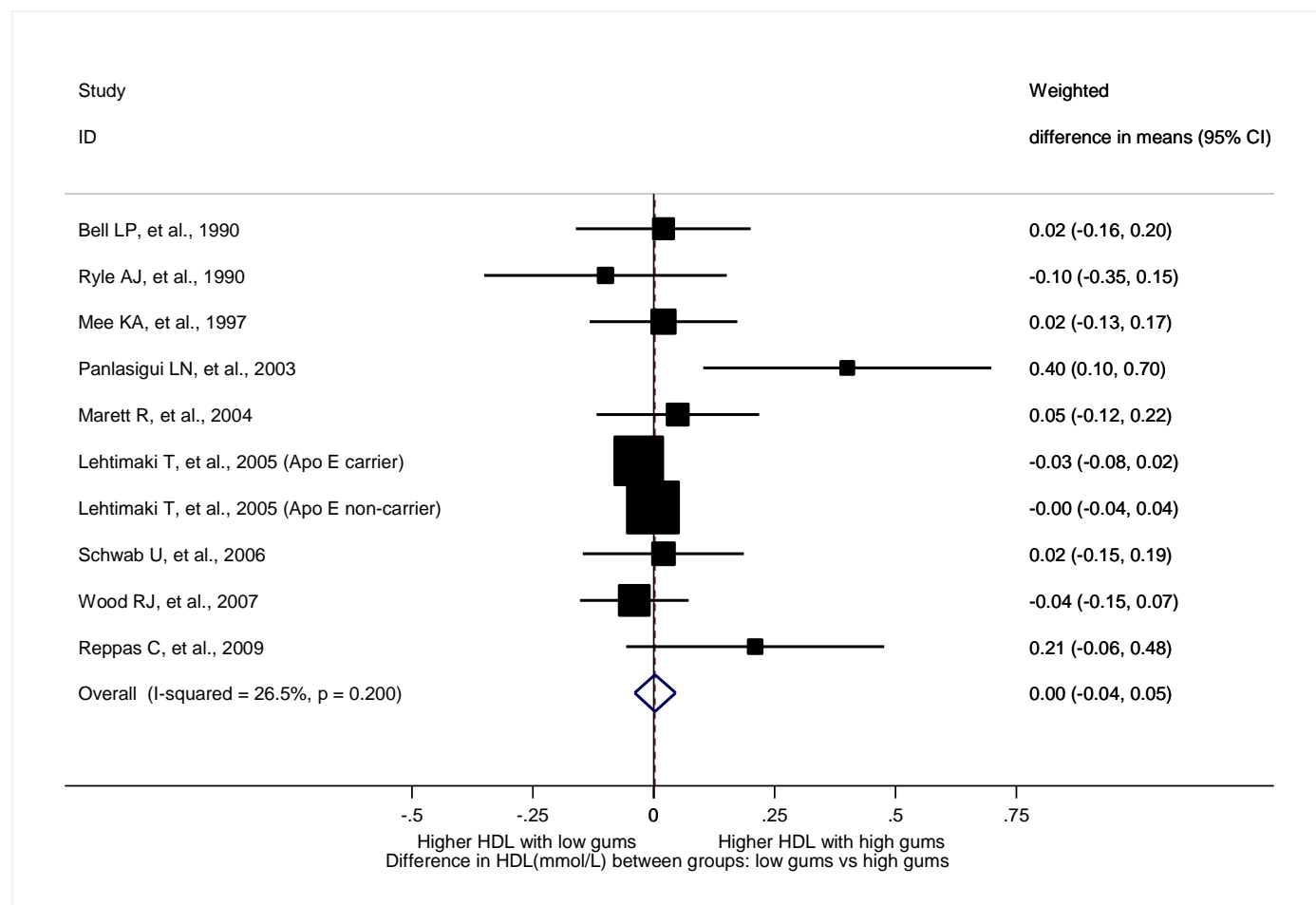


Figure 2.66 Contour-enhanced funnel plot for studies presenting data on dietary fibre isolates as gums and other extracts and HDL cholesterol

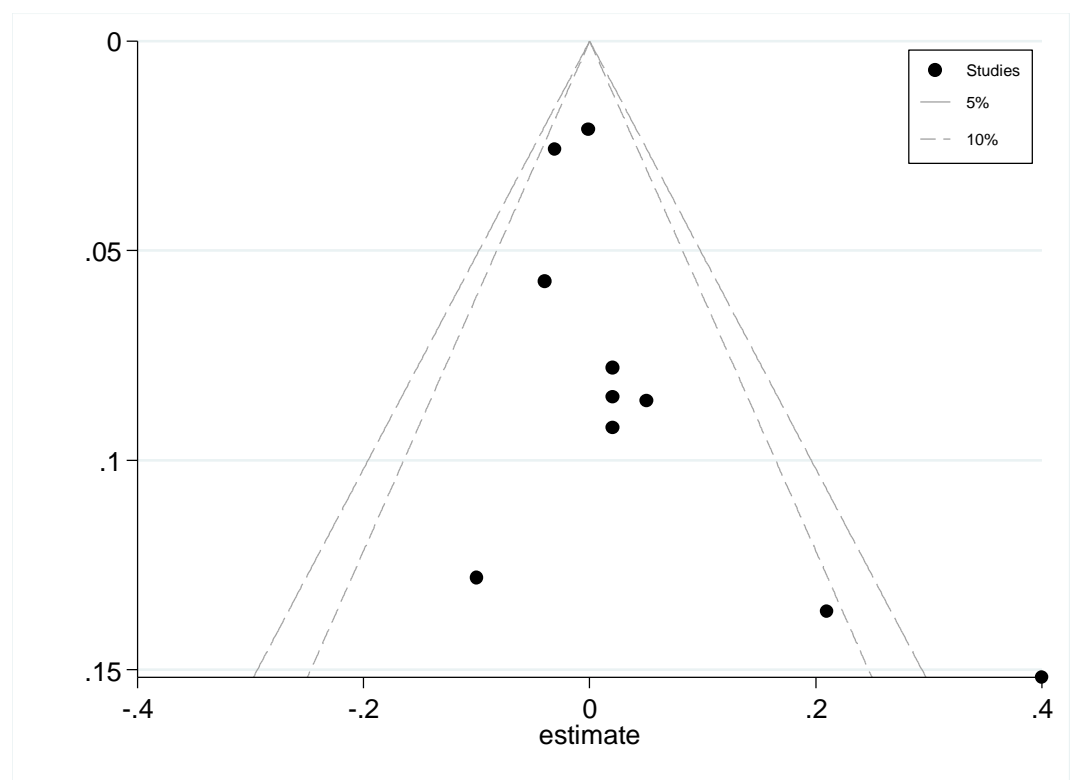


Table 2.123 HDL cholesterol and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Bell <i>et al.</i> , 1990) 17191		Pectin enriched cereal	20/20		1.21 (SE 0.06)	0.03	NS	NS	HDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
		Placebo	19/20		1.19 (SE 0.07)	0.02	NS					No change	
		Psyllium enriched cereal	19/20		1.19 (SE 0.08)	-0.02	NS	NS				No change	
(Lehtimäki <i>et al.</i> , 2005) *17502	Apo E genotype E4 carrier	Encapsulated microcrystalline chitosan	86/96	1.54 (SD 0.39)	1.5 (SD 0.37)	-1.4 (SD 8.6)		NS	HDL-C	Fasting plasma (mmol/L)	3 months	Not reported	No bias
		Starch capsules	85/96	1.54 (SD 0.39)	1.57 (SD 0.41)	0.6 (SD 12.9)						Not reported	
*17503	Apo E genotype E4 non-carrier	Encapsulated microcrystalline chitosan	86/96	1.54 (SD 0.29)	1.53 (SD 0.36)	-1.2 (SD 8.8)		NS	HDL-C	Fasting plasma (mmol/L)	3 months	Not reported	No bias
		Starch capsules	85/96	1.54 (SD 0.29)	1.51 (SD 0.32)	-1.1 (SD 9.1)						Not reported	
(Marett and Slavin, 2004) 16646		Larch arabinogalactan	18/18	1.25 (SD 0.27)	1.21 (SD 0.29)		NS	NS	HDL-C	Fasting plasma (mmol/L)	1 month	No change	No bias
		Placebo	17/17	1.34 (SD 0.26)	1.32 (SD 0.35)		NS	NS				No change	
		Tamarack arabinogalactan	19/19	1.32 (SD 0.22)	1.32 (SD 0.27)		NS	NS				No change	
16647		Larch arabinogalactan	18/18	1.25 (SD 0.27)	1.13 (SD 0.24)		NS	NS	HDL-C	Fasting plasma (mmol/L)	2 months	No change	No bias
		Placebo	17/17	1.34 (SD 0.26)	1.30 (SD 0.3)		NS	NS				No change	
		Tamarack arabinogalactan	19/19	1.32 (SD 0.22)	1.34 (SD 0.37)		NS	NS				No change	
16648		Larch arabinogalactan	18/18	1.25 (SD 0.27)	1.17 (SD 0.3)		NS	NS	HDL-C	Fasting plasma (mmol/L)	3 months	No change	No bias
		Placebo	17/17	1.34 (SD	1.33		NS	NS				No change	

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Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
				0.26)	(SD 0.22)								
		Tamarack arabinogalactan	19/19	1.32 (SD 0.22)	1.35 (SD 0.35)		NS	NS				No change	
16649		Larch arabinogalactan	18/18	1.25 (SD 0.27)	1.22 (SD 0.3)		NS	NS	HDL-C	Fasting plasma (mmol/L)	4 months	No change	No bias
		Placebo	17/17	1.34 (SD 0.26)	1.31 (SD 0.3)		NS	NS				No change	
		Tamarack arabinogalactan	19/19	1.32 (SD 0.22)	1.33 (SD 0.29)		NS	NS				No change	
16650		Larch arabinogalactan	18/18	1.25 (SD 0.27)	1.20 (SD 0.27)		NS	NS	HDL-C	Fasting plasma (mmol/L)	5 months	No change	No bias
		Placebo	17/17	1.34 (SD 0.26)	1.32 (SD 0.28)		NS	NS				No change	
		Tamarack arabinogalactan	19/19	1.32 (SD 0.22)	1.37 (SD 0.35)		NS	NS				No change	
*16651		Larch arabinogalactan	18/18	1.25 (SD 0.27)	1.24 (SD 0.25)		NS	NS	HDL-C	Fasting plasma (mmol/L)	6 months	No change	No bias
		Placebo	17/17	1.34 (SD 0.26)	1.30 (SD 0.22)		NS	NS				No change	
		Tamarack arabinogalactan	19/19	1.32 (SD 0.22)	1.35 (SD 0.29)		NS	NS				No change	
(Mee and Gee, 1997) *15633		Filtered apple juice	25/27	1.14 (SD 0.21)	1.07 (SD 0.26)		<0.05		HDL-C	Fasting serum (mmol/L)	6 weeks	No change	unclear
		Gum arabic- supplemented apple juice	25/27	1.14 (SD 0.21)	1.09 (SD 0.29)		<0.05	NS				No change	
(Panlasig ui <i>et al.</i> , 2003) *15191		Carageenan-added test foods	20/20		1.65 (SD 0.49)				HDL-C	Fasting serum (mmol/L)	8 weeks	No change	unclear
		Usual diet	20/20		1.25 (SD 0.47)			0.0071				No change	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Pasman <i>et al.</i> , 1997a) 15523		Control	11/14				NS		HDL-C	Fasting plasma	14 months	Increase	unclear
		Guar gum - High compliance	10/10				NS	NS				Increase	
		Guar Gum - Low compliance	10/10				NS	NS				Increase	
(Reppas <i>et al.</i> , 2009) 16798		High dose Hydroxypropylmethylcel lulose	completers not reported/10	1.22 (SE 0.1)	1.08 (SE 0.10)		<0.05	No diet X time effect	HDL	Serum (mmol/L)	6 weeks	Not reported	No bias
		Low dose Hydroxypropylmethylcel lulose	completers not reported/10	1.12 (SE 0.05)	1.01 (SE 0.04)		<0.05					Not reported	
		Placebo	completers not reported/10	1.05 (SE 0.08)	0.95 (SE 0.07)		NS					Not reported	
16799		High dose Hydroxypropylmethylcel lulose	completers not reported/10	1.22 (SE 0.1)	1.05 (SE 0.10)		<0.05		HDL	Serum (mmol/L)	7 weeks	Not reported	No bias
		Low dose Hydroxypropylmethylcel lulose	completers not reported/10	1.12 (SE 0.05)	1.03 (SE 0.04)		<0.05					Not reported	
		Placebo	completers not reported/10	1.05 (SE 0.08)	0.98 (SE 0.07)		NS					Not reported	
*16800		High dose Hydroxypropylmethylcel lulose	completers not reported/10	1.22 (SE 0.1)	1.17 (SE 0.11)		<0.05	NS	HDL	Serum (mmol/L)	8 weeks	Not reported	No bias
		Low dose Hydroxypropylmethylcel lulose	completers not reported/10	1.12 (SE 0.05)	1.02 (SE 0.04)		<0.05	NS				Not reported	
		Placebo	completers not reported/10	1.05 (SE 0.08)	0.96 (SE 0.08)		NS					Not reported	
(Ryle <i>et al.</i> , 1990) *16205		High glucose low soluble fibre	11/11	1.1 (SD 0.3)	1.1 (SD 0.3)		NS		HDL-C	Fasting (mmol/L)	6 weeks	No change	unclear
		Low glucose high soluble fibre diet (guar gum)	11/11	1.1 (SD 0.3)	1.0 (SD 0.3)		NS	NS				No change	
(Schwab <i>et al.</i> , 2006) 16488		Pectin	22/22	1.12 (SD 0.23)	1.15 (SD 0.25)			NS	Log HDL-C	Fasting Serum, (mmol/L)	8 weeks	Decrease	No bias
		Placebo	22/22	1.14 (SD 0.32)	1.15 (SD 0.34)							Decrease	

Author/ Result ID	Subgroup detail	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
*16489		Polydextrose	22/22	1.25 (SD 0.32)	1.29 (SD 0.31)			NS				Decrease	
		Pectin	22/22	1.12 (SD 0.23)	1.19 (SD 0.25)		<0.05	NS	Log HDL-C	Fasting Serum, (mmol/L)	12 weeks	Decrease	No bias
		Placebo	22/22	1.14 (SD 0.32)	1.17 (SD 0.31)		NS					Decrease	
		Polydextrose	22/22	1.25 (SD 0.32)	1.32 (SD 0.35)		<0.05	NS				Decrease	
(Wood <i>et al.</i> , 2007) 17229		Low carbohydrate diet + placebo	15/15	1.08 (SD 0.33)	1.18 (SD 0.35)				HDL-C	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	1.07 (SD 0.26)	1.14 (SD 0.27)			NS				Decrease	
*17230		Low carbohydrate diet + placebo	15/15	1.08 (SD 0.33)	1.23 (SD 0.35)	0.15 (SD 0.18)			HDL-C	Fasting plasma (mmol/L)	12 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	1.07 (SD 0.26)	1.17 (SD 0.32)	0.11 (SD 0.13)		NS				Decrease	

*This result was used in the meta-analysis of gums and extracts and HDL cholesterol

LDL cholesterol and fibre isolates, gum and extracts

No cohort studies reported results concerning fibre isolates, gum and extracts and LDL cholesterol.

Summary of RCT data

Nine studies provided data on the effects of a variety of gums and extracts, which included guar gum, arabinogalactan and pectin, on LDL cholesterol (Bell *et al.*, 1990; Pasman *et al.*, 1997a; Wood *et al.*, 2007; Marett and Slavin, 2004; Mee and Gee, 1997; Schwab *et al.*, 2006; Lehtimäki *et al.*, 2005; Panlasigui *et al.*, 2003; Reppas *et al.*, 2009). These studies also provided data on total cholesterol and so study details are provided in the Trial Characteristics table and in the text concerning total cholesterol.

Data from Pasman *et al.* (Pasman *et al.*, 1997a) were not included in the meta-analysis as quantitative data were not reported. In this study, the authors (Pasman *et al.*, 1997a) provided data on the effects of guar gum supplements on blood lipids in weight-reduced subjects for maintenance of weight loss over 16 months. This study compared a 20g water soluble fibre (guar gum) supplement with no treatment condition for 14 months, following a two month very low calorie diet. LDL cholesterol was not affected by supplementation of the diet with guar gum.

Eight studies were included in the meta-analysis comparing different gums intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months. There were too few studies to explore sources of bias through construction of a funnel plot.

The pooled estimate indicated that LDL cholesterol was 0.09mmol/L (95% CI -0.07 to 0.25) lower with consumption of a diet higher in gums and extracts. This was not significantly different from zero ($p=0.29$). Overall heterogeneity denoted by I^2 was 49% (95% CI 0 to 76%). Statistically, there was no evidence that a diet higher in dietary fibre in the form of gums or extracts is associated with changes in LDL cholesterol.

Figure 2.67 Forest plot for fibre isolates, gums and extract and LDL cholesterol (mmol/L)

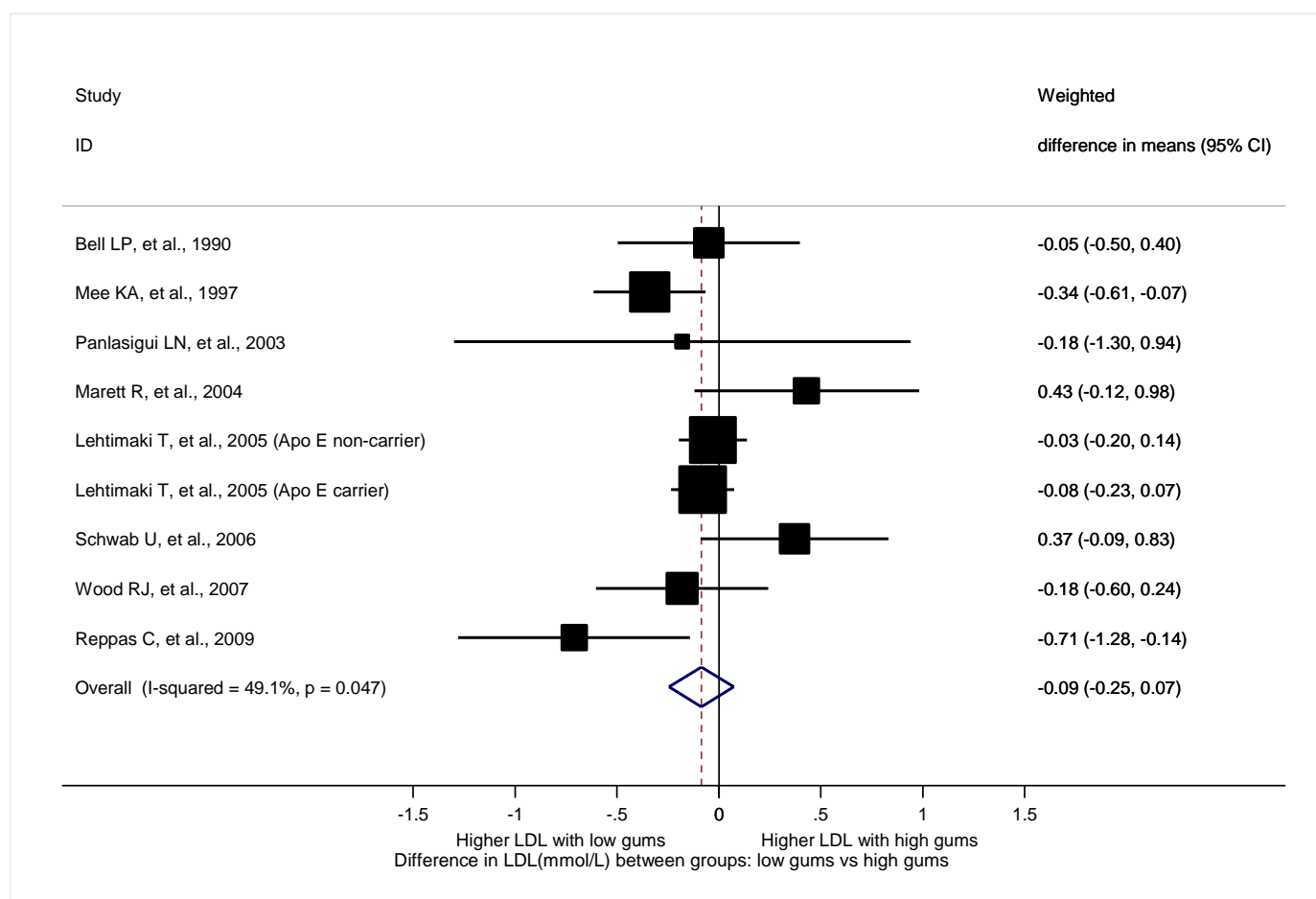


Table 2.124 LDL cholesterol and fibre isolates, gum and extracts: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Bell <i>et al.</i> , 1990) 17170	Pectin enriched cereal	20/20		3.76 (SE 0.14)	-0.16	NS	NS	LDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Placebo	19/20		3.81 (SE 0.18)	-0.02	NS					No change	
	Psyllium enriched cereal	19/20		3.6 (SE 0.12)	-0.23	0.0066	0.034				No change	
(Lehtimäki <i>et al.</i> , 2005) 17500	Encapsulated microcrystalline chitosan	86/96	3.83 (SD 0.58)	3.5 (SD 0.56)	-7.2% (SD 14.3)		NS	LDL-C	Derived by calculatio n Fasting, Plasma (mmol/L)	3 months	Not reported	No bias
	Starch capsules	85/96	3.83 (SD 0.58)	3.6 (SD 0.6)	-5% (SD 12.5)						Not reported	
*17501	Encapsulated microcrystalline chitosan	86/96	3.81 (SD 0.69)	3.49 (SD 0.66)	-7.7% (SD 14.2)		NS	LDL-C	Derived by calculatio n Fasting, Plasma (mmol/L)	3 months	Not reported	No bias
	Starch capsules	85/96	3.81 (SD 0.69)	3.53 (SD 0.72)	-6.9% (SD 15.1)						Not reported	
(Marett and Slavin, 2004) 16641	Larch arabinogalactan	18/18	2.85 (SD 1.0)	2.59 (SD 0.85)		NS	NS	LDL-C	Fasting plasma (mmol/L)	2 months	No change	No bias
	Placebo	17/17	2.73 (SD 0.83)	2.59 (SD 0.86)		NS	NS				No change	
	Tamarack arabinogalactan	19/19	2.84 (SD 0.96)	2.66 (SD 0.73)		NS	NS				No change	
16642	Larch arabinogalactan	18/18	2.85 (SD 1.0)	2.74 (SD 0.93)		NS	NS	LDL-C	Fasting plasma (mmol/L)	3 months	No change	No bias
	Placebo	17/17	2.73 (SD 0.83)	2.74 (SD 0.93)		NS	NS				No change	
	Tamarack arabinogalactan	19/19	2.84 (SD 0.96)	2.75 (SD 0.65)		NS	NS				No change	
16643	Larch arabinogalactan	18/18	2.85 (SD 1.0)	2.62 (SD 0.87)		NS	NS	LDL-C	Fasting plasma (mmol/L)	4 months	No change	No bias

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
16644	Placebo	17/17	2.73 (SD 0.83)	2.62 (SD 0.87)		NS	NS				No change	
	Tamarack arabinogalactan	19/19	2.84 (SD 0.96)	2.72 (SD 0.80)		NS	NS				No change	
	Larch arabinogalactan	18/18	2.85 (SD 1.0)	2.63 (SD 0.92)		NS	NS	LDL-C	Fasting plasma (mmol/L)	5 months	No change	No bias
	Placebo	17/17	2.73 (SD 0.83)	2.63 (SD 0.92)		NS	NS				No change	
	Tamarack arabinogalactan	19/19	2.84 (SD 0.96)	2.90 (SD 1.0)		NS	NS				No change	
	Larch arabinogalactan	18/18	2.85 (SD 1.0)	2.55 (SD 0.76)		NS	NS	LDL-C	Fasting plasma (mmol/L)	6 months	No change	No bias
*16645	Placebo	17/17	2.73 (SD 0.83)	2.55 (SD 0.76)		NS	NS				No change	
	Tamarack arabinogalactan	19/19	2.84 (SD 0.96)	2.98 (SD 0.92)		NS	NS				No change	
	Larch arabinogalactan	18/18	2.85 (SD 1.0)	2.55 (SD 0.76)		NS	NS	LDL-C	Fasting plasma (mmol/L)	6 months	No change	No bias
(Mee and Gee, 1997) *15632	Filtered apple juice	25/27	4.03 (SD 0.60)	3.80 (SD 0.52)		<0.05		LDL-C	Fasting serum (mmol/L)	6 weeks	No change	unclear
	Gum arabic- supplemented apple juice	25/27	4.03 (SD 0.60)	3.46 (SD 0.47)		<0.05	<0.05				No change	
(Panlasigui <i>et al.</i> , 2003) *15192	Carageenan-added test foods	20/20		3.07 (SD 1.64)				LDL-C	Fasting serum (mmol/L)	8 weeks	No change	unclear
	Usual diet	20/20		3.25 (SD 1.96)			NS				No change	
(Pasman <i>et al.</i> , 1997a) 15524	Control	11/14				NS		LDL-C	Fasting plasma	14 months	Increase	unclear
	Guar gum - High compliance	10/10				NS	NS				Increase	
	Guar Gum - Low compliance	10/10				NS	NS				Increase	
(Reppas <i>et al.</i> , 2009) 16790	High dose Hydroxypropylmethylc ellulose	completers not reported/10	3.95 (SE 0.15)	3.25 (SE 0.16)		<0.05	Significant diet x time effect	LDL-C	Serum (mmol/L)	6 weeks	Not reported	No bias
	Low dose Hydroxypropylmethylc ellulose	completers not reported/10	4.15 (SE 0.13)	3.87 (SE 0.12)		<0.05					Not reported	
	Placebo	completers not reported/10	4.16 (SE 0.13)	4.3 (SE 0.23)		NS					Not reported	

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
16791	High dose Hydroxypropylmethylc ellulose	completers not reported/10	3.95 (SE 0.15)	3.24 (SE 0.20)		<0.05		LDL-C	Serum (mmol/L)	7 weeks	Not reported	No bias
	Low dose Hydroxypropylmethylc ellulose	completers not reported/10	4.15 (SE 0.13)	3.81 (SE 0.11)		<0.05					Not reported	
	Placebo	completers not reported/10	4.16 (SE 0.13)	4.46 (SE 0.17)		NS					Not reported	
*16792	High dose Hydroxypropylmethylc ellulose	completers not reported/10	3.95 (SE 0.15)	3.58 (SE 0.20)		<0.05	<0.05	LDL-C	Serum (mmol/L)	8 weeks	Not reported	No bias
	Low dose Hydroxypropylmethylc ellulose	completers not reported/10	4.15 (SE 0.13)	3.78 (SE 0.10)		<0.05	<0.05				Not reported	
	Placebo	completers not reported/10	4.16 (SE 0.13)	4.29 (SE 0.21)		NS					Not reported	
(Schwab <i>et al.</i> , 2006) 16491	Pectin	22/22	3.88 (SD 1.04)	3.7 (SD 0.87)			NS	LDL-C	Fasting serum (mmol/L)	8 weeks	Decrease	No bias
	Placebo	22/22	3.64 (SD 0.71)	3.45 (SD 0.87)							Decrease	
	Polydextrose	22/22	3.58 (SD 0.80)	3.38 (SD 0.85)			NS				Decrease	
*16492	Pectin	22/22	3.88 (SD 1.04)	3.65 (SD 0.77)		NS	NS	LDL-C	Fasting serum (mmol/L)	12 weeks	Decrease	No bias
	Placebo	22/22	3.64 (SD 0.71)	3.28 (SD 0.79)		<0.05					Decrease	
	Polydextrose	22/22	3.58 (SD 0.80)	3.29 (SD 0.71)		<0.05	NS				Decrease	
(Wood <i>et al.</i> , 2007) *17232	Low carbohydrate diet + placebo	15/15	2.89 (SD 1.16)	2.72 (SD 0.95)	-0.18 (SD 0.59)			LDL-C	Derived by calculatio n Fasting, Plasma (mmol/L)	12 weeks	Decrease	No bias
	Low carbohydrate diet + Soluble fibre	14/15	2.98 (SD 0.72)	2.61 (SD 0.72)	-0.36 (SD 0.59)		NS				Decrease	

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

Triacylglycerol and fibre isolates, gums and extracts

No cohort studies reported results concerning gums and extracts and TAG.

Summary of RCT data

Twelve studies, reported in thirteen papers, provided data on the effects of a variety of gums and extracts on TAG (Bell *et al.*, 1990;Vido *et al.*, 1993;Landin *et al.*, 1992;Ryle *et al.*, 1990;Garcia *et al.*, 2007;Garcia *et al.*, 2006;Wood *et al.*, 2007;Marett and Slavin, 2004;Mee and Gee, 1997;Schwab *et al.*, 2006;Lehtimaki *et al.*, 2005;Panlasigui *et al.*, 2003;Reppas *et al.*, 2009). Two also explored the effects of gums and extracts on non-esterified fatty acids (Garcia *et al.*, 2007;Garcia *et al.*, 2006). Other than Landin *et al.* (Landin *et al.*, 1992), which only reported TAG data, these studies also provided data on total cholesterol and so study details are provided in the Trial Characteristics table and in the text concerning total cholesterol.

Two studies were not included in the meta-analysis (Vido *et al.*, 1993;Garcia *et al.*, 2007).

It was not possible to include Vido *et al.* (Vido *et al.*, 1993) in the meta-analysis as the participants used were children less than 15 years of age. In their double blind, placebo-controlled trial, Vido *et al.* randomised children to receive either glucomannan (2g/day) or a placebo product (Vido *et al.*, 1993) for two months whilst consuming their usual diet. After the intervention, TAG levels in the glucomannan supplement group were statistically significantly higher than the placebo group ($p=0.05$). TAG decreased in the placebo group, although there was no statistically significant difference from baseline.

Garcia *et al.* (Garcia *et al.*, 2007) and Garcia *et al.* (Garcia *et al.*, 2006) report results from the same study and therefore results from the former paper were not included in the meta-analysis. The authors used overweight subjects with impaired glucose tolerance to compare an arabinoxylan supplement with an identical placebo intervention. Body weights were unchanged during the trial. After six weeks, TAG AUC post meal response was statistically significantly lower in the intervention group compared with the placebo group ($p=0.001$).

Eleven studies were included in the meta-analysis comparing different gums or fibre isolates and changes in TAG reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six weeks to six months. The pooled estimate indicated that TAG levels were 0.01mmol/L (95% CI -0.07 to 0.09) higher with consumption of the higher fibre diet. This was not significantly different from zero ($p=0.84$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 48%). There is a suggestion of asymmetry in the funnel plot, but this could be the result of chance. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was no evidence that a diet higher in dietary fibre in the form of gums or extracts is associated with differences in TAG.

Figure 2.68 Forest plot for fibre isolates, gums and extract and TAG (mmol/L)

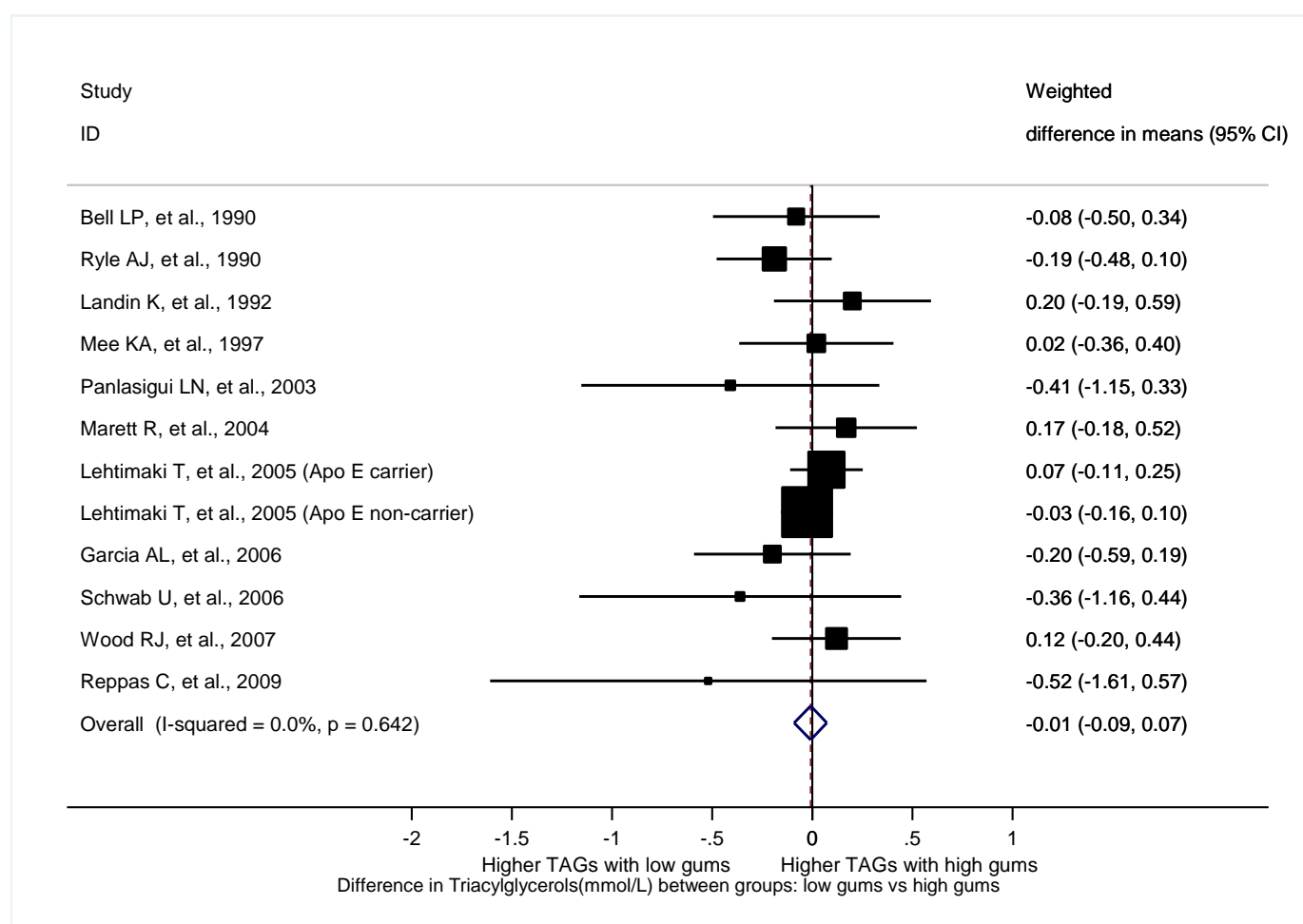
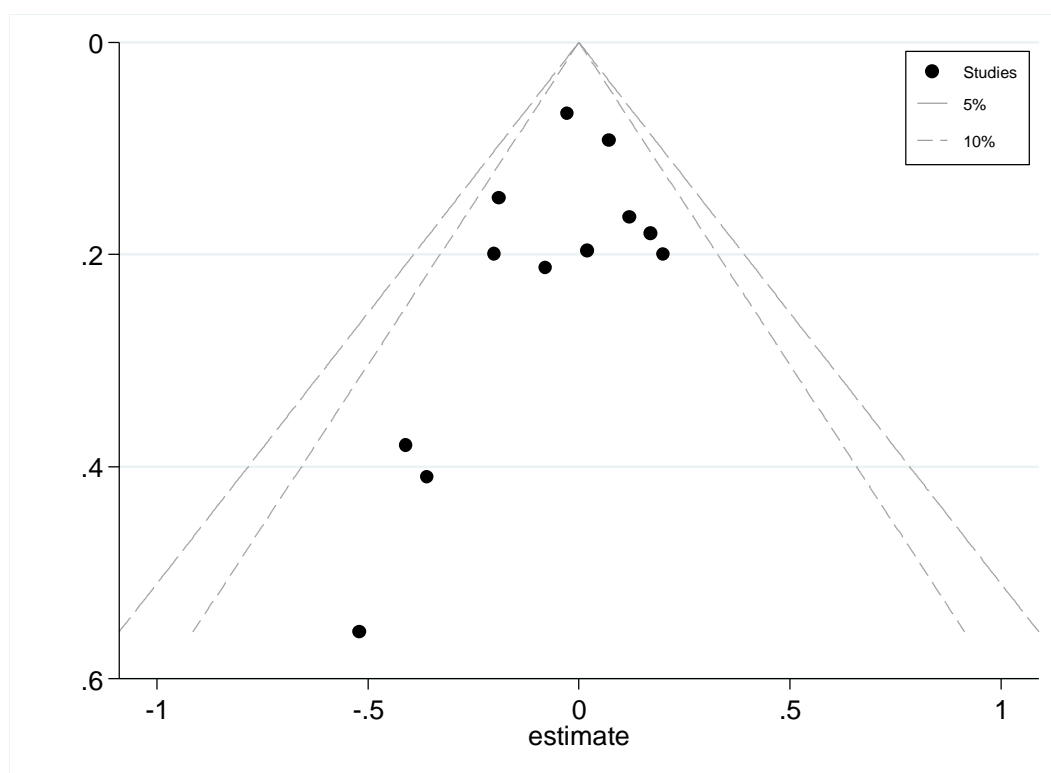


Figure 2.69 Contour-enhanced funnel plot for studies presenting data on dietary fibre isolates as gums and other extracts and TAG



One study also explored the effects of gums intake on non-esterified fatty acids (Garcia *et al.*, 2006). Garcia *et al.* (Garcia *et al.*, 2006) used overweight subjects with impaired glucose tolerance to compare an arabinoxylan supplement with an identical placebo intervention. Body weights were unchanged during the trial. Comparison of non-esterified fatty acid values at six weeks indicated there was no statistically significant difference between the arabinoxylan diet group and the placebo group ($p=0.874$).

Table 2.125 Triacylglycerol and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
Adolescent study															
(Vido <i>et al.</i> , 1993) 16941		Glucomannan supplement	30/30	73.2 (SD 37.9)	96.9 (SD 70.2)		<0.05	<0.05			TAG	Not reported (mg/dL)	2 months	Decrease	unclear
		Placebo	30/30	107 (SD 55.7)	80 (SD 65.8)		NS							Decrease	
Adult studies															
(Bell <i>et al.</i> , 1990) 17192		Pectin enriched cereal	20/20		1.3 (SE 0.16)	0.05	NS	NS			TAG	Fasting serum (mmol/L)	6 weeks	No change	No bias
		Placebo	19/20		1.38 (SE 0.14)	0.03	NS							No change	
		Psyllium enriched cereal	19/20		1.07 (SE 0.15)	-0.135	NS	NS						No change	
(Garcia <i>et al.</i> , 2006) *17375		Arabinoxylan	11/11	1.8 (CI 1.6, 2)	1.6 (CI 1.4, 2)			0.047			TAG	Fasting Geometric mean, Serum (mmol/L)	6 weeks	No change	unclear
		Placebo	11/11	1.5 (CI 1.3, 1.7)	1.8 (CI 1.6, 2.1)									No change	
(Garcia <i>et al.</i> , 2007) 17406		Arabinoxylan	11/11		lower			0.001			TAG AUC post meal response	4 hour AUC Serum (pmol/L)	6 weeks	No change	unclear
		Placebo	11/11											No change	
(Landin <i>et al.</i> , 1992) *17119		Guar gum minus placebo	Crossover: 25/25						0.2 (SD 1)	<0.05	TAG	Fasting serum (mmol/L)	6 weeks	No change in both	No bias
(Lehtimäki <i>et al.</i> , 2005) *17504	Apo E genotype E4 carrier	Encapsulated microcrystalline chitosan	86/96	1.38 (SD 0.61)	1.42 (SD 0.73)	7.1% (SD 44.8)		NS			TAG	Fasting plasma (mmol/L)	3 months	Not reported	No bias
		Starch capsules	85/96	1.38 (SD 0.61)	1.34 (SD 0.48)	2% (SD 42.9)								Not reported	
*17505	Apo E	Encapsulated microcrystalline	86/96	1.29 (SD	1.26	-2.6%		NS			TAG	Fasting	3	Not	No bias

Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
(Marett and Slavin, 2004) 16635	genotype E4 non- carrier	chitosan		0.58)	(SD 0.77)	(SD 35)						plasma (mmol/L)	months	reported	
		Starch capsules	85/96	1.29 (SD 0.58)	1.29 (SD 0.61)	-0.5% (SD 32.8)								Not reported	
		Larch arabinogalactan	18/18	1.17 (SD 0.46)	1.25 (SD 0.74)		NS	NS			TAG	Fasting plasma (mmol/L)	2 months	No change	No bias
		Placebo	17/17	1.21 (SD 0.89)	0.99 (SD 0.37)		NS	NS						No change	
16636		Tamarack arabinogalactan	19/19	1.02 (SD 0.46)	1.00 (SD 0.44)		NS	NS						No change	
		Larch arabinogalactan	18/18	1.17 (SD 0.46)	1.17 (SD 0.76)		NS	NS			TAG	Fasting plasma (mmol/L)	3 months	No change	No bias
		Placebo	17/17	1.21 (SD 0.89)	0.99 (SD 0.46)		NS	NS						No change	
		Tamarack arabinogalactan	19/19	1.02 (SD 0.46)	1.16 (SD 0.54)		NS	NS						No change	
16637		Larch arabinogalactan	18/18	1.17 (SD 0.46)	1.14 (SD 0.68)		NS	NS			TAG	Fasting plasma (mmol/L)	4 months	No change	No bias
		Placebo	17/17	1.21 (SD 0.89)	1.10 (SD 0.76)		NS	NS						No change	
		Tamarack arabinogalactan	19/19	1.02 (SD 0.46)	1.24 (SD 0.67)		NS	NS						No change	
		Larch arabinogalactan	18/18	1.17 (SD 0.46)	1.21 (SD 1.07)		NS	NS			TAG	Fasting plasma (mmol/L)	5 months	No change	No bias
16638		Placebo	17/17	1.21 (SD 0.89)	1.02 (SD 0.47)		NS	NS						No change	
		Tamarack arabinogalactan	19/19	1.02 (SD 0.46)	1.09 (SD 0.54)		NS	NS						No change	
		Larch arabinogalactan	18/18	1.17 (SD 0.46)	1.21 (SD 1.07)		NS	NS			TAG	Fasting plasma (mmol/L)	5 months	No change	No bias
		Placebo	17/17	1.21 (SD 0.89)	1.02 (SD 0.47)		NS	NS						No change	

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
*16639	Larch arabinogalactan	18/18	1.17 (SD 0.46)	1.15 (SD 0.71)		NS	NS				TAG	Fasting plasma (mmol/L)	6 months	No change	No bias
	Placebo	17/17	1.21 (SD 0.89)	1.01 (SD 0.55)		NS	NS							No change	
	Tamarack arabinogalactan	19/19	1.02 (SD 0.46)	1.18 (SD 0.53)		NS	NS							No change	
(Mee and Gee, 1997) *15634	Filtered apple juice	25/27	1.53 (SD 0.68)	1.57 (SD 0.62)		NS					TAG	Fasting serum (mmol/L)	6 weeks	No change	unclear
	Gum arabic-supplemented apple juice	25/27	1.53 (SD 0.68)	1.59 (SD 0.76)		NS	NS							No change	
(Panlasigui <i>et al.</i> , 2003) *15190	Carageenan-added test foods	20/20		0.87 (SD 1.16)							TAG	Fasting serum (mmol/L)	8 weeks	No change	unclear
	Usual diet	20/20		1.28 (SD 1.24)				0.0006						No change	
(Reppas <i>et al.</i> , 2009) 16806	High dose Hydroxypropylmethylcellulose	completers not reported/10	2.01 (SE 0.32)	1.97 (SE 0.38)		NS	No diet X time effect				TAG	Serum (mmol/L)	6 weeks	Not reported	No bias
	Low dose Hydroxypropylmethylcellulose	completers not reported/10	1.74 (SE 0.12)	1.66 (SE 0.13)		NS								Not reported	
	Placebo	completers not reported/10	2.18 (SE 0.08)	2.18 (SE 0.25)		NS								Not reported	
16807	High dose Hydroxypropylmethylcellulose	completers not reported/10	2.01 (SE 0.32)	2.12 (SE 0.42)		NS					TAG	Serum (mmol/L)	7 weeks	Not reported	No bias
	Low dose Hydroxypropylmethylcellulose	completers not reported/10	1.74 (SE 0.12)	1.77 (SE 0.14)		NS								Not reported	
	Placebo	completers not reported/10	2.18 (SE 0.08)	2.04 (SE 0.19)		NS								Not reported	
*16808	High dose Hydroxypropylmethylcellulose	completers not reported/10	2.01 (SE 0.32)	1.78 (SE 0.34)		NS	NS				TAG	Serum (mmol/L)	8 weeks	Not reported	No bias

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
		Low dose Hydroxypropylmethylcellulose	completers not reported/10	1.74 (SE 0.12)	1.95 (SE 0.01)		NS	NS							Not reported
		Placebo	completers not reported/10	2.18 (SE 0.08)	2.3 (SE 0.44)		NS								Not reported
(Ryle <i>et al.</i> , 1990) *16206		High glucose low soluble fibre	11/11	0.77 (SD 0.33)	0.89 (SD 0.39)		NS				TAG	Fasting (mmol/L)	6 weeks	No change	unclear
		Low glucose high soluble fibre diet (guar gum)	11/11	0.77 (SD 0.33)	0.7 (SD 0.29)		NS	NS							No change
(Schwab <i>et al.</i> , 2006) 16497		Pectin	22/22	1.59 (SD 1.21)	1.85 (SD 1.20)			NS			Log TAG	Fasting serum, (mmol/L)	8 weeks	Decrease	No bias
		Placebo	22/22	1.84 (SD 0.95)	1.96 (SD 1.19)									Decrease	
		Polydextrose	22/22	1.30 (SD 0.51)	1.61 (SD 1.03)			NS						Decrease	
*16498		Pectin	22/22	1.59 (SD 1.21)	1.60 (SD 0.25)		NS	NS			Log TAG	Fasting serum, (mmol/L)	12 weeks	Decrease	No bias
		Placebo	22/22	1.84 (SD 0.95)	1.96 (SD 1.46)		NS							Decrease	
		Polydextrose	22/22	1.30 (SD 0.51)	1.46 (SD 0.71)		NS	NS						Decrease	
(Wood <i>et al.</i> , 2007) 17227		Low carbohydrate diet + placebo	15/15	1.34 (SD 0.68)	0.86 (SD 0.47)						TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	1.31 (SD 0.45)	0.94 (SD 0.41)			NS						Decrease	
*17228		Low carbohydrate diet + placebo	15/15	1.34 (SD 0.68)	0.77 (SD 0.35)	-0.57 (SD 0.47)					TAG	Fasting plasma (mmol/L)	12 weeks	Decrease	No bias
		Low carbohydrate diet + Soluble fibre	14/15	1.31 (SD 0.45)	0.86 (SD 0.39)	-0.45 (SD 0.43)		NS						Decrease	
Non-esterified fatty															

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Author/ Result ID	Subgroup detail	Intervention groups	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 at follow-up	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
acids															
(Garcia <i>et al.</i> , 2006) 17376		Arabinoxylan	11/11	489 (CI 437, 547)	565 (CI 478, 668)			0.874			Non- esterified fatty acids	Fasting Geometri c mean, Serum (μmol/L)	6 weeks	No change	unclear
		Placebo	11/11	497 (CI 455, 544)	569 (CI 520, 624)									No change	

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

One paper (Garcia *et al.*, 2007) presented results for TAG and non-esterified fatty acids; however these have not been extracted as they are reported here in another paper (Garcia *et al.*, 2006).

Total cholesterol:HDL ratio and fibre isolates, gums and extracts

No cohort studies reported results concerning fibre isolates, gums and extracts and TC:HDL ratio.

Summary of RCT data

One study provided data. In the study reported by Schwab *et al.* (Schwab *et al.*, 2006), 70 overweight participants were randomly assigned to one of three groups: i) a drink enriched with pectin; ii) a drink enriched with polydextrose; or iii) a drink without fibre enrichment. The TC:HDL ratio, at 12 weeks but not at eight weeks, had statistically significantly decreased from baseline in all three groups ($p < 0.05$). Between groups, however, no differences were observed. It is however noteworthy to highlight that all groups lost weight during the intervention and, since blood lipids are modified by body weight change, any differences in outcome may not be solely attributable to the carbohydrate component of the dietary intervention.

Table 2.126 Total cholesterol:HDL ratio and fibre isolates, gums and extracts: RCT data

Author / Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Schwab <i>et al.</i> , 2006) 16494	Pectin	22/22	5.31 (SD 1.49)	5.13 (SD 1.18)		NS	TC :HDL ratio	8 weeks	Decrease	No bias
	Placebo	22/22	5.20 (SD 1.40)	5.16 (SD 1.71)					Decrease	
	Polydextrose	22/22	4.56 (SD 1.34)	4.34 (SD 1.22)		NS			Decrease	
16495	Pectin	22/22	5.31 (SD 1.49)	4.85 (SD 1.16)	<0.05	NS	TC :HDL ratio	12 weeks	Decrease	No bias
	Placebo	22/22	5.20 (SD 1.40)	4.85 (SD 1.55)	<0.05				Decrease	
	Polydextrose	22/22	4.56 (SD 1.34)	4.22 (SD 1.22)	<0.05	NS			Decrease	

Total cholesterol:LDL ratio and fibre isolates, gums and extracts

No cohort studies reported results concerning TC:LDL ratio.

Summary of RCT data

One trial of healthy subjects provided data on TC:LDL ratio and the effects of arabinogalactan supplementation from two sources (Marett and Slavin, 2004). Body weights were unchanged in this trial. Marett and Slavin (Marett and Slavin, 2004) conducted a six-month randomised, double-blind, parallel trial to explore the physiological effects of arabinogalactan (soluble fibre) supplementation from larch or tamarack. Fifty-four subjects were given 8.4g/day placebo (rice starch), 8.4g/day larch arabinogalactan supplement or 8.4g/day tamarack arabinogalactan supplement and instructed to consume this within a beverage or with food. Quantitative data were not recorded, although the authors concluded that no differences in TC:LDL ratio from baseline were observed.

Table 2.127 Total cholesterol:LDL ratio and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Marett and Slavin, 2004) 16669	Larch arabinogalactan	18/18			NS	NS	TC : LDL ratio		6 months	No change	No bias
	Placebo	17/17			NS	NS				No change	
	Tamarack arabinogalactan	19/19			NS	NS				No change	

LDL:HDL cholesterol ratio and fibre isolates, gums and extracts

No cohort studies reported results concerning gums and extracts and LDL:HDL cholesterol ratio.

Summary of RCT data

(Marett and Slavin, 2004) conducted a six-month randomised, double-blind, parallel trial to explore the physiological effects of arabinogalactan (soluble fibre) supplementation from larch or tamarack. Fifty-four subjects were given 8.4g/day placebo (rice starch), 8.4g/day larch arabinogalactan supplement or 8.4g/day tamarack arabinogalactan supplement and instructed to consume this within a beverage or with food. Body weights were unchanged throughout the trial. Quantitative data were not recorded, but the authors reported that LDL:HDL cholesterol ratio was not differentially affected by the addition of arabinogalactan from larch or tamarack to the diet.

Table 2.128 LDL:HDL cholesterol ratio and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Intervention group	Completers/ Allocated	p-value within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Marett and Slavin, 2004) 16670	Larch arabinogalactan	18/18	NS	NS	LDL:HDL cholesterol ratio	6 months	No change	No bias
	Placebo	17/17	NS	NS			No change	
	Tamarack arabinogalactan	19/19	NS	NS			No change	

Apolipoproteins and fibre isolates, gums and extracts

No cohort studies reported results concerning fibre isolates, gums and extracts and apolipoproteins.

Summary of RCT data

Five studies provided data on the effects of a variety of gums and extracts on apolipoproteins (Pasman *et al.*, 1997a; Wood *et al.*, 2006; Marett and Slavin, 2004; Garcia *et al.*, 2006; Vido *et al.*, 1993).

Of the included studies, four used a parallel group design (Vido *et al.*, 1993; Marett and Slavin, 2004; Pasman *et al.*, 1997a; Wood *et al.*, 2006) and one a crossover design (Garcia *et al.*, 2006). Three studies were double blind (Marett and Slavin, 2004; Vido *et al.*, 1993; Wood *et al.*, 2006) and the remaining were open (Pasman *et al.*, 1997a) or single blind (Garcia *et al.*, 2006). The study durations ranged from six weeks to 14 months.

Studies were conducted in the USA (2), Germany (1), Sweden (1), Italy (1) and the Netherlands (1).

All trials, bar one, studied adults. The exception was the study by Vido *et al.* (Vido *et al.*, 1993) which used adolescents under 15 years. Average BMI tended to fall into the obese ($>30\text{kg/m}^2$) range for three of the five studies; however two studies did not report the mean BMI of participants (Marett and Slavin, 2004; Vido *et al.*, 1993). Three studies included male and female participants, one included males only (Wood *et al.*, 2006) and one females only (Pasman *et al.*, 1997a).

Changes in body weights were inconsistent. Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

As quantitative data were not reported in two studies (Pasman *et al.*, 1997a; Marett and Slavin, 2004) and one trial included children (Vido *et al.*, 1993), there was an insufficient number of trials remaining to combine in a meta-analysis.

In the double blind, placebo-controlled trial by Vido *et al.* children aged less than 15 years were randomised to receive either glucomannan (2g/day) or a placebo product for two months whilst consuming their usual diet (Vido *et al.*, 1993). After the intervention, children in the glucomannan supplement group experienced a statistically significant reduction in apolipoprotein B from baseline ($p=0.01$), but no change in apolipoprotein A. Whether this change was statistically different from the change experienced in the placebo group was not clear.

Four studies of adults also investigated the effects of gums and extracts on apolipoprotein levels. Pasman *et al.* (Pasman *et al.*, 1997a), for example, provided data on the effects of guar gum supplements on body weight, blood pressure and blood lipids in weight-reduced subjects for maintenance of weight loss over 16 months. This study compared a 20g water soluble fibre (guar gum) supplement with no treatment condition for 14 months, following a two month very low calorie diet. Apolipoprotein A-1, apolipoprotein B and lipoprotein (a) were not differentially affected by fibre supplementation.

Marett and Slavin (Marett and Slavin, 2004) conducted a six-month randomised, double-blind, parallel trial to explore the physiological effects of arabinogalactan (soluble fibre) supplementation from larch or tamarack. Fifty-four subjects were given 8.4 g/day placebo (rice starch), 8.4g/day larch arabinogalactan supplement or 8.4g/day tamarack arabinogalactan supplement and instructed to consume this within a beverage or with food. At six months, no statistically significant changes in apolipoprotein A-1 or apolipoprotein B were observed.

The Arabinoxylan and Glucose Metabolism study reported by Garcia *et al.* (Garcia *et al.*, 2006) compared an arabinoxylan supplement with an identical placebo in overweight subjects with impaired glucose tolerance ($n=14$). Comparison of apolipoprotein A-1 values indicated a statistically significant decrease following arabinoxylan consumption compared with placebo ($p=0.029$). Apolipoprotein B levels similarly decreased in the arabinoxylan supplement group, although this observed difference did not reach statistical significance ($p=0.876$).

Finally, one small randomised trial reported lipoprotein (a) values at 12 weeks post-intervention for 30 men that had been randomly allocated to an *ad libitum* low carbohydrate diet plus soluble fibre (3g/d Konjac mannan) supplement or a low carbohydrate diet plus placebo (maltodextrin) (Wood *et al.*, 2006). At follow up, lipoprotein (a) levels were reduced by 11%, with no additional benefit from fibre supplementation.

Collectively, the results of these five studies provide inconsistent evidence concerning the benefits of fibre isolate supplementation on the various apolipoproteins assessed.

Table 2.129 Apolipoproteins and fibre isolates, gums and extracts: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
Adolescent study												
(Vido <i>et al.</i> , 1993) 16934	Glucomannan supplement	30/30	1.38 (SD 0.32)	1.35 (SD 0.32)		NS	Not reported	Apo A-lipoprotein	Not reported (g/L)	2 months	Decrease	unclear
	Placebo	30/30	1.33 (SD 0.23)	1.39 (SD 0.28)		NS					Decrease	
16935	Glucomannan supplement	30/30	1.07 (SD 0.33)	0.99 (SD 0.28)		NS	Not reported	Apo B-lipoprotein	Not reported (g/L)	2 months	Decrease	unclear
	Placebo	30/30	1.02 (SD 0.26)	0.92 (SD 0.22)		0.01					Decrease	
Adult studies												
(Garcia <i>et al.</i> , 2006) 17378	Arabinoxylan	11/11	1.27 (CI 1.21, 1.32)	1.25 (CI 1.19, 1.31)			0.029	Apolipoprotein A- 1	Geometric mean, Fasting serum (g/L)	6 weeks	No change	unclear
	Placebo	11/11	1.26 (CI 1.21, 1.31)	1.30 (CI 1.24, 1.37)							No change	
17379	Arabinoxylan	11/11	1.10 (CI 1, 1.21)	1.09 (CI 1.01, 1.19)			0.876	Apolipoprotein B	Geometric mean, Fasting serum (g/L)	6 weeks	No change	unclear
	Placebo	11/11	1.07 (CI 0.98, 1.17)	1.08 (CI 1, 1.18)							No change	
(Marett and Slavin, 2004) 16667	Larch arabinogalactan	18/18				NS	NS	Apolipoprotein A- 1	Fasting plasma (detail not provided)	6 months	No change	No bias
	Placebo	17/17				NS	NS				No change	
	Tamarack arabinogalactan	19/19				NS	NS				No change	
16668	Larch arabinogalactan	18/18				NS	NS	Apolipoprotein B	Fasting plasma (detail not provided)	6 months	No change	No bias
	Placebo	17/17				NS	NS				No change	
	Tamarack arabinogalactan	19/19				NS	NS				No change	
(Pasman <i>et</i>	Control	11/14	180			NS		Lipoprotein (a)	Fasting	14 months	Increase	unclear

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
<i>al., 1997a)</i> 15527									plasma (mg/dL ⁻¹)			
	Guar gum - High compliance	10/10	243			NS	NS				Increase	
	Guar Gum - Low compliance	10/10	230			NS	NS				Increase	
15525	Control	11/14	122.7			NS		Apolipoprotein A-1	Fasting plasma (mg/dL ⁻¹)	14 months	Increase	unclear
	Guar gum - High compliance	10/10	119.8			NS	NS				Increase	
	Guar Gum - Low compliance	10/10	143.8			NS	NS				Increase	
15526	Control	11/14	71.2			NS		Apolipoprotein B	Fasting plasma (mg/dL ⁻¹)	14 months	Increase	unclear
	Guar gum - High compliance	10/10	85.3			NS	NS				Increase	
	Guar Gum - Low compliance	10/10	85.8			NS	NS				Increase	
(Wood <i>et al.</i> , 2006) 16399	Low carbohydrate diet + placebo	14/15	17.9 (SD 10.3)		Decrease by 11.7% in both groups	<0.05		Lipoprotein (a)	Fasting plasma (mg/dL)	12 weeks	Decrease	No bias
	Low carbohydrate diet + Soluble fibre	14/14	17.9 (SD 10.3)			<0.05	NS				Decrease	

Results – Fibre isolates, beta-glucan

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 studies explored the effects of whole oats, oat bran-supplemented foods or oat-based breakfast cereals compared with similar wheat-based test foods. Smith *et al.* (Smith *et al.*, 2008) and Keenan *et al.* (Keenan *et al.*, 2007) however used beta-glucans derived from barley. Two studies compared wheat and corn (Chen *et al.*, 2006; Davy *et al.*, 2002), whereas three compared wheat and oats (Romero *et al.*, 1998; Swain *et al.*, 1990; Saltzman *et al.*, 2001). The study by Johnson *et al.* compared daily consumption of one serving of a mainly oat-based breakfast cereal with one that was corn-based (Johnston, 1998).

Total cholesterol and fibre isolates, beta-glucan

No cohort studies reported results concerning fibre isolates, beta-glucan and total cholesterol.

Summary of RCT data

Eight studies provided data on the relationship between beta-glucan consumption and total cholesterol (Johnston, 1998; Smith *et al.*, 2008; Keenan *et al.*, 2007; Swain *et al.*, 1990; Chen *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002; Romero *et al.*, 1998). Of these, seven were conducted in the USA and one in Mexico (Romero *et al.*, 1998). Of the included trials, seven used the parallel groups design and one used a crossover design (Swain *et al.*, 1990). The majority of studies were double blind (4); the remaining were either unclear (2) or open (2). Intervention durations ranged from six to 12 weeks.

All studies used adults as participants, with two out of the seven studying males only (Romero *et al.*, 1998; Davy *et al.*, 2002). The remaining studies were mixed gender. Trials ranged in size from reasonably small (24 subjects) (Swain *et al.*, 1990) to large (155 subjects) (Keenan *et al.*, 2007). Mean number of subjects per trial was 78. Of those studies that reported average BMI, subjects generally fell into the overweight category (BMI: 25-30kg/m²).

One study prescribed an energy restriction goal (Saltzman *et al.*, 2001), but the other studies did not limit energy intakes. The low fibre group in the study by Chen *et al.* (Chen *et al.*, 2006), the low molecular weight beta-glucan group in Smith *et al.* (Smith *et al.*, 2008) and all treatment groups in Davy *et al.* (Davy *et al.*, 2002) experienced a weight increase. Saltzman *et al.* (Saltzman *et al.*, 2001) however reported weight loss (the prescribed diets were both hypoenergetic). Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

Smith *et al.* (Smith *et al.*, 2008) was not included in the meta-analysis due to an absence of an appropriate non-glucan control arm. This trial compared the effect of high or low molecular weight beta-glucan supplements on cardiovascular biomarkers and appetite for breakfast, lunch or dinner over a six-week period (Smith *et al.*, 2008). There were no statistically significant differences in fasting total cholesterol within or between groups following the intervention (Smith *et al.*, 2008).

Seven studies were included in the meta-analysis comparing different beta-glucan intakes and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 12 weeks.

The pooled estimate indicated that total cholesterol was 0.27mmol/L (95% CI 0.10 to 0.43) lower with consumption of a diet higher in beta-glucans. This was significantly different from zero ($p=0.002$). Overall heterogeneity denoted by I^2 was high at 67% (95% CI 31 to 85%). Statistically, there was evidence that high beta glucan consumption is associated with lower levels of total cholesterol

Figure 2.70 Forest plot for fibre isolates, beta-glucan and total cholesterol (mmol/L)

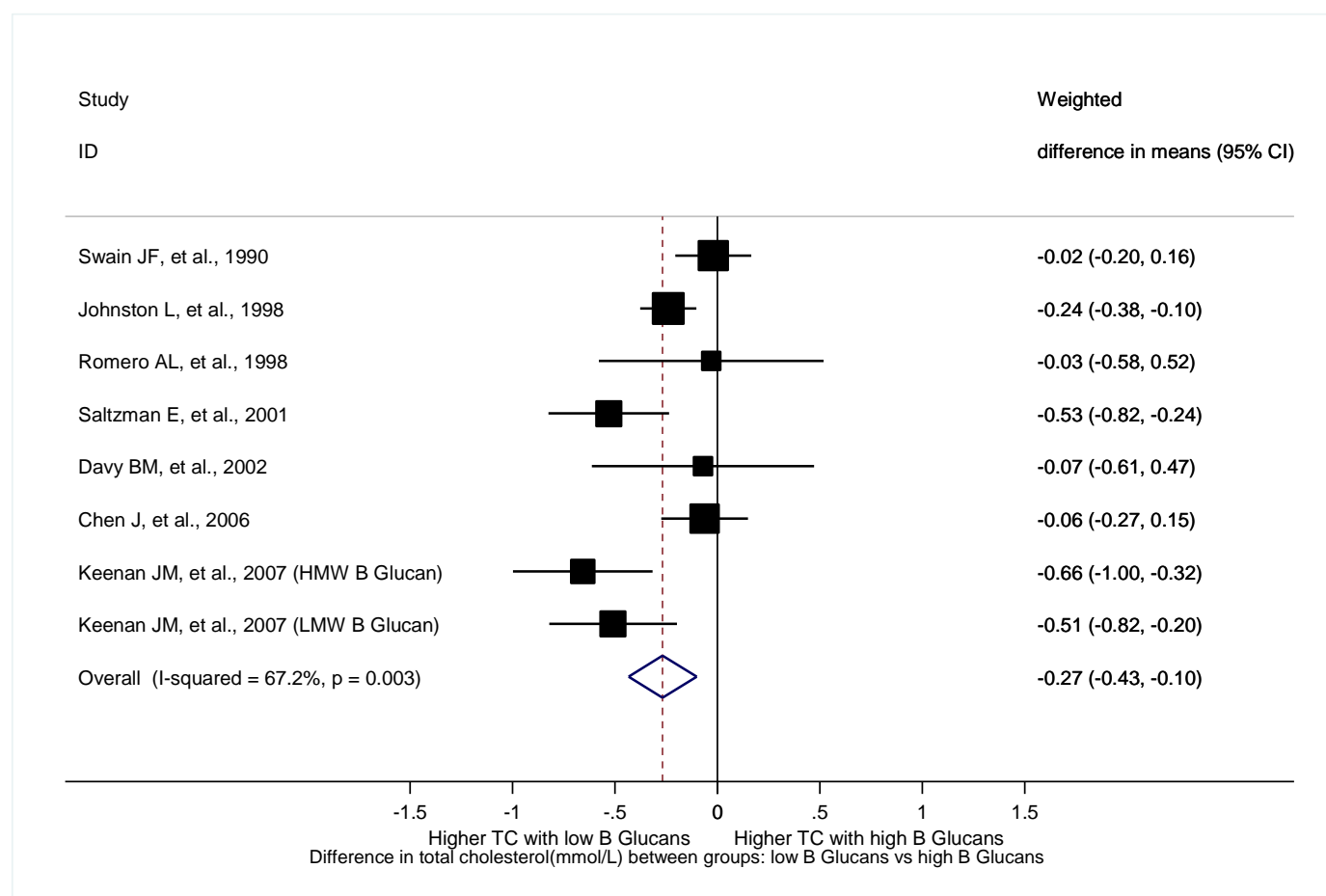


Table 2.130 Total cholesterol and fibre isolates, beta-glucan: RCT data

Author/ Result ID	Intervention groups	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 at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
(Chen <i>et al.</i> , 2006) *1718 0	High fibre minus low fibre	ITT analysis: High fibre:56/56 Low fibre: 54/54							-2.4 (CI - 10.6, 5.82)	NS	Total cholesterol	Fasting (mg/dL)	12 weeks	No change in high fibre group, small increase in low fibre group	No bias
17187	High fibre	54/54			-2.42 (CI - 8.9, 4.05)	NS					Total cholesterol	Fasting (mg/dL)	12 weeks	No change	No bias
	Low fibre	56/56			-0.02 (CI - 5.29, 5.26)	NS								Small increase	
(Davy <i>et al.</i> , 2002) *1543 0	Wheat cereal	18/18	4.91 (SE 0.16)	5.22 (SE 0.18)							Total cholesterol	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	5.28 (SE 0.18)	5.15 (SE 0.21)			0.08							Increase	
(Johns ton, 1998) *1668 6	Control cereal	62/62			0.09 (SD 0.39)						Total cholesterol	Fasting plasma (mg/dL)	6 weeks	No change	unclear
	Wholegrain oat cereal	62/62			-0.14 (SD 0.37)		NS							No change	
(Keenan <i>et al.</i> , 2007) *1632 8	High-dose LMW barley beta- glucan	30/30	238.0 (SD 27.6)	211.6 (SD 20.2)		<0.05	<0.05				Total cholesterol	Fasting (mg/L)	6 weeks	No change	No bias
	High-dose, HMW barley beta-glucan	32/32	235.1 (SD 25.3)	205.9 (SD 25.1)		<0.05	<0.05							No change	
	Low-dose, HMW barley beta-glucan	32/32	233.6 (SD 22.8)	214.5 (SD 21.6)		<0.05	<0.05							No change	
	Low-dose, LMW barley beta- glucan	31/31	235.9 (SD 23.0)	218.8 (SD 20.1)		<0.05	<0.05							No change	
	Placebo	30/30	234.0 (SD 22.7)	231.3 (SD 26.9)		NS								No change	
(Rome ro <i>et al.</i> , 1998)	Oat bran cookies	12/12	214 (SD 13)	184 (SD 22)	-30	<0.05	<0.001				Total cholesterol	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium cookies	10/10	214 (SD 19)	193 (SD 26)	-21	<0.05	<0.001							No change	

*1542 4	Wheat bran cookies	14/14	180 (SD 33)	185 (SD 30)	5	NS					No change	
(Saltzman <i>et al.</i> , 2001)	Control	21/21	4.4 (SD 0.98)		-0.34 (SD 0.5)			Total cholesterol	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
*1619 9	Oats	20/22	4.88 (SD 0.87)		-0.87 (SD 0.47)	0.05	<0.05				Decrease	
(Smith <i>et al.</i> , 2008)	High molecular weight Betaglucan	45/45			0.3 (SE 4.2)	NS	0.56	Total cholesterol	Fasting plasma (mmol/L)	6 weeks	No change	No bias
16553	Low molecular weight Betaglucan	45/45			-2.7 (SE 3.1)	NS					Increase	
(Swain <i>et al.</i> , 1990)	Low fibre wheat supplement	11/11	4.80 (SD 0.80)	4.46 (SD 0.64)	-0.34 (CI - 0.53, -0.15)	<0.05		Total cholesterol	Fasting serum (mmol/L)	6 weeks	No change	No bias
17347	Oat bran supplement	9/9	4.80 (SD 0.80)	4.44 (SD 0.73)	-0.36 (CI - 0.55, -0.17)	<0.05	NS				No change	
*1735 6	Oat bran supplement minus low fibre wheat supplement	High fibre: 9/12 Low fibre: 11/12						Total cholesterol	Fasting serum (mmol/L)	6 weeks	No change in both	No bias
					-0.02 (CI - 0.20, 0.17)	NS						

*This result was used in the meta-analysis of beta-glucan and total cholesterol

HDL cholesterol and fibre isolates, beta-glucan

No cohort studies reported results concerning fibre isolates, beta-glucan and HDL cholesterol.

Summary of RCT data

Eight trials provided data on beta-glucan consumption and HDL cholesterol (Johnston, 1998; Smith *et al.*, 2008; Keenan *et al.*, 2007; Swain *et al.*, 1990; Chen *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002; Romero *et al.*, 1998). These eight trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, beta-glucan.

Seven studies were included in the meta-analysis comparing different beta-glucan intakes and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from eight weeks to six months.

Smith *et al.* (Smith *et al.*, 2008) was not included in the meta-analysis due to an absence of an appropriate non-glucan control arm. This trial compared the effect of high or low molecular weight beta-glucan supplements on cardiovascular biomarkers and appetite for breakfast, lunch or dinner over a six-week period (Smith *et al.*, 2008). There were no statistically significant differences in HDL cholesterol within or between groups following the intervention (Smith *et al.*, 2008).

The pooled estimate indicated that HDL cholesterol was 0.02mmol/L (95% CI -0.04 to 0.08) higher with consumption of a diet higher in beta glucans. This was not significantly different from zero ($p=0.55$). Overall heterogeneity denoted by I^2 was high at 74% (95% CI 48 to 87%). Statistically, there was no evidence that high beta glucan consumption is associated with different levels of HDL cholesterol.

One study had very different results (Romero *et al.*, 1998) due to the fact that HDL levels were very different at baseline in the oats group (27mmol/L) compared with the control (wheat) group (47mmol/L) despite randomisation.

Figure 2.71 Forest plot for fibre isolates, beta-glucan and HDL cholesterol (mmol/L)

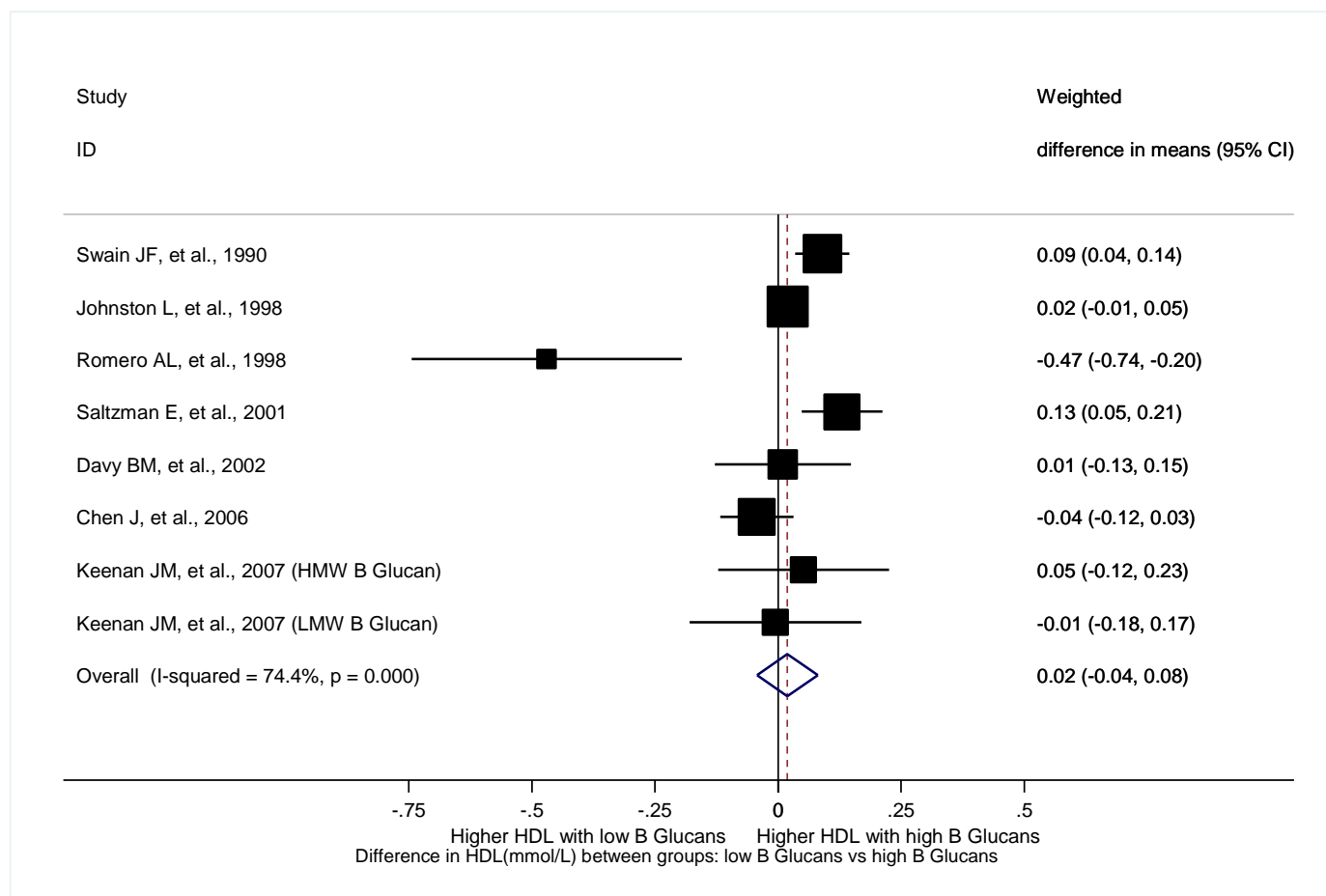


Table 2.131 HDL cholesterol and fibre isolates, beta-glucan: RCT data

Author/ Result ID	Intervention groups	Completer s/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Chen <i>et al.</i> , 2006) *17181	High fibre minus low fibre	ITT analysis: High fibre:56/56 Low fibre: 54/54							-1.66 (CI - 4.55, 1.22)	NS	HDL-C	Fasting (mg/dL)	12 weeks	No change in high fibre group, small increase in low fibre group	No bias
17188	High fibre	54/54			-0.24 (CI - 2.19, 1.71)	NS					HDL-C	Fasting (mg/dL)	12 weeks	No change	No bias
	Low fibre	56/56			1.42 (CI - 0.74, 3.59)	NS								Small increase	
(Davy <i>et al.</i> , 2002) *15434	Wheat cereal	18/18	0.89 (SE 0.04)	0.85 (SE 0.05)							HDL-C	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	0.87 (SE 0.05)	0.86 (SE 0.05)			0.41							Increase	
(Johnston, 1998) *16686	Control cereal	62/62			0.00 (SD 0.10)						HDL-C	Fasting plasma (mg/dL)	6 weeks	No change	unclear
	Wholegrain oat cereal	62/62			0.02 (SD 0.08)		NS							No change	
(Keenan <i>et al.</i> , 2007) *16326	High-dose LMW barley beta-glucan	30/30	50.4 (SD 13.7)	49.7 (SD 12.8)		NS	NS				HDL-C	Fasting (mg/L)	6 weeks	No change	No bias
	High-dose, HMW barley beta-glucan	32/32	50.8 (SD 14.2)	51.9 (SD 12.7)		NS	NS							No change	
	Low-dose, HMW barley beta-glucan	32/32	47.9 (SD 10.7)	47.4 (SD 11.2)		NS	NS							No change	
	Low-dose, LMW barley beta-glucan	31/31	49.6 (SD 14.8)	50.8 (SD 15.8)		NS	NS							No change	
	Placebo	30/30	50.5 (SD 14.4)	49.9 (SD 13.8)		NS								No change	
(Romer <i>et al.</i> , 1998) *15426	Oat bran cookies	12/12	27 (SD 7)	32 (SD 8)		NS	NS				HDL-C	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium cookies	10/10	37 (SD 8)	41 (SD 9)		NS	NS							No change	
	Wheat bran cookies	14/14	47 (SD 19)	50 (SD 17)		NS		NS						No change	
(Saltzman)	Control	21/21	1.17 (SD		-0.04 (SD						HDL-C	Fasting	6 weeks	Decrease	unclear

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Author/ Result ID	Intervention groups	Completer s/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
n <i>et al.</i> , 2001)			0.28)		0.14)							plasma (mmol/L)			
*16203	Oats	20/22	1.15 (SD 0.2)		0.09 (SD 0.13)	0.05	NS							Decrease	
(Smith <i>et al.</i> , 2008)	High molecular weight Betaglucan	45/45			-1 (SE 0.9)	NS	0.11				HDL-C	Fasting plasma (mmol/L)	6 weeks	No change	No bias
16552	Low molecular weight Betaglucan	45/45			0.8 (SE 0.8)	NS								Increase	
(Swain <i>et al.</i> , 1990)	Low fibre wheat supplement	11/11	1.40 (SD 0.43)	1.32 (SD 0.39)	-0.08 (CI - 0.14, - 0.02)	NS					HDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
17349	Oat bran supplement	9/9	1.40 (SD 0.43)	1.40 (SD 0.39)	0.01 (CI - 0.05, 0.06)	NS	NS							No change	
*17358	Oat bran supplement minus low fibre wheat supplement	High fibre: 9/12 Low fibre: 11/12						0.09 (CI 0.03, 0.14)		NS	HDL-C	Fasting plasma (mmol/L)	6 weeks	No change in both	No bias

*This result was used in the meta-analysis of beta-glucan and HDL cholesterol

LDL cholesterol and fibre isolates, beta-glucan

No cohort studies reported results concerning fibre isolates, beta-glucan and LDL cholesterol.

Summary of RCT data

Eight studies provided data on the relationship between beta-glucan consumption and LDL cholesterol (Johnston, 1998; Smith *et al.*, 2008; Keenan *et al.*, 2007; Swain *et al.*, 1990; Chen *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002; Romero *et al.*, 1998). These eight trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, beta-glucan.

Seven studies were included in the meta-analysis comparing different beta-glucan intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 12 weeks.

Smith *et al.* (Smith *et al.*, 2008) was not included in the meta-analysis due to an absence of an appropriate non-glucan control arm. This trial compared the effect of high or low molecular weight beta-glucan supplements on cardiovascular biomarkers and appetite for breakfast, lunch or dinner over a six-week period (Smith *et al.*, 2008). At follow up, the authors reported a decrease of 5.4mmol/L in LDL cholesterol with consumption of low molecular weight barley-derived beta-glucan supplement ($p=0.05$). Between treatments, there was no statistically significant difference in LDL cholesterol.

The pooled estimate indicated that LDL cholesterol was 0.22mmol/L (95% CI 0.10 to 0.34) lower with consumption of a diet higher in beta-glucans. This was significantly different from zero ($p<0.001$). Overall heterogeneity denoted by I^2 was 60% (95% CI 12 to 82%). Statistically, there was evidence that high beta glucan consumption is associated with lower levels of LDL cholesterol.

Figure 2.72 Forest plot for fibre isolates, beta-glucan and LDL cholesterol (mmol/L)

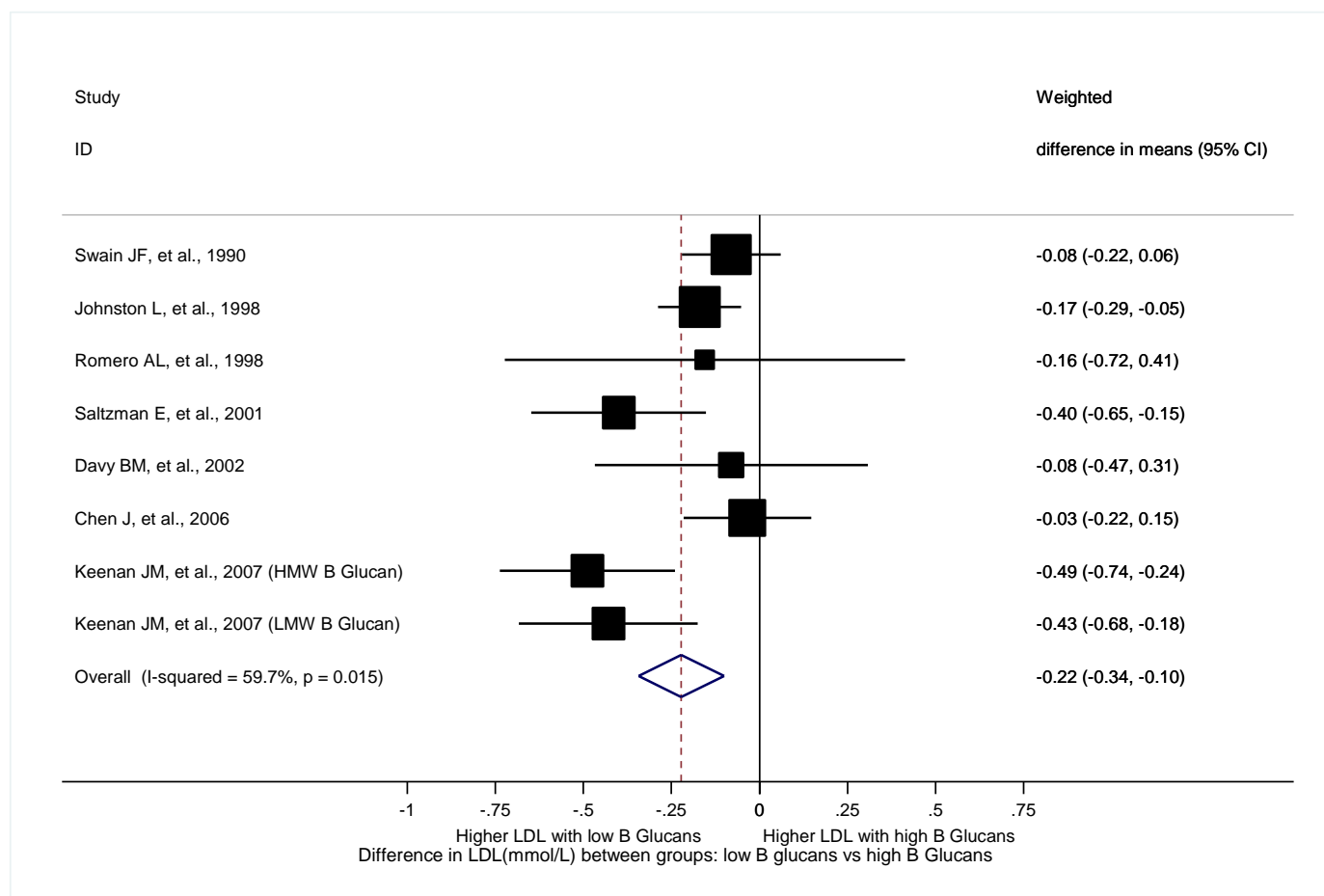


Table 2.132 LDL cholesterol and fibre isolates, beta-glucans: RCT data

Author/ Result ID	Intervention groups	Completer s/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcom e	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Chen <i>et al.</i> , 2006) *17182	High fibre minus low fibre	ITT analysis: High fibre:56/5 6 Low fibre: 54/54							-1.33 (CI -8.33, 5.68)	NS	LDL-C	Fasting (mg/dL)	12 weeks	No change in high fibre group, small increase in low fibre group	No bias
17189	High fibre	54/54			-1.96 (CI -7.32, 3.4)	NS					LDL-C	Fasting (mg/dL)	12 weeks	No change	No bias
	Low fibre	56/56			-0.64 (CI -5.3, 4.03)	NS								Small increase	
(Davy <i>et al.</i> , 2002) *15431	Wheat cereal	18/18	3.3 (SE 0.15)	3.57 (SE 0.14)							LDL-C	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	3.58 (SE 0.16)	3.49 (SE 0.14)			0.02							Increase	
(Johnston, 1998) *16686	Control cereal	62/62	4.15						Control minus oats -0.17 (SE 0.06)	0.006	LDL-C	Fasting plasma (mmol/L)	6 weeks	No change	unclear
	Wholegrain oat cereal	62/62	4.10												
(Keenan <i>et al.</i> , 2007) *16324	High-dose LMW barley beta-glucan	30/30	154.6 (SD 19.9)	134.3 (SD 12.8)		<0.05	<0.05				LDL-C	Fasting (mg/L)	6 weeks	No change	No bias
	High-dose, HMW barley beta-glucan	32/32	154.5 (SD 16.6)	132.0 (SD 11.4)		<0.05	<0.05							No change	
	Low-dose, HMW barley beta-glucan	32/32	152.8 (SD 18.1)	138.8 (SD 20.3)		<0.05	<0.05							No change	
	Low-dose, LMW barley beta-glucan	31/31	153.9 (SD 15.1)	140.5 (SD 15.1)		<0.05	<0.05							No change	
	Placebo	30/30	152.7 (SD 13.9)	150.9 (SD 24.3)		NS								No change	
(Romer <i>et al.</i> , 1998)	Oat bran cookies	12/12	140 (SD 43)	103 (SD 33)	-37	<0.05	<0.001				LDL-C	Plasma (mg/dL)	8 weeks	No change	unclear
	Psyllium	10/10	146 (SD	121 (SD	-25	<0.0001	<0.001							No change	

Author/ Result ID	Intervention groups	Completer s/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Difference between groups at follow-up	Difference between groups in Δ from baseline	p-value difference between groups	Outcom e	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
*15425	cookies		16)	20)											
	Wheat bran cookies	14/14	109 (SD 29)	109 (SD 22)	-0.1	NS								No change	
(Saltzman <i>et al.</i> , 2001) *16202	Control	21/21	2.79 (SD 0.83)		-0.2 (SD 0.41)						LDL-C	Derived by calculation Fasting, Plasma (mmol/L)	6 weeks	Decrease	unclear
	Oats	20/22	3.16 (SD 0.77)		-0.6 (SD 0.41)	0.05	<0.05							Decrease	
(Smith <i>et al.</i> , 2008) 16551	High molecular weight Beta-glucan	45/45			1.6 (SE 3.6)	NS	0.13				LDL-C	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Low molecular weight Beta-glucan	45/45			-5.4 (SE 2.6)	0.05								Increase	
(Swain <i>et al.</i> , 1990) 17348	Low fibre wheat supplement	11/11	2.96 (SD 0.61)	2.77 (SD 0.59)	-0.19 (CI -0.33, - 0.05)	<0.05					LDL-C	Fasting serum (mmol/L)	6 weeks	No change	No bias
	Oat bran supplement	9/9	2.96 (SD 0.61)	2.69 (SD 0.63)	-0.27 (CI -0.41, - 0.13)	<0.05	NS							No change	
*17357	Oat bran supplement minus low fibre wheat supplement	High fibre: 9/12 Low fibre: 11/12						-0.08 (CI -0.22, 0.06)		NS	LDL-C	Fasting plasma (mmol/L)	6 weeks	No change in both	No bias

*This result was used in the meta-analysis for beta-glucan and LDL cholesterol

Triacylglycerol and fibre isolates, beta-glucans

No cohort studies reported results concerning fibre isolates, beta-glucans and TAG.

Summary of RCT data

Seven studies provided data on the relationship between beta-glucan consumption and TAG (Johnston, 1998; Smith *et al.*, 2008; Keenan *et al.*, 2007; Chen *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002; Romero *et al.*, 1998). These seven trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and fibre isolates, beta-glucan.

Six studies were included in the meta-analysis comparing different beta-glucan intakes and changes in TAG reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from eight weeks to six months.

Smith *et al.* (Smith *et al.*, 2008) was not included in the meta-analysis due to an absence of an appropriate non-glucan control arm. This trial compared the effect of high or low molecular weight beta-glucan supplements on cardiovascular biomarkers and appetite for breakfast, lunch or dinner over a six-week period (Smith *et al.*, 2008). No differential effect of beta-glucan supplementation on TAG was observed.

The pooled estimate indicated that TAG level was 0.17mmol/L (95% CI 0.02 to 0.31) lower with consumption of a diet higher in beta-glucans. This was significantly different from zero ($p=0.03$). Overall heterogeneity denoted by I^2 was 53% (95% CI 0 to 80%). Statistically, these studies indicate that high beta-glucan consumption derived from oats or barley is associated with lower levels of TAG.

It should be noted that there was an imbalance in baseline TAG levels between the groups in one study despite randomisation (Romero *et al.*, 1998). Triacylglycerol levels at baseline were reported to be 127mg/dL in the control (wheat) group and 234mg/dL in the oats group (nb. because of this baseline imbalance, the pooled estimate is biased since only the follow up data, and not change from baseline were available). This study contributed relatively little weight to the pooled estimate due to the small number of subjects.

Figure 2.73 Forest plot for fibre isolates, beta-glucan and TAG (mmol/L)

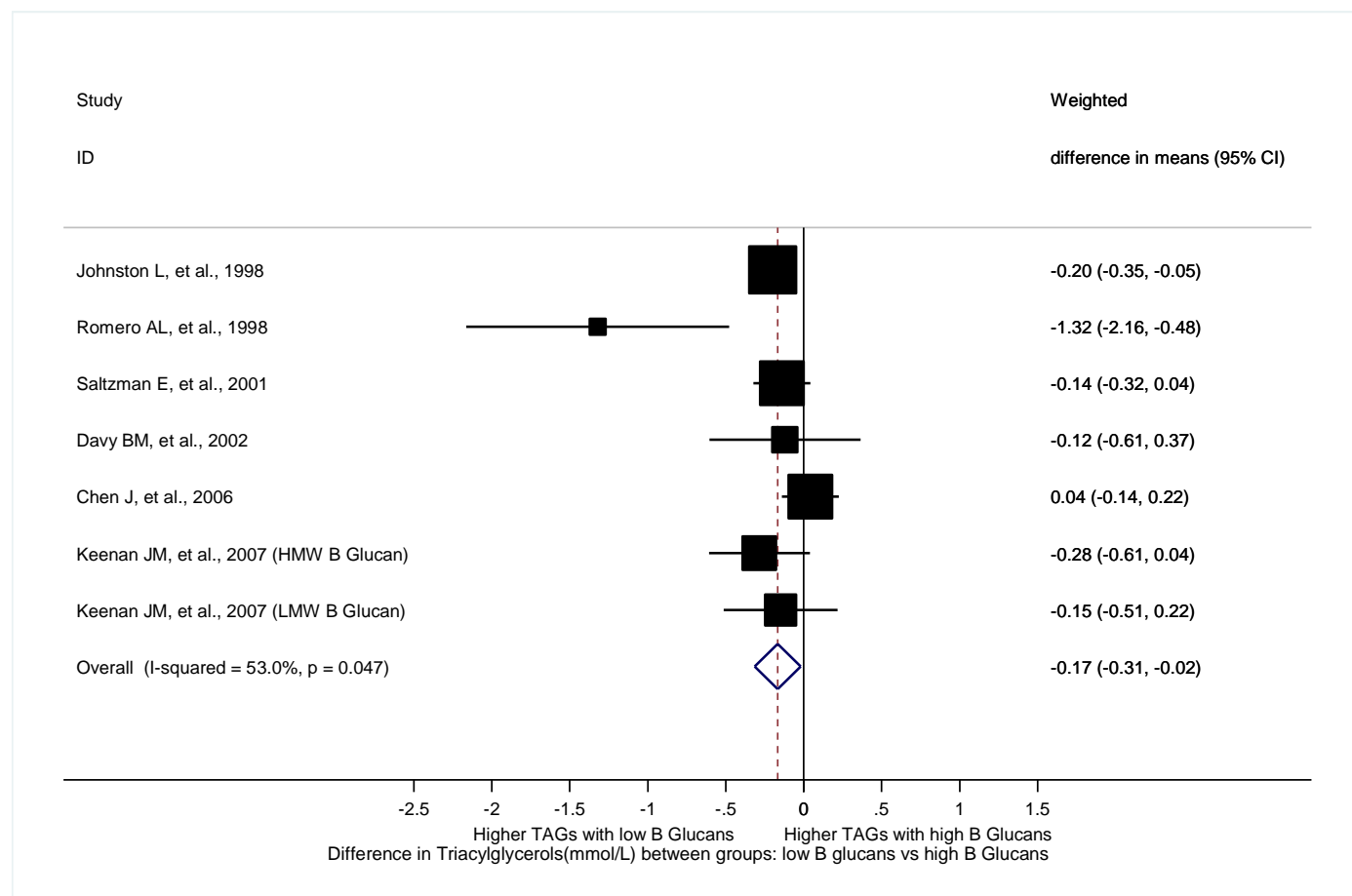


Table 2.133 Triacylglycerol and fibre isolates, beta-glucans: RCT data

Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Chen <i>et al.</i> , 2006) *17183	High fibre minus low fibre	ITT analysis: High fibre:56/56 Low fibre: 54/54						3.76 (CI -12.4, 19.9)	NS	TAG	Fasting (mg/dL)	12 weeks	No change in high fibre group, small increase in low fibre group	No bias
17190	High fibre	54/54			-0.80 (CI - 10.1, 8.5)	NS				TAG	Fasting (mg/dL)	12 weeks	No change	No bias
	Low fibre	56/56			-4.56 (CI - 17.9, 8.75)	NS							Small increase	
(Davy <i>et al.</i> , 2002) *15437	Wheat cereal	18/18	1.5 (SE 0.17)	1.83 (SE 0.17)						TAG	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	1.83 (SE 0.17)	1.71 (SE 0.18)			0.07						Increase	
(Johnston, 1998) *16686	Control cereal	62/62	1.62 (SD 0.7)		0.23 (SD 0.45)				NS	TAG	Fasting plasma (mmol/L)	6 weeks	No change	unclear
	Wholegrain oat cereal	62/62	1.77 (SD 0.69)		0.03 (SD0.39)									
(Keenan <i>et al.</i> , 2007) *16327	High-dose LMW barley beta- glucan	30/30	166.7 (SD 91.7)	145.7 (SD 62.7)		<0.05	NS			TAG	Fasting (mg/L)	6 weeks	No change	No bias
	High-dose, HMW barley beta-glucan	32/32	158.3 (SD 79.2)	133.7 (SD 47.4)		<0.05	<0.05						No change	
	Low-dose, HMW barley beta-glucan	32/32	164.7 (SD 88.7)	152.5 (SD 55.8)		NS	NS						No change	
	Low-dose, LMW barley beta-glucan	31/31	154.9 (SD 61.7)	142.2 (SD 49.2)		<0.05	NS						No change	
	Placebo	30/30	153.9 (SD 75.4)	158.8 (SD 64.7)		NS							No change	
(Romero <i>et al.</i> ,	Oat bran cookies	12/12	234 (SD 187)	246 (SD 97)		NS	NS			TAG	Plasma (mg/dL)	8 weeks	No change	unclear

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Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
1998) *15427	Psyllium cookies	10/10	154 (SD 67)	154 (SD 73)		NS	NS						No change	
	Wheat bran cookies	14/14	127 (SD 83)	130 (SD 91)		NS							No change	
(Saltzman <i>et al.</i> , 2001) *16197	Control	21/21	0.94 (SD 0.36)		-0.22 (SD 0.23)					TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
	Oats	20/22	1.23 (SD 0.6)		-0.36 (SD 0.36)	NS	NS						Decrease	
(Smith <i>et al.</i> , 2008) 16555	High molecular weight Betaglucan	45/45			-1 (SE 7)	NS	0.23			TAG	Fasting plasma (mmol/L)	6 weeks	No change	No bias
	Low molecular weight Betaglucan	45/45			12 (SE 8)	NS							Increase	

*This result was used in the meta-analysis for beta-glucans and TAG

Total cholesterol:HDL ratio and fibre isolates, beta-glucans

No cohort studies reported results concerning fibre isolates, beta-glucans and TC:HDL ratio.

Summary of RCT data

Three studies reported data on the effects of beta-glucan consumption on the TC:HDL ratio (Smith *et al.*, 2008; Keenan *et al.*, 2007; Davy *et al.*, 2002). All were conducted in the USA.

All trials took a parallel group approach and were either double blind (Smith *et al.*, 2008; Keenan *et al.*, 2007) or open (Davy *et al.*, 2002). Participants were adults, and mean BMI was calculated as 29kg/m² in two studies, although the study by Smith *et al.* (Smith *et al.*, 2008) did not report average BMI. One study was identified that recruited males only (Davy *et al.*, 2002) and the remaining two were mixed gender. Final sample sizes ranged from 36 to 155 participants.

Body weights were found to have increased in one study (Davy *et al.*, 2002) and were unchanged in another (Keenan *et al.*, 2007). An increase in weight was also noted in the high molecular weight beta-glucan group in the study by Smith *et al.* (Smith *et al.*, 2008).

In the study by Davy *et al.* (Davy *et al.*, 2002) the effect of wholegrain oat cereal was compared with wheat cereal on plasma lipids in 36 healthy males. Over a period of 12 weeks, the TC:HDL ratio had increased in the wheat cereal group and decreased in the wholegrain oat cereal groups ($p < 0.05$).

Keenan *et al.* (Keenan *et al.*, 2007) compared high and low molecular weight barley derived beta glucans in 5 or 3g/d doses in 155 subjects over a six-week study period. Barley beta-glucans were incorporated into a ready-to-eat cereal and a low energy fruit juice beverage. HDL cholesterol levels were unaffected by treatment, but TC levels fell in all groups with greater reductions in the higher dose groups. Accordingly, the ratio of total to HDL cholesterol decreased to the greatest extent in the higher dose groups compared with the control group ($p < 0.01$), but also decreased relative to control in the low molecular weight, low dose group ($p < 0.01$).

Smith *et al* (Smith *et al.*, 2008) compared the effect of high or low molecular weight beta-glucan supplements on cardiovascular biomarkers and appetite for breakfast, lunch or dinner. Within-group changes in the TC:HDL ratio were not significantly different but there was a difference between the groups ($p<0.03$), with a small decrease in the low molecular weight group and small increase in the high molecular weight group.

These studies suggest some improvement in lipid status as reflected by a decline in the ratio of total to HDL cholesterol with consumption of larger amounts of either oat or barley-derived beta-glucans, although higher molecular weight, which had been suggested as an indicator of efficacy, was less clearly associated with additional benefit in lipid status.

Table 2.134 Total cholesterol:HDL ratio and fibre isolates, beta-glucans: 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	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Smith <i>et al.</i> , 2008) 16554	High molecular weight Betaglucan	45/45			0.13 (SE 0.1)	NS	0.03	TC :HDL ratio	6 weeks	No change	No bias
	Low molecular weight Betaglucan	45/45			-0.1 (SE 0.1)	NS				Increase	
(Keenan <i>et al.</i> , 2007) 16325	High-dose LMW barley beta glucan	30/30	5.0 (SD 1.4)	4.5 (SD 1.2)		<0.05	<0.05	TC :HDL ratio	6 weeks	No change	No bias
	High-dose, HMW barley beta glucan	32/32	4.9 (SD 1.3)	4.2 (SD 1.0)		<0.05	<0.05			No change	
	Low-dose, HMW barley beta glucan	32/32	5.1 (SD 1.2)	4.8 (SD 1.1)		NS	NS			No change	
	Low-dose, LMW barley beta glucan	31/31	5.0 (SD 1.2)	4.6 (SD 1.3)		<0.05	<0.05			No change	
	control	30/30	4.9 (SD 1.2)	5.0 (SD 1.4)		NS				No change	
(Davy <i>et al.</i> , 2002) 15440	Wheat cereal	18/18	5.7 (SE 0.3)	6.4 (SE 0.4)				TC :HDL ratio	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	6.4 (SE 0.4)	6.0 (SE 0.5)			0.05			Increase	

LDL:HDL cholesterol ratio and fibre isolates, beta-glucans

No cohort studies reported results concerning fibre isolates, beta-glucans and LDL:HDL cholesterol ratio.

Summary of RCT data

Three trials provided data on beta-glucan consumption in the form of oat-based breakfast cereals (Davy *et al.*, 2002; Johnston, 1998) or cookies (Romero *et al.*, 1998).

In the study by Davy *et al.* (Davy *et al.*, 2002) the effect of wholegrain oat cereal was compared with wheat cereal on plasma lipids in 36 healthy males. Over a period of 12 weeks, the ratio of LDL:HDL had increased in the wheat cereal group and decreased in the wholegrain oat cereal groups. Despite an increase in body weight in both groups, LDL:HDL was statistically significantly lower in the wholegrain oat cereal group relative to the wheat cereal group ($p=0.02$). In the study by Johnson *et al.* (Johnston, 1998), both intervention cereal groups experienced a decrease in the LDL:HDL cholesterol ratio, with no particular benefit being attributed to the consumption of an oat-based cereal.

Romero *et al.* (Romero *et al.*, 1998) tested the effects of psyllium and oat bran on LDL:HDL cholesterol ratio over an eight-week period. Thirty adults were randomly assigned to a control group consuming wheat bran cookies, a group consuming psyllium cookies or a group consuming oat bran cookies. Body weights did not alter throughout this trial. Over a period of eight weeks, statistically significant reductions in LDL:HDL cholesterol ratio in the psyllium cookie and oat bran cookie groups were noticed; these which differed to the control group ($p<0.01$). It should be noted, however, that the control group had a worse LDL:HDL cholesterol ratio at baseline than the other two intervention groups.

Collectively, these trials provide inconsistent evidence concerning the effect of oat beta-glucan consumption on the ratio of LDL to HDL cholesterol.

Table 2.135 LDL:HDL cholesterol ratio and fibre isolates, beta-glucan: 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	Result- specific follow-up	Weight Change	Outcome Assessment Bias
(Davy <i>et al.</i> , 2002) 15439	Wheat cereal	18/18	3.8 (SE 0.3)	4.3 (SE 0.3)				LDL:HDL cholesterol ratio	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	4.3 (SE 0.3)	4.1 (SE 0.3)			0.02			Increase	
(Johnston, 1998)	Control cereal	61/62	3.48 (SD 0.83)	3.47 (SD 0.83)	-0.01 (SD 0.33)			LDL:HDL cholesterol ratio	6 weeks	No change	unclear
	Wholegrain oat cereal	62/62	3.39 (SD 0.85)	3.21 (SD 0.78)	-0.18 (SD 0.35)		NS				
(Romero <i>et al.</i> , 1998) 15428	Oat bran cookies	12/12	5.2 (SD 1.6)	3.4 (SD 1.5)	-1.8	<0.01	<0.01	Change in LDL:HDL cholesterol ratio	8 weeks	No change	unclear
	Psyllium cookies	10/10	4.2 (SD 1)	3 (SD 1)	-1.2	<0.01	<0.01			No change	
	Wheat bran cookies	14/14	3 (SD 1.6)	2.4 (SD 0.8)	-0.6	NS				No change	

Apolipoproteins and fibre isolates, beta-glucans

No cohort studies reported results concerning fibre isolates, beta-glucans and apolipoproteins.

Summary of RCT data

Data concerning beta-glucan consumption and apolipoproteins were extracted from two randomised controlled trials (Smith *et al.*, 2008; Johnston, 1998). One trial compared the effect of high or low molecular weight beta-glucan supplements on cardiovascular biomarkers and appetite for breakfast, lunch or dinner (Smith *et al.*, 2008) and one compared the effects of a wholegrain oat cereal, containing 3g soluble fibre with a low soluble fibre control breakfast cereal (90g/d cornflakes, delivering 2g fibre (0.1g soluble, 1.9g insoluble)) (Johnston, 1998). Body weights were unchanged in the Johnson *et al.* trial, but increased in the low molecular weight beta-glucan group in Smith *et al.* The two studies lasted six weeks.

In Johnson *et al.* the beta-glucan group experienced a significant decrease in apolipoprotein B levels whilst the control group increased ($p=0.02$). Apolipoprotein A1 levels were not different between groups. Smith *et al.* did not find any differential effect of high and low molecular weight beta-glucans on apolipoprotein levels.

Table 2.136 Apolipoproteins and fibre isolates, beta-glucans: RCT data

Author/ Result ID	Intervention groups	Completers / Allocated	Baseline	Follow- up	Within group Δ from baseline	Difference between groups in Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Johnston, 1998) 16686	Control cereal	59/62	129.7 (SD 19.2)	131.1 (SD 17.5)	1.39 (SD 10.1)				Apolipoprotein A-1	Fasting plasma (mg/dL)	6 weeks	No change	unclear
	Wholegrain oat cereal	59/62	134.8 (SD 22.0)	134.2 (SD 22.4)	-0.6 (SD 11.3)			NS				No change	
16687	Control cereal	59/62	125.6 (SD 17.9)	129.1 (SD 18.7)	3.6 (SD 13.8)	-5.7			Apolipoprotein B	Fasting plasma (mg/dL)	6 weeks	No change	unclear
	Wholegrain oat cereal	59/62	124.2 (SD 15.1)	122.1 (SD 14.7)	-2.1 (SD 12.1)			0.02				No change	
(Smith <i>et al.</i> , 2008) 16557	High molecular weight Beta-glucan	45/45			-0.81 (SE 3)		NS	0.54	Apolipoprotein A-1	Fasting plasma (g/L)	6 weeks	No change	No bias
	Low molecular weight Beta-glucan	45/45			2.14 (SE 3.8)		NS					Increase	
16558	High molecular weight Beta-glucan	45/45			0.21 (SE 2.9)		NS	0.97	Apolipoprotein B	Fasting plasma (g/L)	6 weeks	No change	No bias
	Low molecular weight Beta-glucan	45/45			0.1 (SE 2.2)		NS					Increase	

Results – Breakfast cereals

Total cholesterol and breakfast cereals

Summary of cohort results

Data were extracted from one study of 9-10 year old girls on the association between cereal consumption frequency, assessed using food diaries, and fasting whole blood cholesterol (Albertson *et al.*, 2009). The National Heart, Lung, and Blood Institute Growth and Health Study (Albertson *et al.*, 2009) showed a small statistically significant decrease in total cholesterol with each percentage increase in cereal consumption frequency. Analyses were adjusted for a limited number of variables which included clinical centre, ethnicity and parental education.

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

Summary of RCT data

Three studies provided data on the effects of breakfast cereal on total cholesterol levels (Zaveri and Drummond, 2009; Rosado *et al.*, 2008; Kleemola *et al.*, 1999). One study included children as participants (Rosado *et al.*, 2008) and, as such, there was an insufficient number of adult studies to carry out a meta-analysis.

One study was a crossover trial (Kleemola *et al.*, 1999) and the others employed the parallel group design. Studies were conducted in Finland (Kleemola *et al.*, 1999), Scotland (Zaveri and Drummond, 2009) and Mexico (Rosado *et al.*, 2008). None were single- or double-blind in design.

Both Zaveri and Drummond (Zaveri and Drummond, 2009) and Kleemola *et al.* (Kleemola *et al.*, 1999) studied adults, whereas Rosado *et al.* (Rosado *et al.*, 2008) used children aged six to 12 years as participants. One study included males only (Zaveri and Drummond, 2009) and the remaining two were mixed gender. The average number of participants in the trials was 175 (median=224).

Body weights were unchanged in all trials other than in (Rosado *et al.*, 2008) in which the authors reported that there was a decrease in the 'one serving breakfast cereal and nutrition education' group (Rosado *et al.*, 2008).

Zaveri *et al.* (Zaveri and Drummond, 2009) compared the effects of a conventional cereal bar snack (30g weight; high in carbohydrate) or a control (no snack) on dietary intake, body weight and blood lipids over 12 weeks. The intervention was administered through the provision of two cereal bars per day, which could be consumed at any time. Overall, consumption of cereal bars in this sample did not produce statistically significant differences in total cholesterol within or between groups.

Two studies explored the effects of breakfast cereal as a meal, rather than as a snack (Rosado *et al.*, 2008; Kleemola *et al.*, 1999). In the study reported by Rosado *et al.*, (Rosado *et al.*, 2008) 147 overweight/at risk of overweight children were randomly assigned to one of four groups: i) non-intervention (control); ii) one serving (33+/-7g) of ready-to-eat cereal (RTEC) plus a nutrition education program; iii) one serving (33+/-7g) RTEC for breakfast; or iv) one serving (33+/-7g) RTEC for breakfast and one serving for dinner. At follow up (12 weeks), no statistically significant changes from baseline or between groups in total cholesterol were observed.

Kleemola *et al.* (Kleemola *et al.*, 1999) carried out a six-week randomised controlled crossover trial to test the effects of a high carbohydrate breakfast which included breakfast cereal, on lowering serum cholesterol. The 209 eligible subjects were randomised into intervention breakfast cereal (60g or 80g cereal i.e. Cornflakes or Rice Krispies for women and men, respectively) or usual breakfast (control) groups. After the first six weeks, total cholesterol had decreased in the cereal group and increased in the control group. When comparing groups, this difference in change was statistically significant ($p=0.007$). In contrast, after the second period, both groups experienced a slight reduction in total cholesterol levels, although this difference between groups did not achieve statistical significance.

Overall, these trials do not provide consistent evidence of an impact of breakfast cereals on total cholesterol levels.

Table 2.137 Total cholesterol and breakfast cereal: cohort study in children

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
(Albertson <i>et al.</i> , 2009) 13982 National Heart, Lung, and Blood Institute Growth and Health Study	USA, Multi-ethnic	9-10 %M 0	2379	7 years	Food diary	Cereal consumption frequency (Percentage of days consuming ready- to-eat cereal and cooked cereal)	Total cholesterol Fasting, Whole blood	1 %	-0.08 (0.03)	0.03	Centre, ethnicity, parental education

Table 2.138 Total cholesterol and breakfast cereal: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Rosado <i>et al.</i> , 2008) 14794	Control	27/39	134.6	141.3	6.2 (CI -4.7, 17.1)*			Total cholesterol	Fasting plasma (mg/dL)	12 weeks	No change	unclear
	One serving breakfast cereal + nutrition education	36/45	128.6	136.8	9.5 (CI -0.7, 19.6)*		NS				Decrease	
	One serving breakfast cereal/d	32/46	143.3	149.6	14.7 (CI 4.5, 24.9)*		NS				No change	
	Two servings breakfast cereal/d	34/48	141.3	147.5	14.2 (CI 4.3, 24.1)*		NS				No change	
Adult studies												
(Kleemola <i>et al.</i> , 1999) 15244	Group 1- Cereal diet first	104/allocated not reported	5.97 (SD 0.8)	5.91 (SD 0.8)			0.007	Total cholesterol	Serum (mmol/L)	6 weeks	No change	bias
	Group 2- Control diet first	105/allocated not reported	6 (SD 0.8)	6.1 (SD 0.8)	0.1						No change	
15254	Group 1- Control diet second	104/allocated not reported	6.1 (SD 0.8)	6.03 (SD 0.9)	-0.07						No change	
	Group 2- Cereal diet second	105/allocated not reported	6.04 (SD 0.8)	5.83 (SD 0.8)	-0.21		0.069				No change	
(Zaveri and Drummon d, 2009) 16926	Cereal bar	13/14				NS	NS	Total cholesterol	Fasting	12 weeks	No change	unclear
	Control	12/13				NS					No change	

* Results do not match baseline and follow-up as they have been adjusted for initial value, gender, school random effect and significant interactions.

HDL cholesterol and breakfast cereals

No cohort studies reported results concerning breakfast cereals and HDL cholesterol.

Summary of RCT data

Three studies provided data on the effects of breakfast cereal on HDL cholesterol levels (Zaveri and Drummond, 2009; Rosado *et al.*, 2008; Kleemola *et al.*, 1999). Zaveri *et al.* (Zaveri and Drummond, 2009) included children as participants; consequently there was an insufficient number of studies to combine to perform a meta-analysis.

As the three trials also provided data on total cholesterol, a summary of these can be found in the section on total cholesterol and breakfast cereals section.

Zaveri and Drummond (Zaveri and Drummond, 2009) compared the effects of a conventional cereal bar snack (30g weight; high in carbohydrate) or a control (no snack) on dietary intake, body weight and blood lipids over 12 weeks. In the 45 healthy male subjects who took part, no statistically significant differences in HDL cholesterol within or between groups were observed (data not provided).

Two studies explored the effects of breakfast cereal as a meal, rather than as a snack (Rosado *et al.*, 2008; Kleemola *et al.*, 1999). In the study reported by Rosado *et al.*, (Rosado *et al.*, 2008) 147 overweight/at risk of overweight children were randomly assigned to one of four groups: i) non-intervention (control); ii) one serving (33+/-7g) of ready-to-eat cereal (RTEC) plus a nutrition education program; iii) one serving (33+/-7g) RTEC for breakfast; or iv) one serving (33+/-7g) RTEC for breakfast and one for dinner. At follow up, adjusted changes in HDL cholesterol within and between groups were not statistically significant.

Kleemola *et al.* (Kleemola *et al.*, 1999) carried out a six-week trial to test the effects of a high carbohydrate breakfast which included breakfast cereal on lowering serum cholesterol. The 209 eligible subjects were randomised into intervention breakfast cereal (60g or 80g cereal i.e. Cornflakes or Rice Krispies for women and men, respectively) or usual breakfast (control) groups, with a washout period of six weeks between phases. At follow up, HDL cholesterol was reduced in both groups in the first six weeks and the second six weeks; however changes between groups were only statistically significant during the cereal diet rather than the control diet ($p=0.004$ and $p=0.015$ for the first group and the second group, respectively).

Overall, these three trials are heterogeneous in design, but do not show consistent findings with regard to HDL cholesterol.

Table 2.139 HDL cholesterol and breakfast cereals: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Rosado <i>et al.</i> , 2008) 14816	Control	27/39	47.5	44.8	-3.0 (CI 6.7, 0.7)*			HDL-C	Fasting plasma (mg/dL)	12 weeks	No change	unclear
	One serving breakfast cereal + nutrition education	36/45	43.1	49.7	1.7 (CI -1.7, 5.1)*		NS**				Decrease	
	One serving breakfast cereal/d	32/46	48.4	47	-2.2 (CI -5.7, 1.4)*		NS				No change	
	Two servings breakfast cereal/d	34/48	48.1	48.5	1.0 (CI -2.4, 4.3)*		NS				No change	
Adult studies												
(Kleemola <i>et al.</i> , 1999) 15248	Group 1- Cereal diet first	104/allocated not reported	1.35 (SD 0.3)	1.28 (SD 0.3)	-0.07		0.004	HDL-C	Serum (mmol/L)	6 weeks	No change	bias
	Group 2- Control diet first	105/allocated not reported	1.4 (SD 0.3)	1.38 (SD 0.3)	-0.02						No change	
15256	Group 1- Control diet second	104/allocated not reported	1.36 (SD 0.3)	1.33 (SD 0.3)	-0.03			HDL-C	Serum (mmol/L)	6 weeks	No change	bias
	Group 2- Cereal diet second	105/allocated not reported	1.38 (SD 0.3)	1.3 (SD 0.3)	-0.08		0.015				No change	
(Zaveri and Drummond, 2009) 16929	Cereal bar	13/14				NS	NS	HDL-C	Fasting	12 weeks	No change	unclear
	Control	12/13				NS					No change	

* Results do not match baseline and follow-up as they have been adjusted for initial value, gender, school random effect and significant interactions.

** Unadjusted results show that HDL cholesterol statistically significantly increased when compared with the control group ($p < 0.01$) (Rosado *et al.*, 2008)

LDL cholesterol and breakfast cereals

No cohort studies reported results concerning breakfast cereals and LDL cholesterol.

Summary of RCT data

Two studies provided data on breakfast cereals and LDL cholesterol (Zaveri and Drummond, 2009; Rosado *et al.*, 2008). As there was an insufficient number of studies, it was not possible to combine studies using meta-analysis.

Body weights were unchanged in all trials other than in Rosado *et al.* (Rosado *et al.*, 2008) in which the authors reported that there was a decrease in the 'one serving breakfast cereal plus nutrition education' group only (Rosado *et al.*, 2008).

Zaveri and Drummond (Zaveri and Drummond, 2009) compared the effects of a conventional cereal bar snack (30g weight; high in carbohydrate) or a control (no snack) on dietary intake, body weight and blood lipids over a 12-week period. Forty-five healthy male subjects (BMI 25-35kg/m²) were randomly allocated to the cereal bar group, the control group or a further group which consumed a nonconventional snack (almonds), although data for the latter were not eligible and thus not extracted. The intervention was administered through the provision of two cereal bars per day, which could be consumed at any time. Although quantitative data were not reported, it is evident that LDL cholesterol neither differed markedly from baseline nor differed between groups as the authors concluded that no statistically significant changes were observed.

In the study reported by Rosado *et al.* (Rosado *et al.*, 2008), 147 overweight/at risk of overweight children were randomly assigned to one of four groups: i) non-intervention (control); ii) one serving (33+/-7g) of ready-to-eat cereal (RTEC) plus a nutrition education program; iii) one serving (33+/-7g) RTEC for breakfast; or iv) one serving (33+/-7g) RTEC for breakfast and one for dinner. Results are reported at 12 weeks from baseline randomisation. All groups experienced a slight increase in LDL cholesterol, bar the one serving RTEC plus nutrition education group which saw a reduction; however differences between these means and indeed differences from baseline were not statistically significant.

These two studies indicate no improvement in LDL cholesterol with consumption of breakfast cereal in overweight/obese children and adults.

Table 2.140 LDL cholesterol and breakfast cereals: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Rosado <i>et al.</i> , 2008) 14817	Control	27/39	116.7	125.4	8 (CI -4.8, 20.8)*			LDL-C	Fasting plasma (mg/dL)	12 weeks	No change	unclear
	One serving breakfast cereal + nutrition education	36/45	114.1	112.3	1.8 (CI -10, 13.5)*		NS				Decrease	
	One serving breakfast cereal/d	32/46	122.6	137.1	19.3 (CI 7.3, 31.3)*		NS				No change	
	Two servings breakfast cereal/d	34/48	123.6	125.2	9.2 (CI -2.3, 20.7)*		NS				No change	
Adult study												
(Zaveri and Drummond, 2009) 16928	Cereal bar	13/14				NS	NS	LDL-C	Fasting	12 weeks	No change	unclear
	Control	12/13				NS					No change	

*Results do not match baseline and follow-up as they have been adjusted for initial value, gender, school random effect and significant interactions.

Triacylglycerol and breakfast cereals

No cohort studies reported results concerning breakfast cereals and TAG.

Summary of RCT data

Two trials explored the effects of breakfast cereal intake on TAG levels in overweight children and adults (Zaveri and Drummond, 2009; Rosado *et al.*, 2008). As there was an insufficient number of studies, it was not possible to conduct a meta-analysis.

Zaveri and Drummond (Zaveri and Drummond, 2009) compared the effects of a conventional cereal bar snack (30g weight; high in carbohydrate) or a control (no snack) on dietary intake, body weight and blood lipids over 12 weeks. Forty-five healthy male subjects (BMI 25-35kg/m²) were randomly allocated to these groups; a further group was also included which consumed a nonconventional snack (almonds), although these data were not extracted. The intervention was administered through the provision of two cereal bars per day, which could be consumed at any time. There were no statistically significant differences between or within groups in fasting TAG levels.

In the study reported by Rosado *et al.*, (Rosado *et al.*, 2008) 147 overweight/at risk of overweight children were randomly assigned to one of four groups: i) non-intervention (control); ii) one serving (33+/-7g) of ready-to-eat cereal (RTEC) plus a nutrition education program; iii) one serving (33+/-7g) RTEC for breakfast; or iv) one serving (33+/-7g) RTEC for breakfast and one for dinner. At follow up, adjusted changes in TAG were not statistically significantly different within or between groups.

These trials are heterogeneous in design, but do not demonstrate an impact on TAG levels when comparing cereal consumption as bars or ready-to-eat breakfast cereal with a no cereal control.

Table 2.141 Triacylglycerol and breakfast cereals: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
Children study												
(Rosado <i>et al.</i> , 2008) 14815	Control	27/39	121.9	121.6	-4.3 (CI -24.6, 16)*			TAG	Fasting plasma (mg/dL)	12 weeks	No change	unclear
	One serving breakfast cereal + nutrition education	36/45	129.5	108.7	-18.1 (CI -36.7, 0.6)*		NS				Decrease	
	One serving breakfast cereal/d	32/46	109.5	134.5	13.5 (CI -6.5, 33.4)*		NS				No change	
	Two servings breakfast cereal/d	34/48	134.2	119.4	-10.3 (CI -29, 8.4)*		NS				No change	
Adult study												
(Zaveri and Drummond, 2009) 16927	Cereal bar	13/14				NS	NS	TAG	Fasting	12 weeks	No change	unclear
	Control	12/13				NS					No change	

*Results do not match baseline and follow-up as they have been adjusted for initial value, gender, school random effect and significant interactions.

Results - Legumes

Total cholesterol and legumes

No cohort studies reported results concerning legumes and total cholesterol.

Summary of RCT data

Data from one intervention, which tested the effects of legume intake on lipid peroxidation, are tabulated in Table 2.142

Crujeiras *et al.* (Crujeiras *et al.*, 2007) compared the effects of a hypocaloric diet with non-soybean legumes for four days per week against a hypocaloric diet without legumes (control) in 30 obese subjects. At follow up (eight weeks), subjects in both groups experienced a decrease in total cholesterol levels from baseline, statistically significantly so in the hypocaloric control diet with legumes ($p < 0.001$). Between groups, there was also a difference that achieved statistical significance ($p = 0.01$). The authors concluded that the reduction in total cholesterol was directly related to weight loss (Crujeiras *et al.*, 2007); changes in outcome therefore may not be solely attributable to legume intake.

Table 2.142 Total cholesterol and legumes: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	p-value within group Δ from baseline	p-value difference between groups	Outcome/ Assessment method	Result/ Outcome details	Result- specific follow- up	Weight change	Outcome Assessment Bias
(Crujeiras <i>et al.</i> , 2007)	Hypocaloric control diet	15/15	181 (SD 35)	173 (SD 32)	NS		Total cholesterol	Fasting plasma (mg/dL)	8 weeks	Decrease	unclear
16393	Hypocaloric diet + legumes	15/15	215 (SD 37)	182 (SD 27)	<0.001	0.01				Decrease	

Results - Wholegrains

Total cholesterol and wholegrains

No cohort studies reported results concerning wholegrains and total cholesterol.

Summary of RCT data

Five studies reported data on the effects of wholegrain intake on total cholesterol (Andersson *et al.*, 2007; Kim *et al.*, 2008; Howard *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002). Of these, three studies were conducted in the USA (Howard *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002), one in Sweden (Andersson *et al.*, 2007) and one in Korea (Kim *et al.*, 2008).

For most studies, a parallel group approach was taken and only one study by (Andersson *et al.*, 2007) employed a crossover design. One study was single blind (Howard *et al.*, 2006). The remaining trials either did not provide clear information regarding blinding (Kim *et al.*, 2008; Saltzman *et al.*, 2001) or were open (Andersson *et al.*, 2007; Davy *et al.*, 2002).

With the exception of the very large Women's Health Initiative Dietary Modification Trial (sample size= 2832) (Howard *et al.*, 2006), sample sizes were relatively small and ranged from 34 to 47 subjects. Subjects were all adults and the mean BMI was less than 30kg/m² in four trials. Kim *et al.* (Kim *et al.*, 2008) did not provide an average BMI of subjects. Two studies were identified that studied women only (Howard *et al.*, 2006; Kim *et al.*, 2008) and one that included males only (Davy *et al.*, 2002).

The interventions were somewhat mixed since they compared single wholegrain products such as oats with wheat or white vs. black rice (Davy *et al.*, 2002; Kim *et al.*, 2008; Saltzman *et al.*, 2001), or diets consisting of a range of different grain-based products in whole compared with the refined state (Andersson *et al.*, 2007; Howard *et al.*, 2006).

There was a great deal of heterogeneity between studies in terms of body weight changes. Body weight increased in two out of the five trials (Andersson *et al.*, 2007). Three studies, except a control group in one (Howard *et al.*, 2006), reported a weight decrease in participants. Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

Five studies were included in the meta-analysis comparing different wholegrain intakes and changes in total cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 12 weeks. The pooled estimate indicated that total cholesterol was 0.04mmol/L (95% CI -0.12 to 0.20) lower with consumption of a diet higher in wholegrains. This was not significantly different from zero ($p=0.49$). Overall heterogeneity denoted by I^2 was 75% (95% CI 39 to 90%). Statistically, there was no evidence that high wholegrain consumption is associated with lower levels of total cholesterol.

Figure 2.74 Forest plot for wholegrains and total cholesterol (mmol/L)

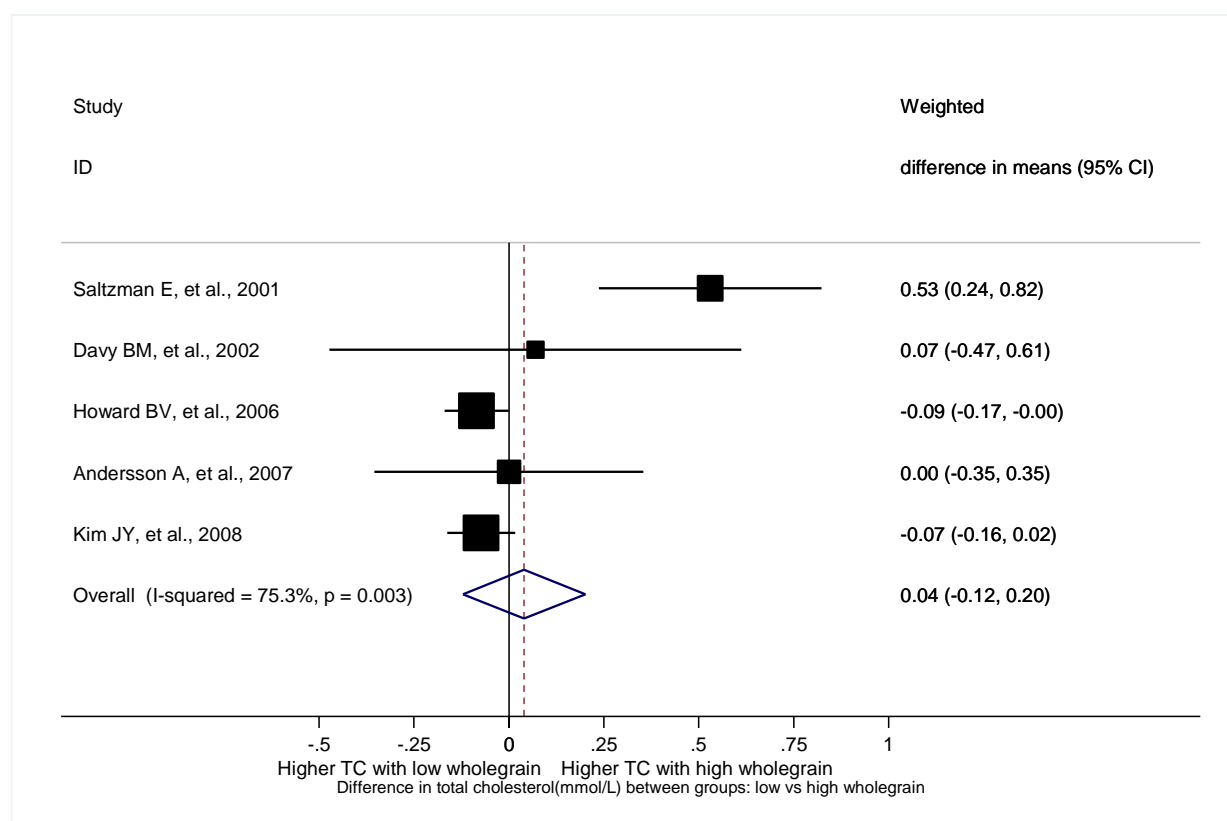


Table 2.143 Total cholesterol and wholegrains: RCT data

Author/ Result ID	Intervention groups	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	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Andersson <i>et al.</i> , 2007) *16300	Refined grain products	30/30	5.5 (SD 0.8)	5.5 (SD 0.7)		NS				Total cholesterol	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	5.5 (SD 0.7)	5.5 (SD 0.7)		NS	0.76						Increase	
(Davy <i>et al.</i> , 2002) 15430	Wheat cereal	18/18	4.91 (SE 0.16)	5.22 (SE 0.18)						Total cholesterol	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	5.28 (SE 0.18)	5.15 (SE 0.21)			0.08						Increase	
(Howard <i>et al.</i> , 2006) *16246	Control	approx n=1699 (5.8% sub-sample of 29294)	224.2 (SD 39.2)	216.6 (SD 35.9)	-6.9 (SD 31.9)					Total cholesterol	Fasting (mg/dL)	3 years	No change	No bias
	Low fat	approx n=1132 (5.8% sub-sample of 19541)	224.0 (SD 36.5)	214.1 (SD 35.3)	-10.2 (SD 32.0)		<0.05						Decrease	
17612	Low fat minus control	As above						-3.26 (CI - 6.53, 0.00)	<0.05	Total cholesterol	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Kim <i>et al.</i> , 2008) *16772	Brown & black rice meal replacement	20/23	184.53 (SD 3.25)	154.2 (SD 3.79)	-30.33 (SD 5.18)	<0.05	NS			Total cholesterol	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
	White rice meal replacement	20/24	184.28 (SD 6.39)	156.78 (SD 3.89)	-27.5 (SD 5.95)	<0.05							Decrease	
(Saltzman <i>et al.</i> , 2001)	Control	21/21	4.4 (SD 0.98)		-0.34 (SD 0.5)					Total cholesterol	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear

Author/ Result ID	Intervention groups	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	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
16199	Oats	20/22	4.88 (SD 0.87)		-0.87 (SD 0.47)	0.05	<0.05						Decrease	

*This result was used in the meta-analysis of wholegrains and total cholesterol

HDL cholesterol and wholegrains

No cohort studies reported results concerning wholegrains and HDL cholesterol.

Summary of RCT data

Five studies reported data on the effects of wholegrain intake on HDL cholesterol (Andersson *et al.*, 2007; Kim *et al.*, 2008; Howard *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002).

The five trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and wholegrains.

Apart from one study that reported a decrease in the intervention group and no weight change in the control group, body weights either increased (Andersson *et al.*, 2007; Davy *et al.*, 2002) or decreased (Kim *et al.*, 2008; Saltzman *et al.*, 2001). Changes in HDL cholesterol therefore may not be solely attributable to the dietary intervention.

Five studies were included in the meta-analysis comparing different wholegrain intake and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 12 weeks. Heterogeneity was high at 77% and therefore the pooled estimate was not displayed. .

Figure 2.75 Forest plot for wholegrains and HDL cholesterol (mmol/L)

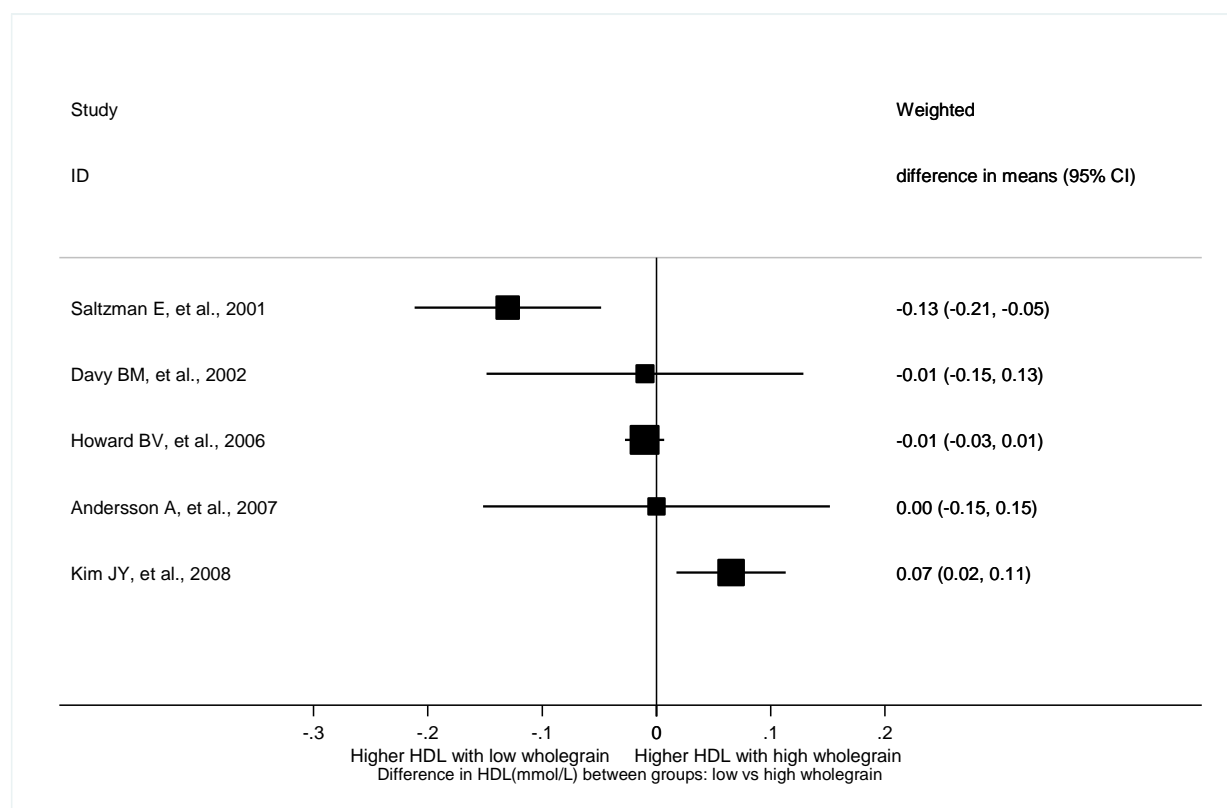


Table 2.144 HDL cholesterol and wholegrains: RCT data

Author/ Result ID	Intervention groups	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	Outcom e details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Anders son <i>et</i> <i>al.</i> , 2007) *16301	Refined grain products	30/30	1.2 (SD 0.2)	1.2 (SD 0.3)		NS				HDL-C	Fasting (mmol/ L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	1.3 (SD 0.3)	1.2 (SD 0.3)		NS	0.15						Increase	
(Davy <i>et</i> <i>al.</i> , 2002) 15434	Wheat cereal	18/18	0.89 (SE 0.04)	0.85 (SE 0.05)						HDL-C	Fasting plasma (mmol/ L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	0.87 (SE 0.05)	0.86 (SE 0.05)			0.41						Increase	
(Howar d <i>et al.</i> , 2006) 16248	Control – healthy diet	approx n=1699 (5.8% sub-sample of 29294)	58.4 (SD 15.4)	58.2 (SD 15.5)	-0.3 (SD 10.2)					HDL-C	Fasting (mg/dL)	3 years	No change	No bias
	Low fat, increased fruit, vegetables and wholegrains	approx n=1132 (5.8% sub-sample of 19541)	60.1 (SD 16.1)	59.7 (SD 15.8)	-0.7 (SD 9.4)		NS						Decrease	
*17614	Low fat minus control	As above						-0.43 (CI - 1.42, 0.57)	NS	HDL-C	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Kim <i>et</i> <i>al.</i> , 2008) *16774	Brown & black rice meal replacement	20/23	50.33 (SD 1.42)	55.87 (SD 1.82)	5.53 (SD 2.08)	<0.05	NS			HDL-C	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
	White rice meal replacement	20/24	51.43 (SD 1.82)	54.43 (SD 2.46)	3.0 (SD 3.67)	NS							Decrease	
(Saltzm an <i>et</i> <i>al.</i> , 2001) 16203	Control	21/21	1.17 (SD 0.28)		-0.04 (SD 0.14)					HDL-C	Fasting plasma (mmol/ L)	6 weeks	Decrease	unclear
	Oats	20/22	1.15 (SD 0.2)		0.09 (SD 0.13)	0.05	NS						Decrease	

*This result was used in the meta-analysis of wholegrains and HDL cholesterol

LDL cholesterol and wholegrains

No cohort studies reported results concerning wholegrains and LDL cholesterol.

Summary of RCT data

Four studies reported data on the effects of wholegrain intake on LDL cholesterol (Andersson *et al.*, 2007; Howard *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002). Of these, three were conducted in the USA (Howard *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002) and one in Sweden (Andersson *et al.*, 2007).

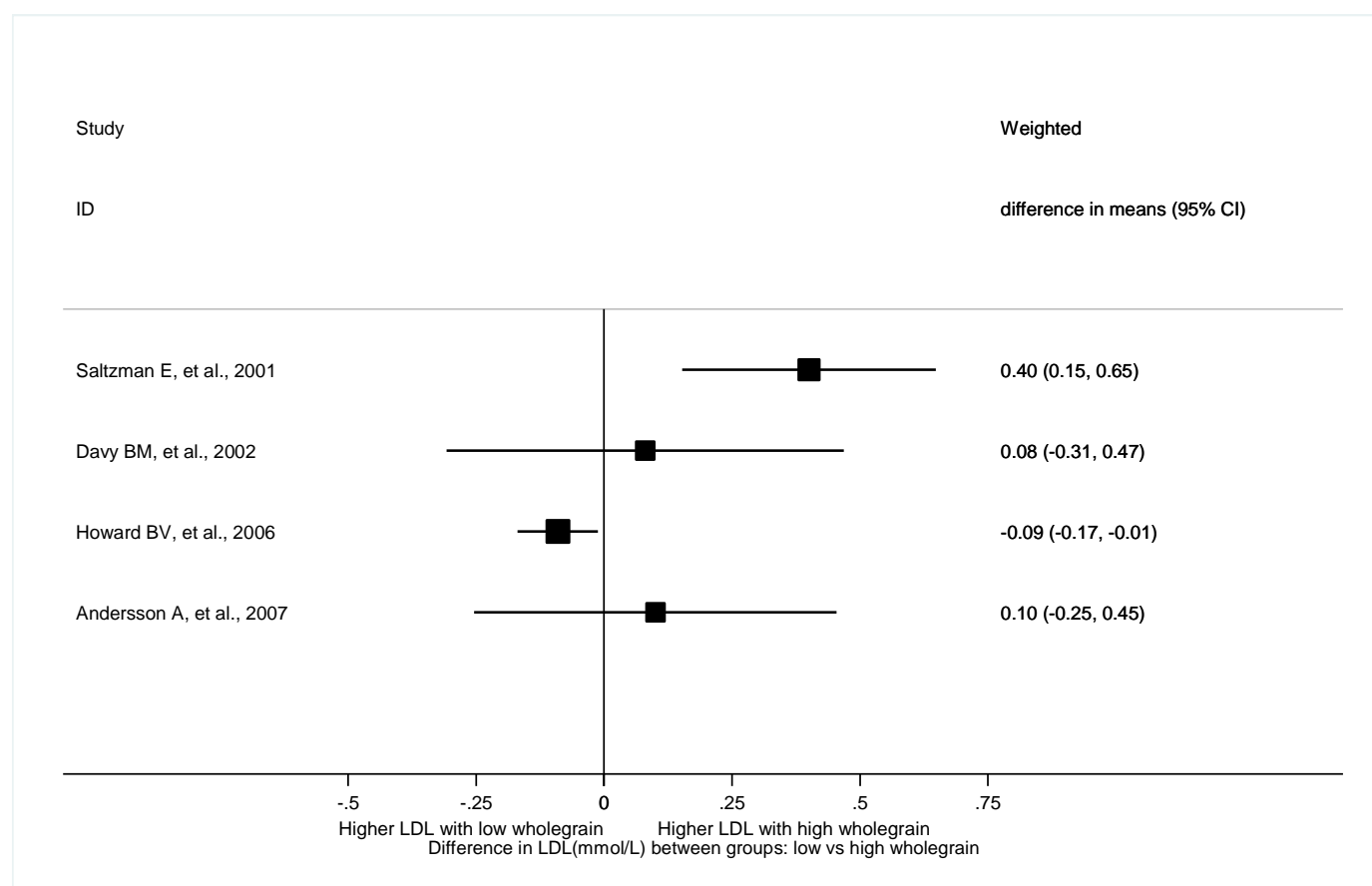
For most studies, a parallel group approach was taken and, in fact, only one study by Andersson *et al.* (Andersson *et al.*, 2007) employed a crossover design. One study was single blind (Howard *et al.*, 2006). The remaining trials either did not provide clear information regarding blinding or were open.

Excluding one large study that reported The Women's Health Initiative Dietary Modification Trial (sample size= 2832) (Howard *et al.*, 2006), sample sizes were relatively small and ranged from 34 to 43 subjects. Subjects were all adults and the mean BMI was between 26 and 29kg/m² in the four trials. One study was identified that studied females only (Howard *et al.*, 2006) and one that included males only (Davy *et al.*, 2002).

Apart from one study that reported a decrease in the intervention group and no weight change in the control group (Howard *et al.*, 2006), body weights either increased (Andersson *et al.*, 2007; Davy *et al.*, 2002) or decreased (Kim *et al.*, 2008; Saltzman *et al.*, 2001). Changes in LDL cholesterol therefore may not be solely attributable to the dietary intervention.

Four studies were included in the meta-analysis comparing different wholegrain intakes and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. Overall heterogeneity denoted by I² was 79%, therefore no pooled estimate was displayed.

Figure 2.76 Forest plot for wholegrains and LDL cholesterol (mmol/L)



One intervention, the Women's Health Initiative Dietary Modification Trial, explored the effects of wholegrain intake on non-HDL cholesterol (Howard *et al.*, 2006). Details concerning the design, participants, duration and nature of the intervention are included in Table 2.145

The Women's Health Initiative Randomized Controlled Dietary Modification Trial was designed to test the hypothesis that a low fat, high fruit and vegetable (F&V), high grain diet would reduce the risk of cardiovascular disease in middle-aged and older women. The goal of the dietary intervention was to decrease total fat to 20% of energy intake, to increase F&V portions to five or more per day and to increase servings of grains to a minimum of six per day. Changes in outcome may therefore not be attributed solely to the increase in wholegrain intake.

At three years, the intervention group had experienced a somewhat greater decrease in non-HDL cholesterol compared with the control group (-9.7 vs. -6.6mg/dL, respectively) but the difference between groups was not statistically significant. It is noteworthy to highlight that the low fat group experienced weight loss, whilst the control group did not. Once again, changes in non-HDL cholesterol may not be solely attributable to the dietary intervention.

Table 2.145 LDL cholesterol and wholegrains: RCT data

Author/ Result ID	Interventi on groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Andersson <i>et al.</i> , 2007)	Refined grain products	30/30	3.7 (SD 0.8)	3.6 (SD 0.7)		NS				LDL-C	Fasting (mmol/L)	6 weeks	Increase	unclear
*16302	Wholegrain products	30/30	3.7 (SD 0.8)	3.7 (SD 0.7)		NS	0.4						Increase	
(Davy <i>et al.</i> , 2002) 15431	Wheat cereal	18/18	3.3 (SE 0.15)	3.57 (SE 0.14)						LDL-C	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	3.58 (SE 0.16)	3.49 (SE 0.14)			0.02						Increase	
(Howard <i>et al.</i> , 2006) 16247	Control	approx n=1699 (5.8% sub-sample of 29294)	134.2 (SD 35.1)	127.0 (SD 34.0)	-6.2 (SD 29.1)					LDL-C	Fasting (mg/dL)	3 years	No change	No bias
	Low fat	approx n=1132 (5.8% sub-sample of 19541)	133.3 (SD 35.3)	123.2 (SD 33.1)	-9.7 (SD 29.3)		<0.05						Decrease	
*17613	Low fat minus control	As above						-3.55 (CI -6.58, -0.52)	<0.05	LDL-C	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Saltzman <i>et al.</i> , 2001) 16202	Control	21/21	2.79 (SD 0.83)		-0.2 (SD 0.41)					LDL-C	Derived by calculation Fasting, Plasma (mmol/L)	6 weeks	Decrease	unclear
	Oats	20/22	3.16 (SD 0.77)		-0.6 (SD 0.41)	0.05	<0.05						Decrease	
Non-HDL-C														
(Howard <i>et al.</i> , 2006) 16250	Control	approx n=1699 (5.8% sub-sample of 29294)	165.8 (SD 41.1)	158.4 (SD 37.0)	-6.6 (SD 32.6)					Non- HDL-C	Fasting (mg/dL)	3 years	No change	No bias
	Low fat	approx n=1132 (5.8%	163.9 (SD 39.5)	154.3 (SD 36.5)	-9.7 (SD 32.0)		NS						Decrease	

Author/ Result ID	Intervention groups	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	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
		sub-sample of 19541)												
17616	Low fat minus control	As above						-3.08 (CI - 6.37, 0.22)	NS	Non- HDL-C	Fasting (mg/dL)	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 LDL cholesterol

Triacylglycerol and wholegrains

No cohort studies reported results concerning wholegrains and TAG.

Summary of RCT data

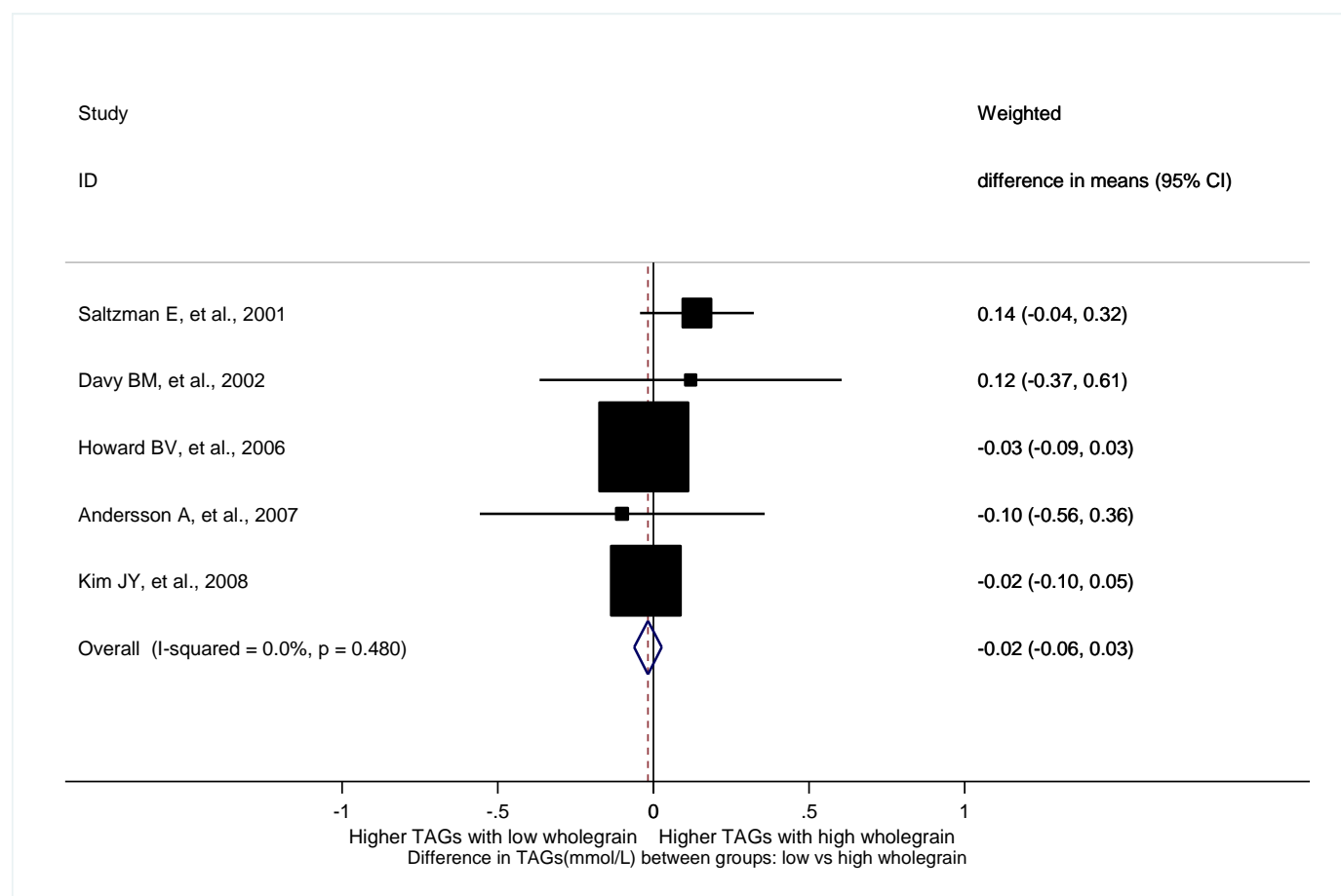
Five studies reported data on the effects of wholegrain intake on TAG (Andersson *et al.*, 2007; Kim *et al.*, 2008; Howard *et al.*, 2006; Saltzman *et al.*, 2001; Davy *et al.*, 2002). Andersson *et al.* (Andersson *et al.*, 2007) also presented results on non-esterified fatty acids.

The five trials also provided data on total cholesterol; a summary of these randomised controlled trials therefore can be found in the section on total cholesterol and wholegrains.

Apart from one study that reported a decrease in the intervention group and no weight change in the control group (Howard *et al.*, 2006), body weights either increased (Andersson *et al.*, 2007; Davy *et al.*, 2002) or decreased (Kim *et al.*, 2008; Saltzman *et al.*, 2001). Changes in TAG therefore may not be solely attributable to the dietary intervention.

Five studies were included in the meta-analysis comparing different wholegrain intakes and changes in TAG reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from six to 12 weeks. The pooled estimate indicated that total TAG levels were 0.02mmol/L (95% CI -0.03 to 0.06) lower with consumption of a diet higher in wholegrains. This was not significantly different from zero ($p=0.46$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 76%). Statistically, there was no evidence that high wholegrain consumption is associated with lower levels of TAG.

Figure 2.77 Forest plot for wholegrains and TAG (mmol/L)



One study also provided data on non-esterified fatty acids and wholegrain intake (Andersson *et al.*, 2007). Andersson *et al.* (Andersson *et al.*, 2007) assessed the effect of wholegrain or refined grain products on various aspects of health in a group of 34 overweight men and women. Body weight in both groups increased throughout the trial. After six weeks, no statistically significant differences in non-esterified fatty acids within or between groups were observed.

Table 2.146 Triacylglycerol, fatty acids and wholegrains: RCT data

Author / Result ID	Intervention groups	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	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Andersson <i>et al.</i> , 2007) *16303	Refined grain products	30/30	1.3 (SD 0.6)	1.6 (SD 1.0)		<0.05				TAG	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	1.4 (SD 0.8)	1.5 (SD 0.8)		NS	0.19						Increase	
(Davy <i>et al.</i> , 2002) 15431	Wheat cereal	18/18	1.5 (SE 0.17)	1.83 (SE 0.17)						TAG	Fasting plasma (mmol/L)	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	1.83 (SE 0.17)	1.71 (SE 0.18)			0.07						Increase	
(Howard <i>et al.</i> , 2006) 16251	Control	approx n=1699 (5.8% sub-sample of 29294)	141.1 (SD 66.3)	144.6 (SD 63.7)	1.0 (SD 0.3)					TAG	Fasting (mg/dL)	3 years	No change	No bias
	Low fat	approx n=1132 (5.8% sub-sample of 19541)	138.6 (SD 65.1)	142.3 (SD 67.5)	1.0 (SD 0.4)		NS						Decrease	
*17617	Low fat minus control	As above						0.00 (CI - 0.03, 0.04)	NS	TAG	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Kim <i>et al.</i> , 2008) *16776	Brown & black rice meal replacement	20/23	140.13 (SD 10.15)	93.13 (SD 5.84)	-47.00 (SD 10.58)	<0.05	NS			TAG	Fasting serum (mg/dL)	6 weeks	Decrease	unclear
	White rice meal replacement	20/24	139 (SD 11.3)	94.07 (SD 9.69)	-44.93 (SD 10.96)	<0.05							Decrease	
(Saltzman <i>et al.</i> , 2001) 16197	Control	21/21	0.94 (SD 0.36)		-0.22 (SD 0.23)					TAG	Fasting plasma (mmol/L)	6 weeks	Decrease	unclear
	Oats	20/22	1.23 (SD 0.6)		-0.36 (SD 0.36)	NS	NS						Decrease	
Fatty acids														

Author / Result ID	Intervention groups	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	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Andersson <i>et al.</i> , 2007) 16304	Refined grain products	30/30	0.63 (SD 0.17)	0.62 (SD 0.18)		NS				Free fatty acid	Fasting (mmol/L)	6 weeks	Increase	unclear
	Wholegrain products	30/30	0.56 (SD 0.19)	0.61 (SD 0.18)		NS	0.99						Increase	

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

Total cholesterol:HDL ratio and wholegrains

No cohort studies reported results concerning wholegrains and TC:HDL ratio.

Summary of RCT data

Two studies provided data on the effects of wholegrain intake on the TC:HDL ratio. Body weights increased in one study (Davy *et al.*, 2002) but varied in another (Howard *et al.*, 2006). That is, weights remained unchanged in the control group of the study conducted by Howard *et al.* (Howard *et al.*, 2006) but the intervention low fat group experienced weight loss.

Davy *et al.* (Davy *et al.*, 2002) tested the effect of breakfast cereals comprised of wheat or wholegrain oats in overweight and obese males. Using a three-month parallel group trial, the consumption of 60g oatmeal and 76g oat-bran ready-to-eat cold cereal for breakfast and as a snack slightly improved the TC:HDL ratio when compared with 60g wholewheat hot natural cereal and 81g frosted mini-wheats ($p=0.05$).

The Women's Health Initiative Dietary Modification Trial reported by Howard *et al.* (Howard *et al.*, 2006) tested the hypothesis that a low fat, high fruit and vegetable, high grain diet would reduce the risk of cardiovascular disease in middle-aged and older women. At three years, TC:HDL ratio had marginally decreased in both dietary groups but not statistically differently between groups. It is important to consider however that compliance in increasing wholegrains intake was only partially achieved, thus any potential associations may have been neglected.

The two studies presented here provide inconsistent evidence concerning the effect of wholegrain intake on the TC:HDL ratio.

Table 2.147 Total cholesterol:HDL ratio and wholegrains: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Outcome	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Davy <i>et al.</i> , 2002) 15440	Wheat cereal	18/18	5.7 (SE 0.3)	6.4 (SE 0.4)			TC:HDL ratio	12 weeks	Increase	bias
	Wholegrain oat cereal	17/18	6.4 (SE 0.4)	6.0 (SE 0.5)		0.05			Increase	
(Howard <i>et al.</i> , 2006) 16249	Control	approx n=1699 (5.8% sub-sample of 29294)	4.1 (SD 1.3)	4.0 (SD 1.2)	-0.1 (SD 1.0)		TC:HDL ratio	3 years	No change	No bias
	Low fat	approx n=1132 (5.8% sub-sample of 19541)	4.0 (SD 1.2)	3.8 (SD 1.1)	-0.2 (SD 0.8)	NS			Decrease	

Apolipoproteins and wholegrains

No cohort studies reported results concerning wholegrains and apolipoproteins.

Summary of RCT data

Two studies of adults investigated the effects of wholegrain consumption on apolipoprotein levels (Johnston, 1998; Howard *et al.*, 2006). As there was an insufficient number of studies, it was not possible to combine studies in a meta-analysis.

One study reported a weight decrease in the intervention group and no weight change in the control group, whereas the other stated no change in body weights during the intervention (Johnston, 1998).

The trial by Johnston *et al.* (Johnston, 1998) compared the effects of a wholegrain oat cereal, containing 3g soluble fibre, with a control breakfast cereal without soluble fibre in 135 participants with mild to moderate hypercholesterolaemia. A Step 1 diet was implemented prior to the treatment. After six weeks, participants in the control cereal group tended to experience an increase in apolipoprotein A-1 and apolipoprotein B whereas the wholegrain oat cereal group experienced a decrease in the respective outcomes. Observed differences in apolipoprotein A-1 were not statistically significant; changes in apolipoprotein B did reach statistical significance however ($p=0.02$).

The Women's Health Initiative Randomized Controlled Dietary Modification Trial reported by Howard *et al.* (Howard *et al.*, 2006) aimed to decrease total fat to 20% of energy intake, to increase F&V portions to five or more per day and to increase servings of grains to a minimum of six per day. This was implemented through a behavioural modification program that ran intensively throughout the first year of the trial and then less intensively thereafter. Results concerning lipoprotein (a) are reported here at three years from baseline randomisation. The low fat intervention did not statistically significantly alter lipoprotein (a) compared with the control group. However, compliance with the goal to increase wholegrains was only partially achieved.

Table 2.148 Apolipoproteins and wholegrains: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value difference between groups	Diff. between groups in Δ from baseline	Outcome	Outcome details	Result- specific follow-up	Weight change	Outcome Assessment Bias
(Howard <i>et al.</i> , 2006) 16255	Control	approx n=1699 (5.8% sub-sample of 29294)	15.4 (SD 17.0)	13.8 (SD 15.5)	0.9 (SD 0.5)			Lipoprotein (a)	Fasting (mg/dL)	3 years	No change	No bias
	Low fat	approx n=1132 (5.8% sub-sample of 19541)	15.7 (SD 17.5)	13.2 (SD 15.1)	0.9 (SD 0.5)	NS					Decrease	
17618	Low fat minus control	As above					-0.01 (CI - 0.07, 0.05)	Lipoprotein (a)	Fasting (mg/dL)	3 years	No change in control group, decrease in low fat group	No bias
(Johnston, 1998) 16686	Control cereal	59/62	129.7 (SD 19.2)	131.1 (SD 17.5)	1.39 (SD 10.1)	NS		Apolipoprotein A-1	Fasting plasma (mg/dL)	6 weeks	No change	unclear
	Wholegrain oat cereal	59/62	134.8 (SD 22.0)	134.2 (SD 22.4)	-0.6 (SD 11.3)						No change	
16687	Control cereal	59/62	125.6 (SD 17.9)	129.1 (SD 18.7)	3.6 (SD 13.8)	0.02		Apolipoprotein B	Fasting plasma (mg/dL)	6 weeks	No change	unclear
	Wholegrain oat cereal	59/62	124.2 (SD 15.1)	122.1 (SD 14.7)	-2.1 (SD 12.1)						No change	

Results – Glycaemic index and load

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

Oxlund (Oxlund and Heitmann, 2006) reported GI and GL calculated from a dietary history using values from the International table of glycaemic index and glycaemic load values: 2002 (Foster-Powell *et al.*, 2002). The reference food used to calculate GI values was glucose or white bread.

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

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

However, within the trials there is some variation in the methods used to calculate GI and GL. Accordingly, the author definitions of high and low GI and GL have been adopted to compare studies, even when the apparent differences between trial arms appear to be quite small or not in accord with notions of what may be viewed as high or low.

It should also be recognised that 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.

Total cholesterol and glycaemic index and load

Summary of cohort results

Data were provided by one cohort study of adults (the MONICA Study) (Oxlund and Heitmann, 2006). The association between baseline dietary GI and GL and the change in fasting total cholesterol during the six years of follow up were presented.

In men only, a small increase in total cholesterol was observed for each unit increase in baseline dietary GI. No association was observed in women, or in both genders for GL.

Analyses on GL were repeated in subgroups of men and women separately aged in their 30s, 40s, 50s and 60s. The association between GL and change in total cholesterol was only significant in the subgroup of men in their 30s (Oxlund and Heitmann, 2006). Results were also presented for women in three different BMI subgroups. Although the association between GL and change in total cholesterol was not significant for women with a BMI of 20-25kg/m² or 25-30kg/m², the association was significant for women with a BMI over 30kg/m² with an increase in GL corresponding with a decrease in change in total cholesterol (Oxlund and Heitmann, 2006). This study adjusted for a number of appropriate variables including age, smoking and BMI.

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

Summary of RCT data

Twelve trials provided data on the effects of high or low glycaemic index/load diets on total cholesterol. All trials used a parallel group design, which ranged in duration from eight to 36 weeks. The majority of papers did not state whether subjects and researchers were blinded to the nature of the intervention, although one paper was double blind (Jensen *et al.*, 2008) and two were open (Philippou *et al.*, 2009a; Maki *et al.*, 2007). The final number of subjects ranged from 18 to 203 and the average number of subjects in the 12 studies was 66 (median= 49). Two of the trials were particularly large with more than 100 subjects (McMillan-Price *et al.*, 2006; Sichieri *et al.*, 2007).

Trials were conducted in an array of countries such as the USA (4) (Wolever and Mehling, 2002; Ebbeling *et al.*, 2005; Maki *et al.*, 2007; Das *et al.*, 2007), the UK (3) (Philippou *et al.*, 2008; Philippou *et al.*, 2009b; Philippou *et al.*, 2009a), Spain (1) (Abete *et al.*, 2008), France (1) (Bellisle *et al.*, 2007), Denmark (1) (Jensen *et al.*, 2008), Brazil (1) (Sichieri *et al.*, 2007) and Australia (1) (McMillan-Price *et al.*, 2006).

All studies used adults as participants, who typically had an average age of between 32 and 57 years. Three studies of women only were identified (Bellisle *et al.*, 2007; Jensen *et al.*, 2008; Sichieri *et al.*, 2007), whereas one study used males (Philippou *et al.*, 2009a). Of those studies that reported mean BMI, subjects were mostly overweight or obese (Wolever and Mehling, 2002; Abete *et al.*, 2008; Jensen *et al.*, 2008; Sichieri *et al.*, 2007; McMillan-Price *et al.*, 2006; Maki *et al.*, 2007; Das *et al.*, 2007).

Four of the 12 trials did not prescribe an energy restriction goal (Jensen *et al.*, 2008; Philippou *et al.*, 2009b; Sichieri *et al.*, 2007; Wolever and Mehling, 2002). Body weight decreased in the majority of trials, but was unchanged in the studies by Philippou *et al.* (Philippou *et al.*, 2009b) and Sichieri *et al.* (Sichieri *et al.*, 2007). Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

Eleven studies were included in the meta-analysis comparing different glycaemic index/load intakes and changes in total cholesterol reported as mmol/L. The first follow up reported at the end of the intervention was used. This varied from eight weeks to 18 months.

One study by Wolever *et al.* (Wolever and Mehling, 2002) could not be included as no quantitative data were reported which could be incorporated into a meta-analysis. The authors reported the results of a parallel group trial with 34 obese subjects with impaired glucose tolerance who had been randomly assigned to one of three groups: a high-carbohydrate, high-GI diet; a high-carbohydrate, low-GI diet; or a low-carbohydrate, high-MUFA diet (Wolever and Mehling, 2002). All diets were *ad libitum* and were intended to be weight maintaining. There were no statistically significant differences in cholesterol within or between groups following the intervention.

The pooled estimate of 11 studies indicated that total cholesterol was 0.13mmol/L (95% CI 0.02 to 0.24) lower with consumption of a low glycaemic index diet. This was significantly different from zero ($p=0.02$). Overall heterogeneity denoted by I^2 was 35% (95% CI 0 to 67%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was evidence that lower glycaemic index diets are associated with lower levels of total cholesterol.

Figure 2.78 Forest plot for glycaemic index and load diets and total cholesterol (mmol/L)

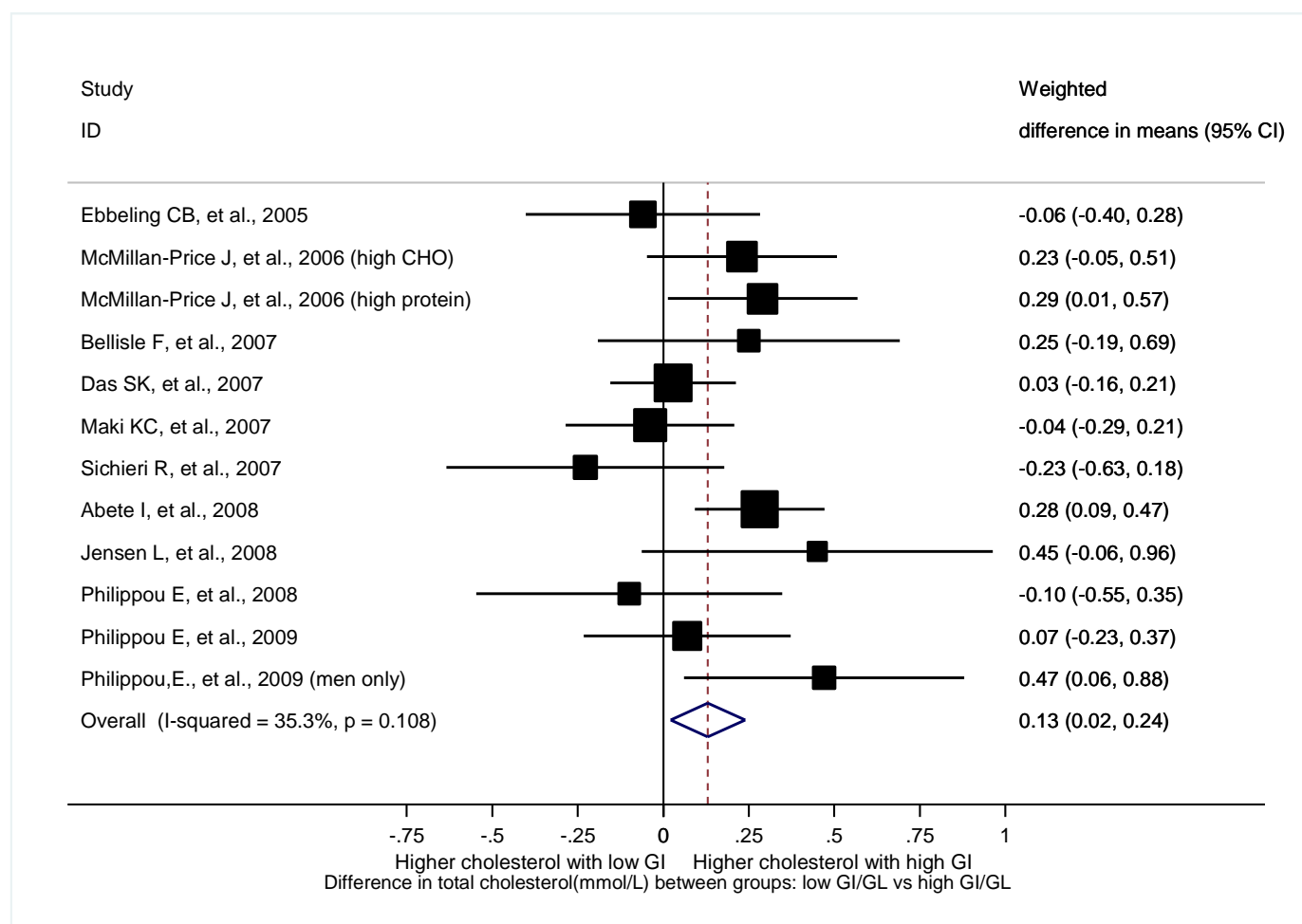


Figure 2.79 Contour-enhanced funnel plot for studies presenting data on dietary glycaemic index or load and total cholesterol

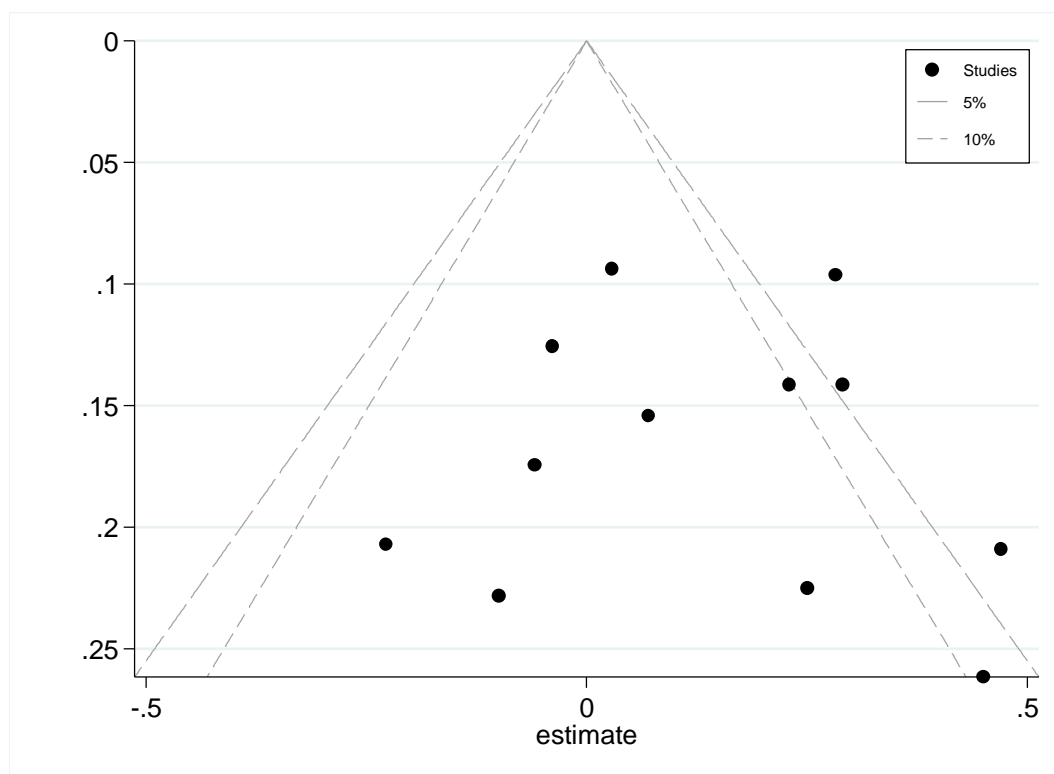


Table 2.149 Total cholesterol and glycaemic index and load: cohort study in adults

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 Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
(Oxlund and Heitmann, 2006) 13808 MONICA	Denmark, Primarily White, Cancer free, No CHD, No medication, No T2DM	30-60 (49) %M 51.3	3608	6 years	Dietary history	GI	Change in TC Fasting	Men	1 Unit/Total energy	0.0044	<0.05	Added sugar, age, alcohol, BMI, CHO, TC intake, education, energy intake, fat intake, physical activity, protein intake, Smoking
13895 MONICA								Women	1 Unit/Total energy	0.0009	NS	As above
13902 MONICA						GL	Change in TC Fasting	Men	1 Unit/Total energy	0.0729	NS	Added sugar, age, alcohol, BMI, TC intake, education, energy intake, dietary fibre, physical activity, smoking
13903 MONICA								Women	1 Unit/Total energy	-0.0645	NS	As above
14329 MONICA								Men, Age 30s	1 Unit/Total energy	0.1636 (0.0232, 0.304)		Added sugar, age, alcohol, Blood TC, BMI, education, energy intake, dietary fibre, physical activity, smoking, SBP
14330 MONICA								Men, Age 40s	1 Unit/Total energy	0.0927 (- 0.0175, 0.203)		As above
14331 MONICA								Men, Age 50s	1 Unit/Total energy	0.0219 (- 0.0976, 0.1414)		As above
14332 MONICA								Men, Age 60s	1 Unit/Total energy	-0.0489 (- 0.2104, 0.1126)		As above
14333 MONICA								Women, Age 30s	1 Unit/Total energy	-0.0264 (- 0.1748, 0.1221)		As above
14334 MONICA								Women, Age 40s	1 Unit/Total energy	-0.0587 (- 0.1699, 0.0526)		As above
14335 MONICA								Women, Age 50s	1 Unit/Total energy	-0.0909 (- 0.2209, 0.039)		As above
14336 MONICA								Women, Age 60s	1 Unit/Total energy	-0.1232 (- 0.3119, 0.0655)		As above
14337 MONICA								BMI 20-25, Women	1 Unit/Total energy	0.0264 (- 0.1118, 0.1647)		As above
14338 MONICA								BMI 25-30, Women	1 Unit/Total energy	-0.0554 (- 0.1646, 0.0537)		As above

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Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub-group Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14339 MONICA								BMI >30, Women	1 Unit/Total energy	-0.1374 (- 0.2658, - 0.0089)		As above

Table 2.150 Total cholesterol (TC) and glycaemic index and load: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Abete <i>et al.</i> , 2008) *15546	Higher GI diet	16/16	181 (SD 34)		-3.5% (SD 10.6%)	NS		TC	(mg/dL)	8 weeks	Decrease	unclear
	Lower GI diet	16/16	215 (SD 37)		-14.4% (SD 10.5%)	NS	0.01				Decrease	
(Bellisle <i>et al.</i> , 2007) *16051	Control	30/45	5.88 (SE 0.16)	5.5 (SE 0.19)				TC	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
	Low GI	35/51	5.64 (SE 0.19)	5.25 (SE 0.13)			NS				Decrease	
(Das <i>et al.</i> , 2007) 15229	Energy restricted high GL diet	15/17	168.4 (SD 25.3)		-11.1% (SD 8.3%)			TC	Fasting plasma (mg/dL)	6 months	Decrease	No bias
	Energy restricted low GL diet	14/17	176.7 (SD 26.7)		-13.4% (SD 12.1%)		NS				Decrease	
*15233	Energy restricted high GL diet	15/17	168.4 (SD 25.3)		-4.2% (SD 9.3%)			TC	Fasting plasma (mg/dL)	1 year	Decrease	No bias
	Energy restricted low GL diet	14/17	176.7 (SD 26.7)		-5.3% (SD 10.5%)		NS				Decrease	
(Ebbeling <i>et al.</i> , 2005) 15421	Low fat diet	12/17	186 (SE 9)		-2.1% (CI -9.2, 5.5)		NS	TC	Fasting (mg/dL)	6 months	Decrease	unclear
	Low GI diet	11/17	191.2 (SE 9.4)		-9.9% (CI -16.7, -2.5)						Decrease	
*15476	Low fat diet	12/17	186 (SE 9)		-6.2%(CI -15, 3.5)		NS	TC	Fasting (mg/dL)	1 year	Decrease	unclear
	Low GI diet	11/17	191.2 (SE 9.4)		-8.5% (CI -17.4, 1.5)						Decrease	
(Jensen <i>et al.</i> , 2008) *15032	High GI diet	22/26	4.79 (SE 0.22)	4.7 (SE 0.18)		NS		TC	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
	Low GI diet	22/29	4.58 (SE 0.22)	4.25 (SE 0.19)		NS	0.06				Decrease	
(Maki <i>et al.</i> , 2007) 17278	Ad libitum low GL diet	39/43	199.3 (SE 4.5)		-12.2 (SE 2.7)		NS	TC	Fasting (mg/dL)	12 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	206.5 (SE 6.5)		-8.3 (SE 3.7)						Decrease	

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
*17279	Ad libitum low GL diet	39/43	199.3 (SE 4.5)		-1.5 (SE 3.9)		NS	TC	Fasting (mg/dL)	36 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	206.5 (SE 6.5)		-3 (SE 2.9)						Decrease	
(McMillan-Price <i>et al.</i> , 2006) *16220	High CHO, high GI diet	32/32	4.79 (SE 0.19)		0.05 (SE 0.10)		0.02	TC	Fasting (mmol/L)	12 weeks	Decrease	unclear
	High CHO, low GI diet	32/32	4.71 (SE 0.19)		-0.18 (SE 0.10)		p=0.01				Decrease	
	High protein, high GI diet	32/32	5.15 (SE 0.18)		0.24 (SE 0.10)						Decrease	
	High protein, low GI diet	33/33	4.83 (SE 0.14)		-0.05 (SE 0.10)						Decrease	
17279	Ad libitum low GL diet	39/43	199.3 (SE 4.5)		-1.5 (SE 3.9)			TC	Fasting (mg/dL)	36 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	206.5 (SE 6.5)		-3 (SE 2.9)						Decrease	
	Ad libitum low GL diet	39/43	199.3 (SE 4.5)		-1.5 (SE 3.9)						Decrease	
	Low fat, energy restricted	38/43	206.5 (SE 6.5)		-3 (SE 2.9)						Decrease	
(Philippou <i>et al.</i> , 2008) *16853	High GI	7/9	5.3	5.2	-0.1 (CI -0.6, 0.2)	NS		TC	Fasting (mmol/L)	12 weeks	Decrease	unclear
	Low GI	6/9	5.7	5.5	0.0 (CI -0.2, 0.2)	NS	NS				Decrease	
(Philippou <i>et al.</i> , 2009a) *14659	High GI	16/28	5.19 (SD 0.91)	5.21 (SD 1.2)	0.02 (SD 0.56)	NS		TC	Fasting (mmol/L)	6 months	Decrease	unclear
	Low GI	22/28	5.61 (SD 0.79)	5.16 (SD 0.95)	-0.45 (SD 0.62)	<0.01	<0.05				Decrease	
(Philippou <i>et al.</i> , 2009b) 15141	High GI	19/19	4.87 (SD 0.67)	5.41 (SD 0.84)	0.54 (SD 0.45)			TC	Fasting plasma (mmol/L)	2 months	No change	unclear
	Low GI	22/23	4.67 (SD 0.93)	5.24 (SD 0.97)	0.54 (SD 0.37)		NS				No change	
*15142	High GI	19/19	4.87 (SD 0.67)	5.26 (SD 0.80)	0.46 (SD 0.34)			TC	Fasting plasma (mmol/L)	4 months	No change	unclear
	Low GI	22/23	4.67 (SD 0.93)	5.05 (SD 0.91)	0.39 (SD 0.58)		0.4				No change	
(Sichieri <i>et al.</i> , 2007) 15792	High GI/GL diet	60/102	194.1 (SD 37.0)	200.9 (SD 43.2)			NS	TC	Fasting plasma (mg/dL)	3 months	No change	unclear
	Low GI/GL diet	73/101	188.8 (SD 34.7)	189.1 (SD 33.5)							No change	
15793	High GI/GL diet	60/102	194.1 (SD	186.5 (SD			NS	TC	Fasting	6 months	No	unclear

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
			37.0)	37.8)					plasma (mg/dL)		change	
	Low GI/GL diet	59/101	188.8 (SD 34.7)	184.1 (SD 34.8)							No change	
15794	High GI/GL diet	42/102	194.1 (SD 37.0)	200.7 (SD 37.1)			NS	TC	Fasting plasma (mg/dL)	1 year	No change	unclear
	Low GI/GL diet	41/101	188.8 (SD 34.7)	185.0 (SD 35.4)							No change	
*15795	High GI/GL diet	53/102	194.1 (SD 37.0)	208.7 (SD 41.6)		0.09	NS	TC	Fasting plasma (mg/dL)	18 months	No change	unclear
	Low GI/GL diet	64/101	188.8 (SD 34.7)	199.9 (SD 40.9)		0.0001					No change	
(Wolever and Mehling, 2002) 17009	High carbohydrate, high GI	11/11					NS	TC	Fasting	16 weeks	Decrease	unclear
	High carbohydrate, low GI	13/13					NS				Decrease	
	Low carbohydrate, high MUFA	11/11					NS				Increase	

*This result was used in the meta-analysis of glycaemic index and load and total cholesterol

HDL cholesterol and glycaemic index and load

Summary of cohort results

Data were extracted from one Danish study (the MONICA study) which provided evidence concerning the association between the GI and GL of the diet and change in HDL cholesterol (Oxlund and Heitmann, 2006). No association was observed between either GI or GL and the change in HDL cholesterol in either men or women over the six year follow up. This study adjusted for a number of different potential confounders including age, BMI, smoking, physical activity and dietary variables.

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

Summary of RCT data

Fourteen trials provided data on the effects of high or low glycaemic index/load diets on HDL cholesterol (Wolever and Mehling, 2003;Ebbeling *et al.*, 2007;Abete *et al.*, 2008;Bellisle *et al.*, 2007;Das *et al.*, 2007;Jensen *et al.*, 2008;Philippou *et al.*, 2008;Philippou *et al.*, 2009b;Philippou *et al.*, 2009a;Sichieri *et al.*, 2007;Pereira *et al.*, 2004;Ebbeling *et al.*, 2005;McMillan-Price *et al.*, 2006;Maki *et al.*, 2007).

All fourteen studies were similar in that they implemented a parallel group design. Of these, one was single blind (Ebbeling *et al.*, 2007), one double blind (Jensen *et al.*, 2008) and two were open (Philippou *et al.*, 2009a;Maki *et al.*, 2007). Ten studies were unclear, with regard to blinding. Trials were conducted in a range of countries such as the UK (3), Spain (1), France (1), Denmark (1) and Brazil (1), although the majority were carried out in the USA (6).

Adults were used as participants in all of the included studies. Three were identified that studied females only (Bellisle *et al.*, 2007;Jensen *et al.*, 2008;Sichieri *et al.*, 2007) and one that studied males only (Philippou *et al.*, 2009a). The remainder were mixed gender.

Excluding two studies that had more than 100 participants in each, sample sizes were relatively small and ranged from 18 to 96 participants (mean=67; median=51). Of the seven studies that reported BMI, participants generally fell into the overweight or obese category (Wolever and Mehling, 2003;Abete *et al.*, 2008;Das *et al.*, 2007;Jensen *et al.*, 2008;Sichieri *et al.*, 2007;McMillan-Price *et al.*, 2006;Maki *et al.*, 2007).

Body weights decreased in all trials other than in Sichieri *et al.* and Philippou *et al.* (Sichieri *et al.*, 2007; Philippou *et al.*, 2009b) in which the authors reported that body weights were unchanged. A slight increase in weight was also noted in the low carbohydrate, high MUFA group in the study by Wolever *et al.* (Wolever and Mehling, 2003). Changes in HDL cholesterol therefore may not be solely attributable to the dietary intervention.

Fourteen studies were included in the meta-analysis comparing different glycaemic index and changes in HDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from eight weeks to 18 months. The pooled estimate indicated that LDL cholesterol was 0.0mmol/L (95% CI -0.06 to 0.06) higher with consumption of a lower glycaemic index diet. This was not significantly different from zero ($p=1$). Overall heterogeneity denoted by I^2 was 67% (95% CI 43 to 81%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was no evidence that HDL was associated with differences in glycaemic index.

Figure 2.80 Forest plot for glycaemic index and load diets and HDL cholesterol (mmol/L)

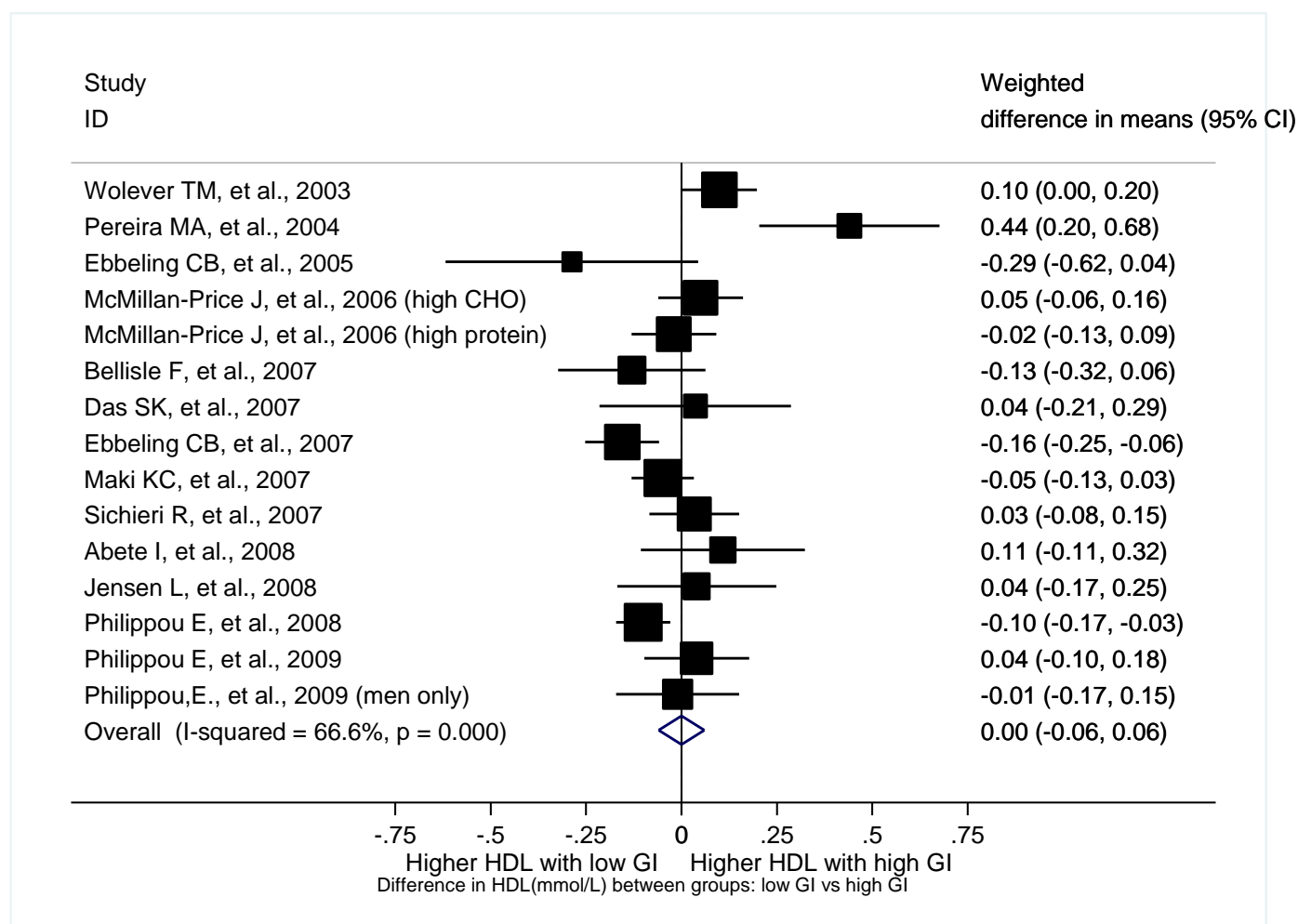


Figure 2.81 Contour-enhanced funnel plot for studies presenting data on dietary glycaemic index or load and HDL cholesterol

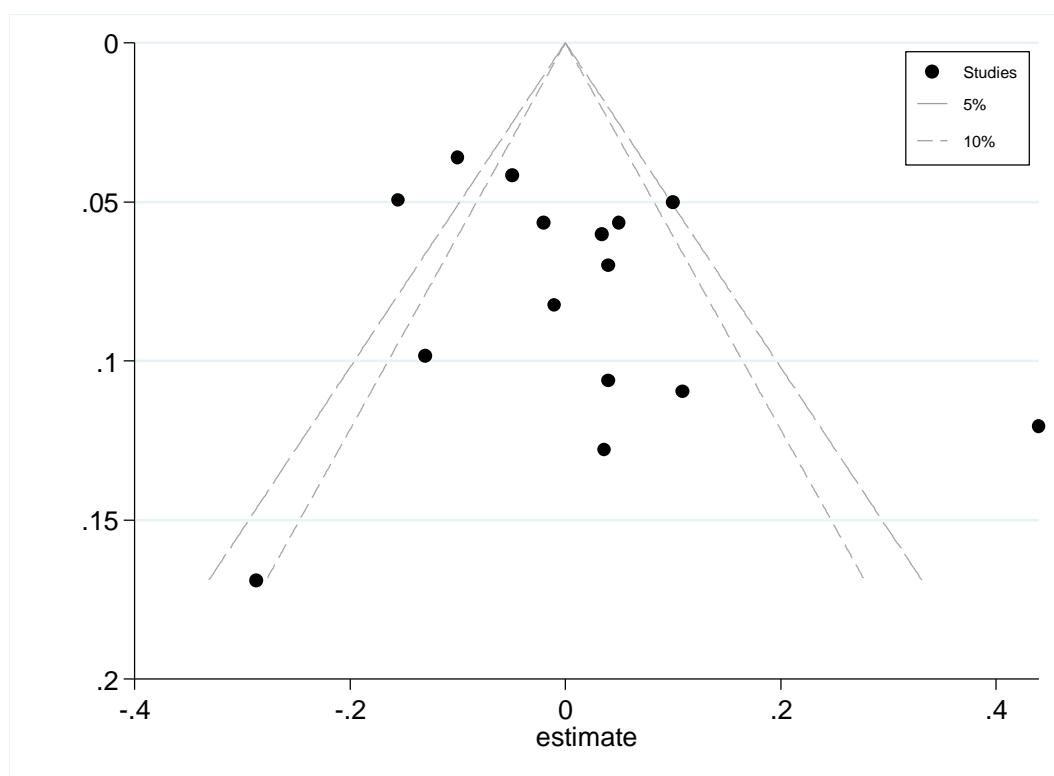


Table 2.151 HDL cholesterol and glycaemic index and load: cohort study in adults

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 Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
(Oxlund and Heitmann, 2006) 13900 MONICA	Denmark, Primarily White, Cancer free, No CHD, No medication, No T2DM	30-60 (49) %M 51.3	3608	6 years	Dietary history	GI	Change in HDL-C Fasting	Men	1 Unit/Total energy	0.0038	NS	Added sugar, age, alcohol, BMI, carbohydrate intake, coffee, eating frequency, education, energy intake, fat, dietary fibre, HDL-C, physical activity, protein intake, smoking, SBP
13901 MONICA								Women	1 Unit/Total energy	0.0007	NS	As above
13908 MONICA						GL	Change in HDL-C Fasting	Men	1 Unit/Total energy	0.0131	NS	Added sugar, age, alcohol, BMI, coffee, eating frequency, education, energy intake, dietary fibre, HDL-C, physical activity, smoking, SBP
13909 MONICA								Women	1 Unit/Total energy	-0.0433	NS	As above

Table 2.152 HDL cholesterol and glycaemic index and load: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Abete <i>et al.</i> , 2008) *15548	Higher GI diet	16/16	51 (SD 9)		-5.5% (SD 14.9%)	NS		HDL-C	(mg/dL)	8 weeks	Decrease	unclear
	Lower GI diet	16/16	50 (SD 12)		-9.7% (SD 8.1%)	NS	0.348				Decrease	
(Bellisle <i>et al.</i> , 2007) *16053	Control	30/45	1.81 (SE 0.09)	1.62 (SE 0.08)				HDL-C	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
	Low GI	35/51	1.9 (SE 0.07)	1.75 (SE 0.06)			NS				Decrease	
(Das <i>et al.</i> ,	Energy restricted	15/17	55.4 (SD		-2.8% (SD 10.6%)			HDL-C	Fasting	6 months	Decrease	No bias

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
2007) 15234	high GL diet		8.5)						plasma (mg/dL)			
	Energy restricted low GL diet	14/17	51.0 (SD 11.5)		-3.1% (SD 19.1%)		NS				Decrease	
*15236	Energy restricted high GL diet	15/17	55.4 (SD 8.5)		13.3% (SD 16.2%)			HDL-C	Fasting plasma (mg/dL)	1 year	Decrease	No bias
	Energy restricted low GL diet	14/17	51.0 (SD 11.5)		11.9% (SD 10.2%)		NS				Decrease	
(Ebbeling <i>et al.</i> , 2005) 15493	Low fat diet	12/17	53.8 (SE 2.7)		-0.3% (CI -8.1, 8.2)		NS	HDL-C	Fasting (mg/dL)	6 months	Decrease	unclear
	Low GI diet	11/17	49 (SE 2.9)		2.3% (CI -6, 11.3)						Decrease	
*15508	Low fat diet	12/17	53.8 (SE 2.7)		1.1% (CI -6.9, 9.8)		NS	HDL-C	Fasting (mg/dL)	1 year	Decrease	unclear
	Low GI diet	11/17	49 (SE 2.9)		12.2% (CI 2.9, 22.3)						Decrease	
(Ebbeling <i>et al.</i> , 2007) *15451	Low fat diet	37/37			-4.4 (SE 1.3)			HDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
	Low GL diet	ITT: 36/36			1.6 (SE 1.4)		0.02				Decrease	
15452	Low fat diet	37/37			-8.2 (SE 1.5)			HDL-C	Fasting plasma (mg/dL)	18 months	Decrease	No bias
	Low GL diet	ITT: 36/36			-3.7 (SE 1.5)		0.3				Decrease	
(Jensen <i>et al.</i> , 2008) *15033	High GI diet	22/26	1.63 (SE 0.07)	1.52 (SE 0.07)		NS		HDL-C	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
	Low GI diet	22/29	1.53 (SE 0.07)	1.48 (SE 0.08)		NS	0.56				Decrease	
(Maki <i>et al.</i> , 2007) 17282	Ad libitum low GL diet	39/43	56.2 (SE 2)		-0.2 (SE 1.2)		NS	HDL-C	Fasting (mg/dL)	12 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	56.4 (SE 2)		-2.1 (SE 0.9)						Decrease	
*17283	Ad libitum low GL diet	39/43	56.2 (SE 2)		3.8 (SE 1.4)		0.037	HDL-C	Fasting (mg/dL)	36 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	56.4 (SE 2)		1.9 (SE 0.8)						Decrease	
(McMillan- Price <i>et al.</i> , 2006) *16221	High CHO, high GI diet	32/32	1.29 (SE 0.07)		0.08 (SE 0.04)		0.82	HDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
	High CHO, low GI diet	32/32	1.17 (SE 0.05)		0.03 (SE 0.04)						Decrease	

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	High protein, high GI diet	32/32	1.16 (SE 0.05)		0.05 (SE 0.04)						Decrease	
	High protein, low GI diet	33/33	1.36 (SE 0.08)		0.07 (SE 0.04)						Decrease	
(Pereira <i>et al.</i> , 2004) *14580	Hypoenergetic low fat diet	11/23	49.4 (SE 3.61)	44.1 (SE 2.41)	-8.1% (SE 3.49%)			HDL-C	Fasting serum (mg/dL)	67 days	Decrease	unclear
	Hypoenergetic low GL diet	14/23	46.9 (SE 3.2)	42.2 (SE 2.14)	-8.9% (SE 3.09%)		0.87				Decrease	
(Philippou <i>et al.</i> , 2008) *16856	High GI	7/9	1.3	1.3	0.0 (CI 0, 0.1)	NS		HDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
	Low GI	6/9	1.5	1.4	0.1 (CI 0, 0.1)	NS	NS				Decrease	
(Philippou <i>et al.</i> , 2009a) *14661	High GI	16/28	1.1	1.07	-0.01 (CI -0.18, 0.12)	NS		HDL-C	Fasting (mmol/L)	6 months	Decrease	unclear
	Low GI	22/28	1.1	1.12	0 (CI -0.08, 0.04)	NS	NS				Decrease	
(Philippou <i>et al.</i> , 2009b) 15145	High GI	19/19	1.16 (SD 0.16)	1.34 (SD 0.23)	0.15 (SD 0.17)			HDL-C	Fasting plasma, (mmol/L)	2 months	No change	unclear
	Low GI	21/23	1.26 (SD 0.21)	1.41 (SD 0.19)	0.15 (SD 0.15)		NS				No change	
*15146	High GI	19/19	1.16 (SD 0.16)	1.34 (SD 0.28)	0.17 (SD 0.23)			HDL-C	Fasting plasma, (mmol/L)	4 months	No change	unclear
	Low GI	23/23	1.26 (SD 0.21)	1.39 (SD 0.21)	0.13 (SD 0.20)		0.3				No change	
(Sichieri <i>et al.</i> , 2007) 15804	High GI/GL diet	60/102	43.2 (SD 15.9)	44.7 (SD 10.2)			NS	HDL-C	Fasting plasma (mg/dL)	3 months	No change	unclear
	Low GI/GL diet	73/101	43.0 (SD 15.4)	46.9 (SD 11.4)							No change	
15805	High GI/GL diet	60/102	43.2 (SD 15.9)	51.6 (SD 12.3)			NS	HDL-C	Fasting plasma (mg/dL)	6 months	No change	unclear
	Low GI/GL diet	59/101	43.0 (SD 15.4)	51.2 (SD 12.7)							No change	
15806	High GI/GL diet	42/102	43.2 (SD 15.9)	55.5 (SD 15.5)			NS	HDL-C	Fasting plasma (mg/dL)	1 year	No change	unclear
	Low GI/GL diet	41/101	43.0 (SD 15.4)	54.4 (SD 13.6)							No change	
*15807	High GI/GL diet	53/102	43.2 (SD 15.9)	52.5 (SD 12.4)		<0.001	NS	HDL-C	Fasting plasma	18 months	No change	unclear

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	Low GI/GL diet	64/101	43.0 (SD 15.4)	51.2 (SD 11.5)		<0.001			(mg/dL)		No change	
(Wolever and Mehling, 2003) *17136	High carbohydrate, high GI	11/13			0.09 (SE 0.04)		NS	HDL-C	Fasting serum (mmol/L)	4 months	Decrease	unclear
	High carbohydrate, low GI	13/13			-0.01 (SE 0.03)		NS				Decrease	
	Low carbohydrate, high MUFA	11/12			0.05 (SE 0.03)		NS				Increase	

*This result was used in the meta-analysis of glycaemic index and load and HDL cholesterol

LDL cholesterol and glycaemic index and load

Summary of cohort results

Data were extracted from the MONICA study which provided evidence concerning the association between the GI and GL of the diet and change in LDL cholesterol (Oxlund and Heitmann, 2006). This study reported no association between GI and the change in LDL cholesterol in either men or women over the six year follow up. This lack of association was seen in all subgroups of men aged in the 30s, 40s, 50s and 60s and in women aged in their 40s, 50s and 60s. There was a small but significant association seen in women in their 30s where an increase in 1 unit/total energy in GI was associated with a small increase in LDL cholesterol. An increase in GL was associated with an increase in LDL cholesterol in men and a decrease in LDL cholesterol in women with a BMI over 30kg/m². No significant association was seen in the subgroup of women with a BMI between 20 and 25kg/m² or between 25 and 30kg/m², or women overall. This study adjusted for a number of different potential confounders including age, BMI, smoking, physical activity and dietary variables.

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

Summary of RCT data

Fourteen trials provided data on the effects of high or low glycaemic index/load diets on LDL cholesterol (Wolever and Mehling, 2002;Ebbeling *et al.*, 2007;Abete *et al.*, 2008;Bellisle *et al.*, 2007;Das *et al.*, 2007;Jensen *et al.*, 2008;Philippou *et al.*, 2008;Philippou *et al.*, 2009b;Philippou *et al.*, 2009a;Sichieri *et al.*, 2007;Pereira *et al.*, 2004;Ebbeling *et al.*, 2005;McMillan-Price *et al.*, 2006;Maki *et al.*, 2007).

These trials also provided data on HDL cholesterol; a summary of these randomised controlled trials therefore can be found in the section on HDL cholesterol and glycaemic index and load.

Body weight decreased in the majority of trials, but was unaffected in the studies by Philippou *et al.* (Philippou *et al.*, 2009b) and Sichieri *et al.* (Sichieri *et al.*, 2007). A slight increase in weight was noted in the low carbohydrate, high MUFA group in one study (Wolever and Mehling, 2002). Any changes in LDL cholesterol therefore may not be attributed solely to the glycaemic index/load of the diets.

Thirteen studies were included in the meta-analysis comparing different Glycaemic index and changes in LDL cholesterol reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from eight weeks to 18 months.

In Wolever and Mehling (Wolever and Mehling, 2002), no quantitative data were included which could be incorporated into the meta-analysis. The authors reported results of a parallel group trial with 34 obese subjects with impaired glucose tolerance (IGT) who had been randomly assigned to one of three groups: a high-carbohydrate, high-GI diet; a high-carbohydrate, low-GI diet; or a low-carbohydrate, high-MUFA diet (Wolever and Mehling, 2002). All diets were *ad libitum* and were intended to be weight maintaining. Comparison of LDL cholesterol did not show statistically significant differences within or between groups following the intervention.

The pooled estimate indicated that LDL cholesterol was 0.14mmol/L (95% CI 0.06 to 0.22) lower with consumption of a lower glycaemic index diet. This was significantly different from zero ($p=0.001$). Overall heterogeneity denoted by I^2 was 0% (95% CI 0 to 53%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. There was strong evidence that a low glycaemic index diet is associated with lower levels of LDL cholesterol.

Figure 2.82 Forest plot for glycaemic index and load diets and LDL cholesterol (mmol/L)

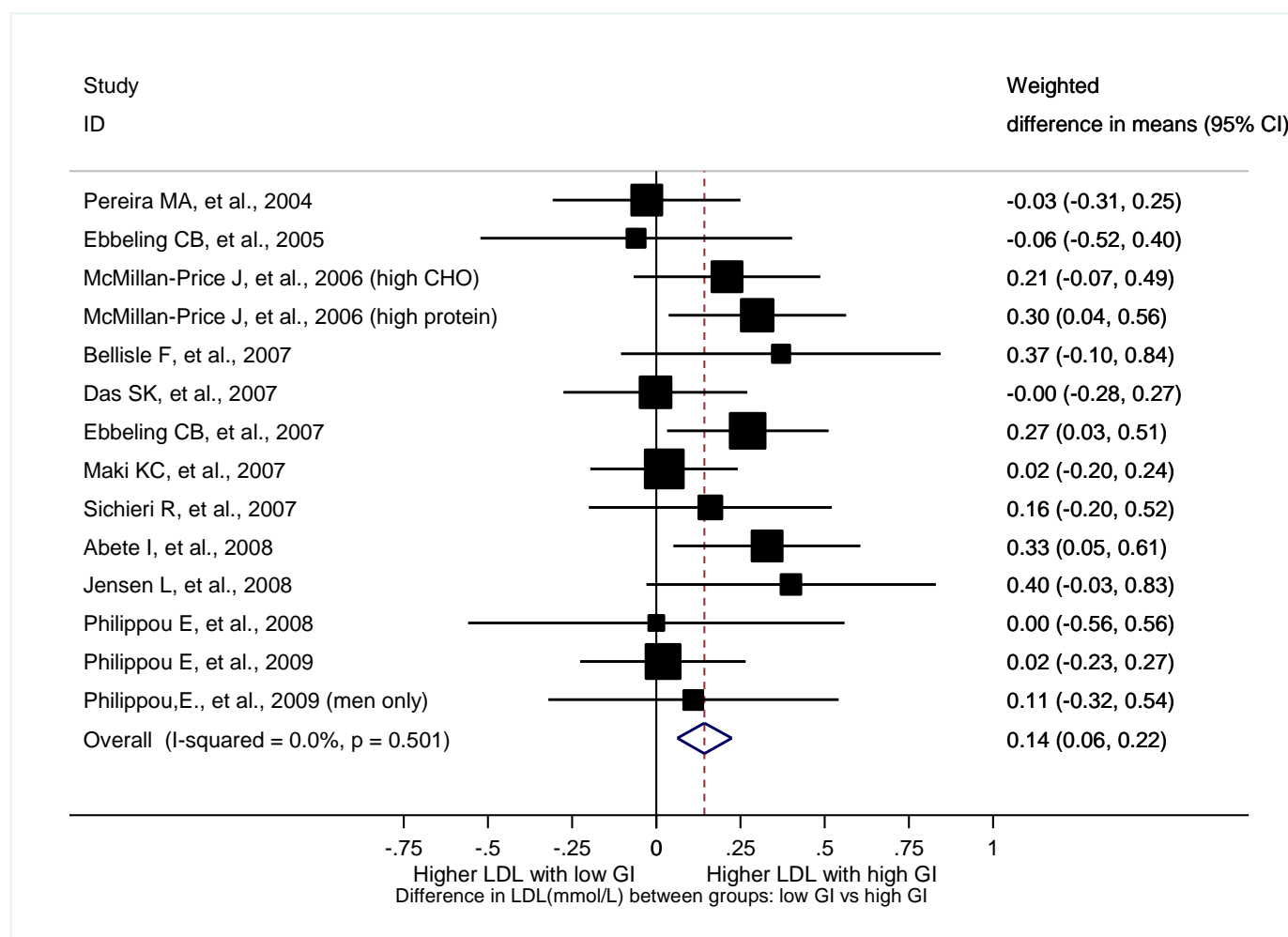


Figure 2.83 Contour-enhanced funnel plot for studies presenting data on dietary glycaemic index or load and LDL cholesterol

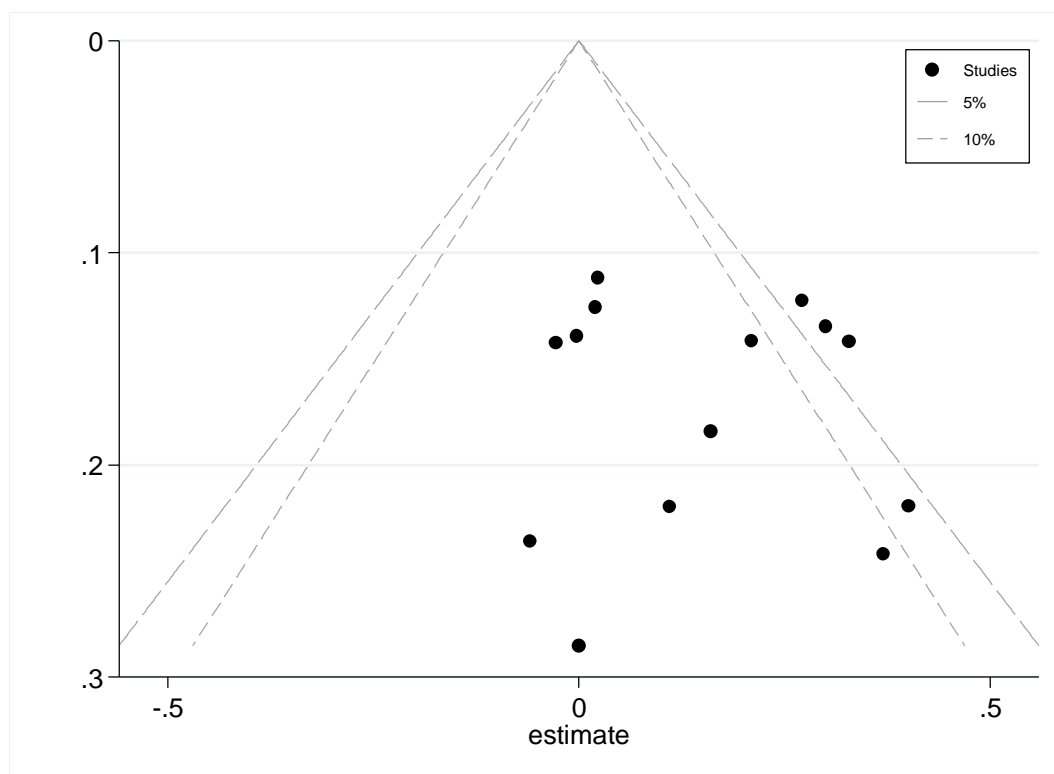


Table 2.153 LDL cholesterol and glycaemic index and load: cohort study in adults

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 Details	Exposure Units	Beta coefficient (SE)/(CI)	P	Adjustments
(Oxlund and Heitmann, 2006) 13896 MONICA	Denmark, Primarily White, Cancer free, No CHD, No medication, No T2DM	30-60 (49) %M 51.3	3608	6 years	Dietary history	GI	Change in LDL-C Fasting	Men	1 Unit/Total energy	0.0038	NS	Added sugar, age, alcohol, BMI, carbohydrate intake, coffee, eating frequency, education, energy intake, fat intake, dietary fibre, LDL-C, physical activity, dietary protein, smoking, SBP
13897 MONICA								Women	1 Unit/Total energy	0.0005	NS	As above
14321 MONICA								Men, Age 30s	1 Unit/Total energy	0.0026 (- 0.0051, 0.0104)		Added sugar, age, alcohol, baseline LDL, BMI, dietary carbohydrate, coffee, education, energy intake, fat intake, dietary fibre, physical activity, protein intake, smoking, SBP
14322 MONICA								Men, Age 40s	1 Unit/Total energy	0.0034 (- 0.0018, 0.0087)		As above
14323 MONICA								Men, Age 40s	1 Unit/Total energy	0.0042 (- 0.0011, 0.0096)		As above
14324 MONICA								Men, Age 60s	1 Unit/Total energy	0.005 (- 0.0028, 0.0128)		As above
14325 MONICA								Women, Age 30s	1 Unit/Total energy	0.0091 (0.0005, 0.0176)		As above
14326 MONICA								Women, Age 40s	1 Unit/Total energy	0.0033 (- 0.0023, 0.009)		As above
14327 MONICA								Men, Age 50s	1 Unit/Total energy	-0.0024 (- 0.008, 0.0033)		As above
14328 MONICA								Women, Age 60s	1 Unit/Total energy	-0.008 (- 0.0165, 0.0004)		As above
13904 MONICA						GL	Change in LDL-C Fasting	Men	1 Unit/Total energy	0.1554	<0.05	Added sugar, age, alcohol, BMI, coffee, education, energy intake, dietary fibre, LDL-C, physical activity, smoking, SBP
13905 MONICA								Women	1 Unit/Total energy	-0.0915	NS	As above

Result ID/ Reference/ Cohort Name	Country, Ethnicity, Inclusion criteria	Age range (mean) %Male	(Cases)/ Total	Follow Up (% loss)	Diet Assessment	Exposure	Outcome/ Assessment Details	Sub- group Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
14340 MONICA								BMI 20- 25, Women	1 Unit/Total energy	0.0467 (- 0.156, 0.2494)		Added sugar, age, alcohol, baseline LDL, BMI, coffee, education, energy intake, dietary fibre, physical activity, smoking, SBP
14341 MONICA								BMI 25- 30, Women	1 Unit/Total energy	-0.075 (- 0.1809, 0.0853)		As above
14342 MONICA								BMI >30, Women	1 Unit/Total energy	-0.1966 (- 0.3836, - 0.01)		As above

Table 2.154 LDL Cholesterol and glycaemic index and load: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Abete <i>et al.</i> , 2008) *15547	Higher GI diet	16/16	112 (SD 29)		-3.2% (SD 14.3%)	NS		LDL-C	(mg/dL)	8 weeks	Decrease	unclear
	Lower GI diet	16/16	136 (SD 5)		-15.9% (SD 16.6%)	NS	0.037				Decrease	
(Bellisle <i>et al.</i> , 2007) *16054	Control	30/45	3.91 (SE 0.14)	3.67 (SE 0.19)				LDL-C	Derived by calculation Fasting, Plasma (mmol/L)	12 weeks	Decrease	unclear
	Low GI	35/51	3.56 (SE 0.19)	3.3 (SE 0.15)			NS				Decrease	
(Das <i>et al.</i> , 2007) 15239	Energy restricted high GL diet	15/17	96.9 (SD 21.5)		-13.2% (SD 11%)			LDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
	Energy restricted low GL diet	14/17	107.6 (SD 24.2)		-13.4% (SD 18.2%)		NS				Decrease	
*15240	Energy restricted high GL diet	15/17	96.9 (SD 21.5)		-7.1% (SD 11.3%)			LDL-C	Fasting plasma (mg/dL)	1 year	Decrease	No bias
	Energy restricted low GL diet	14/17	107.6 (SD 24.2)		-7% (SD 17.5%)		NS				Decrease	
(Ebbeling <i>et al.</i> , 2005) 15491	Low fat diet	12/17	109.4 (SE 7.6)		-2.6% (CI -12.3, 8.2)		NS	LDL-C	Fasting (mg/dL)	6 months	Decrease	unclear
	Low GI diet	11/17	113.1 (SE 6.1)		-9.1% (CI -18.6, 1.4)						Decrease	
*15492	Low fat diet	12/17	109.4 (SE		-7.4% (CI -19.1, 6)		NS	LDL-C	Fasting	1 year	Decrease	unclear

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
			7.6)						(mg/dL)			
	Low GI diet	11/17	113.1 (SE 6.1)		-9.7% (CI -21.6, 3.9)						Decrease	
(Ebbeling <i>et al.</i> , 2007) *15449	Low fat diet	37/37			-16.3 (SE 3.3)			LDL-C	Fasting plasma (mg/dL)	6 months	Decrease	No bias
	Low GL diet	ITT: 36/36			-5.8 (SE 3.4)		0.3				Decrease	
15450	Low fat diet	37/37			-10.6 (SE 3.3)			LDL-C	Fasting plasma (mg/dL)	18 months	Decrease	No bias
	Low GL diet	ITT: 36/36			-0.3 (SE 3.4)		0.3				Decrease	
(Jensen <i>et al.</i> , 2008) *15034	High GI diet	22/26	2.63 (SE 0.18)	2.68 (SE 0.15)		NS		LDL-C	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
	Low GI diet	22/29	2.5 (SE 0.18)	2.28 (SE 0.16)		NS	0.03				Decrease	
(Maki <i>et al.</i> , 2007) 17280	Ad libitum low GL diet	39/43	117.6 (SE 4.2)		-7 (SE 2.2)		NS	LDL-C	Derived by calculation Fasting, (mg/dL)	12 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	123.4 (SE 5.7)		-3.6 (SE 2.9)						Decrease	
*17281	Ad libitum low GL diet	39/43	117.6 (SE 4.2)		-2.8 (SE 3.2)		NS	LDL-C	Derived by calculation Fasting, (mg/dL)	36 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	123.4 (SE 5.7)		-1.9 (SE 2.9)						Decrease	
(McMillan- Price <i>et al.</i> , 2006) *16222	High CHO, high GI diet	32/32	2.87 (SE 0.16)		0.04 (SE 0.10)			LDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
	High CHO, low GI diet	32/32	2.9 (SE 0.14)		-0.17 (SE 0.10)						Decrease	
	High protein, high GI diet	32/32	3.33 (SE 0.15)		0.26 (SE 0.10)		0.01 (compared with high CHO, low Gi diet)				Decrease	
	High protein, low GI diet	33/33	2.89 (SE 0.14)		-0.04 (SE 0.09)						Decrease	
(Pereira <i>et al.</i> , 2004) *14581	Hypoenergetic low fat diet	11/23	124.3 (SE 9.86)	104.6 (SE 9.73)	-15% (SE 4.12%)			LDL-C	Derived by calculation Fasting, Serum (mg/dL)	67 days	Decrease	unclear

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	Hypoenergetic low GL diet	14/23	138.7 (SE 9.75)	115.9 (SE 8.63)	-16.1% (SE 3.65%)		0.84				Decrease	
(Philippou <i>et al.</i> , 2008) *16855	High GI	7/9	3.4	3.5	-0.2 (CI -0.5, 0.5)	NS		LDL-C	Fasting (mmol/L)	12 weeks	Decrease	unclear
	Low GI	6/9	3.7	3.7	-0.2 (CI -0.2, 0.3)	NS	NS				Decrease	
(Philippou <i>et al.</i> , 2009a) *14660	High GI	16/28	3.34 (SD 0.8)	3.23 (SD 1.33)	-0.11 (SD 0.73)	NS		LDL-C	Fasting (mmol/L)	6 months	Decrease	unclear
	Low GI	22/28	3.62 (SD 0.63)	3.4 (SD 0.75)	-0.22 (SD 0.49)	<0.05	NS				Decrease	
(Philippou <i>et al.</i> , 2009b) 15143	High GI	19/19	3.21 (SD 0.58)	3.52 (SD 0.78)	0.31 (SD 0.36)			LDL-C	Fasting plasma (mmol/L)	2 months	No change	unclear
	Low GI	21/23	3.01 (SD 0.81)	3.34 (SD 0.9)	0.33 (SD 0.36)		NS				No change	
*15144	High GI	19/19	3.21 (SD 0.58)	3.42 (SD 0.73)	0.26 (SD 0.31)			LDL-C	Fasting plasma (mmol/L)	4 months	No change	unclear
	Low GI	23/23	3.01 (SD 0.81)	3.24 (SD 0.9)	0.24 (SD 0.45)		0.3				No change	
(Sichieri <i>et al.</i> , 2007) 15800	High GI/GL diet	60/102	133.1 (SD 36.8)	138.0 (SD 41.3)			NS	LDL-C	Fasting plasma (mg/dL)	3 months	No change	unclear
	Low GI/GL diet	59/101	127.7 (SD 32.8)	124.8(SD 33.6)							No change	
15801	High GI/GL diet	60/102	133.1 (SD 36.8)	120.7 (SD 36.6)			NS	LDL-C	Fasting plasma (mg/dL)	6 months	No change	unclear
	Low GI/GL diet	73/101	127.7 (SD 32.8)	117.3 (SD 35.2)							No change	
15802	High GI/GL diet	42/102	133.1 (SD 36.8)	122.6 (SD 37.2)			NS	LDL-C	Fasting plasma (mg/dL)	1 year	No change	unclear
	Low GI/GL diet	41/101	127.7 (SD 32.8)	113.0 (SD 33.6)							No change	
*15803	High GI/GL diet	53/102	133.1 (SD 36.8)	132.0 (SD 34.8)		0.001	NS	LDL-C	Fasting plasma (mg/dL)	18 months	No change	unclear
	Low GI/GL diet	64/101	127.7 (SD 32.8)	125.8 (SD 38.4)		<0.001					No change	
(Wolever and Mehling, 2002)	High carbohydrate, high GI	11/11					NS	LDL-C	Fasting	16 weeks	Decrease	unclear
	High carbohydrate,	13/13					NS				Decrease	

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
17010	low GI Low carbohydrate, high MUFA	11/11					NS				Increase	

*This result was used in the meta-analysis for glycaemic index and load and LDL cholesterol

Triacylglycerol and glycaemic index and load

Summary of cohort results

Data were extracted from one study which provided evidence concerning the association between the GI and GL of the diet and change in TAG levels (Oxlund and Heitmann, 2006). This study found no evidence of an association between either GI or GL and the change in TAG in either men or women over the six year follow up. The MONICA study adjusted for a number of potential confounders including age, BMI, smoking, physical activity and dietary variables.

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

Summary of RCT data

Fifteen trials provided data on the effects of high or low glycaemic index/load diets on TAG (Ebbeling *et al.*, 2007; Abete *et al.*, 2008; Bellisle *et al.*, 2007; Das *et al.*, 2007; Jensen *et al.*, 2008; Philippou *et al.*, 2008; Philippou *et al.*, 2009b; Philippou *et al.*, 2009a; Sichieri *et al.*, 2007; Pereira *et al.*, 2004; Ebbeling *et al.*, 2005; McMillan-Price *et al.*, 2006; Maki *et al.*, 2007; Wolever and Mehling, 2002; Raatz *et al.*, 2005).

All fifteen studies were similar in that they implemented a parallel group design. Of these, one was single blind (Ebbeling *et al.*, 2007), one double blind (Jensen *et al.*, 2008) and two open (Philippou *et al.*, 2009a; Maki *et al.*, 2007). Eleven studies were unclear with regard to blinding. Trials were conducted in a range of countries such as the UK (3), Spain (1), France (1), Denmark (1) Brazil (1) and Australia (1), although the majority were carried out in the USA (7).

Adults were used as participants in all of the included studies. Three studies were identified that studied females only (Bellisle *et al.*, 2007; Jensen *et al.*, 2008; Sichieri *et al.*, 2007) and one that studied males only (Philippou *et al.*, 2009a).

Excluding two studies that had more than 100 participants in each, sample sizes were relatively small and ranged from 18 to 96 participants (mean=50; median=43). Of the eight studies that reported BMI, participants generally fell into the overweight or obese categories (Abete *et al.*, 2008; Das *et al.*, 2007; Jensen *et al.*, 2008; Sichieri *et al.*, 2007; McMillan-Price *et al.*, 2006; Maki *et al.*, 2007; Wolever and Mehling, 2002; Raatz *et al.*, 2005).

Body weights decreased in all trials other than in Sichieri *et al.* and Philippou *et al.* (Sichieri *et al.*, 2007; Philippou *et al.*, 2009b) in which the authors reported that body weights were unchanged. A slight increase in weight was also noted in the low carbohydrate, high MUFA group in the study by Wolever and Mehling (Wolever and Mehling, 2003). Since blood lipids are modified by body weight change, any differences in outcome may therefore not be solely attributable to the carbohydrate component of the dietary intervention.

Fourteen studies were included in the meta-analysis comparing different glycaemic index and changes in TAG reported as mmol/L. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from eight weeks to 18 months.

In Wolever and Mehling (Wolever and Mehling, 2002), no quantitative data were included which could be incorporated into a meta-analysis. The authors reported the results of a parallel group trial with 34 obese subjects with IGT who had been randomly assigned to one of three groups: a high-carbohydrate, high-GI diet; a high-carbohydrate, low-GI diet; or a low-carbohydrate, high-MUFA diet (Wolever and Mehling, 2002). All diets were *ad libitum* and were intended to be weight maintaining. No statistically significant differences in TAG between the three treatment groups were observed.

The pooled estimate indicated that TAG was 0.03mmol/L (95% CI -0.07 to 0.13) lower with consumption of a lower glycaemic index diet. This was not significantly different from zero ($p=0.60$). Overall heterogeneity denoted by I^2 was 64% (95% CI 37 to 79%). The funnel plot does not provide any evidence of asymmetry. A roughly symmetrical funnel plot would indicate an absence of publication bias. Statistically, there was no evidence that TAG level was associated with differences in glycaemic index.

Figure 2.84 Forest plot for glycaemic index and load diets and TAG (mmol/L)

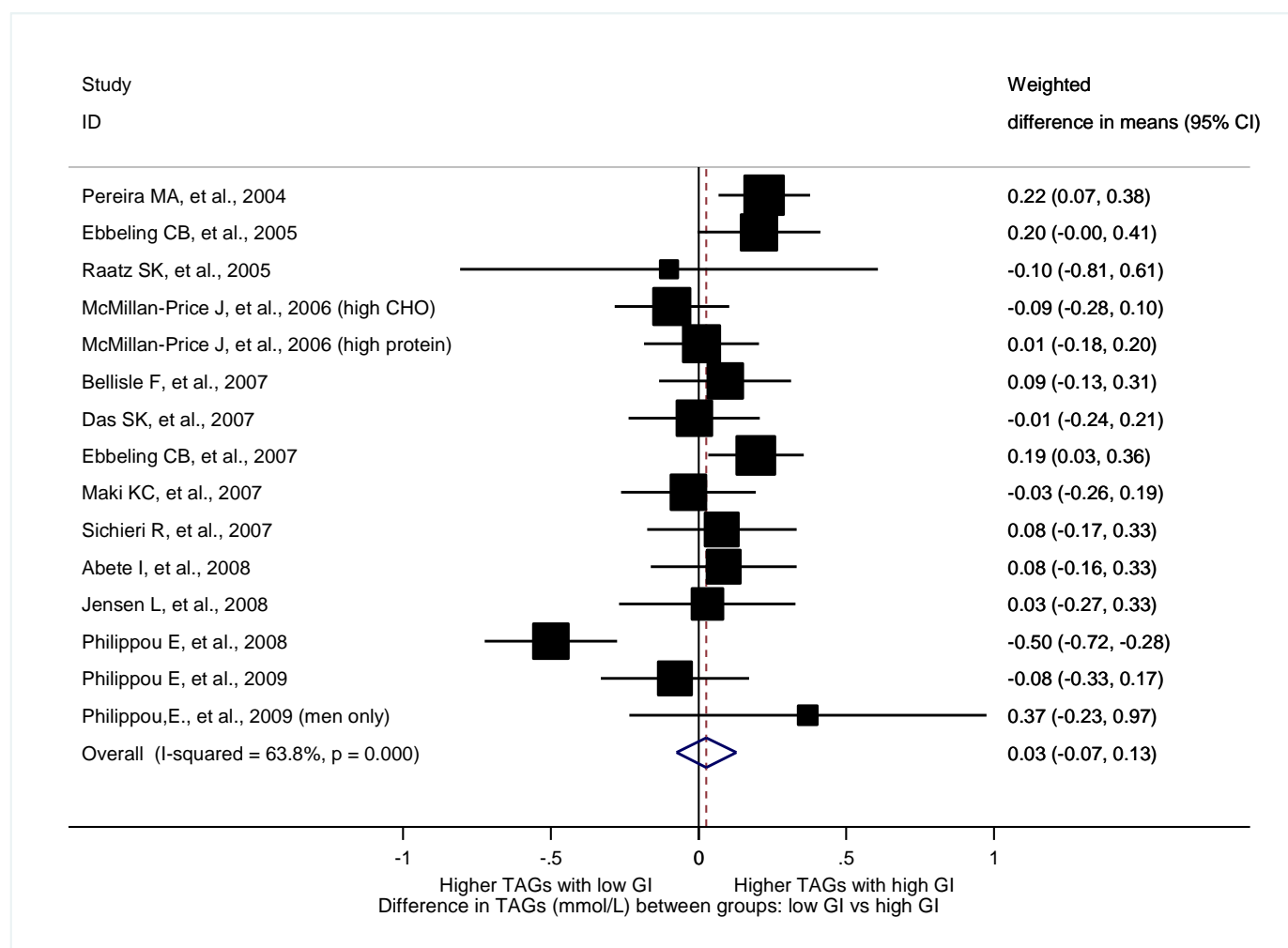


Figure 2.85 Contour-enhanced funnel plot for studies presenting data on dietary glycaemic index or load and TAG

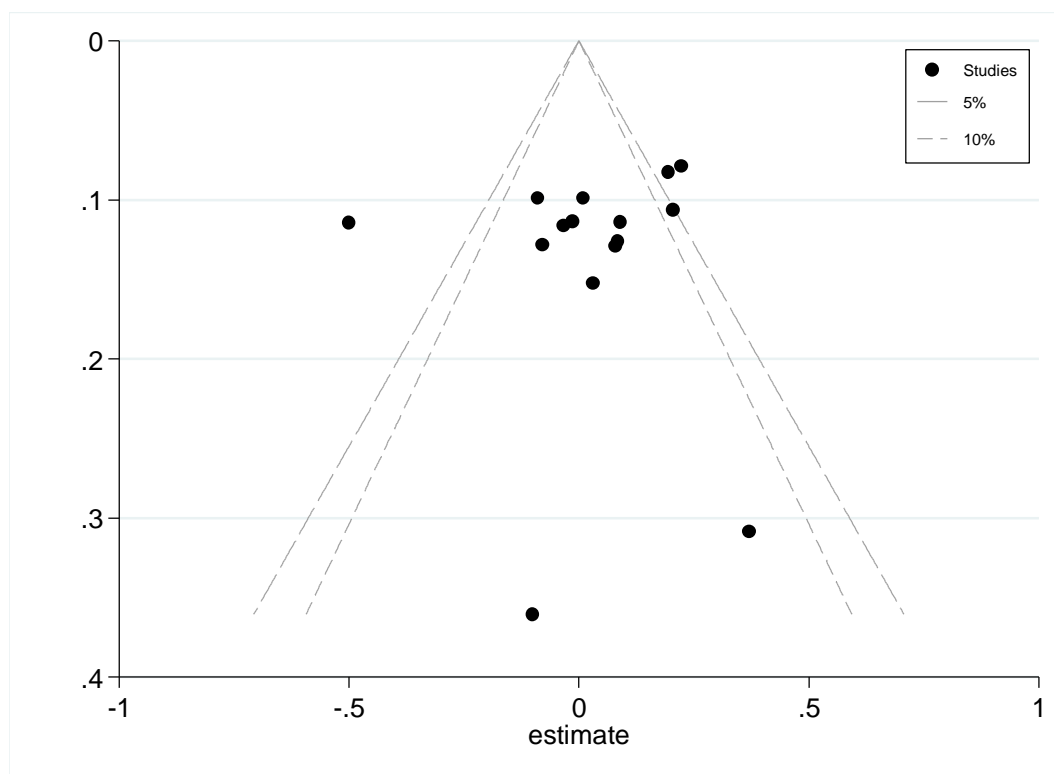


Table 2.155 Triacylglycerol and glycaemic index and load: cohort study in adults

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 Details	Exposure Units	Beta coefficient (SE)/(CI)	p	Adjustments
(Oxlund and Heitmann, 2006) 13898 MONICA	Denmark, Primarily White, Cancer free, No CHD, No medication, No T2DM	30-60 (49) %M 51.3	3608	6 years	Dietary history	GI	Change in TAG Fasting	Men	1 Unit/Total energy	0.0055	NS	Added sugar, age, alcohol, BMI, dietary carbohydrate, coffee, education, energy intake, fat, dietary fibre, physical activity, protein intake, smoking, blood TG
13899 MONICA								Women	1 Unit/Total energy	0.003	NS	As above
13906 MONICA						GL	Change in TAG Fasting	Men	1 Unit/Total energy	-0.1809	NS	Added sugar, age, alcohol, BMI, eating frequency, education, energy intake, dietary fibre, physical activity, smoking, blood TG
13907 MONICA								Women	1 Unit/Total energy	-0.084	NS	As above

Table 2.156 Triacylglycerol and glycaemic index and load: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Abete <i>et al.</i> , 2008) *15549	Higher GI diet	16/16	89 (SD 28)		5.1% (SD 40.8%)	NS		TAG	(mg/dL)	8 weeks	Decrease	unclear
	Lower GI diet	16/16	97 (SD 36)		-2.4% (SD 18%)	NS	0.531				Decrease	
(Bellisle <i>et al.</i> , 2007) *16052	Control	30/45	1.07 (SE 0.08)	1.1 (SE 0.08)				TAG	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
	Low GI	35/51	0.96 (SE 0.09)	1.01 (SE 0.08)			NS				Decrease	
(Das <i>et al.</i> , 2007) 15226	Energy restricted high GL diet	15/17	90.6 (SD 47.2)		-14.3% (SD 21.9%)			TAG	Fasting plasma (mg/dL)	6 months	Decrease	No bias
	Energy restricted low GL diet	14/17	98.6 (SD 33.1)		-24.7% (SD 27.7%)		NS				Decrease	
*15227	Energy restricted high GL diet	15/17	90.6 (SD 47.2)		-16.5% (SD 29.9%)			TAG	Fasting plasma (mg/dL)	1 year	Decrease	No bias

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	Energy restricted low GL diet	14/17	98.6 (SD 33.1)		-15.2% (SD 24.8%)		NS				Decrease	
(Ebbeling <i>et al.</i> , 2005) 15509	Low fat diet	12/17	109 (SE 15)		-7.1% (CI -19.8, 7.6)			TAG	Fasting (mg/dL)	6 months	Decrease	unclear
	Low GI diet	11/17	133 (SE 17)		-35.4% (CI -44.6, - 24.7)						Decrease	
*15510	Low fat diet	12/17	109 (SE 15)		-19.1% (CI -32.2, - 3.6)		0.005	TAG	Fasting (mg/dL)	1 year	Decrease	unclear
	Low GI diet	11/17	133 (SE 17)		-37.2% (CI -47.7, - 24.5)						Decrease	
(Ebbeling <i>et al.</i> , 2007) *15453	Low fat diet	37/37			-4.0 (SE 5.6)			TAG	Fasting plasma (%)	6 months	Decrease	No bias
	Low GL diet	ITT: 36/36			-21.2 (SE 4.7)		0.2				Decrease	
15454	Low fat diet	37/37			2.0 (SE 6.0)			TAG	Fasting plasma (%)	18 months	Decrease	No bias
	Low GL diet	ITT: 36/36			-9.0 (SE 5.4)		0.18				Decrease	
(Jensen <i>et al.</i> , 2008) *15037	High GI diet	22/26	1.18 (SE 0.1)	1.11 (SE 0.06)		NS		TAG	Fasting serum (mmol/L)	10 weeks	Decrease	unclear
	Low GI diet	22/29	1.2 (SE 0.13)	1.08 (SE 0.14)		NS	0.82				Decrease	
(Maki <i>et al.</i> , 2007) 17286	Ad libitum low GL diet	39/43	127.1 (SE 8.3)		-24.8 (SE 5.3)		NS	TAG	Fasting (mg/dL)	12 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	134 (SE 10.6)		-11.5 (SE 6.5)						Decrease	
*17287	Ad libitum low GL diet	39/43	127.1 (SE 8.3)		-12.5 (SE 5.2)		NS	TAG	Fasting (mg/dL)	36 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	134 (SE 10.6)		-15.5 (SE 8.9)						Decrease	
(McMillan- Price <i>et al.</i> , 2006) *16223	High CHO, high GI diet	32/32	1.37 (SE 0.15)		-0.14 (SE 0.07)		0.84	TAG	Fasting (mmol/L)	12 weeks	Decrease	unclear
	High CHO, low GI diet	32/32	1.39 (SE 0.13)		-0.05 (SE 0.07)						Decrease	
	High protein, high GI diet	32/32	1.41 (SE 0.13)		-0.18 (SE 0.07)						Decrease	
	High protein, low GI diet	33/33	1.25 (SE 0.12)		-0.19 (SE 0.07)						Decrease	
(Pereira <i>et al.</i> , 2004) *14579	Hypoenergetic low fat diet	11/23	92.4 (SE 9.47)	102.3 (SE 8.11)	16.2% (SE 5.24%)			TAG	Fasting serum (mg/dL)	67 days	Decrease	unclear
	Hypoenergetic low	14/23	78.3 (SE	72.4 (SE	-3.5% (SE 4.63%)		0.01				Decrease	

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
	GL diet		8.4)	7.19)								
(Philippou <i>et al.</i> , 2008) *16858	High GI	7/9	1.5	1.2	-0.4 (CI -0.6, -0.2)	NS		TAG	Fasting (mmol/L)	12 weeks	Decrease	unclear
	Low GI	6/9	1	1	0.1 (CI -0.1, 0.1)	NS	NS				Decrease	
(Philippou <i>et al.</i> , 2009a) *14662	High GI	16/28	1.29	1.46	-0.02 (CI -0.23, 0.11)	NS		TAG	Fasting (mmol/L)	6 months	Decrease	unclear
	Low GI	22/28	1.63	1.35	-0.39 (CI -1.11, 0.05)	<0.05	NS				Decrease	
(Philippou <i>et al.</i> , 2009b) 15152	High GI	19/19	0.94	1.34	0.09 (CI -0.01, 0.31)			TAG	Fasting plasma (mmol/L)	2 months	No change	unclear
	Low GI	22/23	0.9	1.02	0.12 (CI -0.01, 0.36)		NS				No change	
*15153	High GI	19/19	0.94	1.06	0.04 (CI -0.14, 0.23)			TAG	Fasting plasma (mmol/L)	4 months	No change	unclear
	Low GI	23/23	0.9	0.97	0.12 (CI -0.01, 0.33)		0.2				No change	
(Raatz <i>et al.</i> , 2005) *17235	High fat diet	10/8	1.04 (SE 0.1)		0 (SE 0.1)		0.02	TAG	Fasting plasma (mmol/L)	12 weeks	Decrease	unclear
	High GI diet	9/8	2.04 (SE 0.3)		-0.5 (SE 0.2)						Decrease	
	Low GI diet	10/6	1.79 (SE 0.3)		-0.4 (SE 0.3)						Decrease	
(Sichieri <i>et al.</i> , 2007) 15788	High GI/GL diet	60/102	89.1 (SD 44.2)	89.6 (SD 49.7)			NS	TAG	Fasting plasma (mg/dL)	3 months	No change	unclear
	Low GI/GL diet	73/101	88.9 (SD 46.2)	83.2 (SD 44.8)							No change	
15789	High GI/GL diet	60/102	89.1 (SD 44.2)	79.6 SD 36.5)			NS	TAG	Fasting plasma (mg/dL)	6 months	No change	unclear
	Low GI/GL diet	59/101	88.9 (SD 46.2)	76.9 (SD 36.5)							No change	
15790	High GI/GL diet	42/102	89.1 (SD 44.2)	111.2 (SD 80.9)			NS	TAG	Fasting plasma (mg/dL)	1 year	No change	unclear
	Low GI/GL diet	41/101	88.9 (SD 46.2)	88.0 (SD 48.5)							No change	
*15791	High GI/GL diet	53/102	89.1 (SD 44.2)	120.5 (SD 60.4)		0.0003	NS	TAG	Fasting plasma (mg/dL)	18 months	No change	unclear
	Low GI/GL diet	64/101	88.9 (SD	113.5 (SD		0.0007					No	

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Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow- up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
			46.2)	57.2)							change	
(Wolever and Mehling, 2002) 17011	High carbohydrate, high GI	11/11					NS	TAG	Fasting	16 weeks	Decrease	unclear
	High carbohydrate, low GI	13/13					NS				Decrease	
	Low carbohydrate, high MUFA	11/11					NS				Increase	

*This result was used in the meta-analysis for glycaemic index and load and TAG

Non-esterified fatty acids and glycaemic index and load

No cohort studies reported results concerning glycaemic index/ load and non-esterified fatty acids.

Summary of RCT data

Four papers provided data on three trials that explored the effects of high or low glycaemic index/ load diets on fasting non-esterified fatty acids (Wolever and Mehling, 2002; Jensen *et al.*, 2008; McMillan-Price *et al.*, 2006; Wolever and Mehling, 2003). Two papers from the US trial by Wolever *et al.* reported the same data (Wolever and Mehling, 2002; Wolever and Mehling, 2003). Data only from the earlier publication are included in the table. One study was conducted in Australia (McMillan-Price *et al.*, 2006) and one in Denmark (Jensen *et al.*, 2008).

For all studies a parallel group approach was taken. One study was double blind (Jensen *et al.*, 2008); the remaining trials did not provide clear information regarding blinding. Sample sizes ranged from 37 to 129 participants. Participants were all adults and the mean BMI was between 28 and 31 kg/m². One study was identified that studied women only (Jensen *et al.*, 2008). The remaining trials were mixed gender.

Body weights decreased in all groups in the Australian and Danish trials. However in the study by Wolever and Mehling (Wolever and Mehling, 2003), between-group body weight changes were not statistically different, but tended to increase slightly in the low carbohydrate, high MUFA group. Changes in non-esterified fatty acids therefore may not be attributed purely to the dietary intervention.

Three studies were included in the meta-analysis comparing different glycaemic index/load intakes and changes in non-esterified fatty acids. All studies included adults as participants. The first follow up reported at the end of the intervention was used. This varied from 10 to 16 weeks.

The pooled estimate indicated that the non-esterified fatty acid level was 0.01 mmol/L (95% CI - 0.05 to 0.08) higher with consumption of a higher glycaemic index diet. This was not significantly different from zero ($p=0.74$). Overall heterogeneity denoted by I^2 was 31% (95% CI 0 to 75%). Statistically, there was no evidence that low glycaemic index diets are associated with changes in NEFA.

Figure 2.86 Forest plot for glycaemic index and load diets and non-esterified fatty acids (mmol/L)

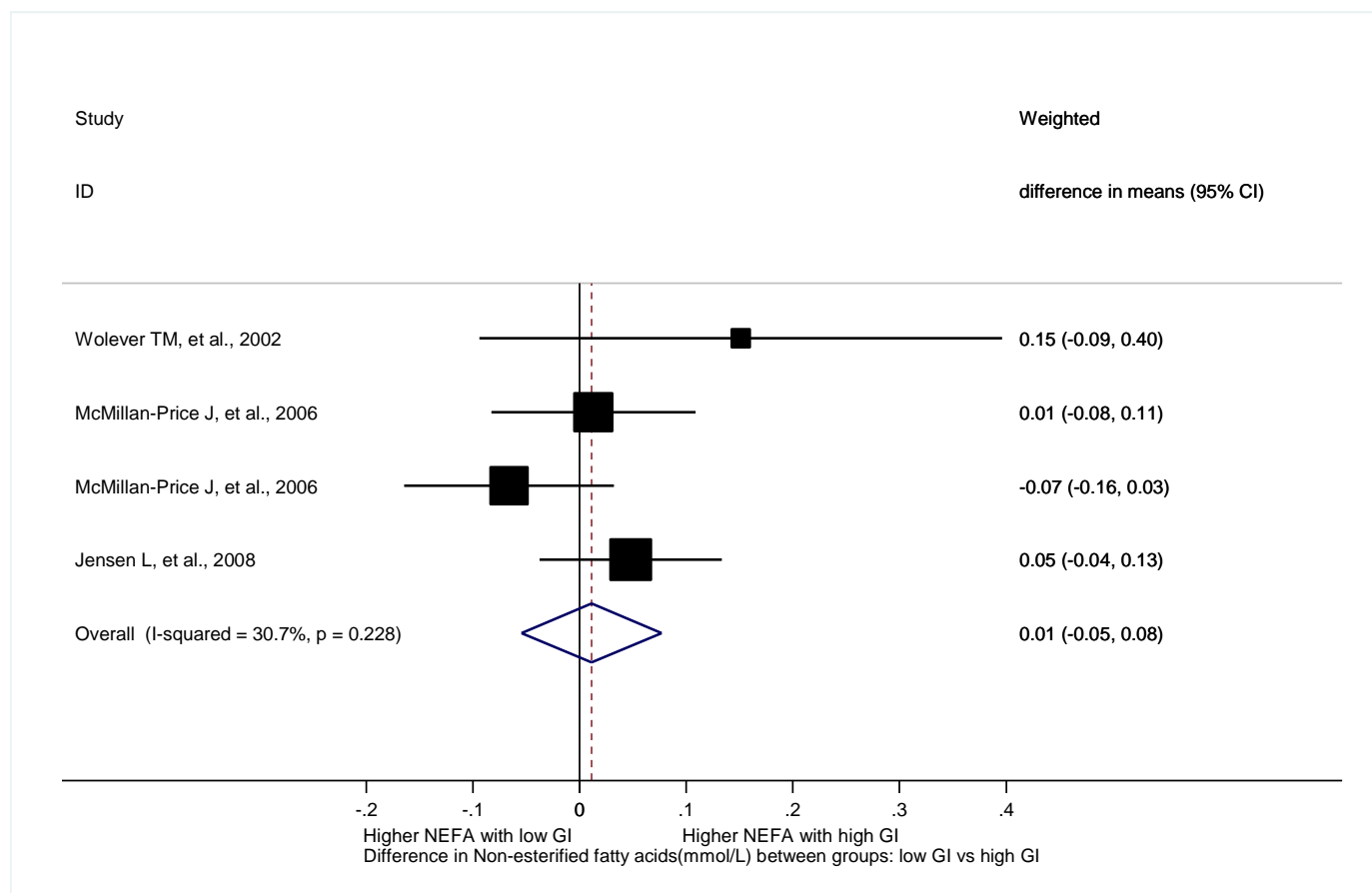


Table 2.157 Non-esterified fatty acids and glycaemic index and load: RCT data

Author/ Result ID	Intervention groups	Completers/ Allocated	Baseline	Follow-up	Within group Δ from baseline	p-value within group Δ from baseline	p-value difference between groups	Outcome	Outcome details	Result-specific follow-up	Weight change	Outcome Assessment Bias
(Jensen <i>et al.</i> , 2008) *15039	High GI diet	22/26	479 (SE 38)	569 (SE 26)		NS		Non-esterified fatty acids	Fasting plasma ($\mu\text{mol/L}$)	10 weeks	Decrease	unclear
	Low GI diet	22/29	444 (SE 30)	521 (SE 35)		NS	0.39				Decrease	
(McMillan-Price <i>et al.</i> , 2006) *16229	High CHO, high GI diet	32/32	510 (SE 33)		-63 (SE 35)		NS	Free fatty acid	Fasting ($\mu\text{mol/L}$)	12 weeks	Decrease	unclear
	High CHO, low GI diet	32/32	436 (SE 32)		3 (SE 36)						Decrease	
	High protein, high GI diet	32/32	545 (SE 42)		-44 (SE 35)						Decrease	
	High protein, low GI diet	33/33	520 (SE 53)		-57 (SE 34)						Decrease	
(Wolever and Mehling, 2002) *17013	High carbohydrate, high GI	11/11			-0.04 (SE 0.05)		NS	Non-esterified fatty acids	Fasting (mEq/l)	16 weeks	Decrease	unclear
	High carbohydrate, low GI	13/13			-0.19 (SE 0.12)		NS				Decrease	
	Low carbohydrate, high MUFA	11/11			-0.14 (SE 0.07)		NS				Increase	

*This result was used in the meta-analysis

Total cholesterol:HDL ratio and glycaemic index and load

No cohort studies reported results concerning glycaemic index/load and the TC:HDL ratio.

Summary of RCT data

Five trials reported on the effects of high or low glycaemic index/load diets on the ratio of total cholesterol:HDL (Philippou *et al.*, 2009b;Bellisle *et al.*, 2007;Philippou *et al.*, 2008;Maki *et al.*, 2007;McMillan-Price *et al.*, 2006). All implemented a parallel group design and were either open (Maki *et al.*, 2007) or unclear regarding the extent of blinding (Bellisle *et al.*, 2007;Philippou *et al.*, 2008;Philippou *et al.*, 2009b;McMillan-Price *et al.*, 2006). Two studies were carried out in the UK (Philippou *et al.*, 2008;Philippou *et al.*, 2009b) and the remaining were conducted in France (Bellisle *et al.*, 2007), Australia (McMillan-Price *et al.*, 2006) or the USA (Maki *et al.*, 2007). The mean number of participants in each trial was 74 and the median was 86. Of the trials that reported mean BMI, participants tended to be obese (Maki *et al.*, 2007;McMillan-Price *et al.*, 2006). All were adults. The majority of studies were mixed gender, but one study was identified that recruited females only (Bellisle *et al.*, 2007).

Body weight decreased in the majority of trials, but was unchanged in the study by Philippou *et al.* (Philippou *et al.*, 2009b).

All five studies were included in the meta-analysis comparing different glycaemic index/load diets and changes in the TC:HDL ratio. The first follow up reported at the end of the intervention was used. This varied from 12 weeks to four months.

The pooled estimate indicated that the TC:HDL ratio was 0.04 (95% CI -0.13 to 0.22) higher with consumption of a higher glycaemic index diet. This was not significantly different from zero ($p=0.62$). Overall heterogeneity denoted by I^2 was 44% (95% CI 0 to 78%). Statistically, there was no evidence that differences in dietary glycaemic index are associated with changes in the TC:HDL ratio.

Figure 2.87 Forest plot for glycaemic index and load diets and total cholesterol:HDL ratio

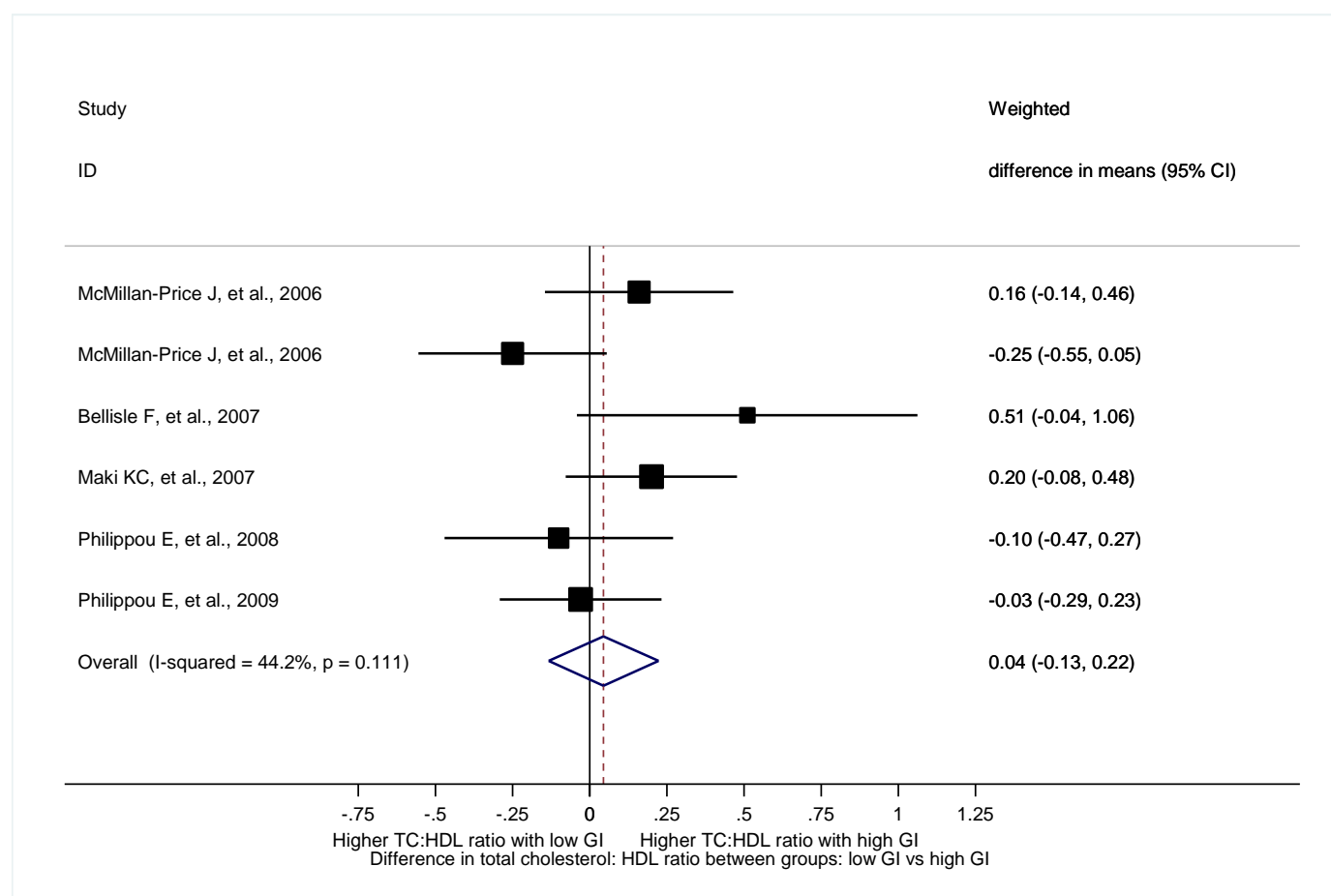


Table 2.158 Total cholesterol:HDL ratio and glycaemic index and load: RCT data

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	Result-specific follow-up	Weight Change	Outcome Assessment Bias
(Philippou <i>et al.</i> , 2009b) *15150	High GI	19/19	4.14 (SD 0.6)	4.14 (SD 0.95)	0 (SD 0.59)			Total cholesterol :HDL ratio	2 months	No change	unclear
	Low GI	21/23	3.78 (SD 0.74)	3.77 (SD 0.7)	-0.01 (SD 0.44)		NS			No change	
15151	High GI	19/19	4.14 (SD 0.6)	4.07 (SD 0.91)	-0.07 (SD 0.33)			Total cholesterol :HDL ratio	4 months	No change	unclear
	Low GI	23/23	3.78 (SD 0.74)	3.74 (SD 0.67)	-0.04 (SD 0.48)		0.1			No change	
(Bellisle <i>et al.</i> , 2007) *16055	Control	30/45	3.54 (SE 0.25 SD)	3.66 (SE 0.24)				Total cholesterol :HDL ratio	12 weeks	Decrease	unclear
	Low GI	35/51	3.12 (SE 0.18)	3.15 (SE 0.16)			NS			Decrease	
(Philippou <i>et al.</i> , 2008) *16857	High GI	7/9	4.4	4.0	-0.2 (CI 0.1, SD -0.4)	NS		Total cholesterol :HDL ratio	12 weeks	Decrease	unclear
	Low GI	6/9	3.9	4	-0.1 (CI 0.1, SD -0.3)	NS	NS			Decrease	
(Maki <i>et al.</i> , 2007) *17284	Ad libitum low GL diet	39/43	3.7 (SE 0.1)		-0.2 (SE 0.1)		NS	Total cholesterol :HDL ratio	12 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	3.8 (SE 0.2)		0 (SE 0.1)		NS			Decrease	
17285	Ad libitum low GL diet	39/43	3.7 (SE 0.1)		-0.3 (SE 0.1)		NS	Total cholesterol :HDL ratio	36 weeks	Decrease	unclear
	Low fat, energy restricted	38/43	3.8 (SE 0.2)		-0.2 (SE 0.1)		NS			Decrease	
(McMillan- Price <i>et al.</i> , 2006) *16958	High CHO, high GI diet	32/32	3.94 (SE 0.25)		-0.23 (SE 0.11)		NS	Total cholesterol : HDL ratio	12 weeks	Decrease	unclear
	High protein, high GI diet	32/32	4.16 (SE 0.24)		-0.21 (SE 0.11)					Decrease	
	High protein, high GI diet	32/32	4.75 (SE 0.32)		0.02 (SE 0.11)					Decrease	
	High protein, low GI diet	33/33	3.83 (SE 0.26)		-0.37 (SE 0.11)					Decrease	

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