Evidence Check

Dietary Patterns and Cardiovascular Disease Outcomes

An Evidence Check rapid review brokered by the Sax Institute for the National Heart Foundation of Australia. April 2017.
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Disclaimer:
This Evidence Check Review was produced using the Evidence Check methodology in response to specific questions from the commissioning agency.

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This report was prepared by Clare Collins, Tracy Burrows, Megan Rollo.

The authors wish to acknowledge the contributions of the following research assistants in producing this work: Alecia Leonard, Amy Ashman, Daisy Coyle, Erin Clarke, Janelle Skinner, Chelsea Carle.
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1 Executive summary

There is a well-established link between dietary intake and chronic disease risk, particularly cardiovascular disease (CVD). Healthy dietary behaviours play an important role in the primary and secondary prevention of CVD.

This systematic review aimed to analyse the evidence from systematic reviews published since 2010 on dietary patterns and the primary and secondary prevention of CVD.

In total, 33 systematic reviews met the inclusion criteria, with 12 reviews on dietary patterns and the primary prevention of CVD outcomes, 3 on dietary patterns and secondary prevention of CVD and 25 reviews on dietary patterns and combined primary and secondary outcomes. A number of systematic reviews reported on multiple dietary patterns and/or CVD outcomes (primary/secondary/combined) and these papers were accounted for in each respective category. Hence, the total number of systematic review publications (n = 33) is less than the total number of reviews for primary, secondary and combined (n=40). Data from the included studies were evaluated using the National Health and Medical Research Council (NHMRC) framework. Using this framework, a total of 16 evidence statements were developed (4 primary, 3 secondary, 9 primary and secondary) and recommendations, graded A–D, were established.

For dietary patterns and primary prevention of CVD, four dietary patterns were investigated with six recommendations generated. The DASH, Dietary Approaches to Stop Hypertension, pattern received two Grade A recommendations for the primary prevention of CVD in relation to reductions in blood pressure and reduced risk of CVD events and/or mortality. Healthy/prudent diet was awarded a Grade B recommendation for reduction in risk associated with CVD events and/or mortality. The Mediterranean diet received two Grade C recommendations for evidence relating to improvements in blood lipids and CVD events and/or mortality. A Low Glycaemic Index/Low Glycaemic Load (Low GI/GL) diet received one Grade C recommendation for improvements in body composition.

In regards to dietary patterns and secondary prevention of CVD, three dietary patterns were evaluated resulting in four recommendations. The Portfolio diet has a Grade B recommendation for reduction in blood lipids for those with pre-existing hyperlipidaemia. The DASH pattern received a Grade C recommendation for improvements in blood pressure. Weight loss/calorie-restricted diets were awarded two Grade C recommendations related to improvements in blood pressure and body weight in those with pre-existing CVD.

Where results for the primary and secondary CVD prevention were reported combined, evidence was evaluated for 10 dietary patterns with 16 recommendations produced. In regards to improving blood pressure in both population groups (i.e. hypertensive and normotensive individuals), dietary patterns defined as DASH, low carbohydrate, and vegetarian had the strongest evidence (four Grade B recommendations), followed by the Mediterranean and Nordic diets (two Grade C recommendations). For weight loss/calorie-restricted dietary patterns, the strength of the recommendations differed between diastolic blood pressure (Grade B) and systolic blood pressure (Grade D). In those with pre-existing CVD, dietary patterns defined as low-fat, DASH and weight loss/calorie-restricted diets had an overall stronger evidence base (three Grade B recommendations) compared to patterns defined as low GI, Mediterranean, high protein or Nordic (four Grade C recommendations). For reducing excess weight in those with or without pre-existing CVD, one Grade B recommendation for the DASH pattern was derived. Finally, for
primary and secondary prevention of CVD events and/or mortality, the Mediterranean diet received one Grade C recommendation.

Of the eight dietary patterns evaluated in this review, all promoted regular consumption of both fruits and vegetables, and whole grains/cereals. In addition, seven patterns included the regular consumption of legumes (including soy), and four patterns included frequent consumption of nuts and seeds. Five patterns included the regular consumption of fish, and specifically the two versions of the Mediterranean dietary patterns also encouraged a low consumption of red meat and meat products. Additional similarities included three dietary patterns that reported the regular consumption of poultry (three patterns), and five patterns that encouraged the regular/moderate consumption of low-fat dairy.

Previously, advice has been predominantly provided in terms of nutrient targets, however there is an increasing shift towards providing food-based dietary guidance. From this rapid review, there is a gap in the existing evidence base concerning the distinct advantages and disadvantages of providing dietary pattern-based advice compared to nutrient-based advice. To answer this question specifically, a direct comparison between providing dietary-pattern advice versus nutrient-based advice is required.

This review concludes that the DASH diet appears to be the most universally beneficial dietary pattern in reducing CVD risk and CVD risk factors in healthy populations. For the exclusive secondary prevention of CVD, the Portfolio diet, followed by the DASH and weight loss/calorie-restricted diet is recommended. However, this is dependent on the pre-existing condition. In groups both with and without CVD, the most consistent evidence was found for the DASH pattern with adherence resulting in improvements in blood pressure, blood lipids and body weight.
2 Background

Various dietary components have been evidenced as important modifiable risk factors for the prevention of chronic diseases, such as cardiovascular disease (CVD). In Australia, CVD is the leading cause of death and disease burden, with recent estimates suggesting that 63.2% of Australian adults have some form of dyslipidaemia. Healthy dietary practices, at all life stages, play an integral part in the primary and secondary prevention of CVD, as well as the mitigation of CVD risk factors. Existing guidelines for the primary and secondary prevention of CVD are based predominantly on single nutrients or foods. Recommendations include decreasing consumption of total fat, saturated fatty acids, cholesterol, salt and/or increasing consumption of fruit, vegetables, polyunsaturated fatty acids, monounsaturated fatty acids, fish, fibre and potassium. Increasingly, literature has emerged that explores whole dietary patterns, rather than single nutrients or foods. Analysing food consumption in the form of dietary patterns offers a perspective different from the traditional single-nutrient focus and it has been suggested that dietary patterns may be more predictive of disease risks compared to specific nutrient or food approaches.

Continuing to identify effective strategies for primary and secondary prevention are imperative for reducing CVD morbidity and mortality, and their associated economic burden.

Primary prevention strategies focus on lifestyle changes in healthy individuals to effectively modify CVD risk factors – such as hypertension, dyslipidaemia and weight gain – to prevent the first occurrence of a cardiovascular event (e.g. heart attack or stroke). Secondary prevention strategies are aimed at reducing the progression of disease in individuals diagnosed with CVD and to prevent the recurrence of further cardiovascular events. Risk factor management, occurring primarily in hospital and community settings, in this population is complex. However, multidisciplinary treatment of risk factors with a combination of lifestyle changes and pharmacologic interventions, can improve an individual’s overall quality of life and survival. Many CVD risk factors are associated with the modern Western diet. These diets are commonly based on low- to moderate- fruit and vegetable intake, full-fat animal and dairy products, and are typically high in refined cereal products, sugar, processed or fried foods. Thus, dietary modification plays an important role in both the primary and secondary prevention of CVD.

Nutritional research has largely focused on the effects of single nutrients or food components (e.g. fruits and vegetables, whole grains, saturated fatty acids, trans-fatty acids, polyunsaturated fatty acids, omega-3 fatty acids) on disease outcomes. However, as these nutrients and foods are consumed in combination, there has been a gradual movement away from nutrient-based approaches to one that considers dietary patterns and the complexity of the overall diet in relation to CVD outcomes. On the basis of current scientific evidence, a healthy dietary pattern can help individuals achieve and maintain a healthy weight, minimise the risk of developing CVD and related risk factors. An evidence report by the Dietary Guidelines Advisory Committee (DGAC), recently released in the US, focused its attention on healthful dietary patterns – instead of single nutrients or foods – in relation to a wide range of disease outcomes. Overall, the findings indicated that a ‘healthy dietary pattern is higher in vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes and nuts; moderate in alcohol (among adults); lower in red- and processed-meat; and low in sugar-sweetened foods and drinks, and refined grains. This healthy dietary pattern described by the DGAC aligns with the current Australian Dietary Guidelines recommendations.

Australians consume many varied dietary patterns and some popular approaches include the Mediterranean diet and the Dietary Approaches to Stop Hypertension (DASH) diet. Recent research, in both primary and secondary populations, have demonstrated that the Mediterranean and DASH diets are associated with moderate decreases in blood pressure and body weight, and an overall reduction in CVD risk and total mortality. Further evaluation of the existing evidence, regarding dietary patterns and CVD outcomes, is
warranted. An approach that considers complete dietary patterns provides the opportunity to offer practical recommendations for CVD risk reduction. Guidelines based on whole diets have the potential to offer a more comprehensive approach for the primary and secondary prevention of CVD. Furthermore, dietary recommendations and interventions may be easier to interpret and implement when initiated as a modification in overall dietary patterns allowing for sustainable lifestyle changes.

Objectives
The Evidence Check aimed to address the following research questions:

- Question 1a: What is the evidence regarding how dietary patterns affect the risk of developing cardiovascular disease?
- Question 1b: What are the characteristics of the dietary patterns that are associated with a reduced risk of developing cardiovascular disease?
- Question 2a: What is the evidence regarding how dietary patterns affect cardiovascular disease outcomes in patients with existing cardiovascular disease?
- Question 2b: What are the characteristics of the dietary patterns that are associated with improved cardiovascular disease outcomes in patients with existing cardiovascular disease?
- Question 3: What is the evidence regarding the advantages and disadvantages of giving dietary pattern advice versus nutrient-based advice for the purpose of achieving better CVD outcomes?
- Question 4: What is the evidence regarding which dietary pattern leads to better CVD outcomes compared to others?
3 Methodology

Purpose and scope
The Evidence Check consisted of a rapid review of the literature relating to the effectiveness of dietary patterns for the primary and secondary prevention of CVD. For Questions 1a, 1b, 2a, 2b and 4 this involved a systematic literature search (outlined below), while evidence for Question 3 was sourced via an intuitive and purposeful electronic search of the literature based on selected key documents identified by the report authors.

Data sources and searches
Data was sourced using the electronic databases: CINAHL, Cochrane Reviews, Cochrane Other Reviews, Cochrane Trials, Embase, Medline (includes Medline in Process), and Scopus electronic databases. Searches were restricted to publications from January 2010 to November 2016 and published in the English language. Search strategies including the key words used in each database are provided in Appendix 1. Following the initial search, retrieved records were then restricted to those containing the key word 'systematic review'. Records were then checked based on title and abstract by one reviewer. Retrieved reviews were then assessed against the inclusion criteria (outlined below) by two reviewers, if there was any discrepancy a third reviewer was used.

Study selection
Systematic literature reviews and meta-analyses on adults with or without CVD were included. No restriction was placed on the type of studies comprising the review and/or meta-analysis, with randomised controlled trials through to observational studies included. Evidence from member countries of the Organisation for Economic Co-operation and Development (OECD) including Australia, New Zealand, Canada, US, UK and Northern European countries were sought, however reviews that may have included some non-OECD countries were included in this Evidence Check. Reviews that aimed to evaluate the primary outcome exclusively in the context of non-OECD countries were excluded from the evidence check.

Dietary patterns were defined ‘as the quantities, proportions, variety or combinations of different foods and beverages in diets, and the frequency with which they are habitually consumed’.

Examples included the Mediterranean and DASH (Dietary Approaches to Stop Hypertension), in addition to diets where the manipulation of macronutrient composition would involve a whole diet approach and restriction of certain foods (e.g. low carbohydrate, low-fat).

For the purpose of this Evidence Check, CVD outcomes of interest to be considered included incidence of coronary heart disease, atrial fibrillation and heart failure; along with risk through incidence of hypertension, lipid profile and weight gain.

Data extraction
Data describing the characteristics and outcomes were extracted into a standardised form for included systematic reviews and meta-analyses. Reviews were categorised according to whether CVD outcomes related to primary prevention, secondary prevention, or combined primary and secondary prevention. To be considered as primary prevention, reviews needed to consist primarily of participants without a pre-existing cardiovascular disease. Individuals with other non-CVD co-morbidities (e.g. type 2 diabetes, obesity) were included as primary prevention studies. Reviews categorised as secondary prevention consisted of individuals with a pre-existing CVD, including past myocardial infarction, heart failure and hyperlipidaemia. In reviews that contained studies on both primary and secondary prevention, but where either a meta-analysis was not performed (i.e. systematic literature review only) or if a meta-analysis was performed,
results for outcomes relating to primary and secondary prevention were not separated, and these studies were categorised as ‘combined primary and secondary prevention’.

**Evaluation of evidence**

Evidence is summarised by dietary pattern and by CVD outcome (grouped into four categories: those effecting blood lipids, blood pressure, weight status and/or body composition, and CVD events and/or mortality). The included reviews and meta-analyses for the body of evidence on each dietary pattern were evaluated using the National Health and Medical Research Council (NHMRC) framework. The framework incorporates grades on the evidence base including number, type and quality of studies; consistency in findings; clinical impact of the recommendations; generalisability to the target population; and applicability of the evidence to the context of the Australian healthcare system. Each component of the framework is summed to establish an overall grade of the evidence and subsequent recommendations for practice.

**Analysis of the evidence**

Following the initial search, 11,442 records were retrieved with 3267 records containing the key term ‘systematic review’. Following title and abstract screening and full-text review, 3165 records were excluded leaving 102 systematic reviews (+/- meta-analysis) included. Of these, 33 reviews incorporated evaluation on the effect of dietary patterns on the primary, secondary or combined primary and secondary prevention of CVD. Subsequently, they were included in this Evidence Review (Appendix 4 and 8). An additional 66, which examined specific food, food group or nutrient were also retrieved and data extracted, however are not included in the current report.
Figure 1. Flow diagram of article identification retrieval and inclusion for the systematic review

Total dietary patterns N = 40

N= 12 primary prevention →
4 evidence statements (Healthy/prudent diets, Mediterranean diet, Low GI/GL diet, DASH diet)

N= 3 secondary prevention →
3 evidence statements (Weight loss/Calorie restricted diet, DASH diet, Portfolio diet)

N= 25 primary + secondary →
9 evidence statements (Low GI/GL diet, Low-fat diet, Low CHO diet, Mediterranean diet, Vegetarian diet, DASH diet, High protein diet, Weight loss/calorie-restricted diet, Healthy diet (Nordic/Tibetan)
4 Results

**Question 1a:** What is the evidence regarding how dietary patterns affect the risk of developing cardiovascular disease?

**Question 2a:** What is the evidence regarding how dietary patterns affect cardiovascular disease outcomes in patients with existing cardiovascular disease?

**Primary prevention of CVD**

This section reviewed the evidence for systematic literature reviews and/or meta-analyses that reported exclusively the primary prevention of CVD outcomes and the risk factor in healthy population groups. Evidence-based statements, as detailed in Appendix 4, were able to be generated for four dietary patterns: Healthy/prudent diets; Mediterranean; Low GI/GL (Glycaemic Index/Glycaemic Load); and DASH. Based on the available evidence, the grade of recommendation was highest for the DASH approach, which was the only dietary pattern from those reviewed to achieve the highest grade of evidence Grade A. This was followed by Healthy/prudent diet (Grade B); then the Mediterranean and Low GI/GL diets, which were both classified with a Grade C recommendation.

The overall grade of recommendation was determined through consideration of five domains: the evidence base, consistency of the findings, generalisability, clinical impact and applicability. The DASH diet was determined as ‘excellent’ across all five domains. The other three dietary patterns were all rated as ‘good’ for generalisability, however varied on the other domains of consistency of the findings, clinical impact and applicability. The lowest grade of clinical impact was found for Low GI/GL diets, which was rated as ‘poor’.

No other dietary patterns on any of the domains were rated as ‘poor’. Of particular note is that in the dietary pattern of Low GI/GL, the evidence-based statement was informed by only one systematic review of 15 randomised controlled trials (RCTs), all other evidence-based statements were informed by greater than or equal to three systematic reviews.

The improvements in CVD outcomes/ risk factors reported in the included reviews varied for each of the dietary patterns. The DASH dietary pattern was found to decrease CVD mortality, the incidence of CVD events, coronary heart disease, heart failure and stroke. Healthy/prudent diets also decreased CVD events and mortality. Mediterranean diets were determined to improve blood lipids, specifically total cholesterol and low-density cholesterol, while Low GI/GL was found to gain improvement in fat-free mass.

Ten of the reviews for primary prevention included a meta-analysis, which compared a dietary pattern to another dietary approach. The comparator group varied across each of the meta-analysis and a detailed table of the comparators can be found in Appendix 7. The comparator diet was described as a ‘Western’ diet in n= 4 reviews, DASH diet (low adherence) (n=2 studies) and Low GI, usual diet, unspecified and no control used in one study each. However, there was a general lack of descriptions for both the dietary pattern being investigated and also the comparator diet. This makes it difficult for direct comparisons to be made and should be considered when interpreting findings. For consideration, when the comparator group is described as ‘Western’ or ‘usual’ diet given the nature of individuals intake this would likely be a highly variable construct.
Secondary prevention of CVD
This section reviewed the evidence for reviews/studies that reported exclusively for secondary prevention of CVD outcomes and risk factors in those populations with an existing or past CVD event (See Appendix 4 for evidence-based statements). Evidence-based statements were generated for three dietary patterns: Weight loss/calorie-restricted diets, DASH and Portfolio dietary patterns. All evidence-based statements were informed by a single systematic review, however the number of studies included in those reviews was variable (weight loss (n= 8 RCTs), DASH (n= 15 RCTs), and Portfolio dietary pattern (n= 3 RCTs)). When compared to primary prevention, the number of reviews and studies within those reviews reflects the overall lower amount of evidence in this area of secondary prevention. Based on the grade of recommendation, no dietary patterns achieved a Grade A rating. The highest grade of evidence was achieved by the Portfolio dietary pattern, which was determined to be a Grade B. Weight loss and the DASH dietary pattern were determined to be Grade C. All reviews used the PRISMA checklist and reported using a study quality checklist that reduced the level of bias in the included reviews.

For each of the included reviews, a meta-analysis was conducted in a sub-group of studies which were comparable. A different comparator group was used in each of the meta-analyses: calorie-restricted diets were compared with a ‘usual’ diet; DASH was compared with a ‘control’ diet, which was not specified in detail in the review; and the Portfolio diet was compared with a ‘usual’ diet.

Primary and secondary prevention
This section reported on the evidence for reviews that discussed both primary and secondary prevention in their outcomes, where primary and secondary preventions were not divisible into separate categories. A total of nine evidence-based statements (see Appendix 4) were generated with three dietary patterns graded as Grade B (DASH, Vegetarian and Low-fat diet), five dietary patterns were rated as Grade C (Low GI/GL, Low carbohydrate, Mediterranean, High protein and Nordic/Tibetan diets). And one dietary pattern of Calorie-restricted diets was rated as Grade D. Overall, six dietary patterns were informed based on a single review.

For seven dietary patterns (Low GI/GL, Low-fat diet, Low carbohydrate, Vegetarian diet, High protein, Calorie-restricted and Nordic/Tibetan diet) one CVD recommendation was able to be determined. However, for both the DASH and Mediterranean diet, three separate CVD recommendations were generated from the evidence. For the Mediterranean diet, a recommendation specific to blood pressure, lipid profiles and overall relative risk was generated. For the DASH diet specific recommendations were generated for blood pressure, weight and BMI and blood lipids. All recommendations for these patterns lead to improvements in CVD risk factors.

Comparators across the 22 reviews included in this section had different comparator groups in their respective meta-analysis. Nine dietary patterns were compared with a ‘usual’ diet. However, in some studies where a brief definition of ‘usual’ diet was reported by authors the definition was highly variable. Definitions ranged from ‘low-fat diets or healthy dietary advice’ to ‘a diet with refined carbohydrates (i.e. no whole grains) while another was described as ‘usual, healthy, low-fat or calorie-restricted diets or higher acid load healthy diet’. The variation of these descriptions highlights the variation in the comparator diets. In addition, a question was also raised in whether these descriptions are truly reflective of a ‘usual’ diet. I.e. how many Australians consume a low-fat diet as their usual diet? The answer is likely very few, given the current prevalence of obesity. Other dietary patterns which were used as comparators were: Omnivorous diet, Low protein, ‘Standard’ protein diet and High GI. Each were used in one meta-analysis. In seven meta-analyses the comparator group was not described.
Question 1b: What are the characteristics of the dietary patterns that are associated with a reduced risk of developing cardiovascular disease?

Question 2b: What are the characteristics of the dietary patterns that are associated with improved cardiovascular disease outcomes in patients with existing cardiovascular disease?

The specific characteristics of the dietary patterns identified and included in this review are provided in Table 1. These patterns are described in three ways: 1) foods and/or food groups, 2) a combination of specific foods with key nutrient targets, or 3) whole diet approaches that focus on manipulation of a single macronutrient.

**Dietary patterns described in terms of foods and/or food groups:**
- Healthy/prudent diet
- Mediterranean diet
- Vegetarian diet
- Nordic and Tibetan diets.

**Dietary patterns described in terms of food plus targets for key nutrients:**
- Dietary Approaches to Stop Hypertension (DASH)
- Portfolio diet.

**Dietary patterns described in terms of a single macronutrient and/or no specific information regarding food and nutrient intakes:**
- High protein diet
- Low-fat diet
- Low carbohydrate diet
- Low GI/GL diet
- Weight loss calorie-restricted diet.
Table 1: Characteristics of all included dietary patterns

<table>
<thead>
<tr>
<th>Dietary Pattern</th>
<th>Description of dietary pattern</th>
</tr>
</thead>
</table>
| High protein diet \(^{16,17}\)      | • Mean protein intake (as % of total diet) for High protein diet 30.5±2.4% (range: 27.0–34.9%) and for standard protein 17.5±1.5% (range: 16.0–21%) \(^{17}\)   
  • Protein intake more than >25% of an individual’s total daily kJ intake \(^{16}\)                     |
| High protein diet V2 \(^{18}\)      | • Protein intake between higher and lower protein diets must have had a 5% difference in total energy  
  • The median protein content of the higher-protein diets was 27% of the total energy intake (range: 16–45%) and 18% (range: 5–23%) in the lower-protein diets \(^{18}\) |
| Low-fat diet \(^{16}\)               | • Less than ≤30% of an individual’s total daily kJ intake comes from fat \(^{16}\)                                                                             |
| Low carbohydrate diet \(^{19}\)     | • Carbohydrate (CHO) intake equal to or less than 120gram/day. \(^{19}\)                                                                                       |
| Low GI/GL diets \(^{20,21}\)        | • No definition regarding classification of low vs high GI and low vs high GL \(^{21}\)                        
  • One study did a sub-group analysis between high and low GI diets; compared GI difference of 0–10 between groups, GI difference of 10.1–20 between groups and GI difference of >20 between groups \(^{20}\) |
| Weight loss/calorie-restricted diets V1 \(^{22}\) | • Diets were defined as weight-reducing diets at least 24 weeks in duration. No further specifics reported \(^{22}\)                                            |
| Weight loss/calorie-restricted diets V2 \(^{23}\) | • Weight loss intervention not specified – included studies must have had dietary pattern intervention longer than 6 months  
  • Net weight change range from −16.0 to +1.4 kg (mean: −4.56 kg) \(^{23}\)                                |
| Healthy/prudent diet \(^{24,28}\)   | Regular consumption of:                                                                                                                                            |
  • Vegetables  
  • Fruits  
  • Whole grains  
  • Fish  
  • Low-fat dairy  
  • Poultry  
  • Soy |
- Olive oil

**Mediterranean diet V1** 29-31

Diet must have at least 2 of the following:

- High monounsaturated/saturated fat ratio (use of olive oil as main cooking ingredient)
- High consumption:
  - Vegetables
  - Fruits
  - Legumes
  - Grains and cereals
- Moderation consumption
  - Milk and dairy products
- Low to moderate consumption
  - Red wine
- Low consumption
  - Meat and meat products
- Increased consumption
  - Fish

**Mediterranean diet V2** 32-38

- High intake of:
  - Vegetables
  - Fruits
  - Cereals and whole grain breads
  - Beans, nuts and seeds
- Low to moderate amounts of:
  - Red wine
  - Cheese and yoghurt
- Low consumption of:
  - Concentrated sugars/honey
  - Red meat (higher quantities of fish)
- Olive oil as a main cooking ingredient and source of fat
**Vegetarian diet** ³¹
- High consumption of fruits and vegetables
- High consumption of grains, legumes
- Little to no consumption of meat

**Portfolio diet** ¹⁶
- Largely vegetarian diet with aim to lower cholesterol:
  - Low in saturated fat
- High intake of:
  - Vegetables
  - Fruits
  - Whole grains
  - Nuts
  - Plant sterols
  - Fibre
  - Soy protein

**DASH** ⁴⁰⁻⁴²
- Foods low in sodium and rich in potassium, magnesium and calcium
- Reduced consumption: saturated fat, total fat, no added salt
- Regular consumption of:
  - Vegetables
  - Fruits
  - Whole grains
  - Legumes, seeds, nuts
  - Fish
  - Poultry
  - Low-fat dairy

**Nordic diet** ⁴³
- Regular consumption of:
  - Whole grains (rye, barley, oats)
  - Vegetables
  - Fruits/berries
  - Nuts
  - Fatty fish,
**Tibetan diet**

- Rapeseed oil
- Low-fat dairy

- Regular consumption of:
  - Cereals (barley, wheat, rye, corn, rice, oats and buckwheat)
  - Meat (beef, mutton, hare, chicken, venison)
  - Vegetables (onion, garlic, radish, fennel, leek, carrots)
  - Beans (soy beans, dark beans)
  - Fruits (pomegranate, banana, pineapple, mango, bramble, apricot and nectarine).

* The above descriptions are based on information collected from the included reviews and dietary expertise of UON authors on broad dietary classifications of these dietary patterns.
Similarities between included dietary patterns

A total of 15 dietary patterns (including dietary variations) were included in the current report. Of these, only eight described dietary patterns in terms of food groups and/or key nutrient targets (Table 2). The other seven dietary patterns either reported on macronutrient targets with no specific food targets or did not report any specific dietary targets at all.

All of the eight dietary patterns that reported on foods and/or key nutrients included the regular/high consumption of both fruits and vegetables and the regular/high consumption of whole grains/cereals (barley, rye, oats and wheat). Seven of the eight included the regular consumption of beans and legumes (including soy), and four dietary patterns included frequent consumption of nuts and seeds (Med diet V2, Portfolio diet, DASH and Nordic diet).

Five dietary patterns included the regular consumption of fish. The two Mediterranean dietary patterns also encouraged a low consumption of red meat and processed meat products. Additionally, three dietary patterns reported the regular consumption of poultry as an aspect of the diet (Tibetan, DASH and Healthy/prudent diet).

Five dietary patterns encouraged the regular/moderate consumption of low-fat dairy (cheese, milk, yoghurt etc.).

Three dietary patterns (2 x Mediterranean diet and 1 x the Healthy/prudent diet) specified olive oil as the main oil for cooking. The Nordic diet also specified an oil (rapeseed oil) for regular consumption.

Standout differences between dietary recommendations

The two versions of the Mediterranean diet were the only diets that encouraged a low to moderate consumption of alcohol (red wine). Alcohol was not mentioned as an aspect of the other dietary patterns.

The Tibetan dietary pattern was the only dietary pattern that encouraged the regular consumption of meat (beef, mutton, hare, chicken, venison) while the vegetarian diet was the only dietary patterns that reported little to no consumption of meat, including fish.

The DASH diet was the only dietary pattern that specified the consumption of particular micronutrients. The DASH diet encouraged the consumption of foods rich in potassium, magnesium and calcium; and low in sodium, saturated fat and total fat. The Portfolio diet was the only other diet to specify a reduced intake of saturated fat and the only diet that specifically encouraged the intake of fibre.

One version of the Mediterranean diet (V2) also specified to consume low amounts of concentrated sugars/honey. This was the only paper to comment on refined and/or natural sugars.
Table 2: Food components of the eight dietary patterns which reported on specific foods and/or key nutrients

<table>
<thead>
<tr>
<th>Food component</th>
<th>Healthy/prudent Diet 24-28</th>
<th>Mediterranean Diet V1 29-31</th>
<th>Mediterranean Diet V2 32-38</th>
<th>Vegetarian Diet 39</th>
<th>DASH Diet 40-42</th>
<th>Nordic Diet 43</th>
<th>Tibetan Diet 43</th>
<th>Portfolio Diet 16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruits</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<td>X</td>
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<td>Vegetables</td>
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<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Whole grains/breads/cereals(^1)</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<td>Fish/seafood</td>
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<td>Low-fat dairy</td>
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<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poultry</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Beans/legumes</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Nuts/seeds</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Olive oil</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Rapeseed oil</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Alcohol (red wine)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meat and meat products</td>
<td></td>
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<td></td>
<td></td>
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<td>Micronutrients(^2)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Plant sterols</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Limit on saturated fat</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Limit on meat/meat products</td>
<td>X</td>
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<td>X</td>
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<td></td>
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<td>Limit on refined sugars</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Limit on sodium</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

1 Whole grains/breads/cereals include: barley, rye, oats, wheat, corn and rice; 2 Micronutrients include: potassium, magnesium and calcium.
Question 3: What is the evidence regarding the advantages and disadvantages of giving dietary pattern advice versus nutrient-based advice for the purpose of achieving better CVD outcomes?

Summary statement
The reviews included in this evidence check did not specifically address this question. This is due to the overall lack of studies identified that directly compare the impact of a specified dietary pattern intervention versus a specific nutrient manipulation. This remains an area for future research to address. However, it should be acknowledged that this type of study would be difficult to achieve in a practical sense. Furthermore, such studies would need clear definitions of how a dietary pattern intervention differed from a nutrient specific intervention as both would involve the provision of food lists for individuals to follow.

Published reports, reviews and studies to inform this discussion below can be found in Appendix 5.

1. Advantages of giving dietary pattern advice to achieve better CVD outcomes, and under what circumstances and for whom (primary versus secondary)
Dietary pattern advice would typically involve providing people with a list of specific included foods and any that should be avoided or excluded. It would also involve advice on how to combine these foods into a meal plan and weekly menus, as well as recipes and cooking techniques.

Individuals want practical advice on what constitutes a healthy dietary pattern and broad advice, such as following a DASH, Mediterranean, Portfolio or Vegetarian pattern that is likely to be appropriate for primary prevention. For secondary prevention, very little research was identified in this rapid review. The current evidence suggests that dietary pattern-based interventions do also facilitate improved CVD risk factors.

To answer the question specifically, a direct comparison of the impact of a dietary pattern intervention versus a nutrient manipulation (e.g. reduced saturated fat and sodium) would be required. At the practical level, any nutrient-based intervention would still need to be translated into meals, menus, recipes and shopping lists, as with dietary pattern-based approaches.

2. Advantages of giving nutrient-based advice to achieve better CVD outcomes, and under what circumstances
To rigorously test the efficacy of the specific nutrient composition of a dietary intervention, researchers/clinicians would need to control the nutrient content of foods that comprise the dietary intervention to ensure that the nutrient profile has been consumed. In an effectiveness study where a dietary intervention is “prescribed” that align with the same nutrient intake targets, this information would need to be translated into food lists, meals, recipes and shopping lists. If this advice is not adhered to then individuals, whether in primary or secondary prevention, would not know or have very little direction on what to eat. It would take an extremely high level of knowledge to translate nutrient data into foods, which is beyond the scope of many individuals in the general Australian population. For example, if an individual is advised to follow a low saturated fat diet, then they need to be told what foods to avoid; what foods to eat instead; how to prepare meals; and what types of fats and oils, and foods that contain fat, can be consumed. As well, recipes should be provided to guide meal planning. Even if the only nutrient manipulation was to lower sodium intake, advice would still need to be given to the individual on which processed foods contain sodium, as added table salt is not the major source of sodium for Australians 44, and then what foods to eat instead. On a research study level, dietary compliance would need to be strictly monitored so that differences between dietary pattern vs nutrient specific interventions could be ascertained.

3. Highlight the conditions under which one approach is superior (i.e. leads to more effective outcomes with CVD outcomes)
A decision to promote nutrient-based advice or dietary pattern advice would be influenced by the user of that advice, i.e. clinician (dietitian to whom a patient has been referred, GP who has identified specific risk
factors in a patient versus a cardiologist who is managing individuals with established CVD), the general population (primary prevention), or those with existing CVD risk factors, or established disease who are seeking information.

Other important factors influencing the choice of approaches include patient literacy and numeracy, and nutrition knowledge. Despite these issues, no matter which approach is selected the key thing is to be able to assess diet and implement the dietary advice. Hence, the more practical the dietary instruction, and the more opportunity for support, the more likely a person is to be able to make and/or sustain changes in their usual dietary intake.

For clinicians prescribing dietary interventions to meet specific nutrient targets, such as sodium or saturated fat intake, it is likely to be easier for a nutrition professional to evaluate an individual’s intake in response to following a set of prescriptive meal plans to meet nutrient intake goals.

For consumers, nutrient-based recommendations alone will not be specific enough on their own, unless accompanied by meals plans, food lists of what to eat and avoid, and how frequently to achieve effective dietary targets that will translate to improvements in CVD. Individuals cannot translate nutrient-based recommendations alone into meals and food combinations, nor can an individual easily self-monitor adherence to a nutrient target unless they use a self-monitoring tool or program to provide feedback on nutrient intake targets.

We recently conducted a systematic review to identify dietary interventions that had translated dietary evidence into advice for people with, or at high risk of CVD, and/or health professionals who are likely to treat patients with established CVD. Of the 43 articles included, related to 35 separate studies, we found none that specifically stated their aim as to translate the scientific evidence on diet and CVD into practice. Of the included studies, 31 were in-patient or high-risk populations, and just four targeted health professionals. In most (n=26), provision of instruction was the most common behaviour change strategy used. We concluded that there is a need to develop specific knowledge translation strategies for the current evidence base on diet for primary and secondary prevention of CVD, for clinicians, policy and decision makers, as well as the end-users. Strategies identified in the published studies to date were of limited help in informing the best approach, as the majority had incomplete details as to how the translation was attempted.

*Data from the included reviews/meta-analyses; reporting information on adherence, including whether it is reported, how it is measured, and whether it impacts on CVD outcomes (if reported).*

Measuring adherence to nutritional interventions is important as inadequate adherence reduces the effectiveness of an intervention. Of the systematic reviews appraised in this current review, adherence measures were used in some randomised controlled trials (RCTs) and adherence rates were mostly reported. However, across the included systematic reviews broadly, there was inconsistent, minimal, or no evidence pertaining to adherence in many of the reviews.

Adherence measures varied among the included studies of each systematic review. Methods used included completion of 24-hour recalls, food diaries (3–7 day records of food intake), food frequency questionnaires (FFQs, either self-administered or interviewer-administered) and checklists of the foods consumed daily.

When appropriate, urinary and plasma biomarkers as objective measures of dietary pattern adherence were conducted in RCTs. For example, some studies (RCTs) had considered urinary sodium levels as a surrogate marker for assessing adherence to the low sodium component of the DASH diet. In addition to sodium levels, urinary or plasma mineral and electrolyte concentrations of magnesium, potassium, phosphate and calcium were measured to evaluate DASH adherence. In some instances, diet effects on urea were used as an objective marker of dietary protein intake; and α-linolenic acid plasma content, or urinary tyrosol and
hydroxytyrosol levels as markers of nut and olive oil intake, respectively. However, actual adherence was not often extracted and only listed as ‘low’ or ‘high’.

Many of the cohort and cross-sectional studies reporting on the Mediterranean diet outcomes assessed individual levels of alignment between reported intake and the Mediterranean diet components based on validated dietary indices [e.g. 9-point index dietary score developed by Trichopoulou (1995)] with linear scoring systems where high scores equated to greater alignment with food groups recommended in the Mediterranean diet dietary pattern. In many cases, these methods were used to further categorise participants’ diets into quantiles of alignment (e.g. high vs low). Other studies used validated food frequency questionnaires (FFQ), 24-hour recall, and more detailed methods to derive a priori definition of Mediterranean diet alignment and corresponding scores. For example, a score based on evaluation of intake frequency of foods included in a Mediterranean diet pyramid and a score that included typical healthy foods of the Mediterranean diet. The use of different scoring for evaluation of an individual’s alignment to the Mediterranean diet and in general the different methodologies used to calculate differing dietary indexes may account for inconsistency in results across some studies, as they each include slightly differing food/beverage components, both within dietary pattern scores for the same dietary pattern [e.g. Mediterranean diet and then between patterns, such as Mediterranean diet versus Healthy Eating Index (HEI)].

Reviews of RCTs of the Mediterranean diet have not reported adherence to the Mediterranean diet as prescribed.

A potential consideration noted in many of the systematic reviews, using FFQs to assess adherence, was the number of food items and/or the factor weightings for individual foods within the different dietary patterns varied across the included studies. In the posteriori diet pattern assessment studies the resulting diet patterns were generally evaluated as ‘healthy’ or ‘less healthy’ (e.g. the Western pattern). Descriptions of the factor loadings for individual food items for the dietary patterns analysed are not equal between studies, and include different food items, based on the statistical methods applied and the population under study. While these posteriori diet patterns cannot be ‘prescribed’ prospectively, there were similarities in the type of foods that generally featured within the healthy patterns (fruits, vegetables, whole grains, fish and poultry) and the unhealthy or Western patterns (meat, processed meat, refined grains, sweets, sugar drinks and fried foods). These food groups are consistent with foods that are included or excluded with the dominant diets identified in this review, i.e. DASH, Mediterranean, Nordic, Tibetan, Vegetarian, Calorie restricted/weight loss, Low-fat, Low carbohydrate, Low GI and High protein diets.

Many of the observational studies indicated that methodological differences and limitations in the degree of dietary pattern alignment within the studies made it difficult to compare results. Additional studies, particularly RCTs, are needed to substantiate the adherence to various dietary patterns and the relationship with efficacy of impact on CVD-related risk and outcomes. Equally, many of the longer term RCTs noted that changing dietary habits along with other factors: such as participants’ health status; variations in types of dietary patterns; duration of intervention; and additional lifestyle interventions (e.g. physical activity), is likely to impact on the level of adherence, efficacy and/or effectiveness of the dietary intervention. Thus, making it difficult to compare the results between different studies and to establish evidence-based recommendations.

While adherence to any lifestyle intervention is of primary importance, the degree of adherence to dietary patterns has not always been addressed and evaluated when comparing results across studies. Thus, the degree of compliance to dietary targets, may have accounted for differences in results across reviews. Furthermore, foods that make up the various dietary patterns and the nutrient composition of the diet actually consumed differs from person to person, and from country to country.
In feeding and supplementation trials, participants are provided with complete meals of known nutrient composition or food/nutrient supplements with specific nutrient compositions/profiles and/or nutrient doses.

However, the majority of the dietary intervention RCTs relied on participants independently following nutritional guidelines and recommendations or menu cycles, which automatically generates issues related to adherence to the intended intervention.\(^5\) Despite this it has been noted that interventions to enhance dietary adherence (including use of contracts, feedback, and telephone follow-up) in various circumstances have been conducted, with no single intervention or group of interventions reviewed being clearly superior than others.\(^4\) The evidence does indicate that some form of support to optimise adherence is better than none.

**Evidence statement for systematic review**

*Is current evidence related to dietary recommendation for CVD translated into practice for the prevention and treatment of CVD in those with or at high risk of CVD?\(^4\)5*

One systematic review was identified that investigated how the best available current evidence on diet for the prevention and treatment of CVD is translated into practice in those with or at high risk of CVD.\(^4\)5 The review considered Knowledge Translation (KT), which describes the process that encompasses stages from the development and synthesis of the evidence-based knowledge and the translation of this knowledge to healthcare providers and consumers in order to change behaviours and hence improve an individual’s health. An evidence-based statement was generated and can be found in the Appendix 4 of this report. The review contained 35 relevant studies from six databases; 31 studies targeted patients with CVD (n = 10,156) or at high risk of CVD (n = 6690), and four studies targeted health professionals (n = 800). The overall grade of evidence for this review was determined to be Grade C, and one recommendation was made. The recommendation was for adults with or at high risk of CVD that received targeted CVD prevention dietary advice for periods of time from ~25 days up to 5 years. Those individuals had healthier eating habits with decreases in daily intake of total energy, total fats, saturated fats, sodium, glycaemic load and increases in daily intake of fruits and vegetables, whole grains and polyunsaturated fats.
**Question 4: What is the evidence regarding which dietary pattern leads to better CVD outcomes compared to others?**

Tables 3, 4 and 5 summarise the evidence on the identified dietary pattern by primary prevention, secondary prevention or combined primary and secondary prevention of CVD outcomes.

A total of 16 evidence statements containing 22 recommendations (Appendix 4) were generated from the rapid review. When result data from meta-analyses were reported separately for the three categories of CVD outcomes, these has been integrated into the evidence-based statement accordingly. However, a number of reviews included individuals both without and with CVD, therefore, a combined primary and secondary prevention section is included.

Of the 16 evidence statements, 10 were informed by only one systematic review (with or without meta-analysis). Of the remaining six evidence statements: two were informed by three reviews, two by four reviews, one by five reviews and one by 11 reviews. For the evidence statements informed by multiple reviews, three were for the dietary patterns of Healthy/prudent, Mediterranean and DASH in relation to primary prevention. The other three were for the patterns of Mediterranean, DASH and High protein for the combined primary and secondary prevention. As the recommendations derived in the report are based on a rapid review of the evidence, for the six statements informed by three or more reviews, the proportion of included studies that were identical between these reviews was determined and are summarised in Appendix 6. This information is provided to assist in the interpretation of the resulting recommendations.

**Summary of evidence relating to primary prevention**

For the **primary prevention** of hypertension, hyperlipidaemia and/or the reduction of CVD events and/or mortality and risk factors relating to body weight, six recommendations were generated with the overall recommendations ranging in strength from Grade A to C. For the prevention of hypertension, the DASH pattern was the only dietary pattern found to have effect on this CVD outcome, with evidence rated an overall grade of A. Evidence for the prevention of CVD events and/or mortality was strongest for the DASH pattern (Grade A), followed by the Healthy/prudent dietary pattern (Grade B), and Mediterranean (Grade C). An overall grading of C was found for both the effectiveness of a Low GI/GL diet on weight status and/or body composition and the effectiveness of a Mediterranean dietary pattern on improving blood lipids.

**I. Blood pressure: one recommendation – Grade A**

- **Dietary Approaches to Stop Hypertension (DASH) diet:** In adults (>18 years), following a DASH dietary pattern for periods of time from 8 to 14 weeks is effective in lowering systolic and diastolic blood pressure compared to usual diets.

**II. Plasma lipids: two recommendations – both Grade C**

- **Mediterranean diet:** In adults, Mediterranean-style dietary interventions lasting from 12 weeks up to 48 months lead to a greater reduction in total cholesterol and LDL cholesterol compared to control interventions
- Greater alignment between the Mediterranean-style dietary interventions with key elements of the traditional Mediterranean diet is associated with a greater reduction in total cholesterol.

**III. Risk of CVD events and/or mortality: three recommendations – one Grade A, one Grade B and one Grade C**

- **DASH diet (Grade A):** Following a DASH dietary pattern is associated with ~20% reduced risk of mortality and incidence from cardiovascular disease, coronary heart disease, heart failure and stroke; in adults (>18 years) in periods of time up to 24 years
• **Healthy/prudent diet (Grade B):** Greater adherence to a Healthy/prudent dietary pattern is associated with a lower risk of CVD events and CVD mortality

• **Mediterranean diet (Grade C):** A greater adherence to a Mediterranean diet is associated with a reduced risk of all-cause mortality and combined CVD incidence and CVD mortality.

**IV. Body weight and/or body composition: one recommendation – Grade C**

• **Low Glycaemic Index /Glycaemic Load (Low GI/GL) diet:** A Low GI/GL diet is more effective in reducing Fat-Free Mass over 6 to 17 months than a high GI/GL diet, however a Low GI/GL diet is not more effective in reducing fasting plasma cholesterol in interventions lasting from 6 to 17 months.

Table 3: Summary of evidence ratings relating to identified dietary patterns for primary prevention of CVD

<table>
<thead>
<tr>
<th>EVIDENCE GRADE</th>
<th>Blood pressure</th>
<th>Lipids</th>
<th>Weight or body composition</th>
<th>CVD events and/or mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>• DASH</td>
<td>X</td>
<td>X</td>
<td>• DASH</td>
</tr>
<tr>
<td>B</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>• Healthy/prudent</td>
</tr>
<tr>
<td>C</td>
<td>X</td>
<td>• Mediterranean</td>
<td>• Low GI/GL</td>
<td>• Mediterranean</td>
</tr>
<tr>
<td>D</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>

Summary of evidence relating to secondary prevention

For the **prevention of secondary CVD outcomes** in those with pre-existing CVD, four recommendations ranging in strength from Grade B to C were generated. For those with existing hyperlipidaemia, the Portfolio diet rated an overall grade of B with regard to improvements in blood lipids. In those with hypertension, a DASH pattern and a calorie-restricted diet showed effects on blood pressure with both receiving an overall C grading. For reducing risk associated with excess body weight in those with pre-existing CVD, a weight loss or calorie-restricted diet showed some support (Grade C).

**I. Blood pressure: two recommendations – both Grade C**

• **DASH diet:** In hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 2 to 26 weeks is effective in lowering systolic and diastolic blood pressure compared to usual diets.
• **Weight loss or Calorie-restricted diet**: Energy-restricted diets of a duration of >24 weeks in adults with primary hypertension resulted in decreases in systolic blood pressure and diastolic blood pressure and in reductions of body weight. It is unclear what amount of energy restriction is optimal for this effect.

II. **Plasma lipids: one recommendation – Grade B**

• **Portfolio diet**: In adults aged >18 years with hyperlipidaemia, following a Portfolio dietary pattern, for periods of time from 4 weeks up to 1 year, leads to modest improvements in total cholesterol and LDL cholesterol levels.

III. **Risk of CVD events and/or mortality: no recommendation**

IV. **Body weight: one recommendation – Grade C**

• **Weight loss or Calorie-restricted diet**: Energy-restricted diets of a duration of >24 weeks in adults with primary hypertension resulted in reductions of body weight. However, the amount of energy restriction for optimal results is inconclusive.

<table>
<thead>
<tr>
<th>Table 4: Summary of evidence ratings relating to dietary patterns for secondary prevention of CVD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
<tr>
<td>C</td>
</tr>
<tr>
<td>D</td>
</tr>
</tbody>
</table>

**EVIDENCE GRADE**
Summary of evidence relating to the combined primary and secondary prevention

Where primary and secondary prevention of CVD outcomes were reported combined, 16 recommendations were generated ranging in strength from Grade B to D. For improving blood pressure in hypertensive and normotensive individuals, dietary patterns defined as Low carbohydrate (CHO), Vegetarian and DASH had the strongest evidence (Grade B), followed by Mediterranean and Nordic diets (Grade C). Interestingly, the evidence for the effect of weight loss or calorie-restricted diets on blood pressure was stronger for diastolic blood pressure (Grade B) compared to systolic (Grade D). In terms of improvements to blood lipids, those with pre-existing hyperlipidaemia, Low-fat, DASH or calorie-restricted diets had an overall stronger evidence base (Grade B) compared to patterns defined as Low GI/GL, Mediterranean, High protein or Nordic (Grade C). For recommendations relating to reducing excess weight in those with or without pre-existing CVD, calorie-restricted diets had an overall grade of B. Finally, for the prevention of primary and secondary prevention of CVD events and/or mortality, recommendations for Mediterranean dietary patterns received an overall C rating.

I. Blood Pressure: seven recommendations – four Grade B, two Grade C and one Grade D

- **Low carbohydrate diet (Grade B):** A low CHO diet (120gram/day or less) is more effective in lowering weight by 5.9–8.2kg, BMI by 2.1–2.8kg/m², blood pressure, and triglycerides of 0.127–0.468 mmol/L than a Low-fat diet in an intervention lasting from 8 weeks up to 2 years.

- **Vegetarian diet (Grade B):** Consumption of a vegetarian diet is associated with lower systolic and diastolic blood pressure, compared with consumption of omnivorous diets in adults who are taking hypertension medication and those not taking medication.

- **DASH diet (Grade B):** In normotensive and hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 1 to 48 months is effective in lowering systolic and diastolic blood pressure compared to usual, healthy, calorie-restricted, Low-fat or low antioxidant diets.

- **Weight loss or Calorie-restricted diet (Grade B):** Energy-restricted diets that adjusted daily caloric intake in order to reduce weight by at least 4.5kg (or 5% of total body weight) of a duration of 6 to 36 months in adults with and without hypertension resulted in decreases in diastolic blood pressure.

- **Weight loss or Calorie-restricted diets (Level D):** Energy restricted diets that adjusted daily caloric intake in order to reduce weight by at least 4.5kg (or 5% of total body weight) of a duration of 6 – 36 months in adults with and without hypertension resulted in decreases in systolic blood pressure.

- **Mediterranean diet (Grade C):** Adherence to a Mediterranean dietary pattern is associated with lower systolic and diastolic blood pressure, compared with control diets, in adults from healthy populations and in adults with existing cardiovascular disease or risk factors.

- **Nordic diet (Grade C):** Adherence to a Nordic dietary pattern including consumption of whole grains, fish, and berries resulted in decreases in systolic blood pressure and diastolic blood pressure in hypertensive and normotensive populations.

II. Plasma Lipids: seven recommendations – three Grade B and four Grade C

- **Low-Fat diet (Grade B):** In adults aged >18 years, following a low-fat dietary pattern, with fat intake ≤30% of total daily energy, for periods of time from 2 weeks to 4 years, leads to modest improvements in total cholesterol and LDL and HDL cholesterol levels compared to usual dietary intakes.

- **Low Carbohydrate diet (Grade B):** A low-fat diet (<30% Cals from fat/day) is more effective in lowering total cholesterol and LDL-cholesterol in an intervention lasting from 8 weeks up to 2 years.

- **DASH diet (Grade B):** In normotensive and hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 2 to 24 weeks leads to modest improvements in total cholesterol and LDL cholesterol levels.
• **Low Glycaemic Index (GI) diet (Grade C):** A low GI diet is more effective than high GI diet in lowering fasting plasma cholesterol in interventions lasting from 1-20 months.

• **Mediterranean diet (Grade C):** Adherence to a Mediterranean dietary pattern may be associated with improvement in lipid profiles, compared with control diets, in adults from healthy populations and in adults with existing cardiovascular disease or risk factors.

• **High Protein diet (Grade C):** Consumption of a high protein diet (>25% daily energy intake from protein) is associated with improvements in triglyceride levels in adults with and without cardiovascular disease risk factors.

• **Nordic diet (Grade C):** Adherence to a Nordic dietary pattern including consumption of whole grains, fish, and berries resulted in decreases in systolic blood pressure and diastolic blood pressure in hypertensive and normotensive populations.

### III. Risk of CVD events and/or mortality: one recommendation – Grade C

• **Mediterranean diet:** Adherence to a Mediterranean dietary pattern may be associated with a relative reduction in CVD risk between 13 – 38%, compared with control diets, in adults from healthy populations and in adults with existing cardiovascular disease or risk factors.

### IV. Body weight and/or composition: one recommendation – Grade B

• **DASH diet:** Following a DASH diet (with or without energy restriction) in intervention from 8 to 52 weeks leads to weight loss, and reductions in **BMI and waist circumference**, with greater effects in overweight/obese participants.
Table 5: Summary of evidence ratings relating to dietary patterns for the combined primary and secondary prevention of CVD*

<table>
<thead>
<tr>
<th>EVIDENCE GRADE</th>
<th>Blood pressure</th>
<th>Lipids</th>
<th>Weight or body composition</th>
<th>CVD events and/or mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>B</td>
<td>Low Carbohydrate • Vegetarian • DASH • Weight loss or calorie restricted (diastolic only)</td>
<td>Low-Fat • DASH • Weight loss or calorie restricted</td>
<td>DASH</td>
<td>X</td>
</tr>
<tr>
<td>C</td>
<td>Mediterranean • Nordic</td>
<td>Low GI • Mediterranean • High Protein • Nordic diet</td>
<td>X</td>
<td>Mediterranean</td>
</tr>
<tr>
<td>D</td>
<td>Weight loss or calorie restricted (systolic only)</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

* This table reflects reviews/studies which reported outcomes combined for primary and secondary prevention, for this reason it is not generalisable to either primary or secondary prevention in isolation as the results from the reviews could not be separated.
5 Conclusion

This rapid review set out to evaluate the current literature regarding the effectiveness of dietary patterns for the primary and secondary prevention of CVD. Based on the included review, evidence-based statements were able to be generated for four dietary patterns exclusively for primary prevention, three dietary patterns for secondary prevention, and nine dietary patterns for primary and secondary prevention.

Overall, the DASH diet was identified in each of the sections for primary prevention (evidence Grade A), secondary prevention (Grade C), and primary and secondary (Grade B). No other dietary approach was identified with CVD outcomes across all three categories. This likely reflects that there is a greater evidence base, which is largely comprised of high-level studies including randomised controlled trials (RCTs).

When the eight identified dietary patterns were considered as a collective group, all promoted regular intakes of fruits and vegetables consistent with the Australian Dietary Guidelines. Other core food groups emphasised were legumes, nuts, seeds, fish, moderate red meat, poultry and consumption of low-fat dairy.

Based on the dietary patterns reviewed there were numerous similarities across the dietary patterns identified. Consideration should be given on the overall similarities of diets for improving CVD outcomes more so than their differences. And future research questions should be focused on what the aspects of dietary patterns for improving CVD outcomes are, rather than investigating and identifying a singular best dietary pattern for improving CVD risk profiles and outcomes. Based on the included evidence of this rapid review it was not possible to ascertain what was driving the protective effects of the key dietary patterns identified (Mediterranean, DASH, Portfolio). It remains to be determined if it is the whole diet approach rather than a specific food and/or nutrient.

Some evidence-based statements in this review were determined using very few reviews. Specifically, all three evidence-based statements in secondary prevention and six out of nine of the statements in the primary and secondary category included only one review. Therefore, it is possible that these statements may not be generalisable to the population until more evidence is generated.

For several of the dietary patterns identified, the information on the specific components of the patterns were not well described within the reviews. This lack of information made it somewhat difficult to evaluate and compare between reviews of a similarly described dietary pattern. For example, two reviews of the Mediterranean diet may not be entirely comparable given variations that can occur in dietary patterns between individuals and between studies.

For this review, most reviews that included a meta-analysis compared the specified dietary pattern to a ‘usual’ diet. However, as identified in each section, the definition of usual diet was highly variable and questions were raised whether these described comparator diets actually reflect population levels of usual dietary intake. The lack of overall description/and definition of the dietary pattern being investigated and also the comparator group in the included studies/reviews highlights that improvements need to be made in the reporting of dietary intervention studies and subsequent published reviews. This point was evident across each of the primary; secondary; and primary and secondary prevention categories. Without this, true comparisons are difficult to ascertain.

This review did not identify any studies that specifically compared nutrient-based advice to dietary-pattern advice. A gap in the evidence base was identified, which showed that direct comparison between dietary
patterns and nutrients is an outstanding question for research. Translating dietary advice into practice is an important consideration for future studies.

There are several limitations to this review that should be acknowledged. This review retrieved only systematic reviews that were from the calendar year 2010 onwards, in published literature from online databases. There is some possibility that key sources of information, such as high-quality original studies, were missed and not included in this review. There is a lack of detail on characteristics of diets in the evidence base, which makes it difficult to articulate which dietary pattern is best for primary and secondary prevention of CVD.

Based on the included reviews in this publication, the dietary pattern identified to be the most beneficial to address multiple CVD risk factors in healthy populations was the DASH diet. The DASH diet was beneficial specifically for improvements in blood pressure, blood lipids and body weight. For secondary prevention or those populations who have experienced a previous CVD event, the Portfolio diet approach was the most beneficial. However, this is dependent on the existing CVD condition.

This review concludes that the DASH diet appears to be the most universally beneficial dietary pattern in reducing CVD risk and CVD risk factors in healthy populations. For the exclusive secondary prevention of CVD, it is the Portfolio diet, followed by the DASH and weight loss/calorie-restricted diet. However, this is dependent on the pre-existing condition. In groups both with and without CVD, the most consistent evidence was found for the DASH pattern with adherence resulting in improvements in blood pressure, blood lipids and body weight.
6 References

42. Schwingshackl L, Hoffmann G. Diet Quality as Assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension Score, and Health Outcomes: A Systematic Review and Meta-Analysis of Cohort Studies. J Acad Nutr Diet. 2015;115(5):780-800.e5.
## 7 Appendices

### Appendix 1: Search Strategy

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<thead>
<tr>
<th>Database</th>
<th>Number of records identified</th>
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<td>4506</td>
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All searches conducted 10 – 11 November 2016

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### Appendix 2: NHMRC Evidence Hierarchy

NHMRC Evidence Hierarchy: used to grade the evidence of studies included in the current review

<table>
<thead>
<tr>
<th>Level</th>
<th>Intervention</th>
<th>Diagnostic accuracy</th>
<th>Prognosis</th>
<th>Aetiology</th>
<th>Screening</th>
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<tr>
<td>I ¹</td>
<td>A systematic review of level II studies</td>
<td>A systematic review of level II studies</td>
<td>A systematic review of level II studies</td>
<td>A systematic review of level II studies</td>
<td>A systematic review of level II studies</td>
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<tr>
<td>II</td>
<td>A randomised controlled trial</td>
<td>A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among consecutive persons with a defined clinical presentation</td>
<td>A prospective cohort study ⁷</td>
<td>A prospective cohort study</td>
<td>A randomised controlled trial</td>
</tr>
<tr>
<td>III-1</td>
<td>A pseudorandomised controlled trial (i.e. alternate allocation or some other method)</td>
<td>A study of test accuracy with: an independent, blinded comparison with a valid reference standard, among non-consecutive persons with a defined clinical presentation ⁶</td>
<td>All or none ⁸</td>
<td>All or none ⁸</td>
<td>A pseudorandomised controlled trial (i.e. alternate allocation or some other method)</td>
</tr>
</tbody>
</table>
| III-2 | A comparative study with concurrent controls:  
- Non-randomised experimental trial ⁹  
- Cohort study  
- Case-control study  
- Interrupted time series with a control group | A comparison with reference standard that does not meet the criteria required for Level II and III-1 evidence | Analysis of prognostic factors amongst persons in a single arm of a randomised controlled trial | A retrospective cohort study | A comparative study with concurrent controls:  
- Non-randomised, experimental trial  
- Cohort study  
- Case-control study |

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¹ National Health and Medical Research Council (2009), NHMRC additional levels of evidence and grades for recommendations for developers of guidelines, NHMRC, p.15
| III-3 | A comparative study without concurrent controls:  
|       | • Historical control study  
|       | • Two or more single arm study  
|       | • Interrupted time series without a parallel control group | Diagnostic case-control study | A retrospective cohort study | A case-control study | A comparative study without concurrent controls:  
|       | • Historical control study  
|       | • Two or more single arm study |
| IV | Case series with either post-test or pre-test/post-test outcomes | Study of diagnostic yield (no reference standard) | Case series, or cohort study of persons at different stages of disease | A cross-sectional study or case series | Case series |
Appendix 3: Grades of Recommendations

Extract from NHMRC additional levels of evidence and grades for recommendations for developers of guidelines.²

How to use the NHMRC Evidence Statement Form

Step 1 — Rate each of the five components
Applying evidence in real clinical situations is not usually straightforward. Consequently, guideline developers find that the body of evidence supporting a recommendation rarely consists of entirely one rating for all the important components (outlined above). For example, a body of evidence may contain a large number of studies with a low risk of bias and consistent findings, but which are not directly applicable to the target population or Australian healthcare context and have only a limited clinical impact.

Alternatively, a body of evidence may only consist of one or two randomised trials with small sample sizes that have a moderate risk of bias but have a very large clinical impact and are directly applicable to the Australian healthcare context and target population. The NHMRC evidence grading system is designed to allow for this mixture of components, while still reflecting the overall body of evidence supporting a guideline recommendation.

The components described above should be rated according to the matrix shown in Table 1. Enter the results into the NHMRC Evidence Statement Form (Attachment 1) along with any further notes relevant to the discussions for each component.

² National Health and Medical Research Council (2009), NHMRC additional levels of evidence and grades for recommendations for developers of guidelines, NHMRC, p.7-8
Table 1 Body of evidence matrix

<table>
<thead>
<tr>
<th>Component</th>
<th>Excellent</th>
<th>Good</th>
<th>Satisfactory</th>
<th>Poor</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Evidence base</strong></td>
<td>one or more level I studies with a low risk of bias or several level II studies with a low risk of bias</td>
<td>one or two level II studies with a low risk of bias or a SR/several level III studies with a low risk of bias</td>
<td>one or two level III studies with a low risk of bias, or level I or II studies with a moderate risk of bias</td>
<td>level IV studies, or level III studies/SRs with a high risk of bias</td>
</tr>
<tr>
<td><strong>Consistency</strong></td>
<td>all studies consistent</td>
<td>most studies consistent and inconsistency may be explained</td>
<td>some inconsistency reflecting genuine uncertainty around clinical question</td>
<td>evidence is inconsistent</td>
</tr>
<tr>
<td><strong>Clinical impact</strong></td>
<td>very large</td>
<td>substantial</td>
<td>moderate</td>
<td>slight or restricted</td>
</tr>
<tr>
<td><strong>Generalisability</strong></td>
<td>population/s studied in body of evidence are the same as the target population for the guideline</td>
<td>population/s studied in the body of evidence are similar to the target population for the guideline</td>
<td>population/s studied in body of evidence differ to target population but it is clinically sensible to apply this evidence to target population</td>
<td>population/s studied in body of evidence differ to target population and hard to judge whether it is sensible to generalise to target population</td>
</tr>
<tr>
<td><strong>Applicability</strong></td>
<td>directly applicable to Australian healthcare context</td>
<td>applicable to Australian healthcare context with few caveats</td>
<td>probably applicable to Australian healthcare context with some caveats</td>
<td>not applicable to Australian healthcare context</td>
</tr>
</tbody>
</table>

SR = systematic review; several = more than two studies

1 Level of evidence determined from the NHMRC evidence hierarchy – Table 3, Part B;
2 If there is only one study, rank this component as ‘not applicable’
3 For example, results in adults that are clinically sensible to apply to children OR psychosocial outcomes for one cancer that may be applicable to patients with another cancer

The Evidence Statement Form also provides space to enter any other relevant factors that were taken into account by the guideline developers when judging the body of evidence and developing the wording of the recommendation.

**Step 2 — Prepare an evidence statement matrix**

In the ‘Evidence statement matrix’ section of the form, summarise the guideline developers’ synthesis of the evidence relating to each component at the right hand side of the form, and fill in the evidence matrix at the left hand side of the form. Each recommendation should be accompanied by this matrix as well as the overall grade given to the recommendation (see Step 3). Developers should indicate dissenting opinions or other relevant issues in the space provided under the evidence matrix.

**Step 3 — Formulate a recommendation based on the body of evidence**

Develop wording for the recommendation. This should address the specific clinical question and ideally be written as an action statement. The wording of the recommendation should reflect the strength of the body of evidence. Words such as ‘must’ or ‘should’ are used when the evidence underpinning the recommendation is strong, and words such as ‘might’ or ‘could’ are used when the evidence base is weaker.
Step 4 — Determine the grade for the recommendation

Determine the overall grade of the recommendation based on a summation of the rating for each individual component of the body of evidence. A recommendation cannot be graded A or B unless the evidence base and consistency of the evidence are both rated A or B.

NHMRC overall grades of recommendation are intended to indicate the strength of the body of evidence underpinning the recommendation. This should assist users of the clinical practice guidelines to make appropriate and informed clinical judgments. Grade A or B recommendations are generally based on a body of evidence that can be trusted to guide clinical practice, whereas Grades C or D recommendations must be applied carefully to individual clinical and organisational circumstances and should be interpreted with care (see Table 2).

Table 2 Definition of NHMRC grades of recommendations

<table>
<thead>
<tr>
<th>Grade of recommendation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>A</td>
<td>Body of evidence can be trusted to guide practice</td>
</tr>
<tr>
<td>B</td>
<td>Body of evidence can be trusted to guide practice in most situations</td>
</tr>
<tr>
<td>C</td>
<td>Body of evidence provides some support for recommendation(s) but care should be taken in its application</td>
</tr>
<tr>
<td>D</td>
<td>Body of evidence is weak and recommendation must be applied with caution</td>
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</tbody>
</table>
Appendix 4: Evidence Statements

The following section contains the evidence statements for primary prevention, secondary prevention, and combined primary and secondary prevention.

<table>
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<tr>
<th>Dietary Pattern: Healthy/prudent diet</th>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
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</tr>
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</table>

**Evidence Base**

One level I systematic review and meta-analysis of level II studies with unclear risk of bias 24, one level III systematic review with unclear level of bias 27, and three level III studies with low risk of bias. 25, 26, 28

- Li et al. 2015 24 Level I with unclear level of bias

**Number and type of studies reviewed**

Systematic review and meta-analysis of 7 prospective cohort studies reported CVD mortality

**Number of databases searched**

1 database searched (PubMed)

**CVD outcomes assessed**

CVD mortality

**Study quality**

1. PRISMA: No but review follows the meta-analysis of observational studies in epidemiology guidelines.
2. Quality assessment of components of included studies was performed, however no evidence a standardised tool was used and the results of the quality assessment are not reported. No statistical evidence of publication bias found.
3. Funding source potential from bias: not reported.

- Oude Griep 27 Level III-2 with unclear level of bias

**Number and type of studies reviewed**

Systematic review (without meta-analysis) of 8 observational cohort studies

**Number of databases searched**

1 database searched (PubMed)

**CVD outcomes assessed**

C-reactive protein (CRP)

**Study quality**

1. PRISMA: No
2. Quality assessment not done and/or not reported, no indication that quality of included studies was assessed.
3. Funding source potential from bias: not reported

**Funding source**

Authors supported by grants from the National Heart, Lung, and Blood Institute.
• Wang 2016 26 Level III-2 with low risk of bias

Number and type of studies reviewed
Systematic review and meta-analysis of 27 studies (16 cohort studies, 11 cross-sectional). Note that in 2 of the studies, participants aged ≥15 years old were eligible.

Number of databases searched
2 databases searched (MEDLINE and EBSCO)

CVD outcomes assessed
Likelihood of hypertension

Study quality
1. PRISMA: No
2. Risk of bias assessed using the Newcastle-Ottawa quality assessment scale. Max score 9. All studies received a ‘high’ methodological quality rating (≥6 score).
3. Funding source potential from bias: not reported.

Funding source
Not reported

• Zhang 2015 25 Level III-2 with low risk of bias

Number and type of studies reviewed
Systematic review and meta-analysis of 37 cohorts (n=27) and case-control (n=10) studies (14 evaluated healthy diets).

Number of databases searched
2 databases searched (MEDLINE and EBSCO)

CVD outcomes assessed
Coronary heart disease (CHD)

Study quality
1. PRISMA: No
2. Newcastle-Ottawa Quality Assessment scale was used for quality assessment. Max score 9. 31/35 studies received a score ≥6 (high methodological quality).
3. Funding source potential from bias: not reported.

Funding source
Study supported by provinces and the ministry of education.

• Rodriguez-Monforte 201528 Level III-2 with low risk of bias

Number and type of studies reviewed
Systematic review and meta-analysis of 22 observational studies (19 cohort studies and 3 case-control studies). In the analysis of Healthy/prudent studies, 18 cohort studies and 3 case-control studies were included.

Number of databases searched
1 database searched (PubMed)

**CVD outcomes assessed**
Cardiovascular events

**Study quality**

1. PRISMA: No
2. Newcastle-Ottawa Assessment Scale used for quality assessment of included articles (maximum 9 score). All included studies had a high methodological quality (score of ≥7).
3. Funding source potential from bias: not reported.

**Funding source**
Review funded by the Foundation IDIAP Jordi Gol (primary care research).

<table>
<thead>
<tr>
<th>Consistency</th>
<th>Good</th>
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<tr>
<td></td>
<td>A systematic review and meta-analysis by Wang et al. (2016) (^{26}) of 27 studies found a decreased likelihood for hypertension in the highest category of healthy pattern compared with the lowest (OR = 0.81; 95% CI: 0.67 to 0.97; P = 0.02; heterogeneity Chi(^2) = 382.37, df = 23, P&lt;0.0001, I(^2) = 94%). (^{26}) Li et al. (2015) (^{24}) found greater adherence to a Healthy/prudent eating dietary pattern was associated with a lower risk of CVD mortality (SRRE(^a) = 0.81, 95% CI: 0.75 to 0.87, heterogeneity: I(^2) = 3.5%, P = 0.399) (^{24}). Rodriguez-Monforte et al(^{28}) (2015) found pooled relative risk (RR) for CVD (including CVD mortality and total CVD(^b)) in a comparison of highest to lowest category of Healthy/prudent dietary pattern in cohort studies (n=5) was RR = 0.69 (95% CI: 0.60 to 0.78, heterogeneity I(^2) = 0%, P = 0.687), and RR = 0.71 (95% CI: 0.63 to 0.80, heterogeneity I(^2) = 0%, P = 0.560) in case-control studies (outcomes included acute myocardial infarction and coronary heart disease [CHD(^c)]). Zhang et al. (2015) (^{25}) found a decreased risk of CHD in the highest category of Healthy/prudent dietary patterns compared with the lowest category (OR = 0.67, 95% CI: 0.60 to 0.75, P&lt;0.00001; heterogeneity was apparent in all studies: Chi(^2) = 30.25, df = 13, P = 0.004, I(^2) = 57%). (^{25}) Rodriguez-Monforte (^{28}) found pooled relative risk (RR) for CHD in a comparison of highest to lowest category of Healthy/prudent dietary pattern in cohort studies (n=11) was RR = 0.83 (95% CI: 0.75 to 0.92, heterogeneity I(^2) = 44.6%, P = 0.054).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical impact</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Greater adherence to a healthy dietary pattern was associated with a lower risk of CVD events and mortality in two reviews. (^{24, 28}) Healthy dietary patterns may also be associated with decreased odds of hypertension (^{26}) and risk of coronary heart disease. (^{25, 28})</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Generalisability</th>
<th>Good</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Population in body of evidence can be contextualised to adult</td>
</tr>
</tbody>
</table>
Australians without existing cardiovascular disease.

<table>
<thead>
<tr>
<th>Applicability</th>
<th>Excellent</th>
<th>The evidence base is relevant to the Australian health care setting generally.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommendation</td>
<td>Greater adherence to a Healthy/prudent dietary pattern is associated with a lower risk of CVD events and CVD mortality.</td>
<td></td>
</tr>
<tr>
<td>Grade of recommendation</td>
<td>B</td>
<td></td>
</tr>
</tbody>
</table>

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### Characteristics of Dietary Pattern

The included reviews characterise a Healthy/prudent dietary pattern as one with a high consumption/dietary loading of vegetables, fruit, whole grains, and fish. Other foods included in this dietary pattern are low-fat dairy, poultry, soy, and olive oil.

---

SRRE Summary Relative Risk Estimates.

In this review clinical CVD was defined as CHD (including myocardial infarction and ischaemic heart disease), stroke (cerebrovascular disease and ischaemic stroke) and overall CVD.

CHD includes myocardial infarction and ischaemic heart disease.
### Dietary Pattern: Mediterranean Diets

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Satisfactory</td>
<td>One level I study including a meta-analysis and had moderate risk of bias(^29) and Two Reviews of level II studies(^30,31) including one meta-analysis(^30) with an unclear risk of bias.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Rees et al. 2013(^28) Review of Level 1 with moderate risk of bias</td>
</tr>
</tbody>
</table>

**Number and type of studies reviewed**

The Cochrane review included 11 RCTs\(^29\). Some studies included in this review were secondary prevention, however this was <25%.

**Number of databases searched**

9 databases searched\(^29\)

**CVD outcomes assessed**

Lipids (Total Chol, LDL, HDL, TG) and blood pressure\(^29\)

**Study quality**

1. Review used PRISMA\(^29\)
2. Assessed review quality using a standardised tool\(^29\). For blinding, risk of bias was unclear in 7 trials and low in 4. In terms of incomplete outcome data, 4 trials were low risk of bias, 3 were high risk, and the remaining trials were unclear. For selective reporting, there was an unclear risk of bias for four studies and low risk for the remaining 7 trials.
3. Potential for bias from funding source of included studies not reported.

**Funding source**

Reported receiving external funding from NIHR Cochrane Programme Grant, UK. No potential bias.

- Sofi et al. 2014\(^30\) Review of level II studies with an unclear risk of bias

**Number and types of studies reviewed**

35 observational studies\(^30\)

**Number of databases searched**

6 databases searched

**CVD outcomes assessed**

Review assessed CVD and incidence of neoplastic disease\(^30\)

**Study quality**

1. PRISMA not used
2. No report of quality assessment
3. Potential for bias from funding sources not reported
Funding source
No funding received

- Tyrovolas et al. 2010
  Review of level II studies with unclear risk of bias

Number and types of studies reviewed
10 studies included in review: 7 prospective cohort studies, 2 cross-sectional, 1 case-control. Of these, 9 evaluated CVD risk

Number of databases searched
2 databases searched

CVD outcomes assessed
CHD mortality, myocardial infarction, lipids, blood pressure, triglycerides

Study quality:
1. PRISMA not used.
2. No report of quality assessment.
3. Potential for bias from funding sources not reported.

Funding source
Funding source not reported

Consistency: Good

One meta-analysis found significant impacts on CVD-related mortality. 30

Total cholesterol reduction (in 8 trials) (-0.16mmol/L 95% CI -0.26 to -0.06; random-effects model, P=0.003, I² = 74%) and low-density lipoprotein (LDL) cholesterol (-0.07 mmol/L, 95% CI -0.13 to -0.01), fixed effect model, I² = 22%. A statistically significantly greater reduction in total cholesterol was observed for interventions describing themselves as a Mediterranean diet (MD -0.23 mmol/L, 95% CI -0.27 to -0.2) compared with those that did not (contained a minimum of two components, but did not increase fruit and vegetables or replace saturated fats with monounsaturated fats (MD -0.06 mmol/L, 95% CI -0.13 to 0.01. Significant heterogeneity was observed for trials involving TGs, HDL-C and BP and meta-analysis was not performed.

The meta-analysis of cohort studies reported a 2-point increased adherence to the Mediterranean diet was associated with a significant reduction of all-cause mortality. (RR=0.92; 95% CI 0.91, 0.93; P<0.00001; I²=47%). A 10% reduced risk in mortality from and incidence of CVD was associated with a 2-point increased adherence to the Mediterranean (RR=0.90; 95% CI 0.87, 0.92; P>0.000001; I² = 38%).

Clinical impact: Good

Greater adherence to Mediterranean style dietary patterns is associated with a greater reduction in total cholesterol (MD -0.23 mmol/L, 95% CI -0.27 to -0.2) compared to those that did not contain...
a minimum of 2 components and did not increase fruit & vegetables or replace saturated fats with monounsaturated fats (MD -0.06 mmol/L, 95% CI -0.13 to 0.01).

Greater adherence to Mediterranean dietary (every 2-point increase in adherence score) patterns is associated with a reduced risk of all-cause mortality (RR 0.92; 95% CI 0.91, 0.93), CVD incidence and/or mortality RR = 0.90; 95% CI 0.87, 0.92; P<0.00001, I² = 38%).

<table>
<thead>
<tr>
<th>Generalisability</th>
<th>Good</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population in body of evidence can be contextualised to Adult Australians at risk of cardiovascular disease.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Applicability</th>
<th>Good</th>
</tr>
</thead>
<tbody>
<tr>
<td>The evidence base is relevant to the Australian health care setting generally.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>In adults, Mediterranean style dietary interventions lasting from 12 weeks up to 48 months lead to a greater reduction in total cholesterol and LDL cholesterol compared to control interventions.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade of recommendation</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greater alignment between the Mediterranean style dietary interventions with key elements of the traditional Mediterranean diet is associated with a greater reduction in total cholesterol.</td>
<td></td>
</tr>
</tbody>
</table>

<p>| |</p>
<table>
<thead>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>A greater adherence to a Mediterranean diet is associated with a reduced risk of all-cause mortality and combined CVD incidence and CVD mortality.</td>
</tr>
</tbody>
</table>
**Characteristics of Dietary Pattern**

Mediterranean style diet or dietary pattern includes the following dietary factors: a high intake of plant foods comprising mainly fruits and vegetables, cereals and whole grain breads, beans, nuts and seeds; locally grown, fresh and seasonal, unprocessed foods; large quantities of fresh fruit consumed daily whereas concentrated sugars or honey are consumed a few times per week in smaller quantities; olive oil as a main cooking ingredient and source of fat; low to moderate amounts of cheese and yogurt; low quantities of red meat and higher quantities of fish; and low to moderate amounts of red wine often accompanying main meals. The original Mediterranean type of diet reflects the common dietary pattern of communities in countries of the Mediterranean region in the early 1960s.

For inclusion in this Cochrane review, studies had to have at least 2 of the following:

1. High monounsaturated/saturated fat ratio (use of olive oil as main cooking ingredient)
2. Low to moderate red wine consumption
3. High consumption of legumes
4. High consumption of grains and cereals
5. High consumption of fruits and vegetables
6. Low consumption of meat and meat products and increased consumption of fish
7. Moderate consumption of milk and dairy products.

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**Dietary Pattern: Low Glycaemic Index/Glycaemic Load (Low GI/GL) diets**

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Satisfactory</td>
<td>One Level I review and meta-analysis with moderate risk of bias in the included studies</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Number and types of studies reviewed</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 RCTS (14 included in meta-analysis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Number of databases searched</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 databases (MEDLINE, EMBASE, Cochrane)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>CVD outcomes assessed</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fat free mass (FFM), weight, waist circumference, lipids (TC, LDL, HDL, TG), glycaemic control, CRP</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Study quality</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. <em>Review</em> used PRISMA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Review assessed independently for methodological quality by both authors using the risk of bias assessment tool by the Cochrane Collaboration. Not all studies provided information on the quality of their respective setup. No other information on results from risk of bias provided (and not in supplementary materials).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Potential for bias from funding not reported.</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Funding source</strong> Review made no mention of funding or conflicts of interest.</td>
</tr>
</tbody>
</table>
The review compared low GI/Low glycaemic load versus high GI/high glycaemic load. This review showed no significant changes in total cholesterol: -0.032mmol/L (95% CI -0.146 to 0.08, p=0.59) \( I^2 = 44\% \); LDL-C: -0.008mmol/L (95% CI -0.115 to 0.10, p=0.89) \( I^2 = 54\% \); HDL-C: 0.019mmol/L (95% CI -0.006 to 0.044, p= 0.14) \( I^2 = 0\% \); Triglycerides: -0.010 mmol/L (95% CI -0.064 to 0.044, p=0.72, \( I^2 = 0\% \)) between LGI/LGL vs. HGI/HGL diets. However, this review reported that decrease in FFM was significantly greater following LGI/LGL diet compared with HGI/HGL = -1.04kg (95% CI -1.73 to -0.35, p = 0.003), \( I^2 = 0\% . \)

There were no differences in fasting plasma cholesterol between groups when comparing low GI/low glycaemic load and high GI/high glycaemic load diets. 

Population in body of evidence can be contextualised to Adult Australians without cardiovascular disease.

The evidence base is relevant to the Australian health care setting generally.

A low GI low GL diet is more effective in reducing Fat-Free Mass over 6-17 months than a high GI high GL diet, however a low GI, low GL diet is not more effective in reducing fasting plasma cholesterol in interventions lasting from 6-17 months.

Glycaemic index: The GI ranks the carbohydrate content of individual foods according to their postprandial glycaemic effects expressed as a percentage of the response to an equivalent carbohydrate portion of a reference food such as 50g of glucose. 

Glycaemic load: was introduced to quantify the glycaemic effect of the food with respect to its specific carbohydrate content in typically consumed quantities.
**Dietary Pattern: Dietary Approaches to Stop Hypertension (DASH) diet**

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Excellent</td>
<td>3 systematic reviews and meta-analyses consisting of level I studies (^{40}), level III-2 studies (^{41}) and level III-2 studies (^{42}). Overall, the quality of the included studies was positive, with low risk of publication bias.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Saneei et al. 2014 (^{40})</td>
</tr>
<tr>
<td>Number and type of studies reviewed</td>
<td></td>
<td>3 RCTs (n = 293)</td>
</tr>
<tr>
<td>Number of databases searched</td>
<td></td>
<td>Minimum of 3 databases for relevant studies</td>
</tr>
<tr>
<td>CVD outcomes assessed</td>
<td></td>
<td>Blood pressure</td>
</tr>
</tbody>
</table>
| Study quality: |             | 1. PRISMA: Yes  
                  2. Cochrane Risk of Bias tool used for quality assessment of included studies.  
                  3. Funding source potential from bias: no                                                                                         |
| Funding source: |             | Food Security Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.                                                     |
|                |             | • Salehi-Abargouei et al. 2013 \(^{41}\)                                                                                                  |
| Number and type of studies reviewed |             | 6 cohorts (n = 259,984)                                                                                                                  |
| Number of databases searched |             | Minimum of 3 databases for relevant studies                                                                                              |
| CVD outcomes assessed |             | CVD incidence &/or mortality and CHD, HF and stroke incidence &/or mortality                                                            |
| Study quality: |             | 1. PRISMA: No  
                  2. Quality of included studies not done and/or not reported: not reported  
                  3. Funding source potential from bias: no                                                                                         |
| Funding source: |             | Food Security Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.                                                     |
### DIETARY PATTERNS AND CARDIOVASCULAR DISEASE OUTCOMES

- **Schwingshackl et al. 2015**

**Number and types of studies:**

9 cohort studies (n = 852,438).

**Number of databases searched**

Minimum of 3 databases for relevant studies

**CVD outcomes assessed**

CVD incidence and/or mortality

**Study quality:**

1. PRISMA: No
2. Quality of included studies assessed via Newcastle-Ottawa Quality Assessment Scale for cohort studies
3. Funding source potential from bias: No (no funding to disclose).

<table>
<thead>
<tr>
<th>Consistency</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Studies were conducted with male and female adults aged 30 – 83 years. Many of the cohort studies were sex specific (female only). Study durations and follow-up ranged from 4 weeks to 24 years. Control diets in RCTs included: usual, healthy, low-fat, moderate sodium and low-fat, low carbohydrate or low antioxidant diets. Overall, there was minimal heterogeneity ($I^2 = 0-30%$, $p = 0.16$) between studies. Reviews reporting CVD and BP outcomes had low levels of heterogeneity ($I^2 = 14.3%$, $p = 0.16$, and $I^2 = 0%$, $p = 0.389 – 14.3%$, $p = 0.312$, respectively). CHD, HF and stroke outcomes showed no heterogeneity between studies ($I^2 = 0%$).</td>
<td></td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Clinical impact</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>DASH-style diet significantly decreased risk for CVD incidence and CVD mortality by 20% [RR 0.80 (95% CI: 0.74–0.86), $p&lt;0.001$]; and RR 0.80 (95% CI: 0.76, 0.85), $p&lt;0.00001$. One meta-analyses also found a significantly reduced risk of CHD, HF and stroke [RR 0.79 (95% CI: 0.71, 0.88), $p &lt; 0.001$; RR 0.71 (95% CI: 0.58, 0.88), $p &lt; 0.001$; and RR = 0.81 (95% CI: 0.72, 0.92), $p &lt; 0.001$, respectively]. DASH diet, compared to control group (intervention duration 8–14 weeks), resulted in significant decreases in both systolic and diastolic BP [SBP -2.44 mmHg (95% CI: -3.84, -1.03); DBP -1.69 mmHg (95% CI: -2.71, -0.67)].</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Generalisability</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population in body of evidence can be contextualised to healthy Australian adults.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Applicability</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>The evidence base is relevant to the Australian population generally.</td>
<td></td>
</tr>
</tbody>
</table>

**Recommendation**

In adults (>18 years), following a DASH dietary pattern for periods of time from 8 to 14 weeks is effective in lowering systolic and diastolic blood pressure compared to usual diets.

**Grade of recommendation**: A
### Recommendation

Following a DASH dietary pattern is associated with ~20% reduced risk of mortality and incidence from cardiovascular disease, coronary heart disease, heart failure and stroke; in adults (>18 years) in periods of time up to 24 years.

| Grade of recommendation | A |

### Characteristics of Dietary Pattern

The Dietary Approaches to Stop Hypertension (DASH) eating pattern is a recognised dietary approach that aims to lower BP. This diet emphasises foods that are lower in sodium, as well as foods that are rich in potassium, magnesium and calcium. This eating pattern is rich in fruit, vegetables, whole grains, legumes, seeds, nuts, fish, poultry and low-fat dairy, with a reduced content of saturated and total fat, and no added salt.
**Evidence Statements – Secondary Prevention**

**Dietary Pattern: Weight loss/ calorie-restricted diets**

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Satisfactory</td>
<td>1 systematic review and meta-analysis of randomized controlled trials (level II studies) with moderate risk of bias.</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Number and type of studies reviewed</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 RCTs</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Number of databases searched</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>7 databases were searched (Database of Abstracts of Reviews of Effectiveness, Cochrane database of systematic reviews, Cochrane Central Register of Controlled Trials (CENTRAL), the Cochrane Hypertension Specialised Register, MEDLINE, EMBASE, ClinicalTrials.gov).</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>CVD outcomes assessed</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cardiovascular morbidity, changes in SBP and DBP and weight reduction</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Study quality</strong></td>
</tr>
</tbody>
</table>
|                      |                 | 1. PRISMA statement used.  
2. Cochrane risk of bias assessment tool used. Risk of bias was unclear or high in 6/8 trials.  
3. Three out of eight included studies did not mention industry sponsoring. |
|                      |                 | **Funding source**                                                                                                                                                                         |
|                      |                 | Medical University of Graz and Institute of General Practice, Goethe University                                                                                                           |
| Consistency          | Excellent       | A systematic review and meta-analysis of RCTs evaluated weight-loss interventions of a minimum 24 weeks duration on weight loss and blood pressure in adults with primary hypertension. For SBP there was a mean difference of -4.5 mmHg (95% CI: -7.2 to -1.8, heterogeneity $I^2 = 21\%$, Chi$^2 = 3.78$, df= 3, $P = 0.3$) (in 3/8 studies that reported this outcome) and for DBP a mean difference of -3.2 mmHg (95% CI: -4.8 to -1.5 heterogeneity $I^2 = 35\%$, Chi$^2 = 7.67$, df = 5, $P = 0.2$) (in 3/8 studies). In weight reduction interventions change was -4.0kg (95% CI: -4.8 to -3.2 heterogeneity $I^2 = 34\%$, Chi$^2=9.14$, $P=0.2$) (in 5/8 studies) compared with controls. One of the included studies evaluated the hazard ratio for participants receiving dietary intervention to reach combined endpoint (reinstate antihypertensive therapy and severe cardiovascular complications); hazard ratio was 0.70 (95% CI: 0.57 to 0.87). |
| Clinical impact      | Good            | Energy-restricted diets resulted in significant decreases in SBP and DBP in adults with existing hypertension (duration of trials >24 weeks). Weight loss diets reduced body weight in people with hypertension, although magnitudes of the effect are unknown. |
Generalisability | Good | Population in body of evidence can be contextualised to adult Australians with primary hypertension.

Applicability | Excellent | The evidence base is relevant to the Australian health care setting generally.

Recommendation | Energy restricted diets of a duration of >24 weeks in adults with primary hypertension resulted in decreases in systolic blood pressure and diastolic blood pressure and in reductions of body weight. It is unclear what amount of energy restriction is optimal for this effect.

Grade of recommendation | C

Characteristics of Dietary Pattern

This review characterised weight-loss diets as dietary interventions with the intention to reduce body weight, but did not elaborate further on characteristics of the dietary pattern.

Dietary Pattern: Dietary Approaches to Stop Hypertension (DASH) diet

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Excellent</td>
<td>1 systematic review and meta-analysis consisting of: level I studies. 40</td>
</tr>
</tbody>
</table>

Number and type of studies reviewed

Systematic review contained relevant studies from 4 databases (15 RCTs, n= 1747).

Number of databases searched

4 databases

CVD outcomes assessed

SBP and DBP

Study quality:

1. PRISMA: Yes
2. Cochrane Risk of Bias tool used to assess quality of included studies. Overall, the quality of the included studies was positive, with low risk of publication bias.
3. Funding source potential from bias: no

Funding source:

Food Security Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.
Studies were conducted in male and female hypertensive adults (mean age 35 – 60 years) with/without T2DM and MetS. Three RCTs were female sex specific. The interventions in the majority of RCTs was dietary advice/education to use DASH diet, other RCTs supplied participants with the food items of the DASH diet. Eight trials applied energy restriction. Control diets were not specified. Study durations ranged from 2 – 26 weeks. Between-study heterogeneity was significant for both SBP ($I^2 = 70.2\%, p = 0.000$) and DBP ($I^2 = 46.5\%, p = 0.025$).

Systematic review found the DASH diet (with/without energy restriction), compared with control diet, reduced SBP [MD $-6.82\text{mmHg} (95\% \text{ CI: } -8.55, -5.09)$] and DBP [MD $-3.59\text{mmHg} (95\% \text{ CI: } -4.41, -2.76)$], regardless of duration of the interventions (below/above 12 weeks).

Countries in which RCTs were conducted was not specified, however the body of evidence can be contextualised to hypertensive Australian adults with/without Type 2 Diabetes Mellitus or Metabolic Syndrome.

The evidence base is relevant to the Australian healthcare context.

In hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 2 to 26 weeks is effective in lowering systolic and diastolic blood pressure compared to usual diets.

The Dietary Approaches to Stop Hypertension (DASH) eating pattern is a recognised dietary approach that aims to lower BP. This diet emphasises foods that are lower in sodium, as well as foods that are rich in potassium, magnesium and calcium. This eating pattern is rich in fruit, vegetables, whole grains, legumes, seeds, nuts, fish, poultry and low-fat dairy, with a reduced content of saturated and total fat, and no added salt.

1 systematic reviews consisting of: level I studies 16

Number and types of studies reviewed
Systematic review contained relevant studies from 2 databases (3 RCTs, n = 245).

CVD outcomes assessed
Lipid levels: TC, LDL, HDL and TG.

Study quality:
Overall, the quality of the included studies was neutral, with low to
Characteristics of Dietary Pattern

The Portfolio eating pattern is a vegetarian diet; low in saturated fat, rich in fruits, vegetables, and whole grains; aimed at lowering cholesterol. The diet emphasises consuming a 'portfolio' of various cholesterol-lowering foods - nuts, plant sterols, fibre and soy protein, as part of the daily diet. 51
<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Satisfactory</td>
<td>One Level I review of level II studies, including meta-analyses and with moderate risk of bias in the included studies</td>
</tr>
<tr>
<td><strong>Evidence Base</strong></td>
<td></td>
<td><strong>Number and types of studies reviewed</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>28 RCTs</td>
</tr>
<tr>
<td><strong>Number of databases searched</strong></td>
<td></td>
<td>3 databases searched (OVID Medline, EMBASE, Cochrane).</td>
</tr>
<tr>
<td><strong>CVD outcomes assessed</strong></td>
<td></td>
<td>Blood lipids (total cholesterol, LDL, HDL, triglycerides)</td>
</tr>
<tr>
<td><strong>Study quality</strong></td>
<td></td>
<td>1. Review used PRISMA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Review assessed quality using two independent researchers to determine the risk of bias using the criteria specified by Jadad and Schulz. The quality of the studies was variable and often difficult to assess due to studies providing insufficient information to assess risk of bias. Thirteen studies reported what the study was powered towards, only one was powered towards a change in blood lipids. Risk of bias was assessed via 5 categories, with 17/28 reviews receiving an ‘unclear’ or ‘high’ bias for 3 out of 5 categories (predominantly unclear not high).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Potential for bias from funding not reported</td>
</tr>
<tr>
<td><strong>Funding source</strong></td>
<td></td>
<td>Review was funded by King’s College, London</td>
</tr>
<tr>
<td><strong>Consistency</strong></td>
<td>Excellent</td>
<td>The review compared low GI diets versus high GI diets. This review found low GI diets led to significant reduction in total cholesterol by -0.13mmol/L (95% CI -0.22 to -0.04, p=0.004), I² = 0% and LDL-C by -0.16mmol/L (95% CI -0.24 to -0.08, p&lt;0.0001) I²=0% compared to high GI diets. GI had no effect on HDL-C concentrations: MD -0.03mmol/L, 95% CI -0.06 to -0.00, p= 0.06) I² = 0%. There was no significant effect of lowering GI on triglycerides (MD 0.01mmol/L, 95% CI -0.06 to 0.08, p=0.69, I² = 0%).</td>
</tr>
<tr>
<td><strong>Clinical impact</strong></td>
<td>Satisfactory</td>
<td>A low GI diet reduced total cholesterol and LDL-cholesterol</td>
</tr>
<tr>
<td><strong>Generalisability</strong></td>
<td>Good</td>
<td>Population in body of evidence can be contextualised to Adult Australians with cardiovascular disease or without cardiovascular disease.</td>
</tr>
<tr>
<td><strong>Applicability</strong></td>
<td>Good</td>
<td>The evidence base is relevant to the Australian health care setting generally.</td>
</tr>
<tr>
<td><strong>Recommendation</strong></td>
<td></td>
<td>A low GI diet is more effective than high GI diet in lowering fasting plasma cholesterol in interventions lasting from 1-20 months.</td>
</tr>
</tbody>
</table>
## Characteristics of Dietary Pattern

Low GI foods stimulate less insulin secretion, and have been shown to limit reductions in insulin sensitivity \(^{20}\).

## Dietary Pattern: Low-Fat Diet

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Good</td>
<td>One level I systematic review with a meta-analysis of randomized controlled trials, with unknown risk of bias (^{16})</td>
</tr>
<tr>
<td>Number and type of studies reviewed</td>
<td></td>
<td>Systematic review of 136 studies, n=16 evaluated low-fat diets (n=4 meta-analysis and n=14 RCTs). Three studies were in healthy populations and the rest in populations with hyperlipidaemia, CAD, or T2DM.</td>
</tr>
<tr>
<td>Number of databases searched</td>
<td></td>
<td>2 databases searched (Medline and Cochrane)</td>
</tr>
<tr>
<td>CVD outcomes assessed</td>
<td></td>
<td>Lipid profile: TC, LDL, HDL and TG.</td>
</tr>
<tr>
<td>Study quality</td>
<td></td>
<td>1. PRISMA: No</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Unclear if quality assessment and risk of bias was performed.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Potential for bias from included studies from funding not reported.</td>
</tr>
<tr>
<td>Funding source</td>
<td></td>
<td>Funding source not reported</td>
</tr>
<tr>
<td>Consistency</td>
<td>Good</td>
<td>Diet with 18-30% of total energy intake as fat significantly reduced total and LDL cholesterol by 5% to 15% in adults in healthy populations and those with hyperlipidaemia, CAD and T2DM. However, there was no clear benefit in most studies for triglycerides and HDL. Heterogeneity was not assessed (^{16}).</td>
</tr>
<tr>
<td>Clinical impact</td>
<td>Satisfactory</td>
<td>Adherence to a low-fat diet (≤30% daily energy from fat) was associated with significantly reduced total and LDL cholesterol by 5% to 15% in adults. There was no further effect from diets with ≤18% total energy from fat.</td>
</tr>
<tr>
<td>Generalisability</td>
<td>Satisfactory</td>
<td>Population in body of evidence can be contextualised to healthy Australian adults and Australian adults with hyperlipidaemia and coronary artery disease.</td>
</tr>
<tr>
<td>Applicability</td>
<td>Satisfactory</td>
<td>The evidence base is relevant to the Australian population generally.</td>
</tr>
<tr>
<td>Recommendation</td>
<td>In adults aged &gt;18 years, following a low-fat dietary pattern, with fat intake ≤30% of total daily energy, for periods of time from 2 weeks to 4 years, leads to modest improvements in total</td>
<td></td>
</tr>
</tbody>
</table>
cholesterol and LDL and HDL cholesterol levels compared to usual dietary intakes.

Grade of recommendation

B

Characteristics of Dietary Pattern

A low-fat dietary pattern is one where ≤30% of an individual’s total daily kJ intake comes from fat.

<table>
<thead>
<tr>
<th>Dietary Pattern: Low CHO diets</th>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Good</td>
<td>One level I review and meta-analysis of type II studies with unknown risk of bias 19</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Number and types of studies reviewed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>The study had 17 RCTs.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Number of databases searched</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>The review included 1 database (PubMed)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CVD outcomes assessed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>The study assessed weight (kg and BMI), fasting plasma cholesterol, HDL, LDL, TG, Systolic BP</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Study quality:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. The study used PRISMA.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Unclear if quality assessment was performed.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Potential for bias from funding of included studies not reported.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Funding source</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>The review was funded by Atkins Nutritionals. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.</td>
<td></td>
</tr>
<tr>
<td>Consistency</td>
<td>Good</td>
<td>Compared with the low-fat diet, the low carbohydrate (low CHO) diet had a greater reduction in weight (pooled mean change = -2.0 kg, 95% CI: -3.1, -0.9), BMI: -0.7 (95% CI: -1.1, -0.3) P=0.0016 and systolic blood pressure -1.7mmHg (95% CI: -3.5, 0.2), P=0.08. The low CHO diet also favourably affected triglycerides compared to the low-fat diet: between group difference -0.33mmol/L (95% CI: -0.44, -0.21) P&lt;0.0001. The low CHO also favourably affected HDL cholesterol compared to the low-fat diet: between group difference 0.13mmol/L (95% CI: 0.09, 0.17) P&lt;0.0001.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low-fat significantly more favourably affected total cholesterol: between group difference -0.24mmol/L (95% CI: -0.07, -0.41) P=0.006 and LDL compared to low CHO: between group difference -0.22mmol/L (95% CI: -0.09, -0.35) P=0.0008.</td>
<td></td>
</tr>
<tr>
<td>Clinical impact</td>
<td>For Low CHO:</td>
<td>The systematic review of RCTs found that both low CHO and low-fat diets reduced weight, BMI, fasting lipids (total cholesterol, LDL-</td>
<td></td>
</tr>
</tbody>
</table>
For Low-fat: Satisfactory

Compared with the low CHO diet, the low-fat diet had a greater reduction in total cholesterol and LDL cholesterol. The low CHO diet had more favourable outcomes than the low-fat diet for HDL cholesterol and triglycerides.

Generalisability Excellent

Population in body of evidence can be contextualised to overweight/obese Adult Australians who may or may not have dyslipidaemia.

Applicability Good

The evidence base is relevant to the Australian health care setting generally.

Recommendation

A low CHO diet (120gram/day or less) is more effective in lowering weight, BMI, blood pressure, and triglycerides than a low-fat diet in an intervention lasting from 8 weeks up to 2 years.

Grade of recommendation B

Recommendation

A low-fat diet (<30% Cals from fat/day) is more effective in lowering total cholesterol and LDL-cholesterol in an intervention lasting from 8 weeks up to 2 years.

Grade of recommendation B

Characteristics of Dietary Pattern

Low CHO diets (120gram/day or less), Low-fat diets (<30% Cals from fat/day)

Dietary Pattern: Mediterranean Diet

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
</table>
| Evidence Base     | Satisfactory | Four level I studies with an unclear risk of bias, 1 level I study with a low-moderate risk of bias, 2 level I studies with a moderate risk of bias, 1 Level II with unclear risk of bias, and 3 Level III with unclear risk of bias.  
  * Gay et al. 2016 14. Level I with low/unclear risk of bias  
  
Number and type of studies reviewed  
5 RCTs in this review evaluated a Mediterranean diet (out of a total 24 included RCTs).  

Number of databases searched  
3 databases searched (PubMed, Embase and Web of Science)  

CVD outcomes assessed  
Systolic blood pressure (SBP) and diastolic blood pressure (DBP)  

Study quality  
1. PRISMA statement used.  
2. Cochrane risk of bias tool was used to assess quality of included studies, assessed for selection bias, performance bias, detection bias, attrition bias and reporting bias. Of the 5 included studies, 3 had 1 component at a high risk of bias (out of 7 components) but other components were low risk of bias. Two studies had either mostly low risk scores for components,
but some components were graded unclear for risk of bias.
3. Potential for risk of bias from funding of included studies not reported.

**Funding source**
Funding source of review not reported.

- **Huang et al. 2011** 16 Level I with unclear risk of bias

**Number and type of studies reviewed**
8 RCTs (2 in healthy populations, the remaining in populations with hyperlipidaemia, CAD, and metabolic syndrome)

**Number of databases searched**
2 databases were searched (Cochrane library and Medline)

**CVD outcomes assessed**
Lipid profile

**Study quality**
1. PRISMA not used.
2. Unclear if quality assessment of included studies occurred.
3. Potential for bias from funding of included studies not reported.

**Funding source**
Funding source of review not reported

- **Ndanuko et al. (2016)** 43 Level I with unclear risk of bias

**Number & types of studies reviewed**
Systematic review and meta-analysis of 17 RCTs. Of these 17, five RCTs evaluated Mediterranean diets (only 3 included in meta-analysis). Participants included in these trials were populations with and without existing cardiovascular disease (Primary and secondary).

**Number of databases searched**
3 databases searched (Scopus, Web of Science, MEDLINE)

**CVD outcomes assessed**
Systolic blood pressure (SBP) and diastolic blood pressure (DBP)

**Study quality:**
1. PRISMA statement used.
2. Cochrane risk of bias assessment tool used. One study was high risk of bias for incomplete outcome data, however it was unclear about the risk of bias in other studies or other components assessed.
3. Potential for bias from funding of included studies not reported.

**Funding source**
No funding received for this review
• Grosso et al. 2014 ³⁷ Level III with unclear risk of bias

**Number and type of studies reviewed**

58 studies included (n=33 cross-sectional, n=9 cohorts, and n=16 intervention studies).

**Number of databases searched**

1 database searched (PubMed)

**CVD outcomes assessed**

Weight, blood pressure, CVD mortality, lipid profile

**Study quality**

1. PRISMA not used
2. Quality assessment of included studies not performed or not reported.
3. Potential for bias from funding of included studies not reported.

**Funding source**

Funding source of review not reported

• Kastorini et al. 2010 ³³ Level III with unclear risk of bias

**Number and type of studies reviewed**

35 studies included (3 were prospective, 11 were cross-sectional studies, and 21 were clinical trials).

**Number of databases searched**

3 databases searched (PubMed, Embase, Scopus)

**CVD outcomes assessed**

Weight, CVD morbidity and mortality, lipid profile

**Study quality**

1. PRISMA not used
2. Quality assessment of included studies not assessed or not reported.
3. Potential for bias from funding of included studies unclear.

**Funding source**

Not reported

• Mancini et al. 2016 ³⁵ Level I with moderate risk of bias

**Number and type of studies reviewed**

5 RCTs

**Number of databases searched**

3 databases searched (Medline, Embase, and Cochrane library)

**CVD outcomes assessed**

Weight, lipid profile, blood pressure

**Study quality**

1. PRISMA used.
2. The Cochrane Collaboration’s tool was used for assessing risk
of bias in RCTs. Most trials had a low or unclear risk of bias for most domains however 2 trials had a high risk of bias for several domains.

3. Potential for bias from funding of included studies not reported.

**Funding source**
No funding received for this review

- Martinez-Gonzalez et al. 2014 38 Level II with unclear risk of bias

**Number and type of studies reviewed**
2 RCTs and 12 observational cohort studies

**Number of databases searched**
2 databases searched (PubMed and Institute for scientific information web of knowledge)

**CVD outcomes assessed**
CVD risk

**Study quality**
1. PRISMA used.
2. Quality assessment of included studies not reported/not assessed.
3. Potential for bias from funding of included studies not reported.

**Funding source**
Funding source of review not reported

- Nissensohn et al. 2016 36 Level I with low-moderate risk of bias

**Number and type of studies reviewed**
6 RCTs included in this review

**Number of databases searched**
2 databases searched (PubMed and Scopus)

**CVD outcomes assessed**
Hypertension

**Study quality**
1. PRISMA used.
2. Risk of bias was assessed using domains adapted from the Cochrane Handbook for Systematic Reviews. 5 studies had a low risk of bias and one study had a high risk.
3. Risk of bias from funding of included studies was assessed as one of the domains of quality assessment. No studies had a potential risk of bias from funding.

**Funding source**
Funding source of review not reported
<table>
<thead>
<tr>
<th>Study</th>
<th>Level of Evidence</th>
<th>Risk of Bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liyanage et al. 2016</td>
<td>Level I</td>
<td>Moderate</td>
</tr>
<tr>
<td>Schwingshackl et al. 2014</td>
<td>Level I</td>
<td>Unclear</td>
</tr>
<tr>
<td>Kastorini et al. 2011</td>
<td>Level III</td>
<td>Unclear</td>
</tr>
</tbody>
</table>

**Number and type of studies reviewed**

- 6 RCTs included
- 17 RCTs
- 50 studies included (n=35 clinical trials, n=2 prospective studies, n=13 cross-sectional studies).

**Number of databases searched**

- 3 databases searched (Medline, Embase and Cochrane)
- 3 databases searched
- 4 databases searched (PubMed, Embase, Web of Science, Cochrane library)

**CVD outcomes assessed**

- CVD events and mortality
- Endothelial function and inflammatory markers including CRP

**Study quality**

1. PRISMA used.
2. Cochrane risk of bias assessment tool was used to assess quality of included studies. The quality of included studies was variable.
3. Potential for bias from funding of included studies not reported.
4. PRISMA used.
5. The risk of bias assessment tool by the Cochrane Collaboration was used to assess quality of included studies. Unclear how many studies were high or low risk of bias.
6. Potential for bias from funding of included studies unknown.

**Funding source**

- Review supported by the NHMRC and individual authors supported by postgraduate scholarships, the NHMRC and Canadian Institutes of Health Research and Alberta Innovates Health Solutions.
- Not reported

- Schwingshackl et al. 2014 supported by the NHMRC and individual authors supported by postgraduate scholarships, the NHMRC and Canadian Institutes of Health Research and Alberta Innovates Health Solutions.
- Not reported

- Kastorini et al. 2011 supported by the NHMRC and individual authors supported by postgraduate scholarships, the NHMRC and Canadian Institutes of Health Research and Alberta Innovates Health Solutions.
- Not reported
Blood pressure, lipid profile, waist circumference

**Study quality**

1. 1.PRISMA not used
2. Quality of studies was assessed according to: number of participants, duration of follow-up, adjustment for potential confounders. However, findings are not reported.
3. Potential for bias from funding of included studies not reported.

**Funding source**

Funding source of review not reported

<table>
<thead>
<tr>
<th>Consistency</th>
<th>For blood pressure: Good</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gay et al. (2016) reviewed the association between a Mediterranean diet pattern and blood pressure, with reductions in SBP and DBP but high heterogeneity between studies. There was a reduction in SBP of MD=-1.17 (95% CI: -2.81 to -0.46, heterogeneity: I²=93%, Chi²=55.00, P=0.00001); and DBP MD=-1.44 (95% CI: -2.11 to -0.76, heterogeneity: I²=82%, Chi²=22.23, P=0.0002) 14. Ndanuko (2016) found a Mediterranean dietary pattern significantly reduced SBP (MD=3.02 mmHg [95% CI: -3.47, -2.58], I²=0%, Chi²=1.80, P=0.41) and DBP (MD=1.99 mmHg [95% CI: -2.28, -1.71], I²=0%, Chi²=0.06, P=0.97) in n=3 studies 43. The review by Nissensohn et al. (2016) concurs that adopting the Mediterranean diet for at least 1 year reduced SBP (MD=1.44 mmHg, 95% CI: -2.88 to 0.01, I²=87%, Chi²=46.22, P&lt;0.00001), and DBP (MD=0.70 mmHg, 95% CI: -1.34 to -0.07, I²=63%, Chi²=16.26, P=0.01) in normotensive populations and those with mild hypertension 36. Grosso et al. reported improvements in blood pressure in three of the included studies in their review. However no meta-analysis was performed in this review or heterogeneity between studies reported 37. Kastorini et al. (2011) revealed the protective role of the Mediterranean diet was associated with lower SDP (-2.35 mm Hg, 95% CI: -3.51 to -1.18) and DBP (-1.58 mm Hg, 95% CI: -2.02 to -1.13). Heterogeneity was observed for SBP (I²=51.8%) but not for DBP (I²=6.7%). Observational studies did not show significant effects of the Mediterranean diet on DBP or SBP. 32</td>
</tr>
<tr>
<td>For lipid profile: Good</td>
<td>Huang et al. (2011) reported reductions in total cholesterol by 5-15%, in LDL by 5-15% and increase in HDL by 3-15% in participants with and without CVD 16. Results from the RCTs included in the study by Kastorini et al. (2011) revealed the protective role of the Mediterranean diet was associated with statistically higher HDL compared with control diet (0.03 mmol/L 95% CI: 0.38 to 1.96), and lower triglycerides (-0.07 mmol/L, 95% CI: -10.35 to -1.93) 32. However, heterogeneity was observed for HDL (I²=63.6%), and TG’s (I²=55.3%). Some observational studies reported beneficial effects of the diet on TGs and HDL 32. Mancini et al. reported that all diets included in their review were similar at improving blood pressure and lipid levels. 35</td>
</tr>
<tr>
<td>For CVD outcomes: Satisfactory</td>
<td>Three studies included in the review by Grosso et al. (2014) reported improvements in lipid profiles associated with consumption of a Mediterranean diet pattern, and that the Mediterranean diet may be better at primary and secondary prevention of coronary artery disease than a conventional prudent diet. However, another included study found no improvement in markers of inflammation or metabolic risk with a Mediterranean diet in patients with established coronary artery disease. 37</td>
</tr>
</tbody>
</table>
In a systematic review comparing the Mediterranean diet with low-fat, low-CHO, and American Diabetes Association diets in overweight populations, there was greater weight loss than for the low-fat diet, but similar weight loss as for the other diets. 35 Kastorini et al. (2010) found in their review that not all studies showed a protective effect of the Mediterranean diet on body weight and obesity, however there was a possible beneficial role of this dietary pattern.33

In the meta-analysis by Martinez-Gonzalez et al. (2014) reported a pooled risk ratio of 0.62 (95% CI: 0.45 - 0.85) for risk of CVD in RCTs and risk ratio of 0.87 (95% CI 0.85 - 0.90, heterogeneity I²=19.8%, P=0.26) amongst observational studies- a 38% and 13% relative reduction in CVD risk, respectively, associated with Mediterranean diets.38

Mediterranean diet was associated with a 37% relative reduction in the risk of major CVD events (RR 0.69, 95% CI 0.55–0.86, p< 0.001) (RR 0.63, 95% CI 0.53–0.75, p< 0.001), compared to control diet 34. For all-cause mortality the Mediterranean diet had no clear effect on all-cause mortality (RR 1.00, 95% CI: 0.86–1.15, p = 0.97) or cardiovascular mortality (RR: 0.90, 95% CI: 0.72–1.11, p = 0.32). Mediterranean diet associated with relative risk of 0.65 (95% CI 0.50–0.85) for coronary events, 0.65 (95% CI 0.48–0.88) for stroke, and 0.30 (95% CI: 0.17–0.56) for heart failure. When a study of low integrity was excluded, coronary events and heart failure lost significance but stoke remained significant (RR: 0.66, 95% CI: 0.48–0.92, p = 0.01).

A Mediterranean dietary pattern significantly improved markers of inflammation CRP (c-reactive protein) by WMD: -0.98mg/l, 95% CI - 1.48 to -0.49, P<0.0001, I² = 91% 52

Adherence to a Mediterranean dietary pattern may significantly lower SDP (between -1.17 and -3.02 mmHg) and DBP (between -0.70 and -1.99 mmHg), and may improve total, HDL, and LDL cholesterol and triglycerides (effect size not clear), and result in a relative reduction in CVD risk between 13 – 38%. Adherence to a Mediterranean dietary pattern is associated with lower systolic and diastolic blood pressure, compared with control diets, in adults from healthy populations and in adults with existing cardiovascular disease or risk factors.

<table>
<thead>
<tr>
<th>Clinical impact</th>
<th>For blood pressure: Satisfactory</th>
<th>For lipid profile: satisfactory</th>
<th>For CVD outcomes: Good</th>
<th>For other outcomes: Unsure</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Generalisability</th>
<th>Good</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Applicability</th>
<th>Good</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Recommendation</th>
</tr>
</thead>
</table>

| Grade of Recommendation | C |

For blood pressure: Satisfactory
For lipid profile: satisfactory
For CVD outcomes: Good
For other outcomes: Unsure

The population in the body of evidence can be contextualised to adult Australians with and without pre-existing cardiovascular disease.

The evidence base is relevant to the Australian healthcare context.
**Recommendation**

Adherence to a Mediterranean dietary pattern may be associated with improvement in lipid profiles, compared with control diets, in adults from healthy populations and in adults with existing cardiovascular disease or risk factors.

**Grade of Recommendation**

C

**Recommendation**

Adherence to a Mediterranean dietary pattern may be associated with a relative reduction in CVD risk between 13 – 38%, compared with control diets, in adults from healthy populations and in adults with existing cardiovascular disease or risk factors.

**Grade of Recommendation**

C

**Characteristics of Dietary Pattern**

Huang et al (2011) characterised the Mediterranean diet as 25% to 35% of total energy from fat; moderate to high intake of fish but low consumption of red meat; moderate alcohol intake; small amounts of dairy products; and high consumption of non-refined grains, legumes, nuts, fruits, and vegetables. In addition to the aforementioned, Ndanuko et al. (2016), Liyange et al. (2016) and Martinez-Gonzalez specified that olive oil is the major source of fat, and that wine is consumed in moderation.
<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Good</td>
<td>1 systematic review with meta-analysis of 7 clinical trials (including 6 RCTs- level II) and 32 observational cross-sectional studies (level III-2), risk of bias unclear.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number and type of studies reviewed</td>
<td></td>
<td>7 clinical trials (including 6 randomized clinical trials) of 6 – 52 week duration, and 32 observational cross-sectional studies (in 22 of these participants had been following vegetarian diets for more than one year, in other studies participants were ‘currently following diet’) included in meta-analysis.</td>
</tr>
<tr>
<td></td>
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</tr>
<tr>
<td>Total 21,915 participants. In 5/7 of the clinical trials no participants were using antihypertensive medications; in one trial 81.8% of participants were using antihypertensive medication; and medication use was not reported in one trial. In the observational studies, 5/32 studies included participants who were taking antihypertensive medications (range of 11.4% – 58.4% of participants using medication); in 12/32 studies participants were not using medications, and in 15/32 studies medication use was not reported. Overall for this review, 15.4% of studies included participants taking antihypertensive medications, 43.6% of studies had no participants taking medication, and 41.0% of studies did not report on medication use.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of databases searched</td>
<td></td>
<td>2 databases searched (MEDLINE and Web of Science)</td>
</tr>
<tr>
<td>CVD Outcomes Assessed</td>
<td></td>
<td>Net differences in systolic and diastolic blood pressure</td>
</tr>
<tr>
<td>Study Quality</td>
<td></td>
<td>1. PRISMA not used.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Methods used to assess quality of included studies are not reported.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Potential for bias from funding of included studies not reported.</td>
</tr>
<tr>
<td>Funding Sources</td>
<td></td>
<td>Grant-in-aid for Japan Society for the Promotion of Science Fellows</td>
</tr>
<tr>
<td>Consistency</td>
<td>Good</td>
<td>1 systematic review and meta-analysis included clinical trials (n=7) and cross-sectional observational studies (n=32) that examined the association between vegetarian diets and blood pressure, compared with omnivorous diets.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>In the clinical trials, following a vegetarian diet (including vegan, lacto, and lacto-ovo) led to a mean reduction in systolic blood pressure (-4.8mmHg; 95% CI: -6.6 to -3.1; P&lt;.001) and diastolic blood pressure (-2.2mmHg; 95% CI: -3.5 to -1.0; P&lt;.001). Heterogeneity was I² = 0, P = .45 and I² = 0, P = .43, respectively.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>In the observational studies, vegetarian diets (including vegan, lacto,</td>
</tr>
</tbody>
</table>
lacto-ovo, and mixed diets) were associated with lower mean systolic blood pressure (-6.9mmHg; 95% CI: -9.1 to -4.7; P<.001) and diastolic blood pressure (-4.7mmHg; 95% CI: -6.3 to -3.1; P<.001). Heterogeneity was high for the observational studies at $I^2 = 91.4$, $P<.001$ and $I^2 = 92.6$, $P<.001$, respectively.

Clinical impact | Good | 1 systematic review and meta-analysis 39 found that vegetarian diets were associated with lower systolic and diastolic blood pressure in both hypertensive and normotensive populations when compared with omnivorous diets in both clinical trials and observational studies.

Generalisability | Good | Population in body of evidence can be contextualised to adult Australians who may be normotensive or have hypertension.

Applicability | Good | The evidence base is relevant to the Australian health care setting generally.

Recommendation | **Consumption of a vegetarian diet is associated with lower systolic and diastolic blood pressure, compared with consumption of omnivorous diets in adults who are taking hypertensive medication and those not taking medication.**

Grade of recommendation | B

**Characteristics of Dietary Pattern**

Vegetarian diets are dietary patterns generally excluding or rarely including meat; including:

**Semi-vegetarian diets:** Diets rarely including meat, **Vegan diets:** Diets omitting all animal products, **Lacto-vegetarian:** Vegetarian diet including dairy products, **Ovo-vegetarian:** Vegetarian diet including eggs and **Pesco-vegetarian:** Vegetarian diet including fish.

All vegetarian diets have an emphasis on plant based foods; particularly vegetables, grains, legumes, and fruit 39.

| Dietary Pattern: Dietary Approaches to Stop Hypertension (DASH) diet | Evidence Base | Excellent | 5 systematic reviews (all included meta-analyses) consisting of level I studies 23, level I studies 46, level I studies 53, level I studies 40, level I studies 43. Overall, the quality of the included studies was positive.

- Gay et al. 2016 23

**Number and type of studies reviewed**

Relevant studies from 3 databases (4 RCTs, n = 408)

**Number of databases searched**

3 databases searched

**CVD outcomes assessed**

Blood pressure

**Study quality**

1. **PRISMA:** Yes
2. Quality assessed via Cochrane Collaboration’s tool
3. Funding source potential from bias: No (No funding to disclose)

- Siervo et al. 2015 46

Number and type of studies reviewed
Relevant studies from 3 databases (20 RCTs, n = 1,917)

Number of databases searched
3 databases searched

CVD outcomes assessed
Blood pressure and lipid profile

Study quality
1. PRISMA: Yes
2. Quality of included studies assessed via modified Jadad score
3. Funding source potential from bias: no

Funding source
Ministry of Higher Education and Scientific Research of Iraq

- Soltani et al. 2016 53

Number and type of studies reviewed
Relevant studies from 4 databases (13 RCTs, n = 2,292)

Number of databases searched
4 databases searched

CVD outcomes assessed
Weight loss

Study quality:
1. PRISMA: Yes
2. Quality of included studies assessed via Cochrane Collaboration’s tool
3. Funding source potential from bias: no

Funding source:
Nutrition and Food Security Research Center, Shahid Sadoughi University of Medical Sciences, Iran

- Saneei et al. 2014 40

Number and type of studies reviewed
Relevant studies from 4 databases (4 RCTs, n = 495)

Number of databases searched
4 databases searched

CVD outcomes assessed
Blood pressure
| Study quality | 1. PRISMA: Yes  
2. Quality of included studies assessed via Cochrane Risk of Bias tool  
3. Funding source potential from bias: no |
|---|---|
| Funding source | Food Security Research Center, Isfahan University of Medical Sciences, Isfahan, Iran.  
- Ndanku et al. 2016 43 |
| Number and type of studies reviewed | Relevant studies from 3 databases (10 RCTs, n = 2798) |
| Number of databases searched | 3 databases searched |
| CVD outcomes assessed | Blood pressure |
| Study quality: | 1. PRISMA: Yes  
2. Quality of included studies assessed via Cochrane Collaboration’s tool  
3. Funding source potential from bias: no (no funding to disclose) |
| Consistency | Good |
| Studies were conducted in normotensive and hypertensive adults (mean age 31 – 67 years) with varying weight statuses, including participants with/without T2DM and MetS. One RCT included participants with heart failure (n = 48) and a small number of RCTs included participants receiving anti-hypertensive medication. Many of RCTs were sex specific. DASH diet interventions were administered with and without energy restrictions. Control diets in RCTs varied between studies and included typical (Western or usual) diet, healthy diet, calorie-restricted diet, low antioxidant diet, exercise only, advice only or standard follow-up. Study durations ranged from 1 to 48 months. Reviews A, B and E reporting BP outcomes had moderate to high levels of heterogeneity (I² = 61, p = 0.005 - 89%, p<0.00001), review D had low levels of heterogeneity (I² = 0%, p = 0.706 – 31.3%, p = 0.224). Review B reporting lipid outcomes had varied levels of heterogeneity (I² = 0 – 75.6%). Review C reporting weight loss outcomes had a high level of heterogeneity (I² = 71.2%, p ≤0.001), but subgroup analysis could largely describe the between-study heterogeneity (I² = 0%, p = 0.392 – 6.4%, p = 0.379). |
| Clinical impact | For blood pressure: Excellent  
Reviews A, B, D and E (58 RCTs; 29,068 participants; intervention duration 1 – 48 months) reporting on BP outcomes found the DASH diet, compared to control group, resulted in significant decreases in both systolic and diastolic BP. Effects on SBP ranged from -4.90
mmHg (95% CI: -6.22, -3.58) to -9.78 mmHg (95% CI: -11.40, -8.17); and effects on DBP ranging from -2.60 mmHg (95% CI: -3.5, -2.17) to -5.51 mmHg (95% CI: -7.30, -3.72). Reviews A and B reported effects were greater in participants with higher baseline BP or BMI.

Review B meta-analysis, comparing a DASH diet (intervention durations 2 – 24 weeks) to control diet (typical diet, healthy diet, calorie-restricted diet, low antioxidant diet or exercise only), reported significant decreases in concentrations of TC [-0.20 mmol/L (95% CI: -0.31, -0.10), p = 0.001] and LDL [-0.10 mmol/L (95% CI: -0.20, -0.01), p = 0.03].

The effect of the intervention was not significant for the concentrations of HDL [0.003mmol/l (95% CI: -0.05, 0.05), p=0.95); or TGs [0.005mmol/l (95% CI: -0.06, 0.05), p=0.87].

Review C meta-analysis of 13 RCTs (n = 2292) found a DASH diet with energy restriction, compared to a typical (Western or usual) diet, resulted in greater differences in weight loss [WMD -1.42 kg (95% CI: -2.03, -0.82) in 8–24 weeks], BMI [WMD -0.42 kg/m2 (95%CI: -0.64, -0.20) in 8–52 weeks] and waist circumference [WMD -1.05 cm (95%CI: -1.61, -0.49) in 24 weeks]. DASH diet without energy restriction also resulted in significant weight loss [WMD - 0.85 kg, (95% CI: -1.27, -0.43)], decrease in BMI and waist circumference. Effects were greater in overweight/obese participants.

### Generalisability
Good

The body of evidence can be contextualised to normotensive and hypertensive Australian adults with/without Type 2 Diabetes Mellitus or Metabolic Syndrome.

### Applicability
Good

The evidence base is relevant to the Australian healthcare context.

### Recommendation
In normotensive and hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 1 to 48 months is effective in lowering systolic and diastolic blood pressure compared to usual, healthy, calorie restricted, low-fat or low antioxidant diets.

**Grade of recommendation**

B

### Recommendation
Following a DASH diet (with or without energy restriction) in intervention from 8 to 52 weeks leads to weight loss, and reductions in BMI and waist circumference, with greater effects in overweight/obese participants.

**Grade of recommendation**

B

### Recommendation
In normotensive and hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 2 to 24 weeks leads to modest improvements in total cholesterol and LDL cholesterol levels.

**Grade of recommendation**

B
In normotensive and hypertensive adults (>18 years), following a DASH dietary pattern for periods of time from 2 to 24 weeks does not lead to significant improvements in Triglyceride or HDL cholesterol levels.

Characteristics of Dietary Pattern

The Dietary Approaches to Stop Hypertension (DASH) eating pattern is a recognised dietary approach that aims to lower BP. This diet emphasises foods that are lower in sodium, as well as foods that are rich in potassium, magnesium and calcium. This eating pattern is rich in fruit, vegetables, whole grains, legumes, seeds, nuts, fish, poultry and low-fat dairy, with a reduced content of saturated and total fat.

Dietary Pattern: High Protein Diet

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Satisfactory</td>
<td>3 Level I studies with unclear risk of bias and 1 Level 1 study with a moderate risk of bias.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Santesso et al. 2012 18</td>
</tr>
</tbody>
</table>

Number and type of studies reviewed

1 systematic review and meta-analysis consisting of: level II studies (n=74 RCTs) 18

Number of databases searched

Systematic review contained relevant studies from 4 databases

CVD outcomes assessed

CVD outcomes assessed included weight loss (38 RCTs, n = 2326), BMI (16 RCTs, n = 887) waist circumference (15 RCTs, n = 1214), BP (15 RCTs, n = 1186), total cholesterol profiles (21 RCTs, n = 1368), HDL and LDL cholesterol (23 RCTs, n=1555-1576) TG (24 RCTs, n = 1623) and CRP (5 RCTs, n = 398).

Study quality

1. PRISMA tool used.
2. GRADE tool (Grading of Recommendations Assessment, Development and Evaluation) was used to assign level of evidence, and this included an assessment of quality of included studies - however these results are not reported separately.
3. Potential for bias from funding of included studies unclear.

Funding source

Barilla (Food Company), Italy. One author was an employee of the sponsor and she was involved in the review and interpretation of the data in the manuscript. The final decision about interpretation did not rest with her, therefore bias unlikely.
• Wycherley et al. 2012

**Number and type of studies reviewed**
Systematic review and meta-analysis of 23 RCTs

**Number of databases searched**
4 databases searched

**CVD outcomes assessed**
Lipid profile, blood pressure and weight loss

**Study quality**
1. PRISMA used
2. Risk of selection bias, performance bias, and detection bias were evaluated using a modified Cochrane risk of bias assessment tool. For most studies, most risk of bias outcomes were graded ‘unclear’. Overall, the risk of selection bias was unclear. Only one study was low risk for detection bias, the rest were unclear. The majority of studies were high risk for performance bias.
3. Potential for bias from funding of included studies not reported

**Funding source**
Authors supported by an NHMRC project grant and NHMRC and National Heart Foundation postdoctoral research fellowship.

• Huang et al. 2011

**Number and type of studies reviewed**
2 RCTs evaluated high protein diets where protein was 25% of total energy.

**Number of databases searched**
2 databases searched

**CVD outcomes assessed**
Lipid profile

**Study quality**
1. PRISMA not used.
2. Unclear if quality assessment of included studies occurred.
3. Potential for bias from funding of included studies not reported.

**Funding source**
Funding source of review not reported.

• Rebholz et al. 2012

**Number and type of studies reviewed**
40 RCTs

**Number of databases searched**
3 databases searched (MEDLINE, EMBASE, Web of Science).
CVD outcomes assessed
SBP and DBP

Study quality
1. PRISMA used.
2. Quality assessment of included studies not assessed/reported.
3. Potential for bias from funding of included studies not reported.

Funding source
Source of funding not reported

<table>
<thead>
<tr>
<th>Consistency</th>
<th>Poor</th>
</tr>
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</table>

A review by Santesso et al (2012) found a small statistically significant decrease in SBP (SMD -0.21mmHg [95% CI: -0.32, -0.09], P = 0.0004) and DBP (SMD -0.18mmHg [95% CI: -0.29, -0.06], P = 0.003) in high protein diets 16. Rebholz et al. (2012) compared animal protein versus vegetable protein and protein with carbohydrate intake (Rebholz 2012) and found increased protein intake changed SBP by -1.76mmHg (95%CI: -2.33 to -1.20, P<0.001) and DBP by -1.15mmHg (95%CI:-1.59 to -0.71, P=0.014) compared with CHO intake (overall heterogeneity I²=0%, P=0.92). There was no statistically significant difference in blood pressure between animal and vegetable protein intake, and there was large heterogeneity in these studies.

In the review by Santesso, HDL was increased in higher-protein diets however there was heterogeneity in included studies (SMD 0.25 [95% CI: 0.07, 0.44] P = 0.007, heterogeneity I²=65%, P<0.00001), and there were moderate statistically significant decrease in TG but heterogeneity was high (MD -0.51 [95% CI: -0.78, -0.24], P<0.00001, heterogeneity I²=85%, P<0.00001). High protein versus low protein diets demonstrated no significant effects on TC, LDL and CRP. Sub-group analysis findings suggest that protein diets emphasising modest rather than large increases in protein content (20 – 30% of daily energy intake) are more likely to have favourable effects on CVD risk factors 18. Wycherley et al. (2012) compared energy-restricted, iso-caloric, high protein, low-fat diets with standard protein, low-fat diets; and found between group differences for TGs [WMD -0.23 mmol/L (95%CI: -0.33 to -0.12), heterogeneity I²=0%, Chi²=7.09, P=0.85]. There was no difference between diets for changes in TC, LDL, or HDL 17. Significant statistical heterogeneity was present for analysis of TC, LDL and HDL 17. Huang et al. (2011) included 2 RCTs, one in overweight populations which found that a HP diet (25% of total energy from protein, 50% to 60% of total energy from carbohydrate) lowered TC by 23%, and TGs by 9.5%, and increased HDL by 8.7%. Due to small sample size in this study (12 participants) results may not be clinically important 16. The other RCT was in pre-hypertensive and hypertensive participants, which found that a high protein diet (25% of total energy from protein, 48% of total energy from carbohydrate) resulted in decreased LDL (11%), TG (16.2%) and HDL (5.2%). 16
### Clinical impact

<table>
<thead>
<tr>
<th>For blood pressure:</th>
<th>SATISFACTORY</th>
</tr>
</thead>
<tbody>
<tr>
<td>For Total-C/HDL/LDL:</td>
<td>POOR</td>
</tr>
<tr>
<td>For TG:</td>
<td>SATISFACTORY</td>
</tr>
</tbody>
</table>

High protein diets may improve systolic and diastolic blood pressure. It is unclear if high protein diets have an effect on lipid profiles, however high protein diets may improve triglyceride levels.

### Generalisability

<table>
<thead>
<tr>
<th>Good</th>
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</table>

The body of evidence can be contextualised to adult Australians with and without pre-existing cardiovascular disease.

### Applicability

<table>
<thead>
<tr>
<th>Good</th>
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</thead>
</table>

The evidence base is relevant to the Australian healthcare context.

### Recommendation

**Consumption of a high protein diet (>25% daily energy intake from protein) is associated with improvements in triglyceride levels in adults with and without cardiovascular disease risk factors.**

**Grade of recommendation**

| D |

**Consumption of a high protein diet (>25% daily energy intake from protein) is associated with improvements in systolic and diastolic blood pressure in adults with and without cardiovascular disease risk factors.**

**Grade of recommendation**

| D |

**Consumption of a high protein diet (>25% daily energy intake from protein) is not associated with improvements in total cholesterol, LDL cholesterol or HDL cholesterol in adults with and without cardiovascular disease risk factors.**

**Grade of recommendation**

| D |

### Characteristics of Dietary Pattern

A high protein dietary pattern is one where >25% of an individual’s total daily kJ intake comes from protein.

### Dietary Pattern: Weight loss/ calorie-restricted diets

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>SATISFACTORY</td>
<td>1 systematic review and meta-analysis of randomized controlled trials with moderate risk of bias.23</td>
</tr>
</tbody>
</table>

**Number and type of studies reviewed**

5 RCTs evaluated low-sodium, low-calorie diets, 11 RCTs (13 comparison groups) evaluated low-calorie diets (with or without low-fat components).

**Number of databases searched**

3 databases searched (PubMed, EMBASE, Web of Science)
**CVD outcomes assessed**

Net change in systolic blood pressure (SBP) and diastolic blood pressure (DBP)

**Study quality**

1. PRISMA statement was used to guide data extraction and reporting of results.

2. How was quality of included studies assessed? ST (standardised tool), not done and/or not reported: Yes, ST used: Cochrane Risk of Bias tool used to evaluate study methodologies (7 quality components). Funnel plots for publication bias. Each trial assessed for selection bias, performance bias, detection bias, attrition bias, and reporting bias and classified as low, high, or unclear on risk of bias scale. Studies generated a low risk of bias for most quality assessment components (7 components) reviewed, though for some components there was either a high risk or risk of bias was unclear.

3. Funding source potential from bias: Information not provided on funding source of included studies.

**Funding source**

Funding source unclear

| Consistency | Systolic blood pressure: | Poor | 1 systematic review found low-sodium, low-calorie diets with a duration ranging 6 – 36 months were associated with significant pooled net SBP decrease of -2.39 mmHg (95% CI: -3.79 to -0.98, heterogeneity: $\chi^2 = 12.01$, df = 4, $P = 0.02$, $I^2 = 67\%$) and DBP decrease of -1.33 mmHg (95% CI: -2.03 to -0.62, heterogeneity: $\chi^2 = 6.39$, df = 4, $P = 0.17$, $I^2 = 37\%$) \(^{23}\). The same review found low-calorie diets (with and without low-fat components) ranging from 6 – 36 months duration were associated with significant pooled net decreases of -3.18 mmHg (95% CI: -4.24 to -2.11, heterogeneity: $\chi^2 = 38.10$, df = 12, $P = 0.0001$, $I^2 = 69\%$) for SBP and -1.28 mmHg (95% CI: -1.88 to -0.69, heterogeneity: $\chi^2 = 29.94$, df = 12, $P = 0.003$, $I^2 = 60\%$) for DBP \(^{23}\). This review included studies where participants were taking antihypertensive medications and those where participants were not taking antihypertensive medication. |
| Diastolic blood pressure: | Good |

| Clinical impact | Satisfactory | One review found that energy-restricted diets resulted in significant decreases in SBP and DBP (duration of trials 6 -36 months) \(^{23}\) |

| Generalisability | Good | Population in body of evidence can be contextualised to adult Australians both with and without cardiovascular disease or indicators for cardiovascular disease. |

| Applicability | Excellent | The evidence base is relevant to the Australian health care setting generally. |
**Recommendation**

Energy restricted diets that adjusted daily caloric intake in order to reduce weight by at least 4.5kg (or 5% of total body weight) of a duration of 6 – 36 months in adults with and without hypertension resulted in decreases in diastolic blood pressure.

**Grade of recommendation**

C

**Recommendation**

Energy restricted diets that adjusted daily caloric intake in order to reduce weight by at least 4.5kg (or 5% of total body weight) of a duration of 6 – 36 months in adults with and without hypertension resulted in decreases in systolic blood pressure.

**Grade of recommendation**

D

**Characteristics of Dietary Pattern**

Low calorie diets were defined as diets which adjusted daily caloric intake in order to reduce weight by at least 4.5kg or 5% of total body weight.

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
</table>
| Evidence Base              | Satisfactory | 1 systematic review and meta-analysis of level II studies with a moderate risk of bias.  
  - Ndanuko et al. 2016 |
| Number of databases searched |         | 3 databases searched (Scopus, Web of Science, MEDLINE)               |
| CVD outcomes assessed      |         | Systolic blood pressure (SBP) and diastolic blood pressure (DBP)     |
| Study quality:             |         | 1. PRISMA statement used.                                            |
|                           |         | 2. Cochrane risk of bias assessment tool used. Two of the `Nordic` dietary pattern RCTs had unclear methods of allocation concealment. One Nordic and the Tibetan RCT did not have enough information on whether outcome assessors were blinded. All 4 RCTs were high risk for incomplete outcome data. |
|                           |         | 3. Potential for bias from funding of included studies not reported. |
| Funding source             |         | No funding received for this review                                   |

**Dietary Pattern: Nordic and Tibetan**

Component | Rating  | Notes
---|---------|----------------------------------
Evidence Base | Satisfactory | 1 systematic review and meta-analysis of level II studies with a moderate risk of bias.  
  - Ndanuko et al. 2016 |
Number of databases searched |         | 3 databases searched (Scopus, Web of Science, MEDLINE)               |
CVD outcomes assessed |         | Systolic blood pressure (SBP) and diastolic blood pressure (DBP)     |
Study quality: |         | 1. PRISMA statement used.                                            |
  2. Cochrane risk of bias assessment tool used. Two of the `Nordic` dietary pattern RCTs had unclear methods of allocation concealment. One Nordic and the Tibetan RCT did not have enough information on whether outcome assessors were blinded. All 4 RCTs were high risk for incomplete outcome data. |
  3. Potential for bias from funding of included studies not reported. |
Funding source |         | No funding received for this review                                   |
One systematic review and meta-analysis examined the effects of dietary patterns in lowering blood pressure in hypertensive and normotensive adults.\(^\text{43}\)

Nordic dietary patterns of an intervention duration ranging from 6 to 26 weeks, compared with usual diets, resulted in a reduction in SBP of -5.20 mmHg (95% CI: -7.30 to -3.11, heterogeneity: \(\chi^2 = 0.88, df = 2, P = 0.64, I^2 = 0\%\)), and DBP of -3.85 mmHg (95% CI: -5.50 to -2.19, heterogeneity: \(\chi^2 = 0.53, df = 2, P = 0.77, I^2 = 0\%\)).

A Tibetan dietary pattern for an intervention duration of 12 months, compared with a usual Western diet, resulted in decrease in SBP of -1.10 mmHg (95% CI: -3.58 to 1.38) and change in DBP of 0.10 mmHg (-1.57 to 1.77) (heterogeneity not applicable, only one RCT examined this dietary pattern).

### Clinical impact

Good

Only the Nordic diet (n=3 RCTs) significantly reduced SBP and DBP. The Tibetan diet (n=1 RCT) was not associated with significant change in blood pressure.

### Generalisability

Good

Population in body of evidence can be contextualised to adult Australians with and without existing cardiovascular disease.

### Applicability

Good

The evidence base is generally applicable to the Australian healthcare context as foods in both dietary patterns are readily available in Australian food supply.

### Recommendation

Adherence to a Nordic dietary pattern including consumption of whole grains, fish, and berries resulted in decreases in systolic blood pressure and diastolic blood pressure in hypertensive and normotensive populations.

### Grade of recommendation

C

### Characteristics of Dietary Pattern

A Nordic dietary pattern was characterised by consumption of traditional Nordic foods, specifically whole grains (rye, barley, oats), berries, nuts, fatty fish, rapeseed oil, and low-fat dairy. One study limited sodium intake for participants, another included soy protein, almonds, and psyllium seeds in the Nordic diet, and a third included wild plants, mushrooms, unlimited fresh herbs, and specified that food be organically grown.

A Tibetan dietary pattern was characterised as including cereals (barley, wheat, rye, corn, rice, oats, and buckwheat); meat (beef, mutton, hare, chicken, venison); vegetables (onion, garlic, radish, fennel, leek, carrots, soy beans, dark beans); and fruit (pomegranate, banana, pineapple, mango, bramble, apricot, and nectarine).

### Evidence statement for Schumacher et al which informed Question 3

<table>
<thead>
<tr>
<th>Component</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence Base</td>
<td>Satisfactory</td>
<td>1 systematic review of level II, III and IV studies (^\text{45}) (24 RCTs, 6 uncontrolled pre–post interventions, 4 pre–post interventions with a control group (two concurrent and two historical control groups),</td>
</tr>
</tbody>
</table>
and 1 case series.

Systematic review contained 35 relevant studies from 6 databases; 31 studies targeted patients with CVD (n = 10,156) or at high risk of CVD (n = 6,690), and 4 studies targeted health professionals (n = 800). Six studies were gender specific.

Outcomes assessed at patient level included: changes in dietary intakes e.g. fats or sodium (n = 31), serum lipid levels (n = 2), urinary Na and urea (n = 1), and weight (n = 1).

Outcomes assessed at health professional level: changes in practice e.g. knowledge and recommendation of CV nutrition therapy (n = 4).

Quality of the included studies varied (positive, negative and neutral); with moderate risk of bias.

**Study quality:**

1. PRISMA: yes
2. How was quality of included studies assessed? ST (standardised tool): American Dietetic Association Evidence Analysis Manual quality criteria checklist
3. Funding source potential from bias: no (No funding to disclose)

| Consistency  | Satisfactory | Studies were conducted in 1) adults with or at high risk of CVD (aged 18 – 85 years); and 2) health care professionals (community dietitians; 4th year medical students; hospital doctors, nurses, therapists; and GPs) in clinical and community settings.
 Dietary interventions for 1) adults included: individualised or group-based education advice/adherence of low-fat diet, Mediterranean diet, heart healthy diet (kJ, fat and Na), vegetarian diet, reduced GI diet and general healthy diet; and 2) knowledge/recommendation of nutrition risk screening, initiation of nutrition support, CV nutrition therapy and soya foods. **Intervention follow-up varied** from 25 days (or hospital stay only) to 6 years.
 Validated dietary intake assessment measurement tools were used in 17 studies. Study results were not combined due to high heterogeneity between outcomes and measures. The summary of results indicate findings across patient studies is consistent for decreases in fat, cholesterol, Na, animal proteins and sugar intakes; increases in fruit and veg, fibre, whole grains and nut intakes. **Findings across** health professional studies is consistent for changes in practice.

| Clinical impact | Satisfactory | 22 studies found statistically significant (p<0.05) improvements for healthier dietary habits i.e. decreases in daily energy, total fat, dietary cholesterol, Na intakes and mean GI load; increases in fruit and vegetable, whole grains, polyunsaturated fat intakes.
 3 studies demonstrated that providing healthcare professionals with CVD nutrition education training (~1 hour session) lead to changes in practice (p<0.05).

| Generalisability | Good | Population in body of evidence can be contextualised to Australian adults with or at high risk of CVD, and healthcare professionals. |
Applicability | Good | The evidence base is relevant to the Australian adult population with or at high risk of CVD, and healthcare professionals providing CVD care.

Recommendation | In adults aged >18 years, with or at high risk of CVD, receiving targeted CVD prevention dietary advice, for periods of time from ~25 days up to 5 years, leads to healthier eating habits with decreases in daily intake of total energy, total fats, saturated fats, sodium, Glycaemic Load and increases in daily intake of fruit and vegetables, whole grains and polyunsaturated fats.

Grade of recommendation | C

Recommendation | For CVD healthcare professionals, receiving targeted CVD prevention nutrition education, for short periods of time ~1-hour session, leads to positive changes in practice.

Grade of recommendation | C
Appendix 5: Studies used to inform Question 3

Addressing barriers and facilitators to dietary change and providing advice


This study qualitatively examines how individuals (n= 17, aged 18-70 years), from 8 different families, with established CVD (n = 5) or at increased risk of CVD, perceive personal risk, their motivations to make dietary changes, their understanding of the role diet plays in heart health, and the influence of this on other family members.

The study findings indicate risk perception varied widely with participants rarely estimating their true level of CVD risk. While all participants implied awareness of the contribution of diet to heart health, their perceived CVD risk perception had an impact on their motivation to make the necessary dietary changes to conform with recommendations, i.e. lower perception was related to lower motivation. Participants’ family members were shown to be either a significant form of support or a significant barrier to improving their own and/or family members’ dietary patterns. Other barriers to healthy eating that the study identified included dealing with other family member’s food preferences, eating away from the home, lack of time and dealing with ‘individualised’ dietary requirements and meals.

The study concludes that risk perception alone may be inadequate to initiate and sustain dietary changes in this population and that strategies are needed to engage those with low risk perception within families where members have established CVD or elevated risk factors. The authors suggest that involving family members to provide support can enhance adherence to dietary recommendations by way of personal accountability.


The Dietary Guidelines for Americans were introduced 35 years ago, although most of the American population fall short of meeting these guidelines. This is reflected in the high rates of overweight and obesity, along with corresponding lifestyle related diseases. Failure to meet these recommendations is likely the result of a combination of “cultural forces, societal norms, family influences, personal food preferences, food availability and accessibility, declining food preparation skills, changes in meal patterns, food marketing practices, time pressures, economic realities, government policies, and the fact that making and sustaining change is hard.”

Consumers report that they seek information on nutrition and healthy eating, but most find the current guidelines confusing due to the overwhelming amount of detail provided within. To improve nutrition communication and to facilitate positive changes in dietary behaviours Webb and Byrd-Bredbenner suggest that nutrition guidance must be targeted to specific consumer groups, easy to understand and take a holistic approach that fits into consumers’ everyday lives. They propose a gradual remodelling of existing eating behaviours, with a “nondiet” approach, would be more beneficial to overcoming barriers and building the self-confidence needed to improve health. These incremental improvements would also promote stronger feelings of self-efficacy, increased values of making changes, and more skilful use of self-regulation strategies.

As consumers reported convenience and taste were the strongest determinants of food choices, Webb and Byrd-Bredbenner further expand on their proposed approach, emphasising eating for enjoyment; allowing for internal regulation, moderation of food intake, regular meals; rather than to achieve dietary recommendations has the potential leads to more positive eating behaviours, along with healthier weights.
and better cardiovascular disease biomarkers. To promote consumer health, Webb and Byrd-Bredbenner advocate for nutrition and health communicators to provide consumers with personalised advice and clearer nutrition guidance messages that are realistic, consistent, positive, and actionable.


This study qualitatively examines the barriers and facilitators to adherence to the Dietary Guidelines for Americans for four nutrient-rich food groups [milk, whole grains, fruit, and vegetable]. Data was collected from a total of 281 unrelated adult caregivers and 321 fifth-grade children via structured focus group sessions.

The core barriers identified by adult caregivers were lack of meal preparation skills or recipes [whole grains, fruit, vegetables]; difficulty in changing eating habits [whole grains, fruit, vegetables], cost [milk, whole grains, fruit, vegetables], lack of knowledge of recommendation/portion/health benefits [milk, vegetables], and taste [milk, whole grains, vegetables]. The core barriers identified by children were competing foods (e.g. drinking soda or eating junk foods/sugary foods rather than consuming [whole grains/ milk/ fruit/vegetables]), health concerns (e.g. milk allergy/upset stomach [milk]), taste/flavour/smell [milk, whole grains, fruit, vegetables], forgetting to eat them [vegetables, fruit], and hard to consume or unsure of the recommended amount [milk, fruit].

The core facilitators identified by adult caregivers were availability, affordability, and accessibility [milk, fruit]; more information about the benefits and intake recommendations (including information on reading Nutrition Facts labels) [milk, whole grains, vegetables]; parents acting as role models for their children [milk, fruit, vegetables]; the need for recipes and pre-packaged servings [milk, whole grains, vegetables]; and the need for education and marketing efforts toward children [whole grains]. The core facilitators identified by the children were consuming specific foods they liked containing nutrient (e.g. smoothies, pizza) [milk, whole grains]; combining the food with something else they like to eat [fruit, vegetables]; knowledge about the health benefits and intake amounts [milk, vegetables]; consuming foods at specific meals (such as milk at every meal, cereal and fruit for breakfast, bread at lunch) [milk, whole grains, fruit]; and encouraging adults (e.g. doctors and parents) to talk with children about health benefits [milk].

For both adult caregivers and children, reported facilitators closely corresponded with the barriers, highlighting modifiable conditions that could help individuals to meet the Dietary Guidelines.


This review evaluates 304 articles (240 intervention studies, 64 reviews) for the use of health behaviour change techniques/theory in technology-enabled interventions targeting risk factors and indicators (physical activity, weight loss, smoking cessation and management of hypertension, lipids and blood glucose) for CVD prevention and treatment.

Among intervention studies focused on healthy eating, using the Internet (n = 47), mobile/smartphones (n = 10), and activity monitors and sensors (n = 8), were the most frequently used types of technologies. Overall, the most frequently used behaviour change techniques for healthy eating were self-monitoring and feedback on performance. Social Cognitive Theory was the most frequently used theory in studies focused on weight loss (n = 27), healthy eating (n = 22), and glucose monitoring (n = 2). Both Social Cognitive Theory and the Social Ecological Model were utilised most often for lipid monitoring (n = 2; n = 2) and BP (n = 2; n = 2).
In weight loss/management reviews the findings indicate that social support, social media, real-time feedback, and greater engagement with intervention technologies contribute to more successful outcomes. Articles focusing broadly on health behaviour and lifestyle changes found small effect sizes – particularly for interventions that were not individually tailored to participants’ needs. It was suggested that technology-mediated interventions combined with face-to-face weight loss interventions may provide greater effect sizes.

The authors conclude there is potential for the use of technology-enabled interventions to improve modifiable CVD behaviours. For optimal outcomes integrated behaviour change theories that incorporate a variety of evidence-based health behaviour change techniques are needed.


A systematic review of 22 qualitative observational studies assessing barriers and facilitators that individuals with CVD or high CVD risk consider to be influential in maintaining changed healthy lifestyle behaviours, such as diet for weight loss, healthy eating and decreasing alcohol consumption.

The most commonly reported influences were those relating to social support (whether provided formally or informally), beliefs (about the self or the causes and management of poor health, and the value of maintaining lifestyle behaviours), and other psychological factors (including attitude, thinking and coping styles, and problem solving skills).

The findings indicate to facilitate maintenance of changed behaviours, good social support was needed early on to ensure that beliefs about the benefits of healthy lifestyles were more stable. Effective planning and problem solving needed to be in place to maintain healthy behaviours within everyday life. Facilitators that individuals applied whilst trying to maintain healthy diets included planning meals, getting into a routine and self-monitoring.

The ‘beliefs’ core theme comprised mostly barriers. The findings suggest individuals tend to underestimate their personal risk, they question the benefits of healthy lifestyles and their perceptions of risk alter through time due to the absence of subsequent CV events.

The study concludes that social support, education and knowledge, and beliefs and emotions are key areas that lifestyle support programmes need to focus on to facilitate lifestyle behaviour change and maintenance in the longer-term.


A systematic evidence review of 71 trials (meta-analyses on 57) evaluating the benefits and harms of behavioural counselling interventions (including healthy eating) to prevent cardiovascular disease (CVD) in persons with established risk factors (including hypertension, dyslipidaemia, metabolic syndrome, and impaired fasting glucose or glucose tolerance) to assist the U.S. Preventive Services Task Force (USPSTF) in updating its previous recommendation statements.

The evidence was qualitatively summarised for effects on health outcomes, behavioural outcomes, and harms. The review concluded that medium to high intensity diet and physical activity behavioural counselling was beneficial in overweight or obese persons with CVD risk factors. It resulted in consistent improvements across a variety of important cardiovascular intermediate health outcomes, such as lipid levels, BP and weight status, for up to 2 years. The applicability of the findings depended largely on the
availability of intensive counselling and adherence to the interventions. After reviewing all included healthy counselling trials for harms, including any inconsistent change in outcomes, they did not find any.

The review indicated the major barriers to healthy eating included: “low income, food marketing, lack of accessible and accurate information on what constitutes a healthy diet, poor accessibility to affordable healthy foods, lack of opportunity to experiment and to develop cooking skills (e.g., poor literacy, reduced access to well-equipped kitchens, homelessness, poor educational attainment), and sociocultural factors (e.g., family or cultural food norms, family resistance, lack of support, and child care demands can all inhibit dietary change)”. 
Appendix 6: Summary of the literature informing the evidence statements

Summary of the literature informing the evidence statements that included more than one systemic review.

**Primary: Healthy/prudent diet**
There were five systematic reviews included in the primary, Healthy/prudent lifestyle evidence base. Of these reviews Li et al 2015 had the highest percentage of duplicate studies at 64% (7/11 studies), Rodriguez-Monforte et al 2015 contained the second most duplicate studies with 55% (12/22 studies), and Oude Griep et al 2013 contained the least with no duplicate studies. Out of the all the individual studies included in these reviews Shimazu et al 2007 and Maruyama 2023 were included most frequently with 4 and 3 reviews respectively using these studies. All other duplicated studies were only included in two reviews.

**Primary: Mediterranean diet**
In total there were three systematic reviews that made up the evidence base on the Mediterranean diet use in primary prevention. Only one study, Knoopes 2004, was duplicated out of the 69 studies included in the three systematic reviews. This study was reported in Sofi et al 2014 and Tyrovologas et al 2010.

**Primary: DASH diet**
There were three systematic reviews that made up the evidence on the primary prevention of the DASH diet. Salehi-Abargouei et al 2013 contained the most duplicate studies with 100% (6/6 studies) being duplicates, Schwinshackl and Hoffmann 2015 contained 66% (6/9) duplicates and Saneei 2014 contained no duplicate studies. All duplicate studies were the same between Salehi-Abargouei et al 2013 and Schwinshackl and Hoffmann 2015.

**Primary + secondary combined: Mediterranean diet**
Of the 11 systematic reviews included in primary and secondary prevention of the Mediterranean diet combined Huang, Mancini, and Kastorini had the highest percentages of duplicate studies with 88%, 80% and 70% respectively. Both Kastorini and Grosso however had the highest number of duplicate studies (35/50 and 23/58, respectively). There were four studies that were included most frequently among all reviews, these were Esposito 2004 (used in 8 reviews), Estruch 2006, Shai 2008 and Vincent-Baudry 2005 (all used in 7 reviews each). Only one systematic review, by Martinez-Gonzalez contained no duplicates.

**Primary + secondary combined: DASH diet**
In total there were five systematic reviews included in the primary and secondary combined prevention using the DASH diet. Of the five reviews Sanei 2014 had the highest percentage of duplicate studies at 100% (3/3), however Siervo 2015 had the highest number of duplicate reviews included (7/20). One study, Azadbakht 2005, was included in all four reviews. All of the other six reviews that were duplicates were only included in two of the reviews.

**Primary + secondary combined: high protein**
Of the four systematic reviews included, Huang had the highest percentage of duplicate studies included at 100% (2/2 studies). However, the other three reviews, Wycherley, Rebholz and Santesso, had a higher number of studies that were duplicates with 13, 11 and 21 duplicate studies respectively. Two of the studies that were duplicates were found in three of the reviews, these studies were Leidy et al 2007 and Farnsworth et al 2003. All of the 20 other duplicates were used in two of the reviews.
Appendix 7: Description of study comparators to meta-analysis

Table: Meta-analyses of dietary patterns and their comparators

<table>
<thead>
<tr>
<th>Primary</th>
<th>Author (Year)</th>
<th>Dietary pattern</th>
<th>Dietary pattern compared to</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Li (2015)</td>
<td>Healthy/prudent diets</td>
<td>Western/unhealthy diets</td>
<td>Effect size of Prudent or ‘Healthy’ diets on CVD outcomes analysed. Effect sizes of Western/unhealthy diets on CVD outcomes analysed.</td>
</tr>
<tr>
<td></td>
<td>Rodriguez-Monforte (2015)</td>
<td>Healthy/prudent diets</td>
<td>Western diet</td>
<td>Highest category compared to the lowest category of prudent/healthy dietary patterns. Highest category compared to lowest category of Western dietary patterns.</td>
</tr>
<tr>
<td></td>
<td>Wang (2016)</td>
<td>Healthy/prudent diets</td>
<td>Western diet</td>
<td>Highest category compared to the lowest category of healthy dietary patterns. Highest category compared to lowest category of Western dietary patterns.</td>
</tr>
<tr>
<td></td>
<td>Zhang (2015)</td>
<td>Healthy/prudent diets</td>
<td>Western/unhealthy diet</td>
<td>Highest category compared to the lowest category of healthy dietary patterns. Highest category compared to lowest category of Western/unhealthy dietary patterns.</td>
</tr>
<tr>
<td></td>
<td>Rees (2013)</td>
<td>Mediterranean</td>
<td>Usual diet or healthy dietary advice</td>
<td>No intervention or minimal intervention</td>
</tr>
<tr>
<td></td>
<td>Schwingshackl (2013)</td>
<td>Low GI/Low GL</td>
<td>High GI/High GL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Saneei (2014)</td>
<td>DASH</td>
<td>Control diet not specified</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salehi-Abargouei (2013)</td>
<td>DASH</td>
<td>DASH (low adherence)</td>
<td>Highest concordance groups compared with reference groups with lowest score from a DASH-style diet</td>
</tr>
<tr>
<td></td>
<td>Schwingshackl (2015)</td>
<td>DASH</td>
<td>DASH (low adherence)</td>
<td>Highest compared to lowest diet quality</td>
</tr>
<tr>
<td>Study</td>
<td>Diet Type</td>
<td>Comparison</td>
<td>Control Group</td>
<td></td>
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<td>-------------------------------</td>
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<tr>
<td>Secondary</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Semlitsch (2016)</td>
<td>Weight loss/calorie-restricted</td>
<td>Usual diet</td>
<td>No intervention for control group</td>
<td></td>
</tr>
<tr>
<td>Saneei (2014)</td>
<td>DASH</td>
<td>Control diet not specified</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Huang (2011)</td>
<td>Portfolio diet</td>
<td>Usual diet or AHA Step I diet</td>
<td></td>
<td></td>
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<tr>
<td>Primary and Secondary</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Goff (2013)</td>
<td>Low GI/GL</td>
<td>High GI/GL</td>
<td></td>
<td></td>
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<tr>
<td>Huang (2011)</td>
<td>Low-fat</td>
<td>Control diet not specified</td>
<td></td>
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<tr>
<td>Sackner-Bernstein (2015)</td>
<td>Low CHO</td>
<td>Low-fat</td>
<td></td>
<td></td>
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<tr>
<td>Gay (2016)</td>
<td>Mediterranean</td>
<td>Control diet not specified</td>
<td>Comparison group - control diet, advice only, or standard follow-up</td>
<td></td>
</tr>
<tr>
<td>Huang (2011)</td>
<td>Mediterranean</td>
<td>Usual diet, low-fat, low-fat + statin, high-fat diet, AHA Step I diet</td>
<td>Control diets varied among included studies</td>
<td></td>
</tr>
<tr>
<td>Ndanuko (2016)</td>
<td>Mediterranean</td>
<td>Usual, healthy, low-fat diets or healthy dietary advice</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Martinez-Gonzalez (2014)</td>
<td>Mediterranean</td>
<td>Control diet not specified for RCTs</td>
<td>Mostly cohort studies</td>
<td></td>
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<tr>
<td>Nissensohn (2016)</td>
<td>Mediterranean</td>
<td>Low-fat diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liyanage (2016)</td>
<td>Mediterranean</td>
<td>Usual diet, low-fat diet or healthy dietary advice</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schwingshackl (2014)</td>
<td>Mediterranean</td>
<td>Usual diet, healthy dietary advice, or diet with refined CHO (i.e. no whole grains)</td>
<td>All except 1 study, usual diet or advice only</td>
<td></td>
</tr>
<tr>
<td>Kastorini (2011)</td>
<td>Mediterranean</td>
<td>Low-fat diet, high-sat fat diet, high CHO diet, low CHO diet, prudent diet, usual diet, American Diabetes Association diet, healthy dietary advice or less counselling on an MD prescription</td>
<td>Control diets varied among included studies (categorized where possible)</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Diet Type</td>
<td>Diet Description</td>
<td>Methodology</td>
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<td>------------------</td>
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<td>----------------------------------------------------------------------------</td>
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<tr>
<td>Yokoyama (2014)</td>
<td>Vegetarian</td>
<td>Omnivorous diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gay (2016)</td>
<td>DASH</td>
<td>Control diet not specified</td>
<td>Comparison group - control diet, advice only, or standard follow-up</td>
<td></td>
</tr>
<tr>
<td>Siervo (2015)</td>
<td>DASH</td>
<td>Usual or healthy diet</td>
<td>Intervention and control diets comparable in terms of energy intake and other lifestyle interventions, e.g. PA</td>
<td></td>
</tr>
<tr>
<td>Soltani (2016)</td>
<td>DASH</td>
<td>Usual, healthy, low-fat or calorie restricted diets. One study = higher acid load healthy diet.</td>
<td>Control diets varied among included studies</td>
<td></td>
</tr>
<tr>
<td>Saneei (2014)</td>
<td>DASH</td>
<td>Control diet not specified</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Santesso (2012)</td>
<td>High protein</td>
<td>Low protein</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Huang (2011)</td>
<td>High protein</td>
<td>Control diet not specified</td>
<td></td>
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<tr>
<td>Rebholz (2012)</td>
<td>High protein</td>
<td>Diet with ↑ intake of CHO and/or fat</td>
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<tr>
<td>Gay (2016)</td>
<td>Weight-loss/Calorie-restriction</td>
<td>Control diet not specified</td>
<td>Comparison group - control diet, advice only, or standard follow-up</td>
<td></td>
</tr>
<tr>
<td>Ndanuko (2016)</td>
<td>Healthy diets (Nordic and Tibetan)</td>
<td>Usual or healthy dietary advice</td>
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<td></td>
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</tbody>
</table>

### Appendix 8: Data extraction spreadsheets

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Dietary pattern</th>
<th>Participants</th>
<th>No. studies included</th>
<th>Gender</th>
<th>CVD outcomes assessed</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Li (2015)</td>
<td>Healthy/prudent diets</td>
<td>338 787</td>
<td>13</td>
<td>M &amp; F</td>
<td>CVD mortality</td>
<td>Healthy/prudent DIET - Risk of all-cause mortality (highest vs lowest categories): SRRE (summary relative risk estimates) = 0.76 (95%CI: 0.67 - 0.86), I²=52.6%, p=0.039. CVD mortality: SRRE = 0.81 (95%CI 0.75 to 0.87), I²=3.5%, p=0.399. WESTERN/UNHEALTHY DIET - Risk of all-cause mortality: SRRE = 1.07 (95%CI: 0.96 - 1.20), I²=48%, p=0.073. CVD mortality: SRRE = 0.99 (95%CI: 0.91 - 1.08), I²=38.6%, p=0.135.</td>
</tr>
<tr>
<td>Oude Griep (2013)</td>
<td>Healthy/prudent diets</td>
<td>26 145</td>
<td>8</td>
<td>M &amp; F</td>
<td>Inflammation</td>
<td>NB. No meta-analysis, results of included studies reported separately. Japanese study: higher healthy dietary pattern score had lower CRP (logarithmically transformed CRP of 0.40 mg/l in men and 0.29 mg/l in women) compared to lower scores (0.45 mg/l in men, p=0.01; 0.30 mg/l in women, p=0.06 in women). Chinese study: diet high in fruits, low in veg had lower prevalence of elevated CRP defined as &gt;3 mg/l (OR for top vs. lowest quintile: 0.68; 95% CI: 0.46–0.99). Higher prevalence of elevated CRP for high score unhealthy diet pattern high in meat (OR for top vs. lowest quintile: 1.34; 95% CI:0.91–1.99). German study: unhealthy diet pattern (high in meat &amp; beer) strongly correlated to CRP (r=0.24), less with IL-6 (r=0.19) and IL-18 (r=0.11). Western dietary pattern positively correlated with CRP (r=0.33) and IL-6 (=0.27), poorly with IL-18 (=0.08). Study in USA young adults: baseline diet high in fruit &amp; veg inversely associated with mean top vs low quintile (p &lt;0.01) and changes (top vs. lowest quintile p&lt;0.01) in F2-isoprostanes</td>
</tr>
</tbody>
</table>
Dietary patterns and cardiovascular disease outcomes

Concentrations after >15 yrs. Diet pattern high in meat positively associated with F2-isoprostanes (p<0.0001). Diet patterns high in fruit & veg associated inversely with concentrations of CRP and other inflammatory markers. Swedish study: no association between diet patterns and CRP. In women WBC counts inversely associated 'fibre-rich bread' diet pattern (OR for top vs lowest quartile: 0.50; 95%CI: 0.33–0.76) and positively associated with diet patterns characterized by 'milk fat' (OR for top vs. lowest quartile: 1.39; 95%CI: 0.97–1.98) and 'sweets and cakes' (OR for top vs. lowest quartile: 1.25; 95%CI: 0.96–1.63). Inverse associations of 'low-fat high fibre' diet with Lp-PLA2 mass were found for men (OR for top vs. lowest tertile: 0.62; 95%CI: 0.40–0.96) and women (OR for top vs. lowest tertile: 0.69; 95%CI: 0.54–0.87). Lp-PLA2 mass positively associated with diet pattern characterized by 'milk fat' in men (OR for top vs. lowest tertile: 1.50; 95%CI: 1.10–2.05) and 'sweets and cakes' in women (OR for top vs. lowest tertile: 1.29; 95%CI: 1.02–1.62). USA study in elderly: healthy diet patterns had significantly lower IL-6 concentrations of 1.7 pg/ml compared to diet patterns characterized by 'sweets and desserts' of 1.9pg/ml (n=289; P≤0.05) or 'high-fat dairy' of 1.9 pg/ml (n=570; P≤0.05). Another study found baseline diet pattern high in vegetables, olive oil, and vegetable oil associated with lower risk of elevated CRP of > 3 mg/l >12 yrs follow-up (OR top vs. lowest tertile 0.69; 95%CI: 0.49–0.95). Contrasting NS findings observed for unhealthy diet high in processed and organ meat, poultry, eggs, and low in fatty fish (OR top vs. lowest tertile 1.21; 95%CI: 0.87–1.67). Canadian study in women: found high physical activity energy expenditure level (>958 kcal/d) and high C-HEI score (>83.3
out of 100) had significantly lower CRP concentrations (logarithmically transformed CRP of 0.26 mg/l) vs those with low physical activity energy expenditure level and low HEI (0.39 mg/l). Healthy diet high in fruit, veg, whole grains, seed/nuts inversely associated with mean (top vs. lowest quintile p<0.0001) & prospectively with changes (top vs low quintile p<0.01) of F2-isoprostane concentrations.

<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>Diet Pattern</th>
<th>Sample Size</th>
<th>Gender</th>
<th>Outcome(s)</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rodriguez-Monforte (2015)</td>
<td>Healthy/prudent diets</td>
<td>610 691</td>
<td>M &amp; F</td>
<td>CVD morbidity, heart disease, stroke</td>
<td>Highest vs lowest category of Healthy/prudent dietary patterns in cohort studies - CVD: RR=0.69 (95% CI: 0.60, 0.78), I²=0%; p=0.687; CHD: RR=0.83 (95% CI: 0.75, 0.92), I²=44.6%, p=0.054 and Stroke: RR 0.86 (95% CI: 0.74, 1.01), I²=59.5%, p=0.008. High vs lowest category of Healthy/prudent dietary patterns in case-control comparison = CHD: RR 0.71 (95% CI: 0.63, 0.80), I²=0 %, p=0.560. Highest vs lowest category of Western dietary patterns in cohort studies = CVD: RR 1.14 (95% CI: 0.92, 1.42), I²=56.9%, p=0.055; CHD: RR 1.03 (95% CI: 0.90, 1.17), I²=59.4%, p=0.012; Stroke: RR 1.05 (95% CI: 0.91, 1.22), I²=27.6%, p=0.190. In case control studies RR=1.61 (95% CI: 1.17, 2.2.1), I²80.5%, p=0.006.</td>
</tr>
<tr>
<td>Wang (2016)</td>
<td>Healthy/prudent diets</td>
<td>295799</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>Decreased likelihood for hypertension in highest vs lowest categories of healthy pattern (OR=0.81; 95%CI: 0.67 to 0.97; P=0.02). Heterogeneity: P&lt;0.00001, I²&lt;94%.</td>
</tr>
<tr>
<td>Zhang (2015)</td>
<td>Healthy/prudent diets</td>
<td>1031836</td>
<td>M &amp; F</td>
<td>Heart disease</td>
<td>Healthy/prudent diet OR = 0.67 (CI: 0.60, 0.75), p &lt; 0.00001, I² = 57%, chi² 30.25, p=0.004. Unhealthy/ Western-type diet OR = 1.45 (CI: 1.05, 2.01), p = 0.02, I² = 96%, chi²=241.57, p&lt;0.0001. Alcohol consumption levels OR = 0.68 (95% CI: 0.59, 0.78), p &lt; 0.00001, I² = 83%, chi²=110.46, p&lt;0.0001.</td>
</tr>
<tr>
<td>Study</td>
<td>Dietary Pattern</td>
<td>N</td>
<td>Gender</td>
<td>Outcomes</td>
<td>Results</td>
</tr>
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<td>----------------------------------------------------------------------------------------------------------------------------------------</td>
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<tr>
<td>Rees (2013)</td>
<td>Mediterranean</td>
<td>52044</td>
<td>M &amp; F</td>
<td>Hypertension, lipid profile</td>
<td>Statistically significant reduction in SBP in 3 trials (MD = 7.8 mmHg, 95% CI: -12.11 to -3.49; MD = 3.0 mmHg, 95% CI: -3.46 to -2.54; MD = 0.7 mmHg, 95% CI: -1.03 to -0.37). Two studies report no significant difference in SBP. Heterogeneity: SBP = I² = 94%. DBP was statistically significant in 3 trials (MD = 3.7 mmHg, 95% CI: -6.1 to -1.3; MD = 2.0 mmHg, 95% CI: -2.29 to -1.71; MD = 0.7 mmHg, 95% CI: -0.88 to -0.52). Two studies report no significant difference in DBP. Heterogeneity for DBP = I² = 93%. TC: intervention = -0.16 mmol/L (95% CI: -0.26 to -0.06; random-effects model, p = 0.003, I² = 74%). LDL: small statistically significant reduction in LDL following intervention (in 6 trials) (MD = 0.07 mmol/L, 95% CI: -0.13 to -0.01, I² = 22%). HDL (in 7 trials): One trial reported an increase in HDL after intervention (MD = 0.08 mmol/L, 95% CI: 0.06 to 0.09). Remaining 6 studies found no significant effect of intervention on HDL levels (heterogeneity for HDL I² = 83%). TG: One study showed statistically significant reduction in TG levels after intervention (MD = 0.21 mmol/L, 95% CI: -0.23 to -0.19). Four studies showed no effect of intervention on TG levels. Heterogeneity between TG trials (I² = 94%), therefore no meta-analysis performed. TC: HDL = n/a.</td>
</tr>
<tr>
<td>Sofi (2014)</td>
<td>Mediterranean</td>
<td>4172 412</td>
<td>M &amp; F</td>
<td>CVD morbidity and mortality, heart failure, CHD</td>
<td>A 2-point increase in adherence score to Med diet reported to determine an 8% reduction of overall mortality (relative risk = 92; 95% CI: 91, 93), a 10% reduced risk of CVD (RR = 90; 95% CI: 87, 92). NB. Update of earlier systematic review; 18 studies added to meta-analysis.</td>
</tr>
<tr>
<td>Tyrovolas</td>
<td>Mediterranean</td>
<td>192828</td>
<td>M &amp; F</td>
<td>Hypertension, CVD mortality</td>
<td>MEDIS study: High fat foods associated with 4.8% higher likelihood of having hypercholesterolemia (OR = 1.048,</td>
</tr>
</tbody>
</table>

Sofi (2014) Mediterranean 4172 412 18 M & F CVD morbidity and mortality, heart failure, CHD A 2-point increase in adherence score to Med diet reported to determine an 8% reduction of overall mortality (relative risk = 92; 95% CI: 91, 93), a 10% reduced risk of CVD (RR = 90; 95% CI: 87, 92). NB. Update of earlier systematic review; 18 studies added to meta-analysis. |
| Tyrovolas        | Mediterranean     | 192828 | M & F  | Hypertension, CVD mortality                   | MEDIS study: High fat foods associated with 4.8% higher likelihood of having hypercholesterolemia (OR = 1.048, |
(2010) p=0.06). Cereal intake was associated with 28.4% lower likelihood of having hypercholesterolemia (OR = 0.716, p=0.001). Fish intake was inversely associated with total CHOL (p=0.012) and TG levels (p=0.024). Fish intake was inversely associated with systolic blood pressure (p=0.026). The Seven countries study: diets rich in butter, dairy products and other animal products were associated with high rates of CHD. Food consumption patterns high in cereals, legumes, vegetable products, fish oils and wine were associated with low mortality rates from CHD. Overall, combined vegetable foods were inversely associated with CHD morality whereas combined animal products were directly correlated. Habits in Later Life Only study: greater adherence to Med Diet associated with 21% lower odds of having one additional risk factor (i.e. hypertension, hyper-cholesterolemia, diabetes, obesity) in women and 14% lower risk in men. HALE study: adherence to Med diet associated with 23% lower risk of death. INTERHEART study: daily consumption of fruit and veg, and regular physical activity gave odds ratio of 0.6 (99% CI 0.51 – 0.71) for risk of acute MI. Consistent fruit and veg consumption was associated with a 30% RR reduction of MI. MEDIS study: reduction of 100g fish per week associated with 19% higher likelihood of having one additional CVD risk factor e.g. hypertension, diabetes and obesity.
<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>Diet Pattern</th>
<th>N</th>
<th>Gender</th>
<th>Outcomes</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schwingshackl (2013)</td>
<td>Low GI/Low GL</td>
<td>2344</td>
<td>M &amp; F</td>
<td>Weight, lipid profile, inflammation</td>
<td>15 RCT’s (14 included in MA) No sig. changes observed for weight [-0.62 kg (95% CI -1.28 to 0.03), p=0.06, I^2= 0%] and Waist Circumference [0.06 cm (95% CI -0.83 to 0.96), p = 0.89, I^2 = 0%]. No sig. changes in total CHOL: -1.22mg/dl (95% CI -5.62 to 3.19, p=0.59) I^2=44%; LDL-C: -0.29mmol/L (95% CI -4.45 to 3.86, p=0.89) I^2=54%; HDL-C: 0.73mg/dl (95% CI -0.23 to 1.69, p = 0.14) I^2=0%; TG: -0.86mg/dl (95% CI -5.65 to 3.93, p=0.72, I^2 = 0%) between LGI/LGL vs. HGI/HGL diets. Decrease in FFM was significantly more prominent following LGI/LGL diet compared with HGI/HGL [-1.04kg (95% CI -1.73 to -0.35, p = 0.003), I^2=0%]. Decreases in CRP more pronounced in LGI/LGL group: CRP = -0.43mg/dl (95% CI -0.78 to -0.09, p=0.01), I^2 = 54%. Fasting insulin better controlled with LGI/LGL diet: -5.16pmol/L (95% CI -8.45 to -1.88, p=0.002), I^2 = 48%. No sig. diff in fasting glucose 0.49 mg/dl (95% CI -1.28 to 2.25, p=0.59), I^2=52% and HbA1c -0.09% (95% CI -0.52 to 0.33, p=0.67), I^2=72%) between groups.</td>
</tr>
<tr>
<td>Saneei (2014)</td>
<td>DASH</td>
<td>293</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>SBP: MD -6.74 (95% CI: -8.26, -5.22), I^2 = 78.9%; DBP: MD -3.59 (95% CI: -4.35, -2.82), I^2 = 58.3%.</td>
</tr>
<tr>
<td>Salehi-Abargouei (2013)</td>
<td>DASH</td>
<td>259 984</td>
<td>M &amp; F</td>
<td>CVD morbidity and mortality, heart failure, CHD</td>
<td>DASH-style diet can significantly decrease risks for CVD (RR, 0.80; 95% CI; 0.74–0.86; p&lt;0.001), HF (RR, 0.71, 95% CI, 0.58–0.88; p&lt;0.001), and HD (RR, 0.79; 95% CI, 0.71–0.88; P&lt;0.001).</td>
</tr>
<tr>
<td>Schwingshackl (2015)</td>
<td>DASH</td>
<td>1 020 642</td>
<td>M &amp; F</td>
<td>CVD mortality</td>
<td>Highest association of diet quality using HEI, AHEI or DASH score significantly associated with reduced risk of all-cause mortality (RR 0.78, 95% CI: 0.76 to 0.80; P&lt;0.00001; I^2=61%, 95% CI 20% to 81%). CV mortality or incidence (RR 0.78, 95% CI 0.75 to 0.81; p&lt;0.00001; I^2=45%, 95% CI 13% to 66%)</td>
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<td><strong>Secondary</strong></td>
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<tr>
<td><strong>Semlitsch (2016)</strong></td>
<td>Weight loss/calorie-restricted</td>
<td>2 100</td>
<td>8</td>
<td>M &amp; F</td>
<td>Weight, hypertension, CVD morbidity, lipid profile</td>
</tr>
<tr>
<td><strong>Saneei (2014)</strong></td>
<td>DASH</td>
<td>1747</td>
<td>15</td>
<td>M &amp; F</td>
<td>Hypertension</td>
</tr>
<tr>
<td><strong>Huang (2011)</strong></td>
<td>Portfolio diet</td>
<td>245</td>
<td>3</td>
<td>M &amp; F</td>
<td>Lipid profile</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Primary/Secondary</strong></th>
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<tbody>
<tr>
<td><strong>Goff (2013)</strong></td>
<td>Low GI/GL</td>
<td>1272</td>
<td>28</td>
<td>n/a</td>
<td>Lipid profile</td>
</tr>
<tr>
<td>Study</td>
<td>Diet</td>
<td>Sample Size</td>
<td>Study Design</td>
<td>Outcome Measure</td>
<td>Results</td>
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<tr>
<td>Huang (2011)</td>
<td>Low-fat</td>
<td>19981</td>
<td>12 RCTs + 6 meta-analyses</td>
<td>Lipid profile</td>
<td>Diet with 18-30% total energy intake as fat significantly reduced TC and LDL by 5% to 15%. No clear benefit in most studies for TG's and HDL. Conclusion: Low-fat diet recommended to improve lipid profile.</td>
</tr>
<tr>
<td>Sackner-Bernstein (2015)</td>
<td>Low CHO</td>
<td>1797</td>
<td>17</td>
<td>Weight, hypertension, lipid profile</td>
<td>Low CHO: Weight decreased 94.8kg (95% CI: 91.6, 97.3) to 86.6 kg (95% CI: 83.6, 89.6), p&lt;0.0001. Low-fat: weight decreased 94.1kg (95% CI: 91.2, 96.9) to 88.2 kg (95% CI: 85.4, 90.9) (p&lt;0.0001). Compared with low-fat diet, low CHO diet greater reduction in weight (Pooled mean change = -2.0 kg, 95% CI: -3.1, -0.9). BMI: Low CHO = -2.8 (95% CI: -3.3, -2.2), p&lt;0.0001), low-fat = -2.1(95% CI: -2.5, -1.7), p&lt;0.0001. Between group low CHO favours BMI compared to low-fat: -0.7 (-1.1, -0.3) p=0.0016. Within-group total CHOL compared to control: Low CHO group -4.2 (-9.4, 1.1 95%CI) p=0.11, low-fat group -13.8 (-21.6, -5.9 95%CI) p = 0.002. Between-group: low-fat favourably affected total CHOL compared to low CHO: 9.1 (2.6, 15.7 95%CI) p=0.006. Within-group HDL compared to control: Low CHO group 4.4 (2.3, 6.5 95%CI) p=0.0004, low-fat group -1.0 (-3.2, 1.2 95%CI) p = 0.35. Between-group: low CHO favourably affected HDL compared to low-fat: 5.1(3.5, 6.7 95%CI) p&lt;0.0001. Within-group LDL compared to control: Low CHO group -1.8 (-6.1, 2.6 95%CI) p=0.39, low-fat group -10.9 (-17.3, -4.4 95%CI) p = 0.0025. Between-group: low CHO favourably affected LDL compared to low CHO: 8.6 (3.6, 13.7 95%CI) p=0.0008. Within-group TG compared to control: Low CHO group -41.1 (-54.7, -27.5 95%CI) p&lt;0.0001, low-fat group -11.3 (-18.8, -3.7 95%CI) p = 0.006. Between-group: low CHO favourably affected TG compared to low-fat: -28.8(-39.1, -18.5 95%CI) p&lt;0.0001. TC: HDL=n/a. SYSTOLIC BP. Within group:</td>
</tr>
<tr>
<td>Study</td>
<td>Diet Pattern</td>
<td>N</td>
<td>Gender</td>
<td>Outcomes</td>
<td>Results</td>
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<tr>
<td>Gay (2016)</td>
<td>Mediterranean</td>
<td>5121</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>SBP = MD - 1.17 (95% CI: -2.81, -0.46), I² = 93%, Chi² = 55.00, p &lt; 0.00001; DBP = -1.44 (95% CI: -2.11, -0.76), I² = 82%, Chi² = 22.23, p = 0.0002). (1) individuals with diabetes mellitus, SBP - 2.14 (95% CI, -5.14 to 0.86); (2) baseline BMI &gt; 35, SBP - 2.14 (95% CI, -5.14 to 0.86); and (3) trials with &gt; 1000 participants, SBP - 1.77 (95% CI, -3.77 to 0.22). Larger net SBP (P = 0.03) and DBP (P = 0.02) reductions noted among hypertensives at baseline, compared with normotensives.</td>
</tr>
<tr>
<td>Huang (2011)</td>
<td>Mediterranean</td>
<td>1571</td>
<td>M &amp; F</td>
<td>Lipid profile</td>
<td>Med diet (5 primary &amp; 3 secondary) can reduce TC by 5-15%, LDL-C by 5-15% and increase HDL-C by 3-15%. Conclusion: Med diet recommended to improve lipid profile.</td>
</tr>
<tr>
<td>Ndanuko (2016)</td>
<td>Mediterranean</td>
<td>1075</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>SBP: MD - 3.02 mmHg (95% CI: -3.47, -2.58), I² = 0%, Chi² = 1.80, P = 0.41. DBP: MD - 1.99 mmHg (95% CI: -2.28, -1.71), I² = 0%, Chi² = 0.06, P = 0.97.</td>
</tr>
<tr>
<td>Grosso (2014)</td>
<td>Mediterranean</td>
<td>1008 902 (unsure)</td>
<td>M &amp; F</td>
<td>Weight, hypertension, CVD mortality, lipid profile</td>
<td>CROSS-SECTIONAL STUDIES (primary): Med diet protective role against obesity (1/12 studies found inconsistent results) and decreased risk of obesity. NB inconsistent findings found between Med diet and body fat, but most studies (especially those with large numbers) found Med diet associated with lower abdominal adiposity/waist circumference, but no change in BMI. Inverse relationship between Med diet, BMI and waist-hip ratio. COHORT STUDIES: (primary) European studies found inverse relationship between Med diet and likelihood of obesity. A Spanish study found no association between BMI.</td>
</tr>
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</table>
and Med diet. INTERVENTION STUDIES: (primary) one study found high adherence to Med diet was associated with lower BMI and weight loss. CROSS-SECTIONAL STUDIES: (primary) Improvement in lipid profile (n=1). INTERVENTION STUDIES: (primary) Med diet led to reduced blood lipids. Another study found Med diet on diabetic patients resulted in improved lipid levels. CROSS-SECTIONAL: (primary) Improvement in BP (n=1). COHORT STUDIES: (primary) EPIC study found inverse relationship between Med diet and BP. A Spanish study found inverse association between Med diet and BP. CROSS-SECTIONAL STUDIES: (primary) Protective role of Med diet against CVD events (n=1). Protective effect Med diet found in MI survivors. INTERVENTION STUDIES: (primary) Med diet had beneficial effects on CVD risk factors. Study in patients with ≥1 risk factor for CVD found Med diet improves biochemical markers. INTERVENTION STUDIES: (secondary) found Med diet may be better at primary and secondary prevention of CAD than conventional prudent diet. Another study found no statistical sig. in secondary prevention.

Kastorini (2010)

Mediterranean 582617 35 M & F Weight, morbidity and mortality, lipid profile

Majority of studies showed Med diet had beneficial effect on body weight, though not all showed a protective effect on body weight and obesity. Concluded that Med diet protects against development of CHD due to possible effect of Med diet on body weight and obesity. Primary (observational studies, n = 5) indicated participants who consumed diets closest to Med diet decreased body weight and CVD risk factors. Primary (observational studies): Overall, adherence to Med diet decreased body weight and CVD risk factors. ATTICA study: Highest tertile of Med Diet Score had 51% lower
probability of being obese and 59% lower probability of central obesity, compared to lowest tertile. Highest tertile had a sig. lower BMI than lowest tertile. Primary (clinical trials): not all studies reported on Med Diet and lipid profile. Of 19 primary clinical trials, n=1 reported improvement in TC, apo-B, HDL, LDL and TG, n=1 reported improvement in total HDL, and n=1 reported improvement to TC, LDL, TG and apo-B. Primary (cross-sectional): PREDIMED trial - higher adherence to Med diet associated with significantly lower probability of obesity and 33% lower odds of having the following four CHD risk factors at the same time (diabetes, hyperlipidaemia, hypertension or obesity). MEDIS Study: Med diet score associated with a 1.2 kg/m² decrease in BMI levels and a 57% lower likelihood of obesity (OR per 1-unit= 0.92, 95%CI: 0.83-1.00). Primary (Clinical trials): Not all studies reported on Med diet and CVD risk. n=3 reported decrease in coronary risk, n=2 reported improvements in endothelial function, n=1 reported reduction in CVD risk after 3 months, compared to control diet. Primary (clinical trials): majority, but not all studies showed protective effect of the Med diet on body weight and obesity.

<p>| Mancini (2016) | Mediterranean | 998 | S | M &amp; F | Weight, hypertension, lipid profile, CHD | Med diet had modest effects on reducing body weight (range of mean changes: 3.8 to 10.1kg) at 12 months. Med diet similar to comparator diets. However, Med diet resulted in greater reductions in triglyceride levels than comparator diets at 12 months (range of mean changes = 0.25 to 1.50 mmol/L vs 0.03 to 0.70 mmol/L). Secondary: Med diet similar to comparator diets. |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Diet Pattern</th>
<th>Adherence</th>
<th>Population</th>
<th>Gender</th>
<th>Outcomes</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Martinez-Gonzalez (2014)</td>
<td>Mediterranean</td>
<td>Not listed</td>
<td>14</td>
<td>M &amp; F</td>
<td>CVD morbidity, lipid profile</td>
<td>CVD risk - RCT’s: 38% relative reduction in CVD risk after intervention with Med Diet; pooled RR 0.62 (95% CI: 0.45 - 0.85). Observational studies: After removing studies that only reported fatal outcomes to remove heterogeneity, each 2-point increase in 0-9 score of adherence to Med Diet (MDS = Mediterranean diet score) was associated with 13% relative reduction in CVD risk (pooled RR 0.87; 95% CI 0.85 - 0.90, I²=19.8%, P=0.26).</td>
</tr>
<tr>
<td>Nissensohn (2016)</td>
<td>Mediterranean</td>
<td>7987</td>
<td>6</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>SBP: MD -1.44 mmHg (95% CI: -2.88, 0.01), I²=87%, Chi²=46.22, P&lt;0.00001, DBP: MD -0.70 mmHg (95% CI: -1.34, -0.07), I² = 63%, Chi²=16.26, P=0.01.</td>
</tr>
<tr>
<td>Liyanage (2016)</td>
<td>Mediterranean</td>
<td>10950</td>
<td>6</td>
<td>M &amp; F</td>
<td>CVD morbidity and mortality, heart failure, heart disease</td>
<td>Med diet associated with 37% relative reduction in the risk of major CVD events (n=3) (RR 0.63, 95% CI 0.53–0.75, p &lt; 0.001), compared to control diet. One study had concerns regarding its integrity - when excluded from meta-analysis (RR 0.69, 95% CI 0.55–0.86, p &lt; 0.001) for CVD events. No clear effect on all-cause mortality (RR 1.00, 95% CI: 0.86–1.15, p = 0.97) or CV mortality (RR: 0.90, 95% CI: 0.72–1.11, p = 0.32) compared to control (n=5). Med diet associated with RR of 0.65 (95% CI 0.50–0.85) for coronary events (n=3), 0.65 (95% CI 0.48–0.88) for stroke (n=3) and RR of 0.30 (95% CI: 0.17–0.56) for heart failure. When study of low integrity excluded: coronary events + heart failure lost significance but stoke remained significant (RR: 0.66, 95% CI: 0.48–0.92, p = 0.01).</td>
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<tr>
<td>Schwingshackl (2014)</td>
<td>Mediterranean</td>
<td>2300</td>
<td>17</td>
<td>M &amp; F</td>
<td>Inflammation</td>
<td>Med dietary pattern significantly improved markers of inflammation CRP, IL-6 and Adiponectin, as well as endothelial function measured as FMD (Flow mediated dilation) and ICAM-1. Med diet reduced CRP = WMD: -0.98mg/l, 95% CI = 1.48, -0.49, p&lt;0.0001, I² = 91% and IL-6 = WMD: -0.42pg/ml, 95%CI =</td>
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<tr>
<td>Study</td>
<td>Diet</td>
<td>Participants</td>
<td>Gender</td>
<td>Primary Outcome</td>
<td>Results</td>
<td>Notes</td>
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<tr>
<td>Kastorini (2011)</td>
<td>Mediterranean</td>
<td>534,906</td>
<td>50</td>
<td>Hypertension, lipid levels, waist circumference</td>
<td>SBP: MD -2.35 mm Hg (95% CI: 3.51 to 1.18) and DBP: MD -1.58 mm Hg (95% CI: 2.02 to 1.13). HDL: MD 1.17 mg/dl (95% CI: 0.38 to 1.96). TGs: MD: -6.14 mg/dl (95% CI: 10.35 to 1.93). WC: MD -0.42 cm (95% CI: 0.82 to 0.02).</td>
<td>adiponectin increased with Med Diet (WMD: 1.69μg/ml, 95% CI 0.27 - 3.11, p=0.02, I² = 78%). FMD increased with Med Diet (WMD:1.86%, 95% CI 0.23, 3.48, p = 0.02, I² = 43%). ICAM-1 concentration decreased with Med Diet (WMD: -23.73ng/ml, 95% CI: -41.21, -6.22. p=0.008, I² = 34%).</td>
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<tr>
<td>Yokoyama (2014)</td>
<td>Vegetarian</td>
<td>21,915</td>
<td>39</td>
<td>Hypertension</td>
<td>Results for clinical studies: SBP −4.8 mmHg (95%CI: −6.6 to −3.1), p &lt; .001, I² = 0%, P=.45. DBP −2.2mmHg (95%CI: −3.5 to −1.0), p &lt;0.001, I²=0%, P=0.43. Observational studies: SBP -6.9 mmHg (95% CI: -9.1 to -4.7), p&lt;.001, I²=91.4%, P&lt;.001. DBP: -4.7 mmHg (95% CI: -6.3 to -3.1), P&lt;0.001, I²=92.6%, p&lt;.001.</td>
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<tr>
<td>Gay (2016)</td>
<td>DASH</td>
<td>408</td>
<td>4</td>
<td>Hypertension</td>
<td>DASH: SBP = MD -7.62 (95%CI: -9.95, -5.28, I²=81%, Chi²=20.86, p=0.0003); DBP = -4.22 (95% CI: -5.88, -2.57, I²=92%, Chi²=48.02, p&lt;0.0001). with &gt;1000 participants, SBP -1.77 (95% CI, −3.77 to 0.22). Larger net SBP (P=0.03) and DBP (P=0.02) reductions were noted among participants with pre-existing hypertension at baseline, compared with normotensives.</td>
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<tr>
<td>Ndanuko (2016)</td>
<td>DASH</td>
<td>2798</td>
<td>10</td>
<td>Hypertension</td>
<td>DASH = SBP: MD -4.90 (95% CI: -6.22, -3.58), I²=70%, Chi² = 33.59, P=0.0002; DBP: MD -2.63 mmHg (95% CI: -3.34, -1.92), I² = 61%, Chi²=25.39, P=0.005</td>
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<tr>
<td>Siervo (2015)</td>
<td>DASH</td>
<td>1 917</td>
<td>20</td>
<td>Hypertension, lipid profile</td>
<td>The mean BMI of the participants ranged from 23 to 37kg/m² in individual studies; BMI not reported in one study. Baseline BMI directly associated with changes in SBP (β=0.1 (SE0.06)</td>
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<tr>
<td>Study</td>
<td>Intervention</td>
<td>Population</td>
<td>Weight Change</td>
<td>Hypertension</td>
<td>Blood Pressure</td>
<td>Lipid Profile</td>
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<tr>
<td>Soltani (2016)</td>
<td>DASH</td>
<td>2,292</td>
<td>13 M &amp; F</td>
<td>Hypertension</td>
<td><strong>SBP:</strong> MD = -2.27 (95% CI: -1.65, -1.88, p&lt;0.001)</td>
<td><strong>DBP:</strong> MD = -0.85 (95% CI: -1.27, -0.41, p&lt;0.001)</td>
</tr>
<tr>
<td>Saneei (2014)</td>
<td>DASH</td>
<td>495</td>
<td>4 M &amp; F</td>
<td>Hypertension</td>
<td><strong>SBP:</strong> MD = -9.78 (95% CI: -11.40, -8.17, I² = 0%)</td>
<td><strong>DBP:</strong> MD = -5.51 (95% CI: -7.30, -3.72, I² = 31.3%)</td>
</tr>
<tr>
<td>Santesso (2012)</td>
<td>High protein</td>
<td>Median 54 (range: 5–405)</td>
<td>74 M &amp; F</td>
<td>Weight, hypertension, lipid profile, inflammation</td>
<td><strong>Weight loss:</strong> MD = 0.36 (95% CI: -0.56, 0.17, p = 0.0002)</td>
<td><strong>BMI:</strong> MD = -0.37 (95% CI: -0.56, 0.19, p &lt;0.0001)</td>
</tr>
<tr>
<td>Study</td>
<td>Macronutrient</td>
<td>Participants</td>
<td>Duration</td>
<td>Sex</td>
<td>Outcomes</td>
<td></td>
</tr>
<tr>
<td>------------------</td>
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<td>--------------</td>
<td>----------</td>
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<td>-----------------------------------</td>
<td></td>
</tr>
<tr>
<td>Wycherley (2012)</td>
<td>High protein</td>
<td>1,063</td>
<td>12 weeks</td>
<td>M &amp; F</td>
<td>Weight, hypertension, lipid profile</td>
<td></td>
</tr>
<tr>
<td>Huang (2011)</td>
<td>High protein</td>
<td>23</td>
<td>2 weeks</td>
<td>M &amp; F</td>
<td>Lipid profile</td>
<td></td>
</tr>
<tr>
<td>Rebholz (2012)</td>
<td>High protein</td>
<td>3,277</td>
<td>40 weeks</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity in included studies. Moderate statistically significant decrease in TG, but high heterogeneity. CRP (5 studies): MD 0.01 (95% CI: -0.23, 0.25), p = 0.95. HbA1c: MD 0.00 (95% CI: -0.19, 0.19), p = 0.96. 

Wycherley (2012) High protein 1,063 23 M & F Weight, hypertension, lipid profile 12+ weeks duration, weight loss -0.97 (95% CI -2.07 to 0.13kg, I²=75%, Chi²= 48.47, P<0.0001). <12 weeks duration, weight loss -0.49 (95% CI -1.34 to 0.37kg, I²=45.2%, Chi²=22.54, P=0.007); TOTAL combined; weight loss -0.79 (95% CI -1.50 to -0.08kg, I²=71%, Chi²=75.64, P<0.00001). SBP (total): -2.09 mmHg (95% CI -5.01 to 0.83), p=0.82. Overall effect p=0.16; I² 0%, Chi²=1.54, P=0.82. DBP: -0.72 mmHg (95% CI -2.67 to 1.23); p=0.55; overall effect p=0.47; I²=0%, Chi²=3.05, P=0.55. TG'S: 12+ weeks: -0.23 mmol/L (95% CI -0.36 to -0.11), I²=0%, Chi²=5.40, P=0.49). <12 weeks: -0.21 mmol/L (95% CI -0.40 to -0.03), I²=0%, Chi²=1.65, p=0.89); total: -0.23 mmol/L (95% CI -0.33 to -0.12), I²=0%, Chi²=7.09, P=0.85). TOTAL CHOLESTEROL (total): -0.09 mmol/L (95% CI -0.23 to 0.05), p<0.01; overall effect p=0.20, I²=52%, Chi²=31.27, P=0.008. LDL-C (total): 0.05 mmol/L (95% CI -0.10 to 0.21), p<0.01; overall effect 0.52; I²=58%, Chi²=26.10. P=0.006) HDL-C (total): 0.00 mmol/L (95% CI -0.08 to 0.09); p<0.01; overall effect 0.97; I²=92%, Chi²=160.88, P<0.00001). TC:HDL=n/a

Huang (2011) High protein 23 2 M & F Lipid profile High protein (25% of total energy from protein) lowered TC, LDL-C, and TGs by 5% to 10%. 

Rebholz (2012) High protein 3,277 40 M & F Hypertension Increased protein intake changed SBP by -1.76 mmHg (95% CI: -2.33 to -1.20, p<0.001) and DBP by -1.15 mmHg (95% CI: -1.59 to -0.71, p=0.014) compared with CHO intake (overall heterogeneity I²=0%, P=0.92). No statistically significant
Dietary Patterns and Cardiovascular Disease Outcomes

<table>
<thead>
<tr>
<th>Study</th>
<th>Dietary Pattern</th>
<th>Design</th>
<th>Gender</th>
<th>Condition</th>
<th>Outcome</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gay (2016)</td>
<td>Weight-loss/ Calorie-restriction</td>
<td>n/a</td>
<td>11</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>Large heterogeneity between studies. Low kJ+/low-fat SBP = MD -3.18 (95%CI: -4.24, -2.11, I²=69%, Chi²=38.10, p=0.0001; DBP = -1.28 (95% CI: -1.88, -0.69), I²=60%, Chi²=29.94, p=0.003). Larger net SBP (p=0.03) and DBP (p=0.02) reductions noted among hypertensives at baseline, vs normotensives.</td>
</tr>
<tr>
<td>Ndanuko (2016)</td>
<td>Healthy diets (Nordic &amp; Tibetan)</td>
<td>830</td>
<td>4</td>
<td>M &amp; F</td>
<td>Hypertension</td>
<td>Nordic = SBP: MD -5.20 mmHg (95% CI: -7.30, -3.11), I²=0%, Chi²=0.88, P=0.64; DBP: MD -3.85 mmHg (95% CI: -5.50, -2.19), I² = 0%, Chi²=0.53, p=0.77. Tibetan = SBP: MD -1.10 mmHg (95% CI: -3.58, 1.38), I² = N/A; DBP: MD 0.10 mmHg (95% CI: -1.57, 1.77), I² = N/A (only 1 study on this diet, n = 524).</td>
</tr>
</tbody>
</table>

Abbreviations for the above data extraction Table

- BMI: Body Mass Index
- BP: Blood pressure
- CHD: Coronary heart disease
- CHO: Carbohydrate
- CI: Confidence Interval
- CRP: C-reactive protein
- CVD: Cardiovascular disease
- DBP: Diastolic blood pressure
- FFM: Free fat mass
- IL-18: Interleukin-18
- LDL: Low-density lipoprotein
- Lp-PLA2: Lipoprotein-associated phospholipase A2
- MD: Mean difference
- MI: Myocardial infarction
- NS: Non-significant
- PA: Physical activity
- RCT: Randomised controlled trial
- SBP: Systolic blood pressure
- DBP: Diastolic blood pressure
- CI: Confidence Interval
- MD: Mean difference
- I²: Heterogeneity index
- 95% CI: 95% Confidence Interval
- kJ: Kilojoule
- PA: Physical activity
- RCT: Randomised controlled trial
- N/A: Not applicable
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>HDL</td>
<td>High-density lipoprotein</td>
</tr>
<tr>
<td>HEI</td>
<td>Healthy eating index</td>
</tr>
<tr>
<td>HF</td>
<td>Heart failure</td>
</tr>
<tr>
<td>I²</td>
<td>Statistic describes the percentage of variation across studies that is due to heterogeneity</td>
</tr>
<tr>
<td>ICAM-1</td>
<td>Intercellular Adhesion Molecule 1</td>
</tr>
<tr>
<td>IL-6</td>
<td>Interleukin-6</td>
</tr>
<tr>
<td>RR</td>
<td>Relative risk</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>Sig.</td>
<td>Significant</td>
</tr>
<tr>
<td>SMD</td>
<td>Standardised mean difference</td>
</tr>
<tr>
<td>TC</td>
<td>Total cholesterol</td>
</tr>
<tr>
<td>TG</td>
<td>Triglycerides</td>
</tr>
<tr>
<td>WMD</td>
<td>Weighted Mean Difference</td>
</tr>
</tbody>
</table>
Appendix 9: Target cut-offs used to inform “clinical impact” component of evidence statement

Using data obtained from a literature research, the following cut-offs were developed. These cut-offs were used to inform the clinical impact component of each evidence statement.

The cut-offs reported in the table represent a change in the respective CVD outcome. The cut-offs were used as a guide only, as the clinical impact component often represented multiple CVD outcomes and all factors were taken into account for the overall grade.

<table>
<thead>
<tr>
<th>CVD Outcome</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic blood pressure</td>
<td>&gt;4mmHg</td>
<td>1-4mmHg</td>
<td>0-1mmHg</td>
<td>&lt;0mmHg</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>&gt;5mmHg</td>
<td>2-5mmHg</td>
<td>1-2mmHg</td>
<td>&lt;1mmHg</td>
</tr>
<tr>
<td>HDL-Cholesterol</td>
<td>&gt;0.50mmol/L</td>
<td>0.25-0.5mmol/L</td>
<td>0-0.25mmol/L</td>
<td>&lt;0mmol/L</td>
</tr>
<tr>
<td>LDL-Cholesterol</td>
<td>&gt;1mmol/L</td>
<td>0.5-1mmol/L</td>
<td>0-0.5mmol/L</td>
<td>&lt;0mmol/L</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>&gt;1mmol/L</td>
<td>0.5-1mmol/L</td>
<td>0.1-0.5mmol/L</td>
<td>&lt;0.1mmol/L</td>
</tr>
</tbody>
</table>