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# Dietary advice for treatment of type 2 diabetes mellitus in adults (Review)

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# TABLE OF CONTENTS

[Intervention Review]

# Dietary advice for treatment of type 2 diabetes mellitus in adults

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## ABSTRACT

### Background

While initial dietary management immediately after formal diagnosis is an 'accepted' cornerstone of treatment of type 2 diabetes mellitus, a formal and systematic overview of its efficacy and method of delivery is not currently available.

### Objectives

To assess the effects of type and frequency of different types of dietary advice for adults with type 2 diabetes.

### Search methods

We carried out a comprehensive search of *The Cochrane Library*, MEDLINE, EMBASE, CINAHL, AMED, bibliographies and contacted relevant experts.

### Selection criteria

All randomised controlled trials, of six months or longer, in which dietary advice was the main intervention.

### Data collection and analysis

The lead investigator performed all data extraction and quality scoring with duplication being carried out by one of the other six investigators independently with discrepancies resolved by discussion and consensus. Authors were contacted for missing data.

### Main results

Thirty-six articles reporting a total of eighteen trials following 1467 participants were included. Dietary approaches assessed in this review were low-fat/high-carbohydrate diets, high-fat/low-carbohydrate diets, low-calorie (1000 kcal per day) and very-low-calorie (500 kcal per day) diets and modified fat diets. Two trials compared the American Diabetes Association exchange diet with a standard reduced fat diet and five studies assessed low-fat diets versus moderate fat or low-carbohydrate diets. Two studies assessed the effect of a very-low-calorie diet versus a low-calorie diet. Six studies compared dietary advice with dietary advice plus exercise and three other studies assessed dietary advice versus dietary advice plus behavioural approaches. The studies all measured weight and measures of glycaemic control although not all studies reported these in the articles published. Other outcomes which were measured in these studies included mortality, blood pressure, serum cholesterol (including LDL and HDL cholesterol), serum triglycerides, maximal exercise capacity and compliance. The

results suggest that adoption of regular exercise is a good way to promote better glycaemic control in type 2 diabetic patients, however all of these studies were at high risk of bias.

**Authors' conclusions**

There are no high quality data on the efficacy of the dietary treatment of type 2 diabetes, however the data available indicate that the adoption of exercise appears to improve glycated haemoglobin at six and twelve months in people with type 2 diabetes. There is an urgent need for well-designed studies which examine a range of interventions, at various points during follow-up, although there is a promising study currently underway.

## PLAIN LANGUAGE SUMMARY

**Dietary advice for treatment of type 2 diabetes mellitus in adults**

No high quality data on the efficacy of diet alone exists for treatment of type 2 diabetes mellitus. This systematic review assesses the effects of studies that examined dietary advice with or without the addition of exercise or behavioural approaches. Eighteen studies were included. No data were found on micro- or macrovascular diabetic complications, mortality or quality of life. It is difficult to draw reliable conclusions from the limited data that are presented in this review, however, the addition of exercise to dietary advice showed improvement of metabolic control after six- and twelve-month follow-up.

## BACKGROUND

Diabetes mellitus is a metabolic disorder resulting from a defect in insulin secretion, insulin action, or both. A consequence of this is chronic hyperglycaemia (i.e. elevated levels of plasma glucose) with disturbances of carbohydrate, fat and protein metabolism. Long-term complications of diabetes mellitus include retinopathy, nephropathy and neuropathy. The risk of cardiovascular disease is increased. For a detailed overview of diabetes mellitus, please see under 'Additional information' in the information on the Metabolic and Endocrine Disorders Group on the Cochrane Library (see 'About the Cochrane Collaboration', 'Collaborative Review Groups'). For an explanation of methodological terms, see the main Glossary on the Cochrane Library. There is now unequivocal evidence that type 2 diabetes can be prevented or at least delayed by dietary effort, generally resulting in weight loss and increased physical activity in those at high risk of progressing to diabetes. Risk of such progression was decreased by over half in the two major trials to date (Tuomilehto 2001; DPP 2002). The United States Diabetes Prevention Program reported a 58% reduction in the incidence of diabetes when participants were treated with the lifestyle intervention compared with a 31% reduction of incidence of diabetes for the metformin-treated participants, (DPP 2002). However the role and type of dietary advice following diagnosis of type 2 diabetes, how often thereafter and its efficacy during longer-term follow-up over subsequent years is much less clear. It may be that using different methods of helping people to learn the principles behind these diets, and different patterns of support and follow-up, could have effects on long term adherence.

This Cochrane review aims to establish the evidence that exists on what kind of dietary advice is effective in treating type 2 diabetes.

There are an estimated 2.35m people with diabetes in England, with up to another 750,000 undiagnosed cases and this is predicted to grow to more than 2.5m by 2010 (DOH website; Diabetes UK website).

Type 2 diabetes mellitus is the more common of the two main types and accounts for between 85 - 95% of all diabetic patients (Diabetes UK website). In the United Kingdom alone, treatment of type 2 diabetes costs around £1.8 billion per annum (Moore 2000).

It has been recommended for many years that people diagnosed with type 2 diabetes should be treated with diet, and in those overweight to achieve weight loss and, independently, to lower blood glucose (glycaemia). On average, there is a 9% relative increase in the risk of type 2 diabetes, for every 1kg weight gain (Mokdad 2000). Eighty to ninety percent of type 2 diabetics are obese (BMI >30kg/$m^2$) (Wing 2000) and obesity worsens the metabolic and physiologic abnormalities associated with diabetes, particularly hyperglycemia, hyperlipidemia, and hypertension (Maggio 1997; Wing 2000). Initiation of medication becomes necessary in addition to diet and exercise when the latter alone fail to control glycaemia adequately, (Nathan 1999). While short-term benefits are assumed, attempting to treat type 2 diabetes using diet alone is not a particularly successful long-term intervention, as illustrated by the United Kingdom Prospective Diabetes Study, (UKPDS 1998; UKPDS 1998b). This study showed that after three years of regularly reinforcing dietary advice in those randomised to the 'diet alone' arm, only 20% of patients, and after nine years only 8%, maintained the target fasting plasma glucose of less than 7.8 mmol/L, (Turner 1999). Part of the uncertainty in this field is whether the dietary and other non-pharmacological measures actually 'fail' or whether patients themselves fail to continue implementing them adequately. Despite this, diet is usually the first treatment implemented on diagnosis of type 2 diabetes and is maintained throughout the addition of other interventions.

Some observational studies have shown that exercise may have an additional benefit for the treatment of type 2 diabetes, possibly through lowering body fat and reducing blood pressure (Wallberg-H. 1998), and several studies have produced some compelling evidence to show that regular physical exercise can serve to protect and decrease severity of type 2 diabetes (Frisch 1986; Dowse 1991; Manson 1992a). The review by Thomas et al, 2006, found that exercise improves blood glucose control and that this effect is evident even without weight loss (Thomas 2006).

Diets commonly used to control blood glucose levels include low fat and high unrefined carbohydrate, (those which take around 25-30% of energy from fat and around 50% of the total energy from unrefined carbohydrate), or low glycaemic index diets (foods that have a low glycaemic index include pasta products, oats, beans and some fruits and vegetables) usually both in combination with weight reducing advice. Recently, research has also looked into the effect of individual macro- and micro-nutrients such as fibre, chromium, and so called 'functional foods', but much of this evidence seems to be incomplete, with relatively few intervention trials carried out long-term (Riccardi 2005). Similar diets may be of value in those with metabolic syndrome in preventing or delaying progression to frank diabetes (Riccardi 2000). Given the prevalence of type 2 diabetes and the potentially serious outcomes of the disease, it is important to establish which type of diet, either alone or in combination with other interventions (the addition of exercise, behavioural approaches and alternative treatments), is most effective.

### This Review

This review is a 2007 update of the initial (Moore 2004) review. It aimed to provide an update of evidence from studies which have employed a study design which sought to examine the effect of dietary advice and interventions to treat type 2 diabetes mellitus in the adult population.

Although no additional included studies were identified, the review referenced further relevant background research and excluded studies, and has generated a QUOROM (quality of reporting of meta-analyses) flow-chart of study selection.

## OBJECTIVES

The aim of this systematic review was to assess the effects of type and frequency of dietary advice to all adults (overweight and normal weight) with type 2 diabetes on morbidity, quality of life, total mortality, weight and measures of diabetic control, using all available randomised clinical trials and meta-analytic techniques where appropriate. The primary objectives were to assess:

a) the effects of different types of dietary advice through weight change, and development of micro- and macro- vascular complications in people with type 2 diabetes, and

b) the effects of dietary advice plus other lifestyle interventions (i.e. the addition of exercise, behavioural approaches and alternative therapies to dietary advice) through weight change, and development of micro- and macro-vascular complications in people with type 2 diabetes.

### Management of hits

We prepared a QUOROM (quality of reporting of meta-analyses) flow-chart of study selection to describe how we processed the references identified through our searches.
The hits identified from the searches of the electronic databases [Medline (492), AMED (2), CINAHL (87) , Cochrane Library (16) and EMBASE (1,110)] were combined (n = 1,707) and de-duplicated (n = 1,413).
These hit lists were then de-duplicated against the hits of the previous version of this review. The reduced list of hits was then screened on titles and abstracts (LN).

# METHODS

## Criteria for considering studies for this review

### Types of studies

All randomised controlled clinical trials of interventions that involved patients for at least six months were included. Randomisation of individuals or clusters of individuals were accepted.

### Types of participants

All studies had to include adult participants (those who were 18 years or older), who were diagnosed with type 2 diabetes. To be consistent with changes that have occurred over time in the classification and diagnostic criteria of type 2 diabetes mellitus, the diagnosis of diabetes should have been established using the standard criteria valid at the time of the beginning of the trial. Ideally, the diagnostic criterion was described in the article. Diagnostic criteria used included those described by the National Diabetes Data Group standards (NDDG 1979), the World Health Organisation standards, (WHO 1980; WHO 1985; WHO 1998), and the American Diabetes Association Standards, (ADA 1997; ADA 1999). Studies performed on participants suffering from impaired glucose tolerance were not included in this review. Where a study reported combined results for participants with type 2 diabetes and participants with impaired glucose tolerance, then efforts were made to contact the authors of the study to obtain individual patient data. Where this was unsuccessful, the trial was excluded. In the protocol for this review we omitted to state how a study looking at a mixture of type 1 and type 2 diabetic participants would be dealt with. We decided that unless a majority of the participants (i.e. 90% or more) were classified as having type 2 diabetes, or the author of the paper could provide the data for the type 2 diabetic participants only, the trial was excluded.
Participants could be of either sex, but those who were acutely ill or pregnant were excluded.

### Types of interventions

Studies where the intervention was dietary advice with an aim of reducing weight and the severity of type 2 diabetes were included in the review. Dietary advice is taken to mean advice given with the intention of improving dietary habits (i.e. to either produce weight loss or to change diet composition). Studies were not included if they included medication that was provided differently in the control and intervention groups. Studies evaluating the effects of fish oils (omega-3) advice or supplementation on type 2 diabetes mellitus were excluded as this had previously been addressed in a recent Cochrane review (Farmer 2001). Studies looking at the effects of Chinese medical herbs on type 2 diabetes mellitus were also excluded as these are addressed by another Cochrane review (Liu 2002).

### Types of outcome measures

#### *Primary outcomes*

- weight (where the main aim of the study was weight loss);
- development of micro and macrovascular diabetic complications (including neuropathies, retinopathy, nephropathy and cardiovascular diseases).

#### *Secondary outcomes*

- quality of life (ideally, measured using a validated instrument);
- change in anti-diabetic medication use (as an indicator of improving or worsening diabetic control);
- overall cardiovascular disease risk assessment (using any of the scales which include at least three risk factors);
- mortality;
- glycated haemoglobin;
- serum cholesterol (LDL and/or HDL) and serum triglycerides;
- maximal exercise capacity ($VO_2$ max);
- blood pressure;
- compliance.

#### Timing of outcome assessment

Randomised controlled trials of interventions that involved participants for a minimum of six months were included in the review. Outcome measures were extracted and assessed at baseline, six months, one year, two year and at one year intervals from that point where available.

## Search methods for identification of studies

### Electronic searches

The following sources were searched to identify relevant literature for the original review (Moore 2004):

*The Cochrane Library*, which includes the Cochrane Central Register of Controlled Trials, the Database of Systematic Reviews and the Database of Abstracts and Reviews of Effectiveness

- *The Cochrane Library*, (Issue 3, 2003);
- MEDLINE (1966 to October Week 1 2003);
- EMBASE (1980 to Week 40 2003);
- CINAHL (1982 to October Week 1 2003);
- AMED (1985 to October 2003), bibliographies and experts.

***For this update review, the following searches were completed :***

*The Cochrane Library,* which includes the Cochrane Central Register of Controlled Trials, the Database of Systematic Reviews and the Database of Abstracts and Reviews of Effectiveness

- *The Cochrane Library*, (October, 2006);
- MEDLINE (October Week 1 2003 to October 2006);
- EMBASE (Week 40 2003 to October 2006);
- CINAHL (October Week 1 2003 to October 2006);
- AMED (October 2003 to October 2006).

There were no language restrictions for either searching or trial inclusion. The search strategy below was used, adapted to suit the individual databases.

NOTE: Unless otherwise stated, search terms are free text terms;

For details of the search strategy see under Appendix 1.

The MEDLINE search was run as above, with the randomised controlled trials and systematic reviews searches added on separately. The CINAHL search was run as above, with randomised controlled trials search added on separately.
The AMED search was run as above, with randomised controlled trials search added on separately.
The EMBASE search was adapted from the original search above.

Once relevant papers were retrieved, their references were analysed and any relevant referenced papers were also sourced and added into the review.

## Data collection and analysis

### Selection of studies

Two reviewers undertook assessment of results data independently. Results data were assessed by the lead reviewer (LN) and duplicated by one of the co-reviewers (HM, CS, VW, LH, KC and AV). Information on a number of measures of methodological quality of the included studies was assessed independently by two reviewers; study design, method of allocation concealment, blinding of outcome assessment and drop out rates. Where there was uncertainty, authors were contacted to clarify aspects of study design. Differences between reviewers' extraction results were resolved by discussion. Multiple publications were collated and assessed as one study.

In the first instance, relevant studies were determined from the initial search of electronic databases, and then through screening by the lead reviewer (LN). Articles were rejected during this initial screening if the reviewer could determine from the title and/or abstract that it did not meet the inclusion criteria, if rejection was not possible, full text copies were retrieved. For the 2007 update, full text copies of 68 papers were retrieved and assessed independently by two reviewers (LN plus one other from HM, CS, VW, LH, KC and AV) using an inclusion/exclusion form. Inter-rater agreement (using Cohen's kappa (Cohen 1960)) was good with all of the reviewers achieving kappa scores of 0.7 or greater in the first review (Moore 2004), Inter-rater agreement of this review achieved a value of 1.0 (using Cohen's kappa). Thirty-six of the original 287 retrieved papers were included in this review, which reported a total of eighteen studies. In the update, none of the sixty-eight papers were thought to fit the inclusion criteria, so no new papers were added. Where full text copies of articles were retrieved, but then were judged to not meet the inclusion criteria, the reasons were reported in the excluded studies table.

### Data extraction and management

Two reviewers (the lead reviewer (LN) plus one other from HM, CS, VW, LH, KC and AV) independently extracted data from each study using a data extraction form based on the one
provided by the Metabolism and Endocrine Disorders Review Group. Differences between reviewers' extraction results were resolved by discussion, and where necessary, in consultation with a third reviewer. Data concerning participants, interventions, and outcomes, as described above in the selection criteria section, were extracted. Trial quality characteristics, including method of randomisation, allocation concealment, blinding of outcome assessors and losses to follow-up, were extracted onto this form. In addition, data were collected on potential effect modifiers including age, presence of diabetic micro or macrovascular disease, blood pressure, lipids, bodyweight, mortality and current use of diabetic medication.

### Assessment of risk of bias in included studies

The quality of each trial was assessed based largely on the quality criteria specified by Jadad and Schulz (Schulz 1995; Jadad 1996). In particular the following factors were assessed:
1. Minimisation of selection bias - score: 0 or 1 or 2.
One point was given if the study could be described as randomized (which included the use of words such as 'random', 'randomly' and 'randomisation').
One additional point was given if the study described the method of randomisation and was an appropriate method.
One point was taken away if the study described the method of randomisation, but was an inappropriate method.
2. Minimisation of detection bias - score: 0 or 1 or 2.
One point was given if the study could be described as blinded (in any capacity).
One additional point was given if the method of blinding was described and was appropriate.
One point was taken away if the method was described as blinding but was inappropriate.
3. Minimisation of attrition bias - score: 0 or 1.
One point was given if the withdrawals and dropouts from the study were described.
We did not score on performance bias.
Based on these criteria, studies were broadly subdivided into the following three categories (see Cochrane Handbook):
A - all quality criteria met: low risk of bias. (Studies with scores of four and five points (and with at least one point allocated from each section) were allocated to this category.)
B - one or more of the quality criteria only partly met: moderate risk of bias. (Studies with scores of three points (and with at least one point in each section) were allocated to this category.)
C - one or more criteria not met: high risk of bias. (Studies with scores of zero, one, two and three points were allocated to this category. (i.e. studies with no points in at least one section))
These subdivisions of the studies were not used as the basis of a sensitivity analysis, as the included studies received similar quality scores, (between one and three points). These quality scores and categories are reported in the 'characteristics of included studies' table.

### Measures of treatment effect

#### *Continuous data*

Endpoint versus change data: Where possible, endpoint data were presented, as change standard deviations were not available for many studies. If both endpoint and change data were available for the same outcomes, only the former were reported in this review. If endpoint data were not available, but change data were, we reported the change data in the tables and text of the review.
However, for inclusion of a study reporting change data in the meta-analysis, we calculated the endpoint mean from the change data given and assumed that the endpoint standard deviation to be equal to the baseline standard deviation.

***Summary statistic***

For continuous outcomes a weighted mean difference (WMD) between groups was estimated.

**Assessment of heterogeneity**

We tested for heterogeneity by using the standard chi2 test and the I2 test (Higgins 2002), as well as by visually inspecting the graphs, and when there was little heterogeneity between trial results, data were summarised statistically by a fixed effect model for continuous data. A significance level less than 0.10 was interpreted as evidence of heterogeneity. If heterogeneity was found, the data were re-analysed using a random effects model to see if this made a substantial difference.

**Subgroup analysis and investigation of heterogeneity**

Subgroup analyses were planned a priori but were not undertaken as they were not applicable to the included studies. These subgroup analyses were planned to assess whether particular groups of people with type 2 diabetes could obtain more benefit from a particular intervention than other groups could. Efficacy of different combinations of types of diets would have also been considered in the subgroup analyses. The main analyses carried out were as follows:

- dietary advice versus different dietary advice;
- dietary advice versus same dietary advice plus exercise;
- dietary advice versus same dietary advice plus behavioural approaches.

Analyses for dietary advice versus same dietary advice plus alternative therapies and for dietary advice versus the same dietary advice delivered at a different frequency were planned, but as no studies were found these analyses could not be carried out.

Subgroup analyses for each of these comparisons would have included:
with or without weight loss advice (outcome measures: development of (further) microvascular disease only) presence/absence of microvascular disease (outcome measures: development of
(further) microvascular disease only).

**Sensitivity analysis**

No sensitivity analyses were performed.

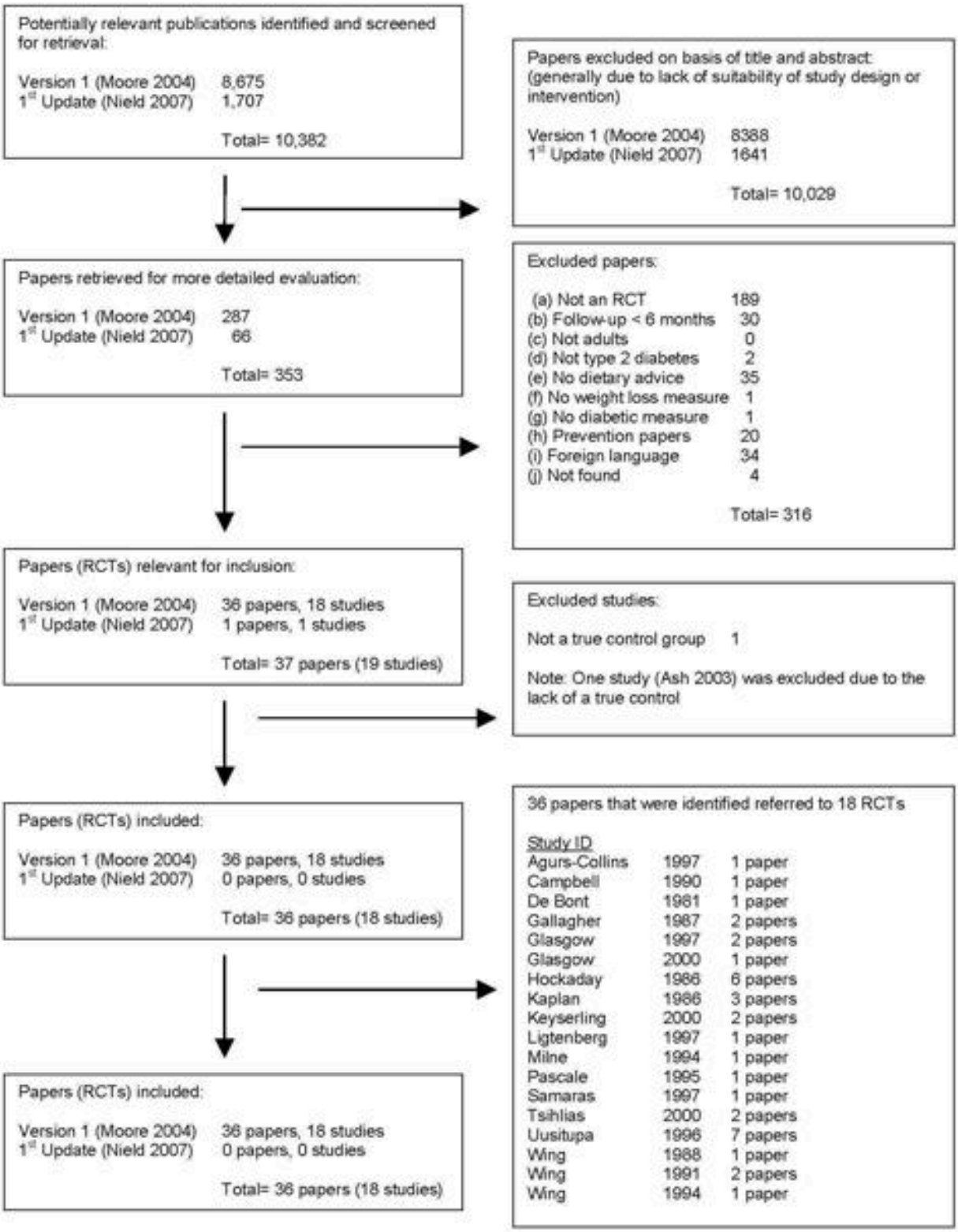
# RESULTS

## Description of studies

**Trials identified**

From the initial searches of electronic databases, 8675 (775 from the Cochrane Library, 4278 from MEDLINE, 3310 from EMBASE, 269 from CINAHL and 43 from AMED) abstracts were screened after de-duplication (HM). For this update review, 1413 articles were screened (16 from the Cochrane Library, 492 from MEDLINE, 1110 from EMBASE, 87 from CINAHL and 2 from AMED) (Figure 1). Articles were rejected on initial screening if the reviewer could determine from the title and/or abstract that it did not meet the inclusion criteria. For the 2007 update review, full text copies of 66 papers were retrieved and assessed independently by two reviewers (LN plus one other from HM, CS, LH, KC, VW and AV), none of which were eligible for inclusion in the study. However, 36 of the original 287 papers, which reported 18 studies, were included in this review. All articles for which hard copies were retrieved but which failed to meet the inclusion criteria were reported in the excluded studies table. Attempts were made to obtain full-text translations and/or evaluations of all relevant non-English articles.

**Figure 1. Adapted QUOROM (quality of reporting of meta-analyses) flow-chart of study selection**




### Interrater agreement

Full text copies of 68 papers were retrieved and assessed independently by two reviewers (LN plus one other from HM, CS, LH, KC, VW and AV) using an inclusion/exclusion form. Interrater agreement (using Cohen's kappa (Cohen 1960)) was good with all of the reviewers achieving kappa scores of 0.7 or greater.

### Missing data

All authors were contacted by the lead reviewer in the event of data which has been reported as recorded but not reported in the text of published articles. Replies were received from Dr Hockaday, (Hockaday 1986), Dr Wolever, (Tsihlias 2000), and Dr Samaras, (Samaras 1997).

### Included studies

Eighteen studies met the inclusion criteria. Nine studies focused on looking at the effects of two types of diabetic dietary advice that did not differ in intent to lose weight, (de Bont 1981; Hockaday 1986; Gallagher 1987; Campbell 1990; Wing 1991; Milne 1994; Wing 1994; Pascale 1995; Tsihlias 2000), three studies focused on looking at dietary advice versus dietary advice plus behavioural approaches, (Glasgow 1997; Glasgow 2000; Keyserling 2000), and six studies concentrated on dietary advice versus dietary advice plus exercise, (Kaplan 1986; Wing 1988; Uusitupa 1996; Agurs-Collins 1997; Ligtenberg 1997; Samaras 1997).

Of the nine studies that compared two types of diabetic diet that did not differ in intent to lose weight, four took place in the United States of America, (Gallagher 1987; Pascale 1995; Wing 1991; Wing 1994), two were based in the United Kingdom, (de Bont 1981 and Hockaday 1986), one in Canada, (Tsihlias 2000), one in New Zealand, (Milne 1994) and one study was based in Australia, (Campbell 1990).

There were three studies that assessed dietary advice versus dietary advice plus behavioural approaches; all of these were conducted in the United States, (Glasgow 1997; Glasgow 2000; Keyserling 2000).

From a total of six dietary advice versus dietary advice plus exercise studies, one took place in Finland, (Uusitupa 1996), one in The Netherlands, (Ligtenberg 1997), one study was conducted in Australia, (Samaras 1997), and three took place in the United States of America, (Kaplan 1986; Wing 1988; Agurs-Collins 1997).

### Studies

Wide ranges of dietary approaches were examined in this review. Within the studies that compared two types of diabetic dietary advice that did not differ in intent to lose weight, there were three distinct groups; those studies that used exchange diets versus those that did not use exchange diets, (Gallagher 1987; Campbell 1990), those studies that used low-fat versus moderate fat or low-carbohydrate diets, (de Bont 1981; Hockaday 1986; Milne 1994; Pascale 1995; Tsihlias 2000), or very-low-calorie diets versus low-calorie diets, (Wing 1991; Wing 1994).

From the studies that assessed dietary advice versus dietary advice plus exercise, it was seen that the types of exercise examined in the studies that are contributing to the review included various forms of aerobic exercise, (in general participants chose their exercise from suggestions that included walking, jogging, swimming, ball games or skiing).

There were similar interventions included within the grouping for dietary advice versus dietary advice plus behavioural approaches. Generally, the mode of imparting the dietary advice was what was changed in these interventions; either through a touch-screen method, (Glasgow 1997), or through community resources, (Glasgow 2000; Keyserling 2000).

### Participants

Thirty-six articles reporting a total of eighteen trials following 1467 participants were included. There were 724 participants in the control groups, and 743 participants in the intervention groups included in this review. The samples used in the trials were generally representative of the overall diabetic population. Diagnostic criteria for the trials in this review included physician confirmed diabetic status, type 2 diabetes classified according to the National Diabetes Data Group, (NDDG 1979), and diabetes mellitus as defined by the World Health Organisation, (WHO 1985). Generally, the diagnostic criteria were not disclosed in the articles.

### Interventions

The participants in the trial run by Campbell, (Campbell 1990), were randomised to one of two groups, to a typical portion exchange diet or to a reduced fat (focusing on reducing fat) diet. de Bont conducted a trial of a low-fat diet versus a low-carbohydrate diet, (de Bont 1981). The Gallagher study, (Gallagher 1987), evaluated the usual American Diabetic Association exchange diet (40% carbohydrate, 40% fat and 20% protein) compared to a reduced fat diet, which was based upon the four basic food groups, (see Results section for more details). Hockaday conducted a trial where participants were randomised to either a low-carbohydrate or a low-fat diet, (Hockaday 1986). Milne randomised the participants into groups that either followed a low-carbohydrate or a low-fat diet, (Milne 1994). Pascale described a trial that randomised the participants to follow either a calorie restricted diet or a calorie restricted and low-fat diet (less than 20% of energy to come from fat), (Pascale 1995). The participants in the Tsihlias trial, (Tsihlias 2000), were randomised to receive either a low-fat or a monounsaturated fat diet.

The participants in the trial described by Wing were randomised to either a balanced low-calorie diet (1000-1200 kcal per day) or a balanced low-calorie diet with two periods of twelve weeks of a very-low-calorie diet (of 400-500 kcal per day), (Wing 1994). Another trial by Wing, (Wing 1991), randomised the participants to either a low-calorie yet balanced diet (with a calorie goal of ˜1000 to 1500 kcal per day) or to a low-calorie (balanced) diet with a period of very-low-calorie diet (˜500 kcal per day) added for eight weeks.

The study conducted by Agurs-Collins randomised participants to either a 'usual care' group or a 'usual diet with exercise' group, (Agurs-Collins 1997). In the study conducted by Uusitupa and colleagues, (Uusitupa 1996), participants were randomised to either a standard dietary treatment group or a diet and exercise group. The trial by Ligtenberg randomised the participants to either continue with their usual diet or to receive their usual diet plus physical training instructions, (Ligtenberg 1997). Samaras randomly assigned participants to receive either their usual diet or to receive their usual diet plus a structured exercise programme, (Samaras 1997). The participants in the Kaplan, (Kaplan 1986), trial were randomly allocated to either weekly sessions of diet education, which focused on promoting a low-fat (30% of total energy), high carbohydrate (55% of total energy) or to weekly sessions of diet and exercise education. Wing conducted a trial which randomised participants to either a diet only group or a diet plus exercise group, (Wing 1988).

Glasgow conducted a trial that randomised the participants to either receive usual care or usual care plus the brief intervention added in, (Glasgow 1997). Another trial by Glasgow, (Glasgow

2000), randomly assigned its participants to receive either a basic intervention (received information about low-fat eating) or the community resources intervention. The trial conducted by Keyserling randomised the participants to receive either a clinic-based intervention or a clinic-based intervention plus community intervention, (Keyserling 2000).

### Outcome measures

The primary outcomes that we intended to assess in this review were:

- weight;
- development of micro and macrovascular diabetic complications (including neuropathies, retinopathy, nephropathy and cardiovascular diseases).

Weight was generally well reported, whereas development of micro and macrovascular diabetic complications was only reported in one trial, (Hockaday 1986), and even here, this was only a brief description.

The secondary outcome measures that we intended to look at were:

1. Quality of life (ideally, measured using a validated instrument),
2. Change in anti-diabetic medication use (as an indicator of improving or worsening diabetic control),
3. Overall cardiovascular disease risk assessment (using any of the scales which include at least three risk factors),
4. Mortality,
5. Glycated haemoglobin,
6. Serum cholesterol (LDL and/or HDL) and serum triglycerides,
7. Maximal exercise capacity ($VO_2$ max),
8. Blood pressure,
9. Compliance.

These additional outcome measures were reported with varying degrees of comprehensiveness. Measures of quality of life and cardiovascular disease risk assessment were not reported in any studies. Change in anti-diabetic medication and maximal exercise capacity were measured and reported in a few trials, although they used different outcome measures. Mortality was briefly reported in a few trials (de Bont 1981; Milne 1994) although details of cause of death were not often disclosed. Serum cholesterol and triglycerides and blood pressure were reported in some trials, although generally only the means (and not their associated standard deviations or standard errors) were reported. Glycated haemoglobin was recorded by nearly all trials and compliance was recorded by some trials, usually in the form of three day food diaries, and although generally not reported in any way, a few studies did report that 'compliance was generally good' (Kaplan 1986; Wing 1988; Samaras 1997).

## Risk of bias in included studies

Further details of the methodological quality of the included studies can be found in the table of characteristics of included studies.

### Minimisation of selection bias

Since randomisation was the first inclusion criterion that studies had to meet, all included studies started with a score of 1. Only two articles described the method of randomisation used, (Hockaday 1986; Glasgow 1997) and therefore were awarded another quality point. Where studies described the method of randomisation, it was appropriate.

### Minimisation of detection bias

One point was assigned for the presence of assessor blinding in only one trial, (de Bont 1981). None of the included articles made any attempt to describe the method of blinding. In dietary trials, it is not possible to blind participants or the study personnel to the intervention or advice they receive (or are imparting).

### Minimisation of attrition bias

More than half of the studies had withdrawals that were not fully described. However, there were seven studies that did describe the drop-outs and withdrawals to an acceptable degree, (de Bont 1981; Hockaday 1986; Wing 1988; Wing 1991; Milne 1994; Pascale 1995; Keyserling 2000) and these studies all received a point to add to their quality scores. Eleven studies reported follow-up data of more than 80% of the baseline sample, (de Bont 1981; Hockaday 1986; Wing 1988; Campbell 1990; Wing 1991; Milne 1994; Pascale 1995; Ligtenberg 1997; Samaras 1997; Glasgow 2000; Keyserling 2000). A number of studies reported higher drop-out rates; 30% at twelve months (Pascale 1995), 32% at six months (Tsihlias 2000), 40% at six months and 44% at twelve months (Keyserling 2000). The study reported by Hockaday (Hockaday 1986) assessed subsections of the initial study population, so analysis of attrition bias was not appropriate. Two other studies did not describe attrition at all (Gallagher 1987; Glasgow 1997).

### Allocation concealment

Allocation concealment to intervention or control was unclear in all but one, (Hockaday 1986), of the eighteen studies. In the published article, allocation concealment was unclear, but as a result of a personal communication from Dr Hockaday, this was then revised to 'adequate'.

The source of funding for more than half of the included studies (12 out of 18) was stated in the published papers. Generally, the funding came from national sources so conflicts of interests were avoided (i.e. operators/providers of treatment/intervention) were not paying for the study to be carried out.

Seventeen studies, (Hockaday 1986; Kaplan 1986; Gallagher 1987; Wing 1988; Campbell 1990; Wing 1991; Milne 1994; Wing 1994; Pascale 1995; Uusitupa 1996; Agurs-Collins 1997; Glasgow 1997; Ligtenberg 1997; Samaras 1997; Glasgow 2000; Keyserling 2000; Tsihlias 2000), were judged to be at high risk of bias; one study, (de Bont 1981), was judged to be at medium risk of bias and no studies were judged to be at a low risk of bias.

## Effects of interventions

All of the primary publications for the included studies were indexed by both MEDLINE and the Cochrane Central Register of Controlled Trials. Five of the primary references were indexed within CINAHL and twelve of the primary publications were indexed within EMBASE. None of the primary references of trials included in this review were indexed by AMED.

**Heterogeneity**

Three tests for heterogeneity were applied for the data in comparison 3 (Dietary Advice versus Dietary Advice plus Physical Activity). These were for

- Comparison 3.02: Weight at twelve months
- Comparison 3.03: Glycated haemoglobin at six months
- Comparison 3.04: Glycated haemoglobin at twelve months

A fixed effects model was used to analyse these data. Significant heterogeneity was found within the studies included in category 3.02 ($I^2$ value of 56.8%, a $chi^2$ value of 4.63 with 2 degrees of freedom and a P-value of 0.10, (when analysed using a random effects model, these values remained the same)). Although in usual circumstances we would take this P-value to be an indicator of significance, we believe that this meta-analysis may have been affected by the large difference between the endpoint data for the two groups in the Samaras trial, (Samaras 1997). Although the participants in this trial were randomly allocated to one of two groups, at baseline the mean weight of the control group was 15kg more than the intervention group, and therefore when we looked at endpoint data, even though the participants in both groups had actually gained a small amount of weight, there was still a large difference between the control, (mean weight at twelve months was 99.0kg) and intervention groups, (mean weight at twelve months was 83.1kg), which suggested that the intervention was particularly successful, when indeed this could not be concluded from the change data provided. The studies included within category 3.03 were not heterogeneous ($I^2$ value of 34.1% and a P-value of 0.21), and showed a statistically significant change in glycated haemoglobin in favour of the addition of exercise to dietary advice. The studies included within category 3.04 were not heterogeneous ($I^2$ value of 0% and $chi^2$ value of 0.16 with 2 degrees of freedom and had a P-value of 0.92) and showed a statistically significant change in glycated haemoglobin in favour of the addition of exercise to dietary advice.

**Effects of the interventions**

***Comparison 01: Two types of diabetic dietary advice that did not differ in intent to lose weight***

Nine studies assessed two types of diabetic dietary advice that did not differ in intent to lose weight (dietary advice versus another form of dietary advice).

Campbell randomised 70 participants to one of two groups, usual diet or a reduced fat diet, (Campbell 1990). Usual diet was a typical portion exchange diet where caloric restriction was discussed. The intervention diet focused only on reducing fat. Change in weight was not reported in the paper using kilograms but through Body Mass Index. Body Mass Index decreased in the intervention group from 30.4 ± 4.8kg/$m^2$ at baseline to 29.6 ± 4.6kg/$m^2$ at six months compared to 32.0 ± 5.5kg/$m^2$ at baseline to 31.1 ± 5.1kg/$m^2$ at the six month follow-up. Glycated haemoglobin was not reported in the paper.

de Bont conducted a trial of a low-fat diet versus a low-carbohydrate diet, (de Bont 1981). One hundred and forty eight type 2 diabetic participants were randomised and 136 were followed up six months later. The primary outcome of the study was body weight, although this was reported separately for obese and non-obese participants. Weight in the obese participants decreased from 84.2kg at baseline to 81.5kg at six months, and in the non-obese participants weight decreased from 60.1kg to 59.7kg, which represents a decrease of 2.7kg for the obese participants and of 0.4kg for the non-obese participants in the low-fat diet. This was compared to the group following the low-carbohydrate diet where the subset of obese participants went from a baseline weight of 84.8kg to 83.9kg at six months, which was a decrease of 0.9kg; the non-obese participants began the trial with a mean weight of 59.0kg which by the end of six months had risen to 59.1kg, a rise of 0.1kg. Glycated haemoglobin was reported for all participants, and at baseline was 10.0% and decreased to 9.3% in the low-fat diet group at six months, whereas glycated haemoglobin was 10.1% at baseline and decreased to 9.5% in the low-carbohydrate group at six months. (Although mean changes were reported, the associated standard deviations/errors were not.)

The papers describing the Gallagher study, (Gallagher 1987), evaluated the American Diabetic Association exchange diet (40% carbohydrate, 40% fat and 20% protein) compared to a reduced fat diet, which was based upon the four basic food groups (which are: 1) fruits and vegetables, 2) whole grains, cereals, and bread, 3) dairy products, 4) meats, fish, poultry, eggs, dried beans, and nuts). Fifty-one male type 2 diabetic participants were randomised to one of the two groups. Weight and glycated haemoglobin was measured but not reported in the published articles. Attempts at personal communication with the authors proved to be unsuccessful.

Hockaday conducted a trial of 250 diabetic participants who were randomised to either a low-carbohydrate or a low-fat diet, (Hockaday 1986). However only subgroups of this trial have been reported in press. In the Hockaday study, the biggest subgroup of the trial was reported which was made up of 93 participants; 39 participants in the low-carbohydrate group and 54 participants in the low-fat group. Between baseline and one year, participants in the low-carbohydrate group had a mean weight loss of 3.8kg compared to a mean weight loss of 4.6kg in the low-fat diet group. (Although mean weight change was reported, the associated standard deviations/errors were not.) Changes in glycated haemoglobin were measured but not reported, although 'changes in glycated haemoglobin were not significant'.

Milne randomised 44 participants into either a low-carbohydrate or a low-fat diet, (Milne 1994). At baseline, participants had a mean weight of 83.1 ± 16.9kg and 80.8 ± 7.8kg for the low-carbohydrate and the low-fat diet groups respectively. At follow-up six months later, mean weight of the low-carbohydrate group had decreased to 82.1 ± 15.0kg (a decrease of 1.0kg) and the low-fat group mean weight had decreased to 80.7 ± 7.8kg (a decrease of 0.1kg). At the 18 month follow-up the mean weight of the low-carbohydrate group had remained stable at 82.1 ± 15.0kg and the low-fat group mean weight had also remained stable at 80.7 ± 13.8kg. Glycated haemoglobin decreased in the low-fat group from a baseline value of 9.8 ± 3.1% to 9.5 ± 2.6% at six months and then rose to 9.7 ± 2.6% at 18 months (an overall decrease of 0.1%) and a decrease in the low-carbohydrate group from a baseline measurement of 8.7 ± 2.3% to 8.5 ± 2.0% at six months and then remained stable at 8.5 ± 2% at the 18 month follow-up (an overall decrease of 0.2%).

Pascale described a trial that randomised 44 participants to follow either a calorie restricted diet (1000-1500 kcal per day) or a calorie restricted and low-fat diet (1000-1500 kcal per day, but less

than 20% of energy to come from fat), (Pascale 1995). Thirty-one participants were followed up (16 in the calorie restricted group and 15 in the calorie restricted and low-fat diet group) one year from baseline. Participants in the calorie restricted diet group decreased their mean weight one year later by 0.96 ± 3.7kg from a baseline measurement of 93.1 ± 13.0kg, compared to the calorie restricted and low-fat diet who decreased their mean weight one year later by 5.2 ± 7.3kg from the baseline measurement of 94.4 ± 9.5kg. At the twelve month follow-up, glycated haemoglobin levels rose in the calorie restricted diet group by 0.2 ± 1.7% (from 10.9% ± 2.7% at baseline). In contrast, at twelve months in the calorie restricted and low-fat diet group, the mean glycated haemoglobin levels remained the same (10.4 ± 1.9% at baseline with a change of 0.0 ± 1.9% at the one year follow-up).

In the study reported by Tsihlias, (Tsihlias 2000), 61 participants were randomised to receive either the low-fat (high-carbohydrate) or the monounsaturated fat diet; six months later 41 participants were followed up. The participants in the monounsaturated fat diet had an overall weight gain of 0.1kg over the six month period (from 78.8 ± 12.5kg at baseline to 78.9 ± 13.0kg at six months), compared with the participants in the low-fat diet group who lost an overall weight of 1.2kg over the six month period (from 77.4 ± 14.7kg at baseline to 76.2 ± 13.8kg at six months). There were 'no significant changes in glycaemic control' during the study period. Glycated haemoglobin data were obtained through a personal communication from Dr Wolever, and at baseline the monounsaturated fat diet group had a measurement of 7.7 ± 1.1%, which rose to 8.1 ± 1.5% at follow-up six months later. The participants in the low-fat (high carbohydrate) diet group at baseline had a blood glycated haemoglobin concentration of 8.1 ± 1.2%, which rose to 8.3 ± 1.5% at the six months follow-up.

As mentioned previously, there are three distinct groups that the above studies can be assigned to, (two groups are assessed here, the third (very-low-calorie diets versus low-calorie diets) is discussed in the next section). Clinical similarity, the decision to pool or not, study quality and an overview of the primary and secondary results will be discussed for each.

**(a) Studies that assessed exchange diets versus not using an exchange diet**

In both of these two studies, (Gallagher 1987; Campbell 1990), the American Diabetes Association exchange diet was compared with a standard reduced fat diet. The two trials recruited similar participants, (the only difference being that the Gallagher trial recruited solely male participants and the Campbell trial had mixed participation). The quality of the these two studies were assessed to be at high risk of being biased. Weight change was measured in the Gallagher study, where 9% of the intervention group lost weight compared to 25% of the control group losing weight, although no hard data were reported. Development of micro and macrovascular diabetic complications were not reported. Body Mass Index (as a measure of weight) was reported in the Campbell study, and the intervention group lost an average 0.8kg/$m^2$ compared to 0.9kg/$m^2$. Both studies measured, but did not report change in glycated haemoglobin.

These studies were clinically similar to permit pooling, but with only two studies in this category, pooling of the data was not possible. There were a total of 121 participants in this comparison grouping, where the quality of the trials was judged to be at high risk of being biased. No firm conclusions could be drawn from this comparison.

**(b) Studies that assessed low-fat diets versus moderate fat or low-carbohydrate diets**

In these five studies, (de Bont 1981; Hockaday 1986; Milne 1994; Pascale 1995; Tsihlias 2000), a low-fat diet was compared with either a moderate fat or a low-carbohydrate (although not necessarily mutually exclusive) diet. The trials recruited similar participants (with regards to age and sex). The quality of the trials were assessed, and five out of the six trials were assessed to be at a high risk of bias, and the other trial was assessed at a moderate risk of bias. Weight change was measured in all of the studies and in general more weight was lost in those groups that were following a low-fat diet. Development of micro and macrovascular diabetic complications were reported in one trial, (Hockaday 1986), although not in great detail, and these outcome measures were not reported in the other five trials. Glycated haemoglobin was reported, but there were only marginal changes in the trials, so no firm conclusions could be drawn from this.

All of these studies were clinically similar enough to permit pooling, but there were only two studies at six months which had data that could be pooled, (two of the other trials reported means without their associated standard deviations, (de Bont 1981; Hockaday 1986)), and one reported change data, (Pascale 1995) therefore it was not possible to look at the statistical heterogeneity in this comparison. There were a total of 378 participants in this comparison grouping, where generally the quality of the trials was judged to be at high risk of being biased. Conclusions could not be drawn from this comparison.

***Comparison 02: very-low-calorie Dietary Advice versus Another Form of Dietary Advice***

Two studies assessed very-low-calorie dietary advice versus another form of dietary advice (low-calorie diet).

Wing described a study in which 93 participants were randomised to either a balanced low-calorie diet (1000-1200 kcal per day) or a balanced low-calorie diet with two periods of twelve weeks of a very-low-calorie diet (of 400-500 kcal per day), (Wing 1994). Glycated haemoglobin was reported at 6 and 12 months, whereas weight was only reported at 12 months. Glycated haemoglobin for the low-calorie diet group was 10.5 ± 2.0% at baseline, decreasing to 8.8 ± 1.8% at six months, which rose to 9.2 ± 2.0% at twelve months and then rose again to 10.7 ± 2.4% at twenty-four months, compared to 10.4 ± 2.0% at baseline, a decrease to 8.4 ± 2.2% at six months, and then a slight rise to 8.9 ± 2.5% at twelve months and a greater rise to 10.4 ± 2.2% at twenty-four months for the very-low-calorie diet group. Mean body weight for the low-calorie diet group at baseline was 107.7 ± 18.7kg, which rose to 118.2 ± 11.6kg at twelve months, whereas participants in the very-low-calorie diet group had a mean baseline weight of 105.8 ± 19.4kg, which decreased to 91.6 ± 10.3kg at twelve months.

Wing randomised 36 people to either a low-calorie yet balanced diet (with a calorie goal of ˜1000 to 1500 kcal per day) or to a low-calorie (balanced) diet with a period of very-low-calorie diet (˜500 kcal per day) added for eight weeks, (Wing 1991). At baseline, the low-calorie group had a mean weight of 104.5 ± 21.5kg and at follow-up twelve months later their mean weight had dropped to 97.7 ± 17.4kg, compared with the very-low-calorie diet group

who at baseline had a mean weight of 102.1 ± 11.7kg and twelve months later had a mean weight of 93.5 ± 10.4kg. Both groups lost a significant amount of weight and weight loss was not improved by use of a very-low-calorie diet. Glycated haemoglobin was 10.4 ± 2.0% at baseline, which rose to 11.8 ± 2.7% at twelve months for the low-calorie group compared with a baseline glycated haemoglobin concentration of 10.4 ± 2.2%, which decreased to 9.2 ± 2.1% at the twelve month follow-up for the very-low-calorie group. The changes in glycated haemoglobin were statistically significant in both groups.

Both studies, (Wing 1991; Wing 1994), assessed the effects of a very-low-calorie diet against the effects of a low-calorie diet. The quality of these trials was judged to be at high risk of bias. Weight change was measured and reported and more weight was lost in the very-low-calorie diet groups at twelve months, although in the first trial, (Wing 1994), at twenty four months more weight loss was recorded in the low-calorie diet group. Development of micro and macrovascular complications were not reported in these studies. Glycated haemoglobin was recorded and reported, at twelve months there was a greater decrease in glycated haemoglobin in the very-low-calorie dietary advice group, compared with the very-low-calorie dietary advice group, however there was very little change from baseline to twenty-four months in either group.

The two studies were clinically similar enough to permit pooling, but as there were only two studies, it was not statistically possible to carry out an analysis on the heterogeneity of the data. One hundred and twenty-nine participants took part in these two trials and overall the trial quality was assessed to be at high risk of bias. The evidence suggests that longer term (twenty-four months) weight loss and glycaemic control is best achieved by low-calorie dietary advice, compared with the very-low-calorie dietary advice, although once again, the high potential for bias should be considered, and no firm conclusions could be drawn from this data.

***Comparison 03: Dietary Advice versus Dietary Advice plus Exercise***

Six studies compared interventions that examined the effect of dietary advice alone or dietary advice plus exercise on participants with type 2 diabetes.

Agurs-Collins randomised 64 participants to either a 'usual care' group or a 'usual diet with exercise' group, (Agurs-Collins 1997). Published details of the usual care control were brief; participants in this group attended a class that discussed methods of glycaemic control and they also received two mailings of nutrition information. Participants in the intervention arm (the diet plus exercise group) were advised to adhere to a diet that had ˜55% of kcal from carbohydrate, 20% from protein and less than 30% from fat. The exercise component consisted of a five minute warm up, 20 minutes of low-impact aerobic activity and 5 minutes of cool-down exercises and was carried out three times a week. Participants were also encouraged to exercise on their own twice more per week. At the end of the trial period, (i.e. six months later) participants in the usual care group had gained a mean weight of 2.0kg, (from 94.9 ± 20.1kg at baseline to 96.9 ± 21.6kg at the six month follow-up) compared to the participants in the diet plus exercise group who lost a mean weight of 2.6kg (from a baseline measurement of 93.3 ± 18.6kg to the six month follow-up value of 90.7 ± 20.1kg). Concentration of glycated haemoglobin in the usual care group rose over the six month period from a mean of 10.0 ± 1.9% to 11.5 ± 4.4%, compared to the diet plus exercise group whose mean glycated haemoglobin concentration decreased from 11.0 ± 1.7% to 9.9 ± 2.0% over the six month period.

In the study conducted by Uusitupa and colleagues, (Uusitupa 1996), 86 participants were randomised to either a standard dietary treatment group or a diet and exercise group. Standard dietary treatment consisted of reduced energy intake with emphasis on reducing intake of total fat and cholesterol. In the diet only group the mean weight changed from 92.2 ± 14.7kg at baseline to a measurement of 90.2 ± 14.3kg at the follow-up appointment at twelve months (which equates to a loss of 2.0 ± 11.4kg), compared to a weight change of 91.6 ± 14.5kg at baseline to a measurement of 86.5 ± 13.7kg at follow-up at twelve months (a loss of 5.1 ± 11.1kg) in the diet plus exercise group. Over the twelve months between baseline and follow-up, blood concentration of glycated haemoglobin decreased by 0.3 ± 1.5% in the diet only group (from 7.8 ± 2.0% at baseline to 7.5 ± 1.7% at twelve months) and by 0.5 ± 1.3% in the diet plus exercise group, (from 7.1 ± 1.8% at baseline to 6.6 ± 1.6% at twelve months).

Ligtenberg describes a study that randomised 58 participants to either continue with their usual diet or to receive physical training instructions for six weeks and then to continue training (with encouragement) at home for a following six weeks with a further fourteen weeks non-encouraged exercise at home, (Ligtenberg 1997). Weight was measured for both groups at the start and the endpoint of the trial, although no data were reported in the article, but the authors did state 'body weight did not change in either group during the whole study period'. Glycated haemoglobin was measured and reported at baseline and follow-up. In the diet only group, glycated haemoglobin rose by 0.2 ± 1.2% from baseline to follow-up at six months (from 8.8 ± 1.5% to 9.0 ± 1.6%) compared to a decrease of 0.2 ± 0.8% from baseline to follow-up for participants in the diet plus exercise group at six months (from 8.9 ± 1.0% to 8.7 ± 1.1%).

Samaras randomly assigned 26 non-exercising participants to receive either their usual diet or to receive their usual diet plus a structured exercise programme that ran for one hour, once a month for six months, (Samaras 1997). This was a moderately paced aerobic exercise session run by an exercise physiologist. This paper reported baseline and change data. Baseline measurement of weight for the diet only group was 98.2 ± 12.3kg which rose by 1.0 ± 2.9kg after six months and then rose (compared to the baseline measurement) at the twelve month follow-up by 0.8 ± 3.9kg compared to the diet plus exercise group where the baseline measurement was 83.0 ± 13.0kg, which decreased by 0.1 ± 2.7kg at the six month follow-up and then rose (compared to the baseline measurement) by 0.1 ± 3.9kg at the twelve month follow-up. Glycated haemoglobin concentrations in both groups of participants rose over the six month period of the trial, by 0.6 ± 0.9% in the control group (from 6.8 ± 2.2% at baseline) and by 0.1 ± 1.1% in the diet and exercise group (from 5.6 ± 1.1% at baseline). Measures of glycated haemoglobin also rose from baseline to the twelve month follow-up in both control and intervention groups, rising by 0.9 ± 1.0% in the diet only group (from 6.8 ± 2.2% at baseline) and by 0.9 ± 1.0% in the diet and exercise group (from 5.6 ± 1.1% at baseline).

In Kaplan's trial, (Kaplan 1986), 76 participants were randomly allocated to either ten weekly sessions of diet education, which focused on promoting a low-fat (30% of total energy), high

carbohydrate (55% of total energy) or to ten weekly sessions of diet and exercise education. Five of these weekly sessions were spent on diet education (as for the control group) and the following five were spent on supervised exercise in an adult fitness programme that included stretching and walking/jogging. Some data on weight change were measured and reported in the text of the article; the mean weight loss over six months for the diet only group was 3.5kg compared to a loss of 0.2kg for the diet plus exercise group from baseline to six months. (Although mean weight change was reported, the associated standard deviations/errors were not.) Glycated haemoglobin was measured but not reported except to say that there was 'no significant difference between the groups at baseline and six month follow up'.

Wing conducted a trial that had 30 participants randomised to either a diet only group or a diet plus exercise group, (Wing 1988). The diet only group had an individualised daily calorie goal and participants were taught to try and increase their complex carbohydrate intake and to decrease their intake of fat. Participants in the diet plus exercise group had the same dietary advice imparted but also walked for three miles a day three times a week and were instructed to exercise (on their own) for another session per week. At the twelve month follow-up, participants in the diet only group had lost a mean weight of 3.8 ± 15.4kg (from 102 ± 19.4kg to 98.2 ± 20.0kg) compared to a mean weight loss of 7.9 ± 18.3kg in the diet and exercise group at twelve months (from 104.1 ± 23.2kg to 96.2 ± 23.4kg). Glycated haemoglobin decreased in a similar fashion, in the diet only group, the concentration decreased by 0.8 ± 1.4% from baseline to follow-up at twelve months, (from 10.9 ± 1.9% to 10.1 ± 1.6%), compared to a decrease of 1.4 ± 1.5% in the diet plus exercise group (from 10.6 ± 1.9% to 9.2 ± 1.8%) over the same time period.

In these six studies, (Kaplan 1986; Wing 1988; Uusitupa 1996; Agurs-Collins 1997; Ligtenberg 1997; Samaras 1997), dietary advice was compared with dietary advice plus exercise. The trials recruited participants with similar characteristics (age and sex) and the quality of the trials were assessed to be at high risk of bias. Weight change was assessed in all trials, although not reported in two trials, (Ligtenberg 1997 and Kaplan 1986), and more weight was lost on average in the diet and exercise groups. The development of micro and macrovascular diabetic complications were not reported. Glycated haemoglobin was reported in most trials and decreased more in the participants in the dietary advice and exercise groups than those in the dietary advice only groups.

The six studies were clinically similar enough to permit pooling, (although the participants in the Samaras intervention group (Samaras 1997) were generally less heavy than the participants in the other trials in this category) and as there were three or more studies (with data that could be entered into the tables) in all but one category, (weight at six months), it was statistically possible to carry out an analysis on the heterogeneity of the data. For weight at twelve months the fixed effect model test for heterogeneity was significant. The test for heterogeneity was not significant for change in glycated haemoglobin at six and twelve months. At six months, dietary advice plus exercise was associated with a statistically significant mean (pooled weighted mean difference) decrease in glycated haemoglobin of 0.9% (with 95% confidence intervals of 0.4 to 1.3), and at twelve months, dietary advice plus exercise was associated with a statistically significant mean (pooled weighted mean difference) decrease in glycated haemoglobin of 1.0% (with 95% confidence intervals of 0.4 to 1.5). Three hundred and forty participants took part in these trials and overall the trial quality was assessed to be at high risk of bias. The evidence suggests that dietary advice plus exercise has the potential to have an impact on weight and glycaemic control, although the high potential for bias should be considered when interpreting the evidence.

***Comparison 04: Dietary Advice versus Dietary Advice plus Behavioural Approaches***

Three studies assessed dietary advice versus dietary advice plus behavioural approaches.

Glasgow conducted a trial that evaluated the efficacy of a personalised medical office-based intervention, (Glasgow 1997). Two hundred and six participants in this study were randomised to either receive usual care or usual care plus the brief intervention added in. All participants went half an hour early to their physician appointment and completed the baseline assessment, which included a touch screen assessment. Usual care participants received a high quality quarterly medical care intervention including regular assessment and follow-up of micro and macrovascular risk factors in addition to the touch screen computer assessment, although there was no focus on behavioural or psychosocial issues. The participants randomised to the 'brief' intervention group completed another five to ten minute touch screen dietary barriers assessment which immediately generated two printed feedback forms (one for the participant and one for the physician). No results were available in the articles, and attempts at personal communication proved to be unsuccessful.

Glasgow randomly assigned 160 participants to receive either the basic intervention or the community resources intervention, (Glasgow 2000). Participants assigned to the basic intervention group received information about low-fat eating. Participants assigned to the community resources group received the same information about low-fat eating as the basic group but in addition they had access to 'community resources' which was comprised of three-ring binder of indexed community resources, four newsletters identifying opportunities for participants to obtain support, a food frequency was mailed and tailored feedback was sent with advice to decrease intake of dietary fat. The participants in the community resources group decreased their mean weight from 96.2 ± 22.2kg at baseline to a follow-up value of 95.3 ± 20.9kg at six months, compared to the participants in the basic care group who decreased their mean weight from 90.3 ± 16.3kg at baseline to a follow-up value of 89.4 ± 16.8kg at six months. The concentration of glycated haemoglobin remained stable in the community resources group (a value of 7.3 ± 1.5% at baseline to a value of 7.3 ± 1.4% at the six month follow-up) compared with the basic intervention group who decreased their mean glycated haemoglobin from 7.6 ± 1.2% at baseline to 7.4 ± 1.2% at the six month follow-up.

Keyserling describes a trial that had 133 participants randomised to receive a clinic-based intervention or a clinic-based intervention plus community intervention, (Keyserling 2000). All participants were given a single loose-leaf notebook from which assessment and monitoring pages could be removed and filed. The clinic-based intervention consisted of four individual visits to a counsellor (sessions lasting for 195 minutes in total) where counsellors would negotiate with the participant on the selection of two or three goals from each assessment area within the provided notebook. The additional community intervention included phone calls to

participants and also three group sessions (two sessions between zero and six months and one between six and twelve months of the trial duration) which specifically addressed issues relating to cultural translation. Measurements were made at baseline, six and twelve months. In the clinic-based arm of the study, mean weight decreased from a baseline of 92.5 ± 22.1kg to 91.6 ± 21.7kg at the six month follow-up (a weight loss of by 0.9kg). Weight then increased by 1.9kg (92.5 ± 22.1kg at baseline to 94.4 ± 23.2kg) at the twelve month follow-up. In the clinic and community resources group, the mean weight remained the same from baseline measurement (93.9 ± 19.3kg) to six months (96.2 ± 19.3kg) and then increased by 2.3kg from baseline (93.9 ± 19.3kg) to the twelve month follow-up measurement (96.2 ± 19.3kg). In the clinic-based group, concentration of glycated haemoglobin increased from a baseline measurement of 11.0 ± 3.2% to the six month follow-up value of 11.1 ± 3.1%; from baseline measurement to the twelve month follow-up, there was a decrease from 11.0 ± 3.2% (baseline) to 10.9 ± 3.8% (twelve month follow-up). In comparison, concentration of glycated haemoglobin in participants in the clinic-based and community resources arm of the study remained stable at the six month follow-up (baseline measurement of 10.7 ± 2.3% compared to a six-month measurement of 10.7 ± 3.1%). At the twelve month follow-up, the glycated haemoglobin had risen from 10.7 ± 2.3% to 10.8 ± 2.9%.

In the three studies, (Glasgow 1997; Glasgow 2000; Keyserling 2000), dietary advice was compared with dietary advice plus a behavioural approach. The trials recruited participants with similar characteristics (age and sex) and the quality of the trials were assessed to be at high risk of bias. Weight change was assessed in all trials, although not reported at all in one of the trials, (Glasgow 1997), and although more weight was lost on average in the usual care (dietary advice only) groups, the amount of weight lost was not substantial. The development of micro and macrovascular diabetic complications was not reported in any of the studies. Glycated haemoglobin was reported in all but one trial, (Glasgow 1997), and generally more improvement in glycaemic control was seen in the usual care groups, although once more the changes were not significant.

Although the three studies were clinically similar enough to permit pooling; data were only available for two of these studies, therefore it was not possible for an analysis for heterogeneity to be carried out. There were a total of 499 participants in these three trials and overall the trial quality was assessed to be at high risk of bias. Firm conclusions could not be drawn from this comparison.

## DISCUSSION

### Summary of main results

This systematic review assessed eighteen randomised controlled trials of dietary advice studying a total of 1467 participants with type 2 diabetes mellitus. Only a minority of the trials examined hard clinical endpoints (such as death or development of macrovascular or microvascular diabetic complications), and those that did offered no details; most articles concerned themselves with the reporting and discussion of the participants' weight and blood glucose control.

Meta-analyses could not be carried out for the data within the dietary advice versus another (different) form of dietary advice, the very-low-calorie dietary advice versus low-calorie dietary advice category and the dietary advice versus dietary advice plus behavioural approaches categories, as there were not sufficient data to allow this.

Within the dietary advice versus dietary advice plus exercise category, there were small, yet significant changes seen in mean glycated haemoglobin at six months and twelve months in the four (Uusitupa 1996; Agurs-Collins 1997; Ligtenberg 1997; Samaras 1997) and three studies (Wing 1988; Uusitupa 1996; Samaras 1997) that contributed data to these analyses. At six months, dietary advice plus exercise was associated with a statistically significant mean (pooled weighted mean difference) decrease in glycated haemoglobin of 0.9% (with 95% confidence intervals of 0.4 to 1.3), and at twelve months, dietary advice plus exercise was associated with a statistically significant mean (pooled weighted mean difference) decrease in glycated haemoglobin of 1.0% (with 95% confidence intervals of 0.4 to 1.5).

A recent systematic review published in the Cochrane Library by Pirozzo, (Pirozzo 2002), suggested that there was no real difference between a low-fat diet and other weight reducing diets (when looking at long term weight loss) in overweight or obese people. The reviewers found that generally there were small, non-significant differences in weight loss between the participants in the control and intervention groups, however this difference was so small it was clinically insignificant. In this review, there were insufficient data to permit a meta-analysis, so conclusions on the effects of low-fat or other weight reducing diets were limited. However, clinically meaningful differences in glucose profile were not achieved.

Recently, some small-scale studies discussed the importance of dietary composition and low-carbohydrate diets in the management of type 2 diabetes mellitus (Boden 2005; Nielsen 2005). Low carbohydrate diets appeared to have a significant effect on decreasing HbA1c and weight reduction. However, more research is required on larger populations and with a strict control group.

This update review yielded no further studies which fit the criteria for inclusion and exclusion, and thus, no further trials have been added to the meta-analyses. However, one study by Ash (Ash 2003), investigated the effectiveness of three isocaloric dietary intervention groups (intermittent energy restriction, pre-portioned meals, and self-selected meals) for a duration of 12 weeks, with a follow-up after 18 months. Despite this study being a randomised control trial, of male adults with type 2 diabetes, it was excluded from any further analyses due to a dubious control arm to the study. However, the study concluded that a moderate energy restriction of 1400-1700kcal/day was effective in achieving a 6% weight loss and an improvement in glycaemic control. The method of implementation was found to be insignificant, whilst the weekly contact with health care professionals was suggested as the facilitator of the successful outcomes.

This review is relevant to physicians treating patients with type 2 diabetes, the results suggest that the addition of exercise alongside a reduced energy diet is the best way to promote better glycaemic control in type 2 diabetic patients, with this achieving consistent success in the trials reported in this review. We found no significant results in relation to weight.

In terms of the relevance of this review to future researchers, it is important to use these findings as a basis from which to generate hypotheses. As this review is high quality, we believe it can form a

firm foundation for research project proposals and identifying gaps in areas of further research.

### Limitations of the review

Diabetes mellitus is a major and growing health problem, and it has been predicted that the number of people with diabetes will double over the next 10 years (WHO/FAO 2003). The adoption of a more affluent and westernised lifestyle (i.e. a lifestyle where energy consumed is not matched or exceeded by energy expended) by some non-Western populations is also contributing to an increase in the diabetic population (Roman 1997). For all diabetic patients, achieving good glycaemic control is central to their well-being. Eighty to ninety percent of type 2 diabetic patients are overweight. With a decrease in weight, an improvement in glycaemic control is often observed.

There were not enough data in the studies that assessed one dietary advice versus another (different) type of dietary advice to enable us to reach any satisfactory conclusions. The data included in the trials in this review which assessed dietary advice plus behavioural approaches did not have the data to allow us to reach any satisfactory substantial conclusions. The studies which examined dietary advice versus dietary advice plus physical activity do suggest benefit from adoption of increasing physical activity levels alongside a reduced energy diet. We found no randomised controlled trials that examined dietary advice versus dietary advice plus alternative therapies.

Despite the frequency and severity of this condition, there are comparatively few trials and participants which have studied the impact of dietary advice and interventions. This may be partly due to type 2 diabetes being diagnosed relatively late by which time islet cell decompensation is reasonably advanced. As a consequence, even impressive degrees of weight loss can result in a rise rather than a fall in glycated haemoglobin. This is not an indication that the dietary intervention has failed but that the patient requires an oral anti-diabetic agent.

There is a need for far more research into effects of dietary change (with and without the addition of physical activity) on micro and macrovascular diabetic complications, weight and glycaemic control. Many of the outcomes we initially wished to look at in this review were not investigated in the included studies. Work carried out in the future should take care to record and publish mortality, change (or delay) in onset of anti-diabetic medication and also quality of life as these are outcomes of importance to people with type 2 diabetes. It would be desirable if measures of compliance would also be recorded and reported in published works.

The new long term study (known as 'look AHEAD' (Action for Health in Diabetes, (Kelley 2002)) sponsored by the National Institutes of Health, Bethesda, Maryland, USA, began in 2002 and aims to look at the long-term health effects of weight loss in men and women who are overweight and have type 2 diabetes who are 45 to 75 years of age. It is envisaged that participants will be assessed up to eleven and a half years after enrolling in the programme, which will reveal the long-term health effects of the trial.

Long-term, high-quality research in this area is necessary as the increase of type 2 diabetes is currently mirroring the recent rise of obesity that has been concerning health care professionals for the last ten years. Diabetes has now reached epidemic proportions and further immediate work is required in an attempt to halt it.

## AUTHORS' CONCLUSIONS

### Implications for practice

Using exercise as an adjunct to dietary advice, compared with dietary advice alone, appears to improve glycated haemoglobin at six and twelve months in people with type 2 diabetes. There were small, yet significant changes in glycated haemoglobin in the four (Uusitupa 1996; Agurs-Collins 1997; Ligtenberg 1997; Samaras 1997) and three (Wing 1988; Uusitupa 1996; Samaras 1997) studies that contributed data to these analyses. Dietary advice plus exercise was associated with a statistically significant mean (pooled weighted mean difference) decrease in glycated haemoglobin of 0.9% (with 95% confidence intervals of 0.4 to 1.3) at six months and of 1.0% (with 95% confidence intervals of 0.4 to 1.5) at twelve months (it should be noted that there is insufficient evidence to suggest what the effects of the dietary advice would be on weight or diabetic micro- and macrovascular diseases).

### Implications for research

The current available evidence assembled in this review points towards the benefit of increasing exercise in people with type 2 diabetes. Further high quality research to examine the addition of exercise alongside a reduced energy diet would be of great importance to either corroborate or contradict these findings. High quality research is needed to identify what types (low-fat/high-carbohydrate diet, modified-fat diet, restricted protein diet etc.), frequency and style (addition of behaviour modification or not) of dietary advice are most efficacious in the long-term for use by people with type 2 diabetes.

This review has also highlighted the lack of outcome studies of effects of dietary change on micro- and macrovascular diabetic complications. Many of the outcomes we wished to look at were not investigated in the included studies and therefore we recommend that any research carried out in the future should take care to note and report mortality, change (or delay) in onset of anti-diabetic medication and also quality of life as these are outcomes of importance to people with type 2 diabetes. Measures of compliance should also be recorded and reported in published works.

### Conclusions

There are no high quality data on the efficacy of the dietary treatment of type 2 diabetes programmes, however the data we do have indicate that the adoption of exercise appears to improve glycated haemoglobin at twelve months in people with type 2 diabetes, although the data presented are at high risk of bias. There is a need for well-designed studies which examine a range of interventions, although there is a promising study currently underway.

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* Indicates the major publication for the study

## CHARACTERISTICS OF STUDIES

### Characteristics of included studies *[ordered by study ID]*

#### Agurs-Collins 1997

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"Assigned randomly (1:1 ratio within the medication strata). Randomisation was supervised by the study statistician."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Urban Hospital<br><br>Number: 64 participants. (32 in IG, 32 in CG)<br><br>Age:<br>CG: 61.0 ± 5.7 years<br>IG: 62.4 ± 5.9 years.<br><br>Sex:<br>CG: 12% male; 88% female<br>IG: 34% male; 66% female<br><br>Diagnostic Details:<br>"Diagnosis of NIDDM by medical history"<br><br>Inclusion Criteria:<br>Should be 120% or greater than Metropolitan Weight Standards.<br>Have HbA1c greater than 8%<br>No medical contraindications<br><br>Other characteristics:<br>1. Smoking<br>CG: 6% smoke<br>IG: 18.8% smoke |
| Interventions | Trial intervention(s):<br>Intervention was promotion of adherence to diet. A weight loss of (around 4.5kgs) 10lbs was the aim. During the first three months, participants attended a single individual session (diet counselling) and twelve weekly group sessions comprising 60 minutes nutrition education plus 30 minutes exercising. For the following three months, participants attended six bi-weekly group sessions. |

**Agurs-Collins 1997** *(Continued)*

| | |
|---|---|
| | Comparison intervention(s): Comparison was a "usual care" control. The participants in this group attended one class on methods of glycaemic control. They also received two nutrition information mailings. |
| Outcomes | Participants in the usual care group went from a baseline mean weight of 94.9 ± 20.1kg to 96.9 ± 21.6kg at the six month follow-up<br>Participants in the diet plus exercise group saw their mean weight change from a baseline measurement of 93.3 ± 18.6kg to the six month follow-up value of 90.7 ± 20.1kg.<br><br>The mean concentration of glycated haemoglobin in the usual care group started at a baseline mean of 10.0 ± 1.9% and ended at a value of 11.5 ± 4.4% at the six month follow-up.<br>In the diet plus exercise group, the mean glycated haemoglobin concentration decreased from a baseline of 11.0 ± 1.7% to 9.9 ± 2% over the six month period.<br><br>1. Weight<br>2. BMI<br>3. HbA1c<br>4. Lipids<br>5. Blood Pressure<br><br>were all measured at six months. |
| Notes | Source of funding of trial: Dissertation research grant AG10361 from the National Institute on Ageing of the NIH. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Campbell 1990**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"subjects were allocated at random in groups of 8-10"<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: Australia<br><br>Setting: Diabetes Centre<br><br>Number: 70 participants. (32 in CG, 38 in IG). Follow-up on 62 participants.<br><br>Age:<br>CG: 59 ± 9 years<br>IG: 58 ± 9 years<br><br>Sex: |

**Campbell 1990** *(Continued)*

| | |
|---|---|
| | CG: 59% males and 41% females<br>IG: 55% males and 45% females.<br><br>Inclusion criteria:<br>1. Age of diabetes onset greater than 30 years old<br>2. Duration of diabetes more than three months<br>3. Duration of current treatment more than a year<br>4. No attendance at an education program in the previous six months<br>5. English speaking<br><br>No diagnostic details explicitly mentioned; just "NIDDM patients". |
| Interventions | Trial intervention(s):<br>An intensive programme which was based on the cognitive motivational theory by Heckhausen and Kuhl. Participants received simplified dietary instructions - they were told to decrease all fats and to increase consumption of all legumes. Caloric restriction was not discussed. Dosage was a total of 22 hours delivered over 11 weeks.<br><br>Comparison intervention(s):<br>A conventional program of diabetes education delivered over three days. This was comprised of simple explanations of food composition that emphasised a calorie-restricted-carbohydrate portion exchange diet with reduced fat intake and increased complex carbohydrate and fibre intake. |
| Outcomes | In the intervention group, BMI decreased from 30.4 ± 4.8kg/m2 at baseline to 29.6 ± 4.6kg/m2 at six months<br>In the control group, BMI decreased from 32.0 ± 5.5kg/m2 at baseline to 31.1 ± 5.1kg/m2 at six months.<br><br>1. BMI<br>2. Lipids<br>3. Fasting Blood Glucose<br><br>were all measured at six months. |
| Notes | Source of funding of trial: Health Services Research and Development Project Grant from the Australian Commonwealth Department of Health.<br><br>Eight participants dropped out, but no indication was given as to which group they were in. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**de Bont 1981**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"They were randomly allocated to receive...."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Yes |

**de Bont 1981** *(Continued)*

| | |
|---|---|
| | Quality Assessment Category: B (three points awarded) - moderate risk of bias. |
| Participants | Country: UK<br><br>Setting: Outpatient Clinic<br><br>Number: 148 randomised. (data presented for 136 participants: CG: 65 and IG: 71)<br><br>Age:<br>CG: 54 ± 8 years<br>IG: 56 ± 7 years<br><br>Sex:<br>Both groups: 100% female<br><br>Inclusion criteria:<br>None mentioned.<br><br>Exclusion criteria:<br>None mentioned.<br><br>Diagnostic criteria:<br>None mentioned.<br><br>Other characteristics:<br>CG: 37% smoke<br>IG: 44% smoke |
| Interventions | Trial intervention(s):<br>Advice was given which attempted to reduce the contribution of fat towards 30% of the prescribed energy intake (low fat diet).<br><br>Comparison intervention(s):<br>Usual care (low carbohydrate diet) - had their current diets reviewed and advice was given to encourage a carbohydrate intake not exceeding 40% of the prescribed energy intake.<br><br>Dose:<br>Participants in both groups had one meeting with a dietitian, followed by three home visits from a nutritionist (to encourage continued adaptation of diet towards dietary targets). |
| Outcomes | Weight was reported for obese and non-obese participants separately; glycated haemoglobin was reported for just the control and intervention groups. Although mean weight change was reported, the associated standard deviations/errors were not.<br><br>In obese participants the mean weight of participants in the low fat diet group went from 84.2kg at baseline to 81.5kg at six months, and for the participants in the low carbohydrate group the mean weight changed from a baseline weight of 84.8kg to 83.9kg at six months.<br><br>In the non-obese participants in the low fat diet group saw their mean weight decrease from 60.1kg to 59.7kg, compared to the group following the low carbohydrate diet who began the trial with a mean weight of 59.0kg which by the end of six months had risen to 59.1kg.<br><br>In the low fat diet group, the participants had a baseline glycated haemoglobin of 10% which decreased to 9.3% at six months.<br>The low carbohydrate group had a baseline glycated haemoglobin measurement of 10.1% which decreased to 9.5% at six months.<br><br>1. Weight<br>2. HbA1c<br>3. Lipids<br>4. Fasting Blood Glucose<br><br>were all measured at six months. |

**de Bont 1981** *(Continued)*

| | |
|---|---|
| Notes | Source of funding of trial:<br>None mentioned.<br><br>Eleven participants dropped out. No mention of which group they had been assigned to. Reasons for dropping out were:<br>a) Six participants had been withdrawn by their physicians.<br>b) Four participants withdrew themselves.<br>c) One patient died. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Gallagher 1987**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"... were randomly assigned..."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Outpatient Clinic<br><br>Number:<br>51 participants. 23 randomised to the CG and 28 randomised to the IG.<br><br>Age:<br>CG: 44.5 ± 12.7 years<br>IG: 47.8 ±16.2 years<br><br>Sex: 100% male in both groups.<br><br>Diagnostic criteria:<br>"... due to such factors as adult age at onset, absence of ketoacidotic episodes, history of treatment by oral agents or by diet alone prior to insulin treatment..."<br><br>Inclusion criteria:<br>1. Male<br>2. No more than 5% over ideal body weight in the past five years<br>3. Without a history of excess weight over 15% of ideal body weight<br>4. No complicating diagnoses affecting glucose or lipid levels |
| Interventions | Trial intervention(s): |

**Gallagher 1987** *(Continued)*

| | |
|---|---|
| | Unmeasured diet - based on the four basic food groups with advice on consistency of caloric intake, avoidance of refined sugars and limitation of saturated fats and cholesterol. Participants were seen by medical residents as necessary but at least every six months.<br><br>Comparison intervention(s):<br>ADA exchange diet - dietary composition advised was 40% carbohydrate, 40% fat and 20% protein. Simple sugars were restricted. Daily calorie levels were calculated considering the participants ideal body weight and level of activity. Participants were seen by medical residents as necessary but at least every three months. |
| Outcomes | Weight and glycated haemoglobin was measured but not reported in the published articles. Attempts at personal communication with the authors proved to be unsuccessful.<br><br>1. Weight<br>2. Fasting Blood Glucose<br><br>were all measured at four years. |
| Notes | Source of funding of trial:<br>None mentioned.<br><br>There was one type 1 diabetic in each group. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Glasgow 1997**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>Used a table of random numbers.<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Not mentioned, probably not<br><br>Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Outpatient Clinic<br><br>Number: 206 participants.<br>(98 in CG and 108 in IG)<br><br>Age:<br>CG: 63.1 ± 10.5 years<br>IG: 61.7 ± 12.1 years<br><br>Sex:<br>CG: 40% males, 60% females |

**Glasgow 1997** *(Continued)*

| | |
|---|---|
| | IG: 37% males, 63% females.<br><br>Diagnostic criteria:<br>None mentioned<br><br>Inclusion criteria:<br>1. Having type 1 or type 2 diabetes<br>2. Must be aged 40 or older |
| Interventions | Trial intervention(s):<br>A brief intervention in addition to their usual care - participants completed a five to ten minutes touch screen dietary barrier assessment, which generated two copies of a feedback form (one for the patient and one for the physician). Also had twenty minutes patient centered goal setting with interventionist.<br><br>Comparison intervention(s):<br>Usual care involved receiving a high quality quarterly medical care intervention. |
| Outcomes | No results were available in the articles, and attempts at personal communication proved to be unsuccessful.<br><br>1. BMI<br>2. HbA1c<br><br>were all measured at 12 months |
| Notes | Source of funding of trial:<br>Grant #3DK-R01-35524 from the National Institutes of Diabetes, Digestive, and Kidney Diseases. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Glasgow 2000**

| | |
|---|---|
| Methods | Trial design:<br>2 x 2 randomised factorial design<br><br>Randomisation procedure:<br>"...Participants were randomly assigned to one of four conditions..."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: Unclear<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Centre for Healthy Living<br><br>Number: 320 participants randomised across the four arms.<br>80 in the CG (67 present at 6 month follow-up) and 80 in the IG (75 present at 6 month follow-up)<br><br>Age: |

**Glasgow 2000** *(Continued)*

| | |
|---|---|
| | CG: 60.6 ± 9.5 years<br>IG: 60.5 ± 8.6 years<br><br>Sex:<br>CG: 33.7% male, 66.3% female.<br>IG: 52.6% male, 47.4% female.<br><br>Diagnostic criteria:<br>Welborn criteria for type 2 diabetes<br><br>Inclusion criteria:<br>1. Type 2 diabetes<br>2. Having a telephone<br>3. Not planning to move out of the area of a year<br><br>Exclusion criteria:<br>1. Type 1 diabetes<br>2. Planning to relocate out of area within one year |
| Interventions | Trial intervention(s): Community Resources Intervention. Comprised of a general pamphlet about low-fat eating and access to community resources which were a three-ring binder full of indexed community resources, four newsletters identifying opportunities for participants to obtain support. A food frequency questionnaire was mailed and tailored feedback was sent with advice to decrease dietary fat intake.<br><br>Comparison intervention(s):<br>A general pamphlet about low fat eating was handed out to the control group participants. |
| Outcomes | Participants in the community resources group decreased their mean weight from 96.2 ± 22.2kg at baseline to a follow-up value of 95.3 ± 20.9kg at six months.<br>Participants in the basic care group decreased their mean weight from 90.3 ± 16.3kg at baseline to a follow-up value of 89.4 ± 16.8kg at six months.<br><br>In the community resources group concentration of glycated haemoglobin remained stable (7.3 ± 1.5% at baseline to 7.3 ± 1.4% at the six month follow-up).<br>In the basic care group, the concentration of glycated haemoglobin decreased from 7.6 ± 1.2% at baseline to 7.4 ± 1.2% at the six month follow-up.<br><br>1. HbA1c<br>2. Weight<br>3. Dietary patterns<br>4. Lipids<br>5. Fat intake<br>6. Patient satisfaction measures<br>7. Quality of life<br><br>were all measured at 6 months. |
| Notes | Source of funding of trial: Supported by grant RO1-DK 35524-13 from the National Institutes of Health. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Hockaday 1986**

| | |
|---|---|
| Methods | Trial design: |

**Hockaday 1986** *(Continued)*

| | |
|---|---|
| | RCT<br><br>Randomisation procedure:<br>Using random number sequence<br><br>Allocation concealment:<br>Adequate<br><br>Blinding:<br>Recipients: No<br>Providers of Care: (a) Dietitians - No; (b) Doctors - Unclear.<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: UK<br><br>Setting: Eye hospital<br><br>Number:<br>At baseline: 250 participants were randomised at the start of the trial to either the low or high carbohydrate diet.<br>At 1 year: 93 followed up (54 in the low-carbohydrate diet group, 39 in the high-carbohydrate/modified-fat diet group)<br><br>Age:<br>At baseline: No mention is given.<br>At 1 year: Mean age of participants in the CG was 53 years (range was 22 to 65) and the mean age of the participants in the IG was 50 years (range was 24 to 65)<br><br>Sex:<br>At baseline: no mention of proportions of gender.<br>At 1 year: 51% male, 49% female.<br><br>Diagnostic criteria:<br>"Attendance at a diabetes mellitus clinic"<br><br>Inclusion criteria:<br>1. Between the recruitment period of 1973 and 1976, the participants had to be younger than 66 years old.<br>2. Participants must have untreated diabetes.<br><br>Exclusion criteria:<br>1. Imminent life-threatening condition.<br>2. Presence or history of endocrine disease, angina pectoris or neurological deficit.<br>3. Presence or past history of liver disease. |
| Interventions | Trial intervention(s):<br>High-carbohydrate/modified-fat diet. Originally designed to be a lipid lowering diet, therefore participants were advised that fats should provide no more than 30% of total energy (with a polyunsaturated fatty acids:saturated fatty acids ratio of ˜0.9). 20% of energy should come from protein and carbohydrate should provide 50% of total energy. Cholesterol was restricted to a maximum of 2.1grams per week. The advice to participants was given once only at the time of diagnosis.<br><br>Comparison intervention(s):<br>Standard low-carbohydrate diet. The advice given to participants was to restrict carbohydrate up to a maximum of 40% of total energy. Protein should provide up to 20% and fat to provide about 40% (with a ratio of polyunsaturated fatty acids:saturated fatty acids of ˜0.3) The advice to participants was given once only at the time of diagnosis. |
| Outcomes | Although mean weight change was reported, the associated standard deviations/errors were not. |

**Hockaday 1986** *(Continued)*

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| | Between baseline and one year, participants in the low carbohydrate group had a mean weight loss of 3.8kg compared to a mean weight loss of 4.6kg in the low fat diet. Changes in glycated haemoglobin were measured but not reported, although "changes in glycated haemoglobin were not significant".<br><br>1. Weight<br>2. BMI<br>3. Fasting plasma glucose<br><br>were all measured at twelve months. |
| Notes | Source of funding of trial: financial support received from the British Diabetic Association and from the International Sugar Research Foundation Inc.<br><br>NB:<br>1. It is important to note that different subsets of participants were reported on in the various papers that comprise this study. It has been very difficult to try and track these participants through.<br>2. The data which was reported was quite fragmented. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Low risk | A - Adequate |

**Kaplan 1986**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...were randomly assigned..."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Out-patient clinic<br><br>Number: 76 participants randomised to one of four groups (No indication of how many people assigned to each group was given)<br>70 participants were assessed at the 18 month follow-up.<br><br>Age:<br>CG: 54.87 ± 12.32 years<br>IG: 56.96 ± 8.95 years<br><br>Sex: Overall, 42% male, 58% women. No indication of the male:female split in the individual groups.<br><br>Diagnostic criteria:<br>1. Physicians had to complete a referral questionnaire providing confirmation of NIDDM status.<br><br>Inclusion criteria: |

**Kaplan 1986** *(Continued)*

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|---|---|
| | 1. Physician confirmed NIDDM.<br>2. Adult.<br><br>Exclusion criteria:<br>1. Heart problems. |
| Interventions | Trial intervention(s):<br>Participants assigned to this group followed the exchange diet recommended by the American Dietetic Association (around 1200 kcalories per day) and an exercise prescription based on the results of the graded exercise test. Over the ten week programme, participants received five (two hour) sessions on diet and five (two hour) sessions on exercise.<br><br>Comparison intervention(s):<br>Participants assigned to this group followed the exchange diet recommended by the American Dietetic Association (around 1200 kcalories per day). Participants attended ten (two hour) weekly sessions. |
| Outcomes | The mean weight loss over six months for the diet only group was 3.5kg.<br>The mean weight loss over six months for the diet plus exercise group was 0.2kg.<br><br>Although mean weight change was reported, the associated standard deviations/errors were not.)<br><br>Glycated haemoglobin was measured but not reported except to say that there was "no significant difference between the groups at baseline and six month follow up".<br><br>1. HbA1c<br>2. Body Composition<br>3. Lipids<br><br>were all measured at the six month follow-up. |
| Notes | Source of funding of trial: Supported by grants R01 AM27901 and K04 908098 from National Institutes of Health.<br><br>Financial Incentives:<br>A deposit of $40 was requested, some of which was returned as a result of attendance. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Keyserling 2000**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"were randomised"<br>"randomisation of individuals to treatment groups was stratified by practice site"<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear |

**Keyserling 2000** *(Continued)*

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|---|---|
| | Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Clinic and community based<br><br>Number: 200 participants were randomised in total (IG = 67; CG = 66)<br>At six month follow-up, 60 participants were followed-up in IG, 60 in CG.<br>At 1 year follow-up, 54 participants were followed-up in IG, 59 in CG.<br><br>Age:<br>IG: 58.5 years<br>CG: 59.8 years<br><br>(Means only given - no SD/SE given for this data)<br><br>Sex: 100% female in all groups<br><br>Diagnostic criteria:<br>1. Type 2 diabetes, defined as diagnosis of type 2 diabetes at age 20 or older with no history of ketoacidosis.<br><br>Inclusion criteria:<br>1. African-American women<br>2. Aged 40 years or older<br>3. Type 2 diabetes |
| Interventions | Trial intervention(s):<br>IG: A clinic and community based intervention designed to reduce total dietary fat. The clinic based intervention included individual counselling visits at month 1 (60 minutes), 2 (45 minutes), 3 (45 minutes) and 4 (45 minutes). All counselling and educational materials were included in a single loose-leaf notebook from which assessment and monitoring pages could be removed for filing and subsequent follow-up. Counsellors were encouraged to negotiate with the participant on the selection of two or three goals from each assessment area to accomplish before the next visit. The goal sheet was designed so it could be placed on the refrigerator where it would be seen frequently. The community intervention included phone calls to participants and group sessions. Alongside the four individual meetings IG participants had during the first six months, participants also had two group sessions of 90 minutes each and monthly telephone calls from the Community Diabetes Advisor. During the second six months, participants met for one group session of 90 minutes and maintaining monthly telephone contact with their Community Diabetes Advisor.<br><br>Comparison intervention(s):<br>CG: A clinic based intervention designed to reduce total dietary fat consisting of individual counselling visits at months 1 (60 minutes), 2 (45 minutes), 3 (45 minutes) and 4 (45 minutes). (These details are the same as for IG). |
| Outcomes | In the control (clinic) arm of the study, mean weight decreased from a baseline of 92.5 ± 22.1kg to 91.6 ± 21.7kg at the six month follow-up (a weight loss of by 0.9kg). Weight then increased by 1.9kg (92.5 ± 22.1kg at baseline to 94.4 ± 23.2kg) at the twelve month follow-up.<br>In the intervention (clinic and community resources) group, the mean weight remained the same from baseline measurement (93.9 ± 19.3kg) to six months (96.2 ± 19.3kg) and then increased by 2.3kg from baseline (93.9 ± 19.3kg) to the twelve month follow-up measurement (96.2 ± 19.3kg).<br><br>Concentration of glycated haemoglobin increased in the clinic-based group, from a baseline measurement of 11.0 ± 3.2% to the six month follow-up value of 11.1 ± 3.1%; and from baseline measurement to the twelve month follow-up, there was a decrease from 11.0 ± 3.2% (baseline) to 10.9 ± 3.8% (twelve month follow-up).<br>Concentration of glycated haemoglobin in participants in the clinic-based and community resources arm of the study remained stable at the six month follow-up (baseline measurement of 10.7 ± 2.3% compared to a six-month measurement of 10.7 ± 3.1%). At the twelve month follow-up, the glycated haemoglobin had risen from 10.7 ± 2.3% to 10.8 ± 2.9%. |

**Keyserling 2000** *(Continued)*

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|---|---|
| | 1. Weight<br>2. HbA1c<br>3. Physical activity<br>4. Lipids<br>5. Psychosocial assessment<br><br>were all measured at six months and twelve months from baseline. |
| Notes | Source of funding of trial: Supported in part by a cooperative agreement (U48/CCU409660) with the Centers for Disease Control and Prevention.<br><br>Drop-outs:<br>At six months, seven dropped out of the IG: two participants withdrew, three participants had medical illnesses, one did not return for the follow-up and one dropped out for other reasons. At six months, six participants dropped out of the CG: two participants withdrew, one moved and three did not return for the follow-up.<br><br>At one year, thirteen participants had dropped out of the IG, three participants withdrew, three had medical illnesses, six participants did not return and one dropped out for other reasons. Seven had dropped out of the CG, three withdrew, two participants moved, one did not return for the follow-up and one had medical illness. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Ligtenberg 1997**

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|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...patients were randomised to either.."<br><br>Allocation concealment: Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: The Netherlands<br><br>Setting: Unclear<br><br>Number: 58 participants randomised (28 in CG, 30 in IG). 51 participants completed the trial.<br><br>Age:<br>CG: 61 ± 5 years<br>IG: 63 ± 5 years<br><br>Diagnostic criteria:<br>Classified according to NDDG<br><br>Inclusion criteria:<br>1. Diabetes for at least a year |

**Ligtenberg 1997** *(Continued)*

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|---|---|
| | 2. BMI greater than 25<br>3. Diabetes treated with drugs/insulin<br>4. Older than 55 years old<br><br>Exclusion criteria:<br>1. Angina pectoris grades II-IV<br>2. Silent ischaemia<br>3. Autonomic neuropathy<br>4. Moderate/severe intermittent claudication<br>5. Impaired renal function<br>6. Presence of other major diseases<br><br>Sex:<br>CG: 36% male, 64% female<br>IG: 33% male, 67% female<br><br>Other characteristics:<br>All women were post-menopausal.<br><br>Smoking Status: 14% of participants in CG smoked and 30% of participants in IG smoked. |
| Interventions | Trial intervention(s):<br>A physical training programme, consisting of six weeks of supervised physical training, six weeks of physical training according to personalised training advice (encouragement given every two weeks) and fourteen weeks of training at home (without encouragement).<br><br>Comparison intervention(s):<br>Participants received a concise education programme, but no exercise advice. |
| Outcomes | Weight was measured but not reported in the article; the authors stated instead "body weight did not change in either group during the whole study period".<br><br>In the diet only group, glycated haemoglobin rose from 8.8 ± 1.5% at baseline to 9.0 ± 1.6% at follow-up at six months.<br>In the diet plus exercise group , glycated haemoglobin decreased from 8.9 ± 1.0% at the baseline measurement to 8.7 ± 1.1% at the six month follow-up.<br><br>1. HbA1c<br>2. Weight<br>3. VO2 Max<br><br>were recorded at six months. |
| Notes | Source of funding of trial: Supported financially by the Dutch Diabetes Fund |

***Risk of bias***

| **Bias** | **Authors' judgement** | **Support for judgement** |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Milne 1994**

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|---|---|
| Methods | Trial design:<br>3-armed RCT of which we are looking at both interventions (high carbohydrate diet as intervention and modified lipid diet as control)<br><br>Randomisation procedure:<br>"Randomly assigned" |

**Milne 1994** *(Continued)*

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|---|---|
| | "After stratification for age, sex, BMI and diabetes duration, participants were randomly allocated to one of three groups"<br><br>Allocation concealment: Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: New Zealand<br><br>Setting: Diabetes Clinic<br><br>Number: 70 participants randomised. (Unclear as to which groups they were assigned to)<br>44 participants began the trial.<br>43 participants completed the trial; IG: 22, CG: 21<br><br>Age:<br>IG: 59 ± 9.4 years<br>CG: 60 ± 9.2 years<br><br>Sex:<br>IG: 45% male, 55% female<br>CG: 48% male, 53% female<br><br>Diagnostic criteria:<br>None given.<br><br>Inclusion criteria:<br>Participants must have NIDDM<br>No other major illnesses |
| Interventions | Trial intervention(s):<br>IG: High carbohydrate/fibre diet which aimed to achieve/maintain a BMI less than or equal to 25, to derive 15% of energy from protein, 30% of energy from fat and 55% of energy from carbohydrate. There was also an aim to consume 10 or more grams of fibre per day and to increase the consumption of soluble fibre.<br><br>Comparison intervention(s):<br>Modified lipid diet which aimed to achieve/maintain a BMI less than or equal to 25, to derive 19% of energy from protein, 36% energy from fat. A saturated fatty acids:polyunsaturated fatty acids:monounsaturated fatty acids ratio of 1 and to derive 45% of energy from carbohydrate.<br><br>For all three arms of the study, for participants with a BMI greater than 25, the diets had an energy deficit of 500 kcalories. |
| Outcomes | In the low carbohydrate group the participants had a baseline mean weight of 83.1 ± 16.9kg and at follow-up six months later, the mean weight of this group had decreased to 82.1 ± 15.0kg. At the 18 month follow-up the mean weight of the low carbohydrate group had remained stable at 82.1 ± 15.0kg.<br><br>In the low fat group, the participants had a baseline mean weight of 80.8 ± 7.8kg and at follow-up six months later, the mean weight of this group had decreased to 80.7 ± 7.8kg. At the 18 month follow-up the mean weight had remained stable at 80.7 ± 13.8kg.<br><br>Glycated haemoglobin decreased in the low carbohydrate group from a baseline measurement of 8.7 ± 2.3% to 8.5 ± 2.0% at six months and then remained stable at 8.5 ± 2.0% at the 18 month follow-up.<br><br>Glycated haemoglobin decreased in the low fat group from a baseline value of 9.8 ± 3.1% to 9.5 ± 2.6% at six months and then rose to 9.7 ± 2.6% at 18 months.<br><br>1. HbA1c |

**Milne 1994** *(Continued)*

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|---|---|
| | 2. Lipids<br>3. Weight<br><br>were all measured at 18 months. |
| Notes | Source of funding of trial: Supported by a grant from the Health Research Council of New Zealand.<br><br>Drop-outs:<br>Six participants dropped out during the trial. (Two moved from the region, two started insulin treatment, one participant was diagnosed with cancer and one died.) |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Pascale 1995**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...subjects were randomly assigned..."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: Unclear<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Unclear<br><br>Number: 44 participants randomised (22 to CG; 22 to IG)<br>31 participants completed the trial (16 in CG; 15 in IG)<br><br>Age:<br>Overall, 56.5 ± 8.4 years<br><br>Sex: 100% women in both groups.<br><br>Diagnostic criteria:<br>1. Participants had to meet the criteria specified by the NDDG.<br>2. Diabetes controlled only by diet or oral medications.<br><br>Inclusion criteria:<br>1. Must be 20% or more above their ideal body weight.<br>2. Must be type 2 diabetic. |
| Interventions | Trial intervention(s):<br>Calorie and fat restriction - participants aimed to consume between 1000 and 1500 kcalories per day, (calorie goal was set depending on current weight). This diet has the same goals as the control group, but aimed to take less than 20% of calories from fat. |

**Pascale 1995** *(Continued)*

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| | Comparison intervention(s):<br>A calorie-restricted diet, of 1000-1500 kcalories per day goal, (calorie goal was set depending on current weight). Emphasis was on staying below the calorie goal prescribed, although participants were encouraged to keep fat intake at less than 30% of total calories per day.<br><br>Both groups had sixteen weekly group meetings, where information about healthy eating was disseminated. Follow-up meetings were held at one, two, four and six months after treatment. |
| Outcomes | Participants in the calorie restricted diet group decreased their mean weight by 0.96 ± 3.7kg at twelve months from a baseline of 93.1 ± 13.0kg.<br>Participants in the calorie restricted and low fat diet decreased their mean weight by 5.2 ± 7.3kg (at the one year follow-up) from their baseline of 94.4 ± 9.5kg.<br><br>At one year, glycated haemoglobin levels rose in the calorie restricted diet group by 0.2 ± 1.7% from 10.9% ± 2.7% at baseline.<br>At one year, glycated haemoglobin levels remained the same in the calorie restricted and low fat diet group, (a change of 0.0 ± 1.9% from 10.4 ± 1.9% at baseline).<br><br>1. Weight loss<br>2. Lipids<br>3. Glucose<br><br>were all measured at twelve months. |
| Notes | Source of funding of trial: Not mentioned<br><br>Drop-outs:<br>CG: Six participants dropped out.<br>IG: Seven participants dropped out. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Samaras 1997**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>Simple randomisation without stratification<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: Australia<br><br>Setting: Council run leisure centre<br><br>Number: 26 participants started the trial (13 in the CG, 13 in the IG)<br>No mention of dropouts, so assume all 26 participants completed the trial. |

**Samaras 1997** *(Continued)*

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| | Age:<br>CG: 60.5 ± 7.6 years<br>IG: 60.5 ± 7.8 years<br><br>Sex:<br>CG: 46% male; 54% female<br>IG: 31% male; 69% female<br><br>Diagnostic criteria:<br>"...all patients with NIDDM..."<br>No other details given.<br><br>Inclusion criteria:<br>1. Participants must have NIDDM<br>2. Participants must be aged between 40 and 70 years<br><br>Exclusion criteria:<br>1. A history, symptoms or signs of ischaemic heart disease<br>2. Current smoking<br>3. Poor comprehension of English<br><br>Other characteristics:<br>1. Smoking - all smokers were excluded from the study |
| Interventions | Trial intervention(s):<br>Participants followed their usual diet plus the Exercise Support Group Programme, which was conducted at the local leisure centre run by the local council. Staff involved included a nurse educator, exercise physiologist, dietitian, group facilitator and physician. Monthly sessions were of an hour with the group facilitator and one other team member dealing with various issues including safe exercise, exercise specific education to improve confidence, coping with diabetes and exercise and self-esteem issues. A moderately paced aerobic exercise group session followed with emphasis placed on exercising up to 50% of VO2max. The intervention phase lasted for six months, with the exercise sessions remaining available to intervention group participants after this time.<br><br>Comparison intervention(s):<br>Participants followed their usual diet, but received no additional encouragement to exercise beyond that which was routinely given at clinic appointments. The participants made visits to the clinic at baseline, six and twelve months for assessment. |
| Outcomes | Participants in the diet only group had a baseline weight of 98.2 ± 12.3kg which rose by 1.0 ± 2.9kg after six months and then rose (compared to the baseline measurement) at the twelve month follow-up by 0.8 ± 3.9kg.<br>Participants in the diet plus exercise group had a baseline weight of 83.0 ± 13.0kg, which decreased by 0.1 ± 2.7kg at the six month follow-up and then rose (compared to the baseline measurement) by 0.1 ± 3.9kg at the twelve month follow-up.<br><br>In the diet only group, the participants' glycated haemoglobin concentrations rose over the six month period of the trial, by 0.6 ± 0.9% from a baseline value of 6.8 ± 2.2%, and then rose again from the baseline value by 0.9 ± 1.0% at the twelve month follow-up.<br>In the diet and exercise group glycated haemoglobin rose by 0.1 ± 1.1% over the six month period of the trial, from 5.6 ± 1.1% at baseline and then at the twelve month follow-up rose from the baseline value by 0.9 ± 1.0%.<br><br>1. Weight<br>2. BMI<br>3. HbA1c<br>4. Lipids<br>5. SF-36 questionnaire<br>6. Activity levels<br><br>were all reported at six and twelve months. |

**Samaras 1997** *(Continued)*

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| Notes | Source of funding of trial: None mentioned.<br><br>Note:<br>The intervention used a model for changing behaviour (precede-proceed health promotion model). It has been shown that interventions based on such models can be more effective. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Tsihlias 2000**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...randomly assigned..."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: Unclear<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: Canada<br><br>Setting: Outpatient clinic<br><br>Number: 91 participants randomised (32 to CG; 29 to IG). 62 participants were followed up at six months (20 in CG; 21 in IG)<br><br>Age:<br>CG: 63.0 ± 6.7 years<br>IG: 62.9 ± 7.33 years<br><br>Sex:<br>At baseline:<br>CG: 53% male; 47% female<br>IG: 59% male; 41% female<br><br>At 6 months:<br>CG: 62% male; 38% female<br>IG: 45% male; 55% female<br><br>Diagnostic criteria:<br>None specifically mentioned just "subjects with type 2 diabetes"<br><br>Inclusion criteria:<br>1. Not pregnant.<br>2. Aged 40 to 80 years.<br>3. Have had diabetes for more than six months.<br>4. No biochemical evidence of impaired renal or hepatic function.<br>5. BMI greater than 36.<br>6. HbA1c greater than 6.5% and less than 10.1%. |

**Tsihlias 2000** *(Continued)*

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| | 7. Serum triglyceride concentration less than 10mmol/L.<br><br>Exclusion criteria:<br>1. Insulin or acarbose treatment. |
| Interventions | Trial intervention(s):<br>IG: High GI breakfast cereal (Corn Flakes, Puffed Rice, or Crispy Rice) was consumed for breakfast every day for six months. No other breakfast food was permitted.<br><br>Comparison intervention(s):<br>CG: High monounsaturated fatty acids intake without breakfast cereal. Not permitted to eat breakfast cereals and were given non-hydrogenated salted, or un-salted, soft-tub margarine or olive oil or both. |
| Outcomes | Participants in the monounsaturated fat diet saw their mean weight change from 78.8 ± 12.5kg at baseline to 78.9 ± 13.0kg at six months.<br>The mean weight for the participants in the low fat diet group decreased from 77.4 ± 14.7kg at baseline to 76.2 ± 13.8kg at six months.<br><br>At baseline the monounsaturated fat diet group had a glycated haemoglobin measurement of 7.7 ± 1.1%, which rose to 8.1 ± 1.5% at follow-up six months later.<br>The participants in the low fat diet group at baseline had a glycated haemoglobin concentration of 8.1 ± 1.2%, which rose to 8.3 ± 1.5% at the six months follow-up.<br><br>1. Body weight<br>2. HbA1c<br>3. Lipids<br><br>were all measured at six months. |
| Notes | Source of funding of trial: None mentioned. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Uusitupa 1996**

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|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...patients were randomised into one of two groups..."<br>"...were randomly placed into one of two groups..."<br>"...patients were randomised into the intervention or conventional groups..."<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: Finland |

**Uusitupa 1996** *(Continued)*

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|---|---|
| | Setting: Out-patient Clinic<br><br>Number: 86 participants were initially randomised. (40 participants in IG, 46 participants in CG)<br><br>Age: (Reported separately as male and female ages)<br>CG: 54.0 ± 6.6 years (male); 54.4 ± 6.4 years (female)<br>IG: 50.7 ± 6.7 years (male); 53.7 ± 6.3 years (female)<br><br>Sex:<br>CG: 61% male, 39% female<br>IG: 53% male, 47% female<br><br>Diagnostic criteria:<br>"Newly diagnosed NIDDM"<br>Diabetes mellitus as defined by WHO 1985<br><br>Inclusion criteria:<br>1. 40-64 years old<br>2. Having NIDDM as defined by WHO 1985<br><br>Exclusion criteria:<br>1. Unwilling to participate<br><br>Other characteristics:<br>18% of all participants were current smokers. |
| Interventions | Trial intervention(s):<br>After a common three month basic education programme, the intensified treatment participants were treated at the outpatient clinic. They visited it six times over the year (at two month intervals) where they were seen by a physician, dietitian and a nurse. Participants received printed and oral instructions for effective exercise training. Physical activity was measured by daily exercise records. The diet was implemented through individually planned energy-restriction.<br><br>Comparison intervention(s):<br>After a common three month basic education programme, the conventional "standard" treatment participants were treated at community health centres (that had originally referred the patients to the trial) for twelve months. They were advised to visit the health centres at two monthly intervals. Participants received basic information regarding the potential benefits of physical activity and also dietary instructions on how to reduce intake of total fat, total energy and dietary cholesterol. |
| Outcomes | Mean weight for the participants in the diet only group changed from 92.2 ± 14.7kg at baseline to a measurement of 90.2 ± 14.3kg at the twelve month follow-up.<br>Mean weight change for the participants in the diet plus exercise group was from 91.6 ± 14.5kg at baseline to a measurement of 86.5 ± 13.7kg at the twelve month follow-up.<br><br>Over the twelve months between baseline and follow-up, the mean blood concentration of glycated haemoglobin decreased by 0.3 ± 1.5% in the diet only group (from 7.8 ± 2% at baseline to 7.5 ± 1.7% at twelve months) and by 0.5 ± 1.3% in the diet plus exercise group, (from 7.1 ± 1.8% at baseline to 6.6 ± 1.6% at twelve months).<br><br>1. Weight<br>2. HbA1c<br>3. VO2 Max<br>4. Lipids<br><br>were all measured at twelve months. |
| Notes | Source of funding of trial: Supported financially by grants from the Juho Vainio Foundation, the Yrjo Jahnsson Foundation and in part by the Academy of Finland.<br><br>Drop-outs: |

**Uusitupa 1996** *(Continued)*

| | |
|---|---|
| | At 1 year 8 patients had dropped out (40 remained in conventional group, 38 remained in intervention group) |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Wing 1988**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...randomly assigned..."<br><br>Allocation concealment: Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Yes<br><br>Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: School of Medicine<br><br>Number: 30 participants were randomised to each arm of the study (15 participants to the CG and 15 participants to the IG).<br><br>Age:<br>CG: 55.1 ± 7.2 years<br>IG: 56.1 ± 6.4 years<br><br>Sex: Overall, 70% of participants were female and 30% of participants were male.<br><br>Diagnostic criteria:<br>Participants had to be diagnosed according to the criteria laid down by the NDDG.<br><br>Inclusion criteria:<br>1. Participants had to be more than 20% above ideal body weight.<br>2. Aged between 30 and 65.<br><br>Exclusion criteria:<br>1. Known history of CHD.<br>2. Taking medication that would interfere with weight loss or measuring heart rates.<br>3. Orthopaedic problems that would limit walking. |
| Interventions | Trial intervention(s):<br>A diet and exercise programme. The diet component of the intervention remained the same as diet for the control group (i.e. low fat high/carbohydrate diet). The exercise component was made up of walking a three mile route with therapists three times a week and were instructed to exercise once more (on their own) during the week.<br><br>Comparison intervention(s):<br>Participants were taught to increase their intake of complex carbohydrates, and to decrease their intake of fat in keeping with the revised American Diabetes Association dietary guidelines. A calorie goal |

**Wing 1988** *(Continued)*

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|---|---|
| | was prescribed, calculated by taking the patients pre-intervention weight, multiplying by 26 and subtracting 1000 calories.<br><br>Participants in both groups attended three meetings a week, for ten weeks, where they were shown demonstrations and films of low calorie cooking techniques, and how to practise behaviours such as portion size estimation and learning through role-play. |
| Outcomes | Participants in the diet only group had a mean weight of 102 ± 19.4kg at baseline and a mean weight of 98.2 ± 20kg twelve months later.<br>Participants in the diet and exercise group had a mean weight of 104.1 ± 23.2kg at baseline and a mean weight of 96.2 ± 23.4kg at the twelve month follow-up.<br><br>Glycated haemoglobin decreased in the diet only group, from a baseline value of 10.9 ± 1.9% to a follow-up value (at twelve months) of 10.1 ± 1.6%.<br>In the diet plus exercise group, glycated haemoglobin also decreased from a baseline value of 10.6 ± 1.9% to a twelve month follow-up value of 9.2 ± 1.8%.<br><br>1. Weight<br>2. Blood pressure<br>3. Lipids<br>4. HbA1c<br><br>were all measured at 12 months. |
| Notes | Source of funding of trial: Trial was supported by grant AM 29757-07 from the National Institutes of Health.<br><br>Drop-outs:<br>Two participants dropped out of the intervention group, although no reasons were given for these drop-outs. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Wing 1991**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...subjects were randomly assigned to either..."<br><br>Allocation concealment: Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (two points awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Unclear - possibly university based.<br><br>Number: 36 participants were randomised, 19 to CG and 17 to IG. (Note that the data that is presented is for 33 participants, 16 in CG and 17 in IG.) |

**Wing 1991** *(Continued)*

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|---|---|
| | Age:<br>CG: 51.9 ± 9.9 years<br>IG: 50.6 ± 7.7 years<br><br>Sex:<br>CG: 25% male, 75% female.<br>IG: 24% male, 76% female.<br><br>Diagnostic criteria:<br>Participants had type 2 diabetes as defined by the NDDG criteria.<br><br>Inclusion criteria:<br>1. Participants must be between 35 and 70 years old.<br>2. Must be 30% or more above ideal body weight.<br>3. Having type 2 diabetes as defined by the NDDG criteria.<br>4. Having no evidence of liver, renal or heart disease that would contraindicate the use of VLCD's. |
| Interventions | Trial intervention(s):<br>Participants were given a calorie goal of 4200 to 6300 joules per day (depending on their initial body weight) . Information was presented regarding the different caloric content of protein, fat and carbohydrate. Subjects were encouraged to increase their complex carbohydrate intake especially fibre, while decreasing dietary fat. Participants allocated to the IG followed this diet for one month, and then switched to a very-low-calorie diet (VCLD) for the second and third months of this program. While following the VCLD, participants were instructed to consume 1680 joules per day of lean fish, meat or fowl and had the option of using a liquid meal for occasional meals. After the two months, other foods were gradually re-introduced so that by week 17 all subjects were back onto a 4200 to 6300 joules per day diet.<br><br>Comparison intervention(s):<br>Participants were given a calorie goal of 4200 to 6300 joules per day (depending on their initial body weight) . Information was presented regarding the different caloric content of protein, fat and carbohydrate. Subjects were encouraged to increase their complex carbohydrate intake especially fibre, while decreasing dietary fat.<br><br>The control and intervention lasted for twenty weeks in total. |
| Outcomes | Mean body weight for the participants in the low calorie diet group at baseline was 107.7 ± 18.7kg, which rose to 118.2 ± 11.6kg at twelve months.<br>Participants in the very low calorie diet group had a mean baseline weight of 105.8 ± 19.4kg, which decreased to 91.6 ± 10.3kg at twelve months.<br><br>Glycated haemoglobin for the low calorie diet group was 10.5 ± 2.0% at baseline, decreasing to 8.8 ± 1.8% at six months, which rose to 9.2 ± 2.0% at twelve months and then rose again to 10.7 ± 2.4% at twenty-four months.<br><br>Glycated haemoglobin in the very low calorie diet group, was measured at 10.4 ± 2.0% at baseline, decreased to 8.4 ± 2.2% at six months, and then slightly rose to 8.9 ± 2.5% at twelve months and a greater rise to 10.4 ± 2.2% at twenty-four months for the very low calorie diet group.<br><br>1. Weight<br>2. BMI<br>3. Fasting plasma glucose<br>4. Fasting insulin<br>5. Lipids<br><br>were all measured at one year. |
| Notes | Source of funding of trial: Supported by grants from Western Pennsylvania Affiliate of the American Diabetes Association and the National Institutes of Health (NIDDK 29757),<br><br>Drop-outs:<br>Three participants dropped out of the CG. |

**Wing 1991** *(Continued)*

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

**Wing 1994**

| | |
|---|---|
| Methods | Trial design:<br>RCT<br><br>Randomisation procedure:<br>"...randomly assigned.."<br>No further detail given.<br><br>Allocation concealment:<br>Unclear<br><br>Blinding:<br>Recipients: No<br>Providers of Care: No<br>Outcome Assessors: Unclear<br><br>Quality Assessment Category: C (one point awarded) - high risk of bias. |
| Participants | Country: USA<br><br>Setting: Out-patient clinic<br><br>Number: 93 participants in total were randomised (48 to IG and 45 to CG) at the start of the trial.<br>At six months: No data reported.<br>At one year: 38 participants in IG and 41 participants in CG were followed up.<br>At two years: 36 participants in IG and 37 participants in CG were followed up.<br><br>Age:<br>At baseline:<br>IG: 52.3 ± 10.7 years<br>CG: 51.3 ± 8.7 years<br><br>Sex:<br>At baseline:<br>CG: 38% male; 62% female.<br>IG: 33% male; 67% female.<br><br>Diagnostic criteria:<br>1. All participants must meet NDDG criteria for type 2 diabetes mellitus.<br><br>Inclusion criteria:<br>1. More than 30% or 18kg above their ideal body weight (based on Metropolitan Life Insurance norms).<br>2. Aged 30 to 70 years.<br>3. No health problems that would preclude use of a VCLD. |
| Interventions | Trial intervention(s):<br>Low calorie diet (LCD) with two x twelve week periods of very low calorie diet (VCLD). Participants were prescribed a diet of 400 to 500 kcalories per day for two periods of twelve weeks (weeks 1 to 12 and weeks 24 to 36) interspersed with a low calorie diet (LCD: 1000 to 1200 kcalories per day as in the control diet). During the VLCD period of the study, participants could either consume up to 500 kcalories per day in liquid formula or from lean meat or fish. The LCD was slowly reintroduced after the VCLD period had finished, slowly increasing the calories over four weeks. |

**Wing 1994** *(Continued)*

| | |
|---|---|
| | Comparison intervention(s):<br>Low calorie diet of 1000 to 1200 kcalories per day. Participants were able to select the food items they wished but they were encouraged to spread their calories over the day and to limit dietary fat to less than 30% of calories. |
| Outcomes | The low calorie group had a baseline mean weight of 104.5 ± 21.5kg and a follow-up mean weight of 97.7 ± 17.4kg twelve months later.<br>The very low calorie diet group had a baseline mean weight of 102.1 ± 11.7kg and twelve months later had a mean weight of 93.5 ± 10.4kg.<br><br>The mean blood concentration of glycated haemoglobin in the low calorie group was 10.4 ± 2.0% at baseline, and 11.8 ± 2.7% at follow-up twelve months later.<br>The mean glycated haemoglobin concentration in the participants of the very low calorie group was 10.4 ± 2.2% at baseline and 9.2 ± 2.1% at the twelve month follow-up.<br><br>1. Weight<br>2. Glycaemic control<br>3. Blood pressure<br>4. Lipids<br><br>were all measured at six, twelve and twenty-four months. |
| Notes | Source of funding of trial: None mentioned<br><br>A deposit of US$150 per participant was required at the start of the study, but was refunded in full for reaching behavioural goals and attending assessment sessions.<br><br>Drop-outs:<br>At one year, ten participants from the IG and four from the CG had dropped out.<br>At two years, twelve participants from the IG and eight from the CG had dropped out. |

***Risk of bias***

| Bias | Authors' judgement | Support for judgement |
|---|---|---|
| Allocation concealment? | Unclear risk | B - Unclear |

HbA1c = Glycated Haemoglobin
BMI = Body Mass Index
CG = Control Group
IG = Intervention Group
NDDG = National Diabetes Data Group
WHO = World Health Organisation
Unless specified, ## ± ## is mean ± SD

## Characteristics of excluded studies *[ordered by study ID]*

| Study | Reason for exclusion |
|---|---|
| ADA 2000 | (a) Was not a randomised controlled trial |
| ADA 2002 | (a) Was not a randomised controlled trial |
| Aizawa 2001 | (a) Was not a randomised controlled trial |
| Andersen 1987 | (a) Was not a randomised controlled trial |
| Anderson 2003 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Anonymous 1988 | (a) Was not a randomised controlled trial |
| Anonymous 1990 | (a) Was not a randomised controlled trial |
| Arky 1983 | (a) Was not a randomised controlled trial |
| Arora 2005 | (a) Was not a randomised controlled trial |
| Ash 2003 | (a)<br>(b)<br>(c)<br>(d)<br>(e)<br>(f)<br>(g)<br>(h)<br>(i) Was not a true control |
| Barnard 1982 | (a)<br>(b) Was not a six month intervention |
| Barnard 1983 | (a) Was not a randomised controlled trial |
| Barnard 1992 | (a)<br>(b) Was not a six month intervention |
| Barnard 1994 | (a)<br>(b) Was not a six month intervention |
| Barnard 1997 | (a)<br>(b) Was not a six month intervention |
| Barnett 2001 | (a) Was not a randomised controlled trial |
| Bassand 2006 | (a) Was not a randomised controlled trial |
| Bennett 1997 | (a) Was not a randomised controlled trial |
| Berg 2003 | (a) Was not a randomised controlled trial |
| Bergenstal 1999 | (a) Was not a randomised controlled trial |
| Bhaskarabhatla 2004 | (a) Was not a randomised controlled trial |
| Bjorntorp 1992 | (a) Was not a randomised controlled trial |
| Blake 1992 | (a) Was not a randomised controlled trial |
| Blonk 1994 | (a) Was not a randomised controlled trial |
| Bloomgarden 2000 | (a) Was not a randomised controlled trial |
| Bloomgarden 2004 | (a) Was not a randomised controlled trial |
| Bloomgarden 2005 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Boden 2005 | (a) Was not a randomised controlled trial |
| Bonanome 1991 | (a)<br>(b) Was not a six month intervention |
| Bonnefont-Rousselot | (a) Was not a randomised controlled trial |
| Borghouts 2000 | (a) Was not a randomised controlled trial |
| Boule 2001 | (a) Was not a randomised controlled trial |
| Boule 2002 | (a) Was not a randomised controlled trial |
| Bourn 1996a | (a) Was not a randomised controlled trial |
| Bourn 1996b | (a) Was not a randomised controlled trial |
| Brand-Miller 2003 | (a) Was not a randomised controlled trial |
| Brown 1996 | (a) Was a randomised controlled trial<br>(b)<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Brown 2002 | (a) Was a randomised controlled trial<br>(b)<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Brownell 1998 | (a) Was not a randomised controlled trial |
| Calle-Pascual 1992 | (a) Was not a randomised controlled trial |
| Campbell 2001 | (a) Was not a randomised controlled trial |
| Capstick 1997 | (a)<br>(b) Was not a six month intervention |
| Carr 2005 | (a)<br>(b)<br>(c)<br>(d) Type 2 diabetes not involved- Impaired Glucose Tolerance |
| Cefalu 2005 | (a) Was not a randomised controlled trial |
| Chakravarthy 2002 | (a) Was not a randomised controlled trial |
| Charatan 2001 | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Chen 1987 | (a)<br>(b) |

| Study | Reason for exclusion |
|---|---|
| | (c)<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Chen 2001 | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Chipkin 2001 | (a) Was not a randomised controlled trial |
| Christensen 1998 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control not measured |
| Ciliska 1995 | (a) Was not a randomised controlled trial |
| Clark 2001 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Coleman 2003 | (a) Was not a randomised controlled trial |
| Collins 1995 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice (was a pre-prepared meal plan) |
| Colman 1995 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Comi 1995 | (a)<br>(b) Was not a six month intervention |
| CRD 2002 | (a) Was not a randomised controlled trial |
| Creviston 2001 | (a) Was not a randomised controlled trial |
| de Fine Olivarius 06 | (a) Was not a randomised controlled trial |
| de Sonnaville 1997 | (a) Was not a randomised controlled trial |
| Delahanty 1995 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Dengel 1998 | (a)<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Devlin 1986 | (a) Was not a randomised controlled trial |
| Dey 2002 | (a) Was not a randomised controlled trial |
| Dhindsa 2003 | (a) Was not a randomised controlled trial |
| Dunstan 2002 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Elson 1998 | (a) Was not a randomised controlled trial |
| Eriksson 1991 | (a) Was a randomised controlled trial, but was a cross-over trial which were not included |
| Eriksson 1997 | (a) Was not a randomised controlled trial |
| Eriksson 1998 | (a) Was not a randomised controlled trial |
| Eriksson 1999a | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Eriksson 1999b | (a) Was not a randomised controlled trial |
| Ernst 2001 | (a) Was not a randomised controlled trial |
| Evans 1995 | (a) Was not a randomised controlled trial |
| Evans 1997 | (a) Was not a randomised controlled trial |
| Evans 2002 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Fletcher 2002 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Franz 1995 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Frati 1990 | (a)<br>(b)<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Fujinuma 1999 | (a)<br>(b) Was not a six month intervention |
| Funnell 2004 | (a) Was not a randomised controlled trial |
| Gaede 2001 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not only dietary advice (was a multifactorial intervention) |
| Gaede 2006 | (a) Was not a randomised controlled trial |
| Gannon 2006 | (a) Was not a randomised controlled trial |
| Garg 1990 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Garg 1993 | (a) Was not a randomised controlled trial |
| Garg 1998 | (a) Was not a randomised controlled trial |
| Gautier 1995 | (a) Was not a randomised controlled trial |
| Genuth 2003 | (a) Was not a randomised controlled trial |
| Glasgow 1989 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Goldberg 1998 | (a) Was not a randomised controlled trial |
| Gonzelez 1994 | (a) Was not a randomised controlled trial |
| Grundy 1991 | (a) Was not a randomised controlled trial |
| Grundy 1999b | (a) Was not a randomised controlled trial |
| Guerrero-Romero 2005 | (a) Was not a randomised controlled trial |
| Gumbiner 1998 | (a)<br>(b) Was not a six month intervention |
| Gumbiner 1999 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Hadden 1982 | (a) Was not a randomised controlled trial |
| Hamdy 2001 | (a) Was not a randomised controlled trial |
| Hamilton 1992 | (a) Was not a randomised controlled trial |
| Hanefeld 1989 | (a)<br>(b) Was not a six month intervention |
| Hart 2006 | (a) Was not a randomised controlled trial |
| Heath 1987 | (a) Was not a randomised controlled trial |
| Heath 1991 | (a) Was not a randomised controlled trial |
| Heine 1989 | (a) Was a randomised controlled trial, but was a cross-over trial which were not included |
| Held 1991 | (a) Was not a randomised controlled trial |
| Helmrich 1994 | (a) Was not a randomised controlled trial |
| Henry 1991 | (a) Was not a randomised controlled trial |
| Hensrud 2001 | (a) Was not a randomised controlled trial |
| Holmes 1987 | (a) Was not a randomised controlled trial |
| Horrocks 1987 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Hu 1999 | (a) Was not a randomised controlled trial |
| Hu 2001a | (a) Was not a randomised controlled trial |
| Hu 2001b | (a) Was not a randomised controlled trial |
| Huh 1996 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Hutton 2004 | (a) Was not a randomised controlled trial |
| Ismail 2004 | (a) Was not a randomised controlled trial |
| Ito 2001 | (a) Was not a randomised controlled trial |
| Ivy 1997 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| James 1998 | (a) Was not a randomised controlled trial |
| Janssen 2002 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Jenkins 2003 | (a) Was not a randomised controlled trial |
| Jenkins 2004 | (a) Was not a randomised controlled trial |
| Jeppesen 1997 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Kao 2000 | (a) Was not a randomised controlled trial |
| Karlstrom 1989 | (a) Was not a randomised controlled trial |
| Kelley 1995 | (a) Was not a randomised controlled trial |
| Kelley 1999 | (a) Was not a randomised controlled trial |
| Kelley 2001 | (a) Was not a randomised controlled trial |
| Kelly 2000 | (a) Was not a randomised controlled trial |
| Kennedy 1982 | (a) Was not a randomised controlled trial |
| Kirk 2003 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c)<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Kirkman 1994 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Kligler 2003 | (a) Was not a randomised controlled trial |
| Knowler 1994 | (a) Was not a randomised controlled trial |
| Kraus 2001 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Kriska 2000 | (a) Was not a randomised controlled trial |
| Kriska 2002 | (a) Was a randomised controlled trial<br>(b)<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |

| Study | Reason for exclusion |
|---|---|
| Kulkarni 2006 | (a) Was not a randomised controlled trial |
| Lehman 2005 | (a) Was not a randomised controlled trial |
| Lehmann 1998 | (a) Was not a randomised controlled trial |
| Liao 2003 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Ligtenberg 1998 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Lindstrom 2003 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Ling 1994 | (a) Was not a randomised controlled trial |
| Little 1996 | (a) Was not a randomised controlled trial |
| Lodha 2000 | (a) Was not a randomised controlled trial |
| Lomasky 1990 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Maggio 1997 | (a) Was not a randomised controlled trial |
| Manson 1992b | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Manson 1994 | (a) Was not a randomised controlled trial |
| Mazzeo 2001 | (a) Was not a randomised controlled trial |
| McCarty 1997 | (a) Was not a randomised controlled trial |
| Meinders 1992 | (a) Was not a randomised controlled trial |
| Melander 1996 | (a) Was not a randomised controlled trial |
| Mensink 2003a | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice, but the intervention and control groups did not received different advice |
| Mensink 2003b | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study |

| Study | Reason for exclusion |
|---|---|
| | (e) Main intervention was dietary advice, but the intervention and control groups did not received different advice |
| Metz 2000 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice (was a pre-prepared meal plan) |
| Miles 2000 | (a) Was not a randomised controlled trial |
| Miller 1996 | (a) Was not a randomised controlled trial |
| Monteiro 2005 | (a) Was not a randomised controlled trial |
| Montori 2001 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Morelli 2000 | (a) Was not a randomised controlled trial |
| Mustajoki 2001 | (a) Was not a randomised controlled trial |
| Myers 2001 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Namdul 2001 | (a) Was a randomised controlled trial<br>(b)<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Narayan 1998 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Narayan 2001 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |

| Study | Reason for exclusion |
|---|---|
| Neff 2003 | (a) Was not a randomised controlled trial |
| Nicollerat 2000 | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Nielsen 2005 | (a) Was not a randomised controlled trial |
| Nilsson 1992 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Norris 2004 | (a) Was not a randomised controlled trial |
| Odegaard 2006 | (a) Was not a randomised controlled trial |
| Oli 1984 | (a) Was not a randomised controlled trial |
| Paffenbarger 1997 | (a) Was not a randomised controlled trial |
| Paisey 1995 | (a) Was not a randomised controlled trial |
| Paisey 1998 | (a) Was not a randomised controlled trial |
| Paisey 2002 | (a) Was not a randomised controlled trial |
| Pan 1997 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Parfitt 1994 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Pascale 1992 | (a) Was not a randomised controlled trial |
| Pejic 2006 | (a) Was not a randomised controlled trial |
| Perez-Martin 2001 | (a) Was not a randomised controlled trial |
| Perri 1993 | (a) Was not a randomised controlled trial |
| Pfohl 2001 | (a) Was not a randomised controlled trial |
| Phillips 2006 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Pigman 2002 | (a) Was not a randomised controlled trial |
| Pischke 2006 | (a) Was not a randomised controlled trial |
| Poppitt 2002 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Pratley 2000 | (a)<br>(b)<br>(c) Participants were over 18 years old and human<br>(d)<br>(e) Main intervention was not dietary advice |
| Pritchard 1999 | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Rabkin 1983 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Racette 2001 | (a) Was not a randomised controlled trial |
| Ratner 2006 | (a) Was not a randomised controlled trial |
| Raz 1994 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Reaven 1995 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Rendell 2006 | (a) Was not a randomised controlled trial |
| Riccardi 2005 | (a) Was not a randomised controlled trial |
| Ronnemaa 1986 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Rosell 1999 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d)<br>(e) Main intervention was dietary advice<br>(f) Weight loss not measured |
| Rowley 2000 | (a) Was a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Rubin 2002 | (a) Was not a randomised controlled trial<br>(b)<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Rukgauer 2006 | (a) Was not a randomised controlled trial |
| Ryan 2003 | (a) Was not a randomised controlled trial |
| Rybka 1987 | (a) Was not a randomised controlled trial |
| Sato 2000a | (a)<br>(b) Was not a six month intervention |
| Scheen 2000 | (a) Was not a randomised controlled trial |
| Schwartz 2006 | (a) Was not a randomised controlled trial |
| Shahid 2000 | (a) Was not a randomised controlled trial |
| Sherwin 2003 | (a) Was not a randomised controlled trial |
| Shintani 2001 | (a) Was not a randomised controlled trial |
| Siddiqui 2004 | (a) Was not a randomised controlled trial |
| Sigal 2004 | (a) Was not a randomised controlled trial |
| Simmons 1997 | (a) Was not a randomised controlled trial |
| Smith 1993 | (a) Was not a randomised controlled trial |
| Solano 2006 | (a) Was not a randomised controlled trial |
| Solano 2006b | (a) Was not a randomised controlled trial |
| Sone 2002 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Song 2006 | (a) Was not a randomised controlled trial |
| Spelsberg 1995 | (a) Was not a randomised controlled trial |
| Srimanunthiphol 2000 | (a) Was not a randomised controlled trial |
| Starke 1994 | (a) Was not a randomised controlled trial |
| Steyn 2004 | (a) Was not a randomised controlled trial |
| Stone 2001 | (a) Was not a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| Strandberg 2000 | (a) Was not a randomised controlled trial |
| Sutherland 2004 | (a) Was not a randomised controlled trial |
| Svendsen 1996 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was not involved in study |
| Swinburn 2001 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Takekoshi 1987 | (a)<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Tamler 2006 | (a) Was not a randomised controlled trial |
| Taniguchi 2000 | (a) Was not a randomised controlled trial |
| Tariq 2001 | (a)<br>(b) Was not a six month intervention |
| Thomson 2001 | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Toeller 1993 | (a) Was not a randomised controlled trial |
| Toeller 2005 | (a) Was not a randomised controlled trial |
| Torjesen 1997 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Trento 2002 | (a)<br>(b)<br>(c)<br>(d)<br>(e) Dietary advice was not main intervention- general lifestyle advice |
| Tsujiuchi 2002 | (a) Was a randomised controlled trial |

| Study | Reason for exclusion |
|---|---|
| | (b) Was not a six month intervention |
| Tudor-Locke 2000 | (a) Was not a randomised controlled trial |
| Tuomilehto 1992 | (a) Was not a randomised controlled trial |
| Tuomilehto 2001b | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Turner 1990 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Turner 1995 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was not dietary advice |
| Turner 1996a | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Turner 1996b | (a)<br>(b)<br>(c) Participants were over 18 years old and human<br>(d)<br>(e) Main intervention was not dietary advice |
| UKPDS 1993 | (a) Was a randomised controlled trial<br>(b)<br>(c)<br>(d)<br>(e) Main intervention was not dietary advice |
| Ullom-Minnich 2004 | (a) Was not a randomised controlled trial |
| Uusitupa 1989 | (a) Was not a randomised controlled trial |
| Uusitupa 2000 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |

| Study | Reason for exclusion |
|---|---|
| Valensi 2006 | (a) Was not a randomised controlled trial |
| Vuksan 2001 | (a) Was not a randomised controlled trial |
| Vuori 2001 | (a) Was not a randomised controlled trial |
| Walker 1996 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Walker 2001 | (a) Was not a randomised controlled trial |
| Wall 1973 | (a) Was not a randomised controlled trial |
| Wallberg-H 1998 | (a) Was not a randomised controlled trial |
| Warnken 2005 | (a) Was not a randomised controlled trial |
| Wei 2000 | (a) Was not a randomised controlled trial |
| Wein 1999 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Weinstock 1998 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study<br>(e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Welch 2006 | (a) Was not a randomised controlled trial |
| Wild 2004 | (a) Was not a randomised controlled trial |
| Williams 2000 | (a) Was not a randomised controlled trial |
| Williamson 2000 | (a)<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d)<br>(e) Main intervention was not dietary advice |
| Wing 1993 | (a) Was not a randomised controlled trial |
| Wing 1995 | (a) Was not a randomised controlled trial |
| Wing 1998 | (a) Was a randomised controlled trial<br>(b) Was a six month intervention<br>(c) Participants were over 18 years old and human<br>(d) Type 2 diabetes was involved in study |

| Study | Reason for exclusion |
|---|---|
| | (e) Main intervention was dietary advice<br>(f) Weight loss measured<br>(g) Diabetic control measured<br>(h) Prevention study |
| Wing 2001 | (a) Was not a randomised controlled trial |
| Wolever 2002 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Wolever 2003 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Wolffenbuttel 1989 | (a) Was not a randomised controlled trial |
| Wylie-Rosett 2006 | (a) Was not a randomised controlled trial |
| Yamamoto 2001 | (a) Was a randomised controlled trial<br>(b) Was not a six month intervention |
| Yamaoka 2005 | (a) Was not a randomised controlled trial |
| Yeh 2003 | (a) Was not a randomised controlled trial |
| Yoo 2005 | (a) Was not a randomised controlled trial |
| Yoshioka 1989 | (a)<br>(b) Was not a six month intervention |
| Zhi-cheng 1994 | (a) Was not a randomised controlled trial |

NB1. Any "no" disqualifies study from inclusion.
NB2. If excluded because "prevention" these will be included in a second prevention review.

## Characteristics of ongoing studies *[ordered by study ID]*

### Kelley 2002

| | |
|---|---|
| Trial name or title | Look AHEAD<br>(Action in Health in Diabetes) |
| Methods | |
| Participants | Men and women who are overweight and have type 2 diabetes. Participants must be aged between 45 and 75 years of age. |
| Interventions | Participants will receive either a diabetes support and education program or a long-term lifestyle change program for weight loss and weight maintenance.<br>Both programs are offered at no cost to the participants. |
| Outcomes | "Long term effects of weight loss" |
| Starting date | During 2002, exact date unspecified. |
| Contact information | |

**Kelley 2002** *(Continued)*

| | |
|---|---|
| Notes | The study is sponsored by the National Institutes of Health, Bethesda, Maryland, USA,<br>It is envisaged that participants will be assessed up to eleven and a half years after enrolling in the programme, which will reveal the long-term health effects of the trial. |

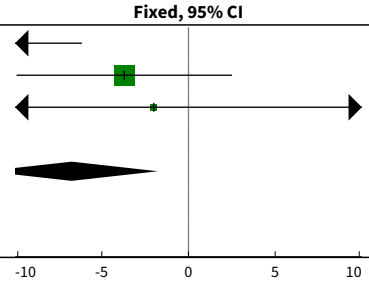
## DATA AND ANALYSES

### Comparison 3. Dietary Advice versus Dietary Advice plus Exercise

| Outcome or subgroup title | No. of studies | No. of participants | Statistical method | Effect size |
|---|---|---|---|---|
| 1 Weight at 12 months | 3 | 132 | Mean Difference (IV, Fixed, 95% CI) | -6.74 [-11.72, -1.76] |
| 2 HbA1c at 6 months | 4 | 227 | Mean Difference (IV, Fixed, 95% CI) | -0.86 [-1.33, -0.38] |
| 3 HbA1c at 12 months | 3 | 132 | Mean Difference (IV, Fixed, 95% CI) | -0.96 [-1.53, -0.39] |

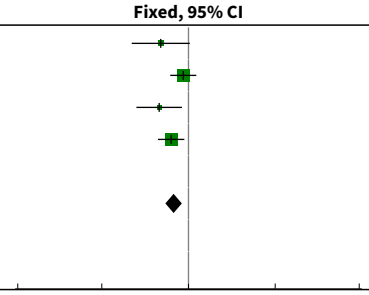
### Analysis 3.1. Comparison 3 Dietary Advice versus Dietary Advice plus Exercise, Outcome 1 Weight at 12 months.

| Study or subgroup | Treatment N | Treatment Mean(SD) | Control N | Control Mean(SD) | Mean Difference Fixed, 95% CI | Weight | Mean Difference Fixed, 95% CI |
|---|---|---|---|---|---|---|---|
| Samaras 1997 | 13 | 83.1 (13) | 13 | 99 (12.3) | | 26.35% | -15.85[-25.56,-6.14] |
| Uusitupa 1996 | 38 | 86.5 (13.7) | 40 | 90.2 (14.3) | | 64.27% | -3.7[-9.91,2.51] |
| Wing 1988 | 13 | 96.2 (23.4) | 15 | 98.2 (20) | | 9.38% | -2[-18.27,14.27] |
| **Total ***** | **64** | | **68** | | | **100%** | **-6.74[-11.72,-1.76]** |
| Heterogeneity: $Tau^2$=0; $Chi^2$=4.63, df=2(P=0.1); $I^2$=56.8% | | | | | | | |
| Test for overall effect: Z=2.65(P=0.01) | | | | | | | |



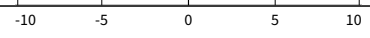

### Analysis 3.2. Comparison 3 Dietary Advice versus Dietary Advice plus Exercise, Outcome 2 HbA1c at 6 months.

| Study or subgroup | Treatment N | Treatment Mean(SD) | Control N | Control Mean(SD) | Mean Difference Fixed, 95% CI | Weight | Mean Difference Fixed, 95% CI |
|---|---|---|---|---|---|---|---|
| Agurs-Collins 1997 | 32 | 9.9 (2) | 32 | 11.5 (4.4) | | 8.04% | -1.6[-3.27,0.07] |
| Ligtenberg 1997 | 25 | 8.7 (1.1) | 26 | 9 (1.6) | | 39.98% | -0.3[-1.05,0.45] |
| Samaras 1997 | 13 | 5.7 (1.1) | 13 | 7.4 (2.2) | | 13.09% | -1.69[-3,-0.38] |
| Uusitupa 1996 | 40 | 6.8 (1.6) | 46 | 7.8 (2) | | 38.89% | -1[-1.76,-0.24] |
| **Total ***** | **110** | | **117** | | | **100%** | **-0.86[-1.33,-0.38]** |
| Heterogeneity: $Tau^2$=0; $Chi^2$=4.55, df=3(P=0.21); $I^2$=34.08% | | | | | | | |
| Test for overall effect: Z=3.54(P=0) | | | | | | | |



**Analysis 3.3. Comparison 3 Dietary Advice versus Dietary Advice plus Exercise, Outcome 3 HbA1c at 12 months.**

| Study or subgroup | Treatment N | Treatment Mean(SD) | Control N | Control Mean(SD) | Mean Difference Fixed, 95% CI | Weight | Mean Difference Fixed, 95% CI |
|---|---|---|---|---|---|---|---|
| Samaras 1997 | 13 | 6.5 (1.1) | 13 | 7.7 (2.2) | | 18.84% | -1.2[-2.51,0.11] |
| Uusitupa 1996 | 38 | 6.6 (1.6) | 40 | 7.5 (1.7) | | 60.52% | -0.9[-1.63,-0.17] |
| Wing 1988 | 13 | 9.2 (1.8) | 15 | 10.1 (1.6) | | 20.64% | -0.9[-2.15,0.35] |
| **Total ***** | **64** | | **68** | | | **100%** | **-0.96[-1.53,-0.39]** |
| Heterogeneity: Tau²=0; Chi²=0.16, df=2(P=0.92); I²=0% | | | | | | | |
| Test for overall effect: Z=3.29(P=0) | | | | | | | |




Favours treatment — Favours control

## APPENDICES

### Appendix 1. Search strategy

**Search terms**

Unless otherwise stated, search terms are free text terms; MeSH = Medical subject heading (Medline medical index term); exp = exploded MeSH; the dollar sign ($) stands for any character(s); the question mark (?) = to substitute for one or no characters; tw = text word; pt = publication type; sh = MeSH; adj = adjacent.

TYPE 2 DIABETES MELLITUS
1. See Cochrane Metabolic and Endocrine Disorders Group search strategy.

DIET INTERVENTIONS
2. explode Diet Therapy/ [MeSH, all subheadings]
3. (diet$ adj5 diabet$).ab,ti.
4. (diet$ adj5 carbohydrat$).ab,ti.
5. (diet$ adj5 fat$).ab,ti.
6. (diet$ adj5 weigh$).ab,ti.
7. (diet$ adj5 sugar$).ab,ti.
8. (diet$ adj5 glyc?em$).ab,ti.
9. 2 or 3 or 4 or 5 or 6 or 7 or 8

EXERCISE INTERVENTIONS
10. explode Exercise Therapy/ [MeSH, all subheadings]
11. (walk$ or jog$ or swim$).ab,ti.
12. (exerci$ or (physic$ and activ$) or exert$ or (physic$ and fit$) or sports).ab,ti.
13. ((weight and lift$) or (strength and train$) or (resistance and train$) or (circuit and weight and train$) or (aerob$ and train $)).ab,ti.
14. Exercise/ [MeSH, all subheadings]
15. explode Exertion/ [MeSH, all subheadings]
16. explode "Physical Education and Training"/ [MeSH, all subheadings]
17. Physical Fitness/ [MeSH, all subheadings]
18. explode Sports/ [MeSH, all subheadings]
19. 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18

BEHAVIOURAL APPROACHES
20. explode Behavio?r Therapy/ [MeSH, all subheadings]
21. Cognitive Therapy/ [MeSH, all subheadings]
22. explode Hypnosis/ [MeSH, all subheadings]
23. explode Psychotherapy/ [MeSH, all subheadings]
24. behavio?r therap$.ab,ti.

(Continued)
25. cognitive therap$.ab,ti.
26. hypno$.ab,ti.
27. psychotherap$.ab,ti.
28. 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27

RANDOMISED CONTROLLED TRIALS
29. See randomised controlled trials search strategy - based on the Cochrane Pregnancy and Childbirth Review Group strategy.

SYSTEMATIC REVIEWS/META-ANALYSIS
30. See systematic review and meta-analysis search strategy - based on the Metabolism and Endocrine Disorders Review Group strategy.

ALL INTERVENTIONS
31. 9 or 19 or 28

TYPE 2 DIABETES AND ALL INTERVENTIONS
32. 1 and 31

TYPE 2 DIABETES AND ALL INTERVENTIONS AND RANDOMISED CONTROLLED TRIAL
32. 32 and 29

TYPE 2 DIABETES AND ALL INTERVENTIONS AND SYSTEMATIC REVIEW/META-ANALYSES
33. 32 and 30

## WHAT'S NEW

| Date | Event | Description |
|---|---|---|
| 29 September 2008 | Amended | Converted to new review format. |

## HISTORY

Protocol first published: Issue 1, 2003
Review first published: Issue 2, 2004

| Date | Event | Description |
|---|---|---|
| 23 May 2007 | New search has been performed | No new studies have been added.<br>The bulk of the background remains the same but some new references have been included and some sections reworded.<br>The main thrust of the review remains the same.<br>No changes have been made to the methodology. The search strategies used were the same, and the dates over which the databases were searched were simply updated.<br>The results have not changed since the first publication as there were no further studies added.<br>The discussion has been amended slightly, with the discussion of a few more recent studies added (e.g. Ash 2003).<br>The conclusions remain the same - more high quality evidence for the treatment of type 2 diabetes mellitus using dietary advice are required before further conclusions can be made. |

## CONTRIBUTIONS OF AUTHORS

LUCIE NIELD: co-ordinating the 2007 update review, data collection for the review, undertaking searches, organising retrieval of papers, screening retrieved papers against inclusion criteria, abstracting data from papers, data management of the review, entering data into RevMan, analysis of data, writing the review.

HELEN MOORE: designing the original review, co-ordinating the review, data collection for the review, developing search strategy, undertaking searches, organising retrieval of papers, screening retrieved papers against inclusion criteria, appraising quality of papers, abstracting data from papers, writing to authors of papers for additional information, providing additional data about papers, obtaining and screening data on unpublished studies, data management for the review, entering data into RevMan, analysis of data, writing the review.

CAROLYN SUMMERBELL: conceiving the review, designing the review, screening search results, screening retrieved papers against inclusion criteria, appraising quality of papers, abstracting data from papers, obtaining and screening data on unpublished studies, interpretation of data, providing general advice on the review, securing funding for the review, performing previous work that was the foundation of current study.

LEE HOOPER: conceiving the review, screening of retrieved papers against inclusion criteria, appraising quality of papers, abstracting data from papers, providing a clinical perspective, performing previous work that was the foundation of the current study, providing general advice on the review.

KENNEDY CRUICKSHANK: conceiving the review, screening of retrieved papers against inclusion criteria, abstracting data from papers, appraising quality of papers, providing a methodological perspective, providing a clinical perspective, performing work that was the foundation of the current study.

AVNI VYAS: conceiving the review, screening of retrieved papers against inclusion criteria, abstracting data from papers, appraising quality of papers, providing a clinical perspective, performing previous work that was the foundation of the current study.

VICKI WHITTAKER: screening retrieved papers against the inclusion criteria, data management for the review, entering data into RevMan, analysis of data, interpretation of data.

## DECLARATIONS OF INTEREST

None known.

## SOURCES OF SUPPORT

### Internal sources

- University of Manchester, UK.
- University of Teesside, UK.

### External sources

- No sources of support supplied

## NOTES

Currencies converted on 29th October 2003
Exchange rate: 1 US dollar = 0.855505 EUR ( $1 = €0.855505)
www.x-rates.com used to convert

lbs to kgs converted using http://annica.in-cyberspace.net/en/lbs_kg.html

## INDEX TERMS

### Medical Subject Headings (MeSH)

Diabetes Mellitus, Type 2 [*diet therapy]; Diet, Fat-Restricted; Dietary Carbohydrates [administration & dosage]; Dietary Fats [administration & dosage]; Energy Intake; Exercise; Randomized Controlled Trials as Topic; Weight Loss

### MeSH check words

Adult; Humans