Comparing dietary strategies to manage cardiovascular risk in primary care: Narrative review of systematic reviews

Greenwood, Hannah; Barnes, Katelyn; Ball, Lauren; Glasziou, Paul

DOI: https://doi.org/10.3399/BJGP.2022.0564

To access the most recent version of this article, please click the DOI URL in the line above.

Received 16 November 2022
Revised 14 September 2023
Accepted 19 September 2023

© 2023 The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 License (http://creativecommons.org/licenses/by/4.0/). Published by British Journal of General Practice. For editorial process and policies, see: https://bjgp.org/authors/bjgp-editorial-process-and-policies

When citing this article please include the DOI provided above.
Comparing dietary strategies to manage cardiovascular risk in primary care:

Narrative review of systematic reviews

Hannah Greenwood (BPsycSci Hons I) Research assistant and PhD student, Institute for Evidence-Based Healthcare, Faculty of Health Science & Medicine, Bond University, Gold Coast, Australia.
ORCID ID: 0000-0001-5127-4667
Email: h.greenwo@bond.edu.au.

Katelyn Barnes (BAppSci, MND, PhD) Research Fellow, Menzies Health Institute Queensland, Griffith University, Brisbane, Australia; Senior Research Officer, Academic Unit of General Practice, ACT Health Directorate and Australian National University Medical School, Canberra, ACT, Australia.
ORCID ID: 0000-0001-5891-1331
Email: k.barnes@griffith.edu.au

Lauren Ball (BAppSci, MND, PhD) Professor in Community Health and Wellbeing, Centre for Community Health and Wellbeing, The University of Queensland, Brisbane, Australia. ORCID ID: 0000-0002-5394-0931
Email: l.ball@griffith.edu.au

Paul Glasziou (MBBS, PhD) Professor of Evidence-Based Practice and Director, Institute for Evidence-Based Healthcare, Faculty of Health Science & Medicine, Bond University, Gold Coast, Australia.
ORCID ID: 0000-0001-7564-073X
Email: pglaszio@bond.edu.au

Corresponding author

Mrs Hannah Greenwood
Institute for Evidence-Based Healthcare, Faculty of Health Science & Medicine, Bond University, 14 University Drive, Robina, Australia, 4226
ORCID ID: 0000-0001-5127-4667
Email: hgreenwo@bond.edu.au

Keywords
Nutritional sciences; cardiovascular diseases; general practice; review; primary health care; lifestyle

How this fits in
Diet is a key factor in preventing cardiovascular disease (CVD) and managing absolute CVD risk, but the comparative effectiveness of different dietary strategies to reduce absolute CVD risk is unclear. By examining current best available evidence, this paper finds that energy reduction, Mediterranean-style diets, and salt substitution are most promising to reduce CVD events, though all examined strategies can help absolute CVD risk reduction. Using behaviour change principles, clinicians can work with patients to select the dietary strategy/ies most aligned with their specific personal and clinical circumstances.

Contributors
All authors contributed to the conception and design of the work. HG, LB and KB conducted the majority of data acquisition and analysis. HG and KB conducted new GRADE assessments. All authors were equally involved in the interpretation of the data. All authors participated in drafting the work (KB led introduction, LB led discussion, HG and PG jointly led methods and results; all authors provided critical revision to all sections of the manuscript). All authors give approval of this manuscript for publication and accept responsibility for the accuracy and integrity of the work.

Abbreviations
CHD = coronary heart disease
CI = confidence interval
CVD = cardiovascular disease
DASH = Dietary Approaches to Stop Hypertension
DBP = diastolic blood pressure
HDL = high density lipoprotein
LDL = low density lipoprotein
MD = mean difference  
RCT = randomised controlled trial  
RR = risk ratio  
RRR = relative risk ratio  
SBP = systolic blood pressure
Abstract

Background

Nutrition care in general practice is crucial for cardiovascular disease (CVD) prevention and management, though comparison between dietary strategies is lacking.

Aim

To compare best-available (most recent, relevant, and high-quality) evidence for six dietary strategies effective for primary prevention/absolute risk reduction of CVD.

Design and setting

A pragmatic narrative review of systematic reviews of randomised trials

Methods

We included studies of: (i) adults without history of cardiovascular events; (ii) target dietary strategies postulated to reduce CVD risk, and (iii) direct cardiovascular or all-cause mortality outcomes. Six dietary strategies were examined: energy deficit, Mediterranean-like diet, sodium reduction (salt reduction and substitution), DASH diet, alcohol reduction and fish/fish oil consumption. Reviews were selected based on quality, recency, and relevance. Quality and certainty of evidence was assessed using GRADE.

Results

Twenty-five reviews met inclusion criteria; eight were selected as the highest quality, recent, and relevant. Three dietary strategies showed modest, significant reductions in cardiovascular events: energy deficit (relative risk reduction [RRR] 30%, 95%CI 13%-43%), Mediterranean-like diet (RRR 40%, 95%CI 20%-55%), and salt substitution (RRR 30%, 95%CI 7%-48%). Salt reduction, DASH diet,
and alcohol reduction showed small, significant reductions in blood pressure, but no reduction in cardiovascular events. Fish/fish oil consumption showed little or no effect; supplementation of fish oil alone showed small reductions in CVD events.

**Conclusion**

For primary prevention, energy deficit, Mediterranean-like diets and sodium substitution have modest evidence for risk reduction of CVD events. Strategies incorporated into clinical nutrition care should ensure guidance is person-centered and tailored to clinical circumstances.
Introduction

The World Health Organisation estimates 75% of cardiovascular events may be preventable. Still, cardiovascular disease (CVD) is a major source of morbidity and mortality globally (1). Appropriate primary care management of absolute CVD risk reduces associated death and disability (2), and contemporary CVD risk management focuses on reducing absolute risk (i.e., 5-year risk of CVD including multiple risk factors), rather than individual risk factors (3, 4).

Diet is a key factor in managing absolute CVD risk (4), and is recognized for the primary prevention of CVD (1, 5, 6), but the relative effectiveness of different dietary strategies remains unclear. Direct comparison of different dietary strategies will support clinicians to make evidence-informed diet recommendations for patients looking to manage CVD risk. While other dietary approaches (e.g., vegetarian or vegan diets (7)) may influence CVD risk, this narrative review of systematic reviews focusses on six strategies identified as potentially influencing absolute cardiovascular risk by the National Heart Foundation of Australia and an expert advisory panel (8, 9). These are outlined in table 1 along with their proposed cardiovascular effects. Given the heterogeneous nature of these interventions, we conduct a narrative review to allow top-level comparison between dietary strategies to inform clinical decisions.

Table 1: Target dietary strategies and their cardiovascular effects

<table>
<thead>
<tr>
<th>Dietary Strategy</th>
<th>Description of dietary strategy</th>
<th>Cardiovascular effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy deficit</td>
<td>Diets specifically formulated to reduce calorie intake (e.g. Very low energy diets (&lt;800 kcal/day) or lifestyle change to induce energy deficit (including diet with or without exercise)</td>
<td>Decreased body weight lowers blood pressure (10)</td>
</tr>
<tr>
<td>Mediterranean diet</td>
<td>A naturalistic dietary pattern that promotes, high intakes of wholegrains, vegetables and fruits, moderate intakes of seafood, unsaturated fats and red wine, limited intake of red meats. Regular exercise is promoted as part of the lifestyle.</td>
<td>Combination of lifestyle factors thought to be cardioprotective via reduced blood pressure, and reduced blood lipids, among other mechanisms (11)</td>
</tr>
</tbody>
</table>
Table salt (NaCl) intake is decreased through reduction of added table salt to foods, or manipulation of table salt intake to allow for comparison between higher and lower salt intake groups.

Sodium reduction

Salt reduction

Table salt (NaCl) intake is decreased through reduction of added table salt to foods, or manipulation of table salt intake to allow for comparison between higher and lower salt intake groups.

Decreased sodium lowers blood pressure (12)

Salt substitution

Sodium in regular table salt or other high sodium produces is replaced with potassium (KCl).

Decreased sodium lowers blood pressure (12); Increased potassium may have cardioprotective effects (13)

Dietary Approaches to Stop Hypertension (DASH diet)

Dietary pattern designed specifically to reduce hypertension that promotes high intakes of fruits and vegetables, moderate intake of wholegrains and low-fat dairy, moderate to limited intakes of meats, and limited intake of fats and salt.

Combination of lifestyle factors thought to reduce blood pressure (14)

Alcohol reduction

Reduction in usual alcohol intake or elimination of alcohol from diet.

Decreased alcohol is thought to lower blood pressure. Alcohol consumption has a complex relationship with cardiovascular health and excess consumption is associated with many CV diseases, so reduction may reduce CV disease risk (15, 16).

Fish/fish oil consumption

Diets high in fish, or supplemental fish oils.

Omega chain fatty acids, commonly found in fish, are though to be cardioprotective (17)

In this pragmatic narrative review of systematic reviews, we aimed to identify and descriptively compare the most relevant, best available, highest quality systematic reviews for six dietary strategies postulated to be effective for primary prevention/absolute risk reduction of CVD: energy deficit, Mediterranean-like diet, sodium reduction (salt reduction and substitution), DASH diet, alcohol reduction and fish/fish oil consumption (8).
Methods

This narrative review has been reported in line with the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines (18). A broad methodological approach was prospectively developed as part of larger program of commissioned work to update Australian absolute CVD risk guidelines. Due to this, the protocol was not registered. Pragmatic decisions were made to enable identification of the most recent, relevant, and high-quality available evidence. In brief, we included studies published after 2014 to limit screening and identify the most recent evidence, limited our review to 6 dietary strategies in line with the funder’s priorities (8, 9), utilised a targeted search approach (i.e., two prong approach) to identify included reviews, and used AMSTAR 1 rather than AMSTAR 2 to assess quality as it allows for an overall score. These decisions and full justifications are summarised in Supplementary Appendix 1.

Identification of target dietary interventions

Target dietary interventions (outlined in Table 1) relevant to clinical practice were identified jointly by general practice, cardiovascular and nutrition & dietetics experts from the authorship team, their networks, and the National Heart Foundation cardiovascular expert committee (8).

Search strategy

We used a two-pronged citation analysis approach to identify reviews for each dietary strategy (19). First, “similar articles” searches in PubMed were conducted from known seed articles (20). Second, for each review identified, we conducted a forward and backward citation analysis using the SpiderCite automation tool (21), and filtered results to include only systematic reviews.

Screening and study selection

Systematic reviews of RCTs published January 2014 to May 2022 were screened for eligibility against PICO criteria in Table 2. If a review for a target diet reporting direct cardiovascular or mortality
outcomes was not found, reviews with indirect outcomes (e.g., blood pressure, blood lipids) were considered. At prong 1, one reviewer (HG) screened systematic reviews for those relevant to the PICO of each target dietary interventions. At prong 2, two reviewers (HG and LB or KB) independently screened title and abstract to select eligible reviews, including those identified in prong 1. Full text was obtained and were independently assessed for eligibility and risk of bias by two reviewers (HG and LB or KB). Discrepancies in screening were resolved by discussion or referral to another author (PG). Selection of key systematic review/s for each dietary intervention was made jointly by all authors, accounting for recency, quality, and relevance (i.e., how closely the review question matched the PICO of 6 selected dietary approaches). If the outcomes in a review did not include both indirect (e.g., blood pressure) and direct (e.g., CVD events or mortality) measures of cardiovascular risk, we considered more than one review to summarise. If studies were related and included additional information (e.g., subsequent trials or re-analysis), we also report these results.

Table 2: PICO inclusion and exclusion criteria

<table>
<thead>
<tr>
<th>Inclusion</th>
<th>Exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population: General population/primary prevention cohort (including hypertension) of any age, if adults are included</td>
<td>Population: Secondary prevention cohort (participants had existing CVD)</td>
</tr>
<tr>
<td>Intervention: Dietary non-drug interventions (energy deficit, Mediterranean diet, sodium reduction [salt reduction or substitution], DASH diet, alcohol reduction, and fish/fish oil consumption)</td>
<td>Intervention: Other diet intervention not pre-specified by expert advisors</td>
</tr>
<tr>
<td>Comparator: Any other intervention or control</td>
<td>Comparator: N/A</td>
</tr>
<tr>
<td>Outcome: CVD event or all-cause mortality.</td>
<td>Outcome: Direct nor indirect cardiovascular outcomes reported.</td>
</tr>
</tbody>
</table>

Review methodological quality (risk of bias)

AMSTAR 1 was used to assess methodological quality of systematic reviews across 11 domains, and it is considered a pragmatic, time-efficient method for comparing quality between studies (22). Two authors (HG and KB or LB) independently assessed risk of bias for all full-text results.
Data extraction

Where available, the following outcome data were extracted: dietary strategy, number of included trials and participants, participant hypertension status (hypertensive, mixed), intervention, length of intervention, adherence to intervention, comparator, all relevant outcomes as reported (i.e., total (all-cause) mortality), CVD mortality, CVD events, blood pressure (systolic, diastolic), body weight, blood lipid concentration, change in alcohol consumption), length of study follow up, and effect estimate (hazard ratio or risk ratio) with 95% confidence intervals and/or p-values.

Evaluation of evidence certainty

We assessed the certainty or quality of the body of evidence for each reported outcome using GRADE (23). GRADE provides a rating reflecting how certain the authors are that estimated effect aligns with the true effect: very low, low, moderate, or high. Where original authors had conducted GRADE, results were reviewed and retained if sufficiently detailed and applicable. For reviews without existing GRADE assessment, two authors (HG and KB) completed GRADE independently, with disputes resolved by discussion or referral to another author (PG or LB).

Data synthesis

Existing outcomes for the dietary strategies were tabulated to allow direct descriptive comparison between strategies. A narrative overview of findings compares the different strategies for primary CVD prevention. No new statistical analyses were planned due to heterogeneity of interventions, timeframes, review questions, and populations.
Results

The selection process is presented in Figure 1. Eight reviews were included for six dietary strategies. Supplementary Table 1 describes each included review, including interventions. Table 3 provides a summary of key direct and indirect cardiovascular outcomes alongside certainty of evidence. See Supplementary Appendix 2 for new GRADE assessments and summaries of existing GRADE assessments and Supplementary Table 2 for description and AMSTAR quality rating of all studies assessed at full text, and reasons for inclusion/exclusion.

Figure 1: PRISMA study flow diagram to select key reviews
**Energy deficit**

Semlitsch 2021 (24) reported low-certainty evidence that energy deficit strategies (with or without exercise) lead to modest reductions in blood pressure (MD -4.5 mmHg, 95%CI -7.2 to -1.5) amongst people with hypertension who have overweight, but had clinically meaningful weight loss compared to control (MD -3.98kg, 95%CI -4.79 to -3.17). One included trial found energy deficit meaningfully lowered risk of CVD events (HR 0.70, 95%CI 0.57 to 0.87), but evidence certainty was low. No evidence was available regarding mortality.

**Mediterranean Diet**

Rees 2019 (25) reported that compared to low-fat diets, there is moderate-certainty evidence that Mediterranean-like diets meaningfully reduce strokes (HR 0.60, 95%CI 0.45 to 0.80). Evidence is inconclusive for CVD mortality; effect estimates show reduced risk but are imprecise with wide confidence intervals (RR 0.81, 95%CI 0.50 to 1.32). There is little or no evidence of effect of Mediterranean-like diet on all-cause mortality (HR 1.00, 95%CI 0.81 to 1.24). Moderate-certainty evidence suggests that compared to no/minimal intervention, Mediterranean-like diets had a small but meaningful effect on blood pressure (SBP MD -3.0mmHg, 95%CI -3.5 to -2.5; DBP MD -2.0 mmHg 95%CI -2.3 to -1.7), but little to no effect on blood lipids (low-certainty; LDL: MD -0.08mmol/L, 95%CI -0.26 to 0.09; HDL: MD 0.02mmol/L, 95% CI -0.04 to 0.08). Compared to other dietary interventions, Mediterranean-like diet had little to no clear effect on blood pressure or lipids.

**Sodium reduction**

Adler 2014 (26) reported that sodium reduction either through salt reduction or substitution of <70-100 mmol/day for people with hypertension appears to reduce blood pressure (SBP MD -4.1mmHg, 95%CI -5.8 to -2.4; DBP MD -3.7mmHg, 95%CI -8.4 to 0.93), CVD mortality (RR 0.67, 95%CI 0.45 to
1.01) and CVD events (RR 0.77, 95%CI 0.58 to 1.02). Although confidence intervals are uncertain and evidence certainty is low, the risk reductions are clinically meaningful. For people with normotensive blood pressure, there is moderate to high-certainty evidence that sodium reduction doesn’t meaningfully reduce CVD events (RR 0.84, 95%CI 0.64 to 1.10) or all-cause mortality (RR 0.90, 95%CI 0.58 to 1.40). These findings are driven by one trial assessing large scale salt reduction by institutional low-sodium alternative substitution (salt substitution).

Salt substitution
Hernandez 2019 (27) reported high-certainty evidence that salt substitution alone meaningfully reduces all-cause mortality (RR 0.89, 95%CI 0.77 to 1.03), though confidence intervals remain inconclusive. In the mixed hyper- and normotensive sample, salt substitution meaningfully reduced systolic blood pressure (SBP MD -7.8mmHg, 95%CI -9.5 to -6.2), though diastolic reductions were more modest (DBP MD -3.96mmHg; 95%CI -5.17 to -2.74). While no meta-analysis of CVD mortality or events were reported, a large subsequent randomised trial of salt substitution (75% sodium chloride; 25% potassium chloride) versus regular salt found significant and clinically important reductions in systolic blood pressure, stroke (Rate Ratio 0.86, 95%CI 0.77 to 0.96), major CVD events (Rate Ratio 0.87, 95%CI 0.80 to 0.94) and death (Rate Ratio 0.87, 95%CI 0.79 to 0.96) (28). Importantly, adverse events from high potassium were not significantly higher in the substitution group (Rate Ratio 1.04, 95%CI 0.80 to 1.37).

DASH diet
Filippou 2020 (29) reported moderate-certainty evidence that over an average 15.3 weeks, the DASH diet (compared to control diet) had a small effect on blood pressure reduction (SBP MD -3.2mmHg, 95%CI -4.2 to -2.3; DBP MD -2.5mmHg, 95%CI -3.5 to -1.5), consistent regardless of baseline blood pressure. The blood pressure lowering effect of DASH diet was more pronounced when participants’ baseline sodium intake was >2400mg/day (P=0.003), and when participants were aged <50 years.
(P<0.001). There was no moderating effect of weight, and no evidence was reported for the impact of DASH diet for primary prevention of cardiovascular events or mortality.

Alcohol reduction

Acin 2020 (30) reported little to no effect of alcohol reduction on all-cause mortality (RR 0.70, 95%CI 0.20 to 3.20) or cardiovascular events (RR 0.80, 95%CI 0.36 to 1.79), but evidence was of low-certainty and drawn from a subgroup of one trial with mostly male participants. No included studies reported CVD mortality or blood pressure. Roerecke 2017 (31) reported low-certainty evidence that reduced alcohol consumption meaningfully decreased blood pressure (SBP MD -3.1 mmHg, 95%CI -3.9 to -2.3, DBP MD -2.0 mmHg, 95%CI -2.7 to -1.4). Substantial heterogeneity was largely explained when stratified by alcohol consumption at baseline. When consuming <2 drinks/day at baseline, reduction had no significant effect on blood pressure, whereas for those consuming >3 drinks/day at baseline, alcohol reduction resulted in meaningful reductions in blood pressure. This effect was strongest for those consuming 6+ drinks/day at baseline (SBP MD -5.5 mmHg, 95%CI -6.7 to -4.3).

Fish/fish oil

Adbelhamid 2020 (32) reported high-certainty evidence that fish or fish oil has very small effect on CVD mortality (RR 0.92, 95%CI 0.86 to 0.99), but little to no effect on all-cause mortality (RR 0.97, 95%CI 0.93 to 1.01), CVD events (RR 0.96, 95%CI 0.92 to 1.01), or cholesterol (LDL: MD 0.01mmol/L, 95%CI -0.01 to 0.03; HDL: MD 0.03mmol/L 95%CI 0.01 to 0.05). There is low-certainty evidence of a small reduction in coronary heart disease (CHD) mortality (RR 0.90; 95%CI 0.81 to 1.00) and CHD events (RR 0.91; 95%CI 0.85 to 0.97), but numbers needed to treat are high. Bernasconi’s 2020 (33) re-analysis of supplementation alone (average dose 1221mg/day) found reduced risk of CHD events, fatal and non-fatal myocardial infarction, and CHD mortality, but not CVD events. Some outcomes were dose dependent, whereby higher dosages of fish oil increased protection from CVD events and myocardial infarction. Although there is no clear evidence for CVD impacts including fish in the diet, when supplementation alone is considered, there is a small, dose-dependent, protective
cardiovascular effect of fish oil supplementation, though number needed to treat for additional benefit is high (32, 33).
### Table 3: Summary of key cardiovascular outcomes for target dietary strategies

<table>
<thead>
<tr>
<th>Dietary strategy</th>
<th>Participant BP</th>
<th>Indirect outcomes</th>
<th>Direct outcomes</th>
<th>CVD events forest plot</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>SBP (mmHg)</td>
<td>CVD mortality</td>
<td>All-cause mortality</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy deficit (24)</td>
<td>High</td>
<td>MD -4.5 (-7.2 to -1.5)</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Mixed</td>
<td>MD -3.0 (-3.5 to -2.5)</td>
<td>RR 0.81 (0.50 to 1.32)</td>
<td>HR 1.00 (0.81 to 1.24)</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>MD -1.2 (-2.3 to 0.02)</td>
<td>NR</td>
<td>RR 0.90 (0.58 to 1.40)</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>MD -4.1 (-5.8 to -2.4)</td>
<td>RR 0.67 (0.45 to 1.01)</td>
<td>RR 0.99 (0.87 to 1.14)</td>
</tr>
<tr>
<td></td>
<td>Mixed</td>
<td>MD -7.8 (-9.5 to -6.2)</td>
<td>RaR 0.87b (0.79 to 0.96)</td>
<td>RR 0.89 (0.77 to 1.03)</td>
</tr>
<tr>
<td>Sodium reduction (any) (26)</td>
<td>Normal</td>
<td>MD -3.2 (-4.2 to -2.3)</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Sodium reduction via substitution (27, 28)</td>
<td>Mixed</td>
<td>MD -3.1 (-3.9 to -2.3)</td>
<td>RR 0.92 (0.86 to 0.99)</td>
<td>RR 0.97 (0.93 to 1.01)</td>
</tr>
<tr>
<td>DASH diet (29)</td>
<td>Mixed</td>
<td>MD -3.2 (-4.2 to -2.3)</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Alcohol reduction (30, 31)</td>
<td>High</td>
<td>MD -3.1 (-3.9 to -2.3)</td>
<td>RR 0.92 (0.86 to 0.99)</td>
<td>RR 0.97 (0.93 to 1.01)</td>
</tr>
<tr>
<td>Fish/fish Oil (32)</td>
<td>Mixed</td>
<td>NR</td>
<td>RR 0.92 (0.86 to 0.99)</td>
<td>RR 0.97 (0.93 to 1.01)</td>
</tr>
</tbody>
</table>

**BP** = blood pressure; **CVD** = cardiovascular disease; **SBP** = systolic blood pressure; **mmHg** = millimetres of mercury; **RR** = Risk ratio; **RaR** = rate ratio; **HR** = Hazard ratio; **NR** = not reported; **MD** = mean difference

*Stroke only, **NR** in systematic review, results from subsequent RCT Neal 2021 (28); **combined endpoint including CVD complications**

**GRADE Certainty of evidence:** 
- □ = not GRADED; □ = VERY LOW; □ = LOW; □ = MODERATE; □ =HIGH
Discussion

Summary
This pragmatic narrative review of systematic reviews descriptively compares the effectiveness of six dietary strategies on cardiovascular outcomes in primary prevention populations. While no singular dietary strategy produced significant risk reductions across all CVD risk or mortality outcomes, all dietary strategies showed some potential benefit to meaningfully reduce CVD events and improve indirect CVD outcomes (e.g., blood pressure), overall reducing CVD risk; though there are some caveats to note within and across dietary strategies.

Energy deficit, Mediterranean-like diets and salt substitution are impactful dietary strategies for reducing CVD event risk, with some caveats. The evidence certainty for energy deficit was very low, and the composite outcome used (cardiovascular complications + recommencing antihypertensives) may inflate the estimate (24). Mediterranean-like diets compared to low-fat diets showed hazard reduction, but this was for stroke only, and from one large study (reanalysis of PREDIMED). The salt substitution effect estimate is from a large trial (28) and is not a pooled estimate. While the point estimates for sodium reduction (via salt reduction or substitution), alcohol reduction or fish/fish oil also suggest reduced CVD event risk, the confidence intervals were too imprecise to confirm or exclude important differences. Most strategies that reported blood pressure showed small but meaningful reductions in systolic blood pressure (24, 25, 27, 31, 34). The most promising strategy reviewed is salt substitution, a type of sodium reduction strategy (27). The reported systolic blood pressure reduction (6mmHg to 9mmHg) is sufficient to reduce CVD mortality, based on estimates that systolic reductions of 1mmHg and 4mmHg translate to reductions in CVD mortality of 2% and 8%, respectively (35). Mortality outcomes were not available for all dietary strategies. For CVD mortality, salt substitution shows a significant reduction in CVD mortality, though this estimate is from a large trial, not meta-analysis (28). Other available point estimates show small reductions in CVD mortality risk, but confidence intervals are too imprecise to determine if these are meaningful.
For all-cause mortality, while available point estimates show a small risk reduction, no strategy was associated with significantly reduced all-cause mortality.

Some caveats across all dietary approaches should be highlighted. First, the length of the studies in each of the included reviews are arguably not long enough to capture lifelong prevention of CVD, and the distinction between intervention and follow-up can be ambiguous due to the assumption of permanent dietary change (36). Second, very few studies within the included reviews reported adherence to the dietary intervention or assessed dietary intake. Dietary intake is an important indication of intervention fidelity and is required to differentiate a dietary intervention’s CVD risk reduction effects compared to other physiological effects. Finally, very few RCTs reported outcome measures that reflect potential mechanisms of action of the dietary intervention, for example, whether energy deficit lowers CVD event risk via indirect lowering of blood pressure, or via direct mechanisms.

**Strengths and limitations**

First, we elected to take a pragmatic approach to identify the most recent, highest quality reviews for a selection of 6 relevant dietary strategies, with the goal to approximate effect sizes of each dietary strategy using *best* available evidence, not *all* available evidence. However, we recognize that this approach does not provide a definitive measure of effect for each dietary strategy considering all available evidence, which would be a substantially larger, more resource intensive project. Second, the six dietary strategies were pragmatically selected by an expert committee for being relevant to clinical practice and postulated to effectively reduce absolute cardiovascular risk. However, we highlight that this is not an exhaustive list of dietary strategies which may reduce cardiovascular risk. Other examples of dietary strategies touted for beneficial cardiovascular effects which were outside the scope of this work include low carbohydrate and intermittent fasting, though recent Cochrane reviews suggest no major cardiovascular benefit (37, 38). Third, we only examined reviews of RCTs due to quality limitations inherent to observational studies. However, RCT
designs for dietary studies also have limitations, such as difficulties with blinding, control of intervention fidelity, long-term follow up, uniformity, independence of effects, and control over comparator groups (39, 40). Assessing long-term outcomes is required to understand cardiovascular impacts of diet, but dietary interventions are challenging to implement long term, particularly using RCT designs. Fourth, as this is a narrative review of heterogeneous interventions, meta-analysis and subgroup analysis was precluded. Although conclusions cannot be drawn for specific population subgroups, this review allows for top-level comparison between different dietary strategies to enable evidence-informed clinical decisions for recommending dietary strategies to manage cardiovascular risk in primary prevention populations. Finally, there may be some overlap between dietary strategies, for example Mediterranean diet may be used as an energy deficit strategy. However, we suggest there is a distinction between dietary strategies with the primary aim of reducing weight via energy deficit (such as the reduced-calorie diets in the included energy deficit study (24)) and approaches like Mediterranean diet or DASH, which may incidentally result in weight loss, but this is not the primary aim. While this justifies our inclusion of both dietary strategies, it does not erase possible overlapping effects which should be considered in interpretation of findings.

Comparison with existing literature
Given the social and economic burden of CVD, and the central role diet plays in the prevention and management of CVD (1, 5, 6), consideration of how to support primary care patients to optimize their diet is essential. To provide positive, cost-effective, and sustainable dietary change, current evidence supports individual, community and system level strategies (41, 42). The complexity of dietary interventions examined in this narrative review varied from simple behaviour change (e.g., salt substitution which requires only direct replacement of regular salt with salt substitute) to more complex behaviour change (e.g., Mediterranean or DASH diets which require adopting a comprehensive lifestyle approach). Dietary interventions that use behaviour change science are likely to better facilitate improvements in health outcomes (43). The selected reviews rarely considered the complexity of behaviour change required to successfully implement different dietary
strategies, and clinicians should be guided by behaviour change theories when supporting patients to make dietary changes. There is inherent complexity in the variability of dietary behaviors, which are highly individual and influenced by personal (e.g., taste, food preference), social (e.g., familial/cultural preferences) and environmental factors (e.g., ability to obtain, store, prepare and cook foods appropriately) (44). For general practitioners exploring diet change for cardiovascular health with patients, selecting dietary strategies to match their patients’ needs, preferences, access, and social determinants of behaviour may maximise person-centred improvements in diet and health outcomes (45).

Nutrition care is not provided as often as clinically beneficial, and patients expect primary care clinicians to be competent in supporting them to optimise their diet (46). This narrative review shows that several dietary approaches are protective for cardiovascular health, so improving primary care clinicians’ skills to support patient dietary change is worth pursuing. Clinicians can also advocate for positive nutrition policies at the community and system levels (47). This may include identifying misinformation, promoting the relevance and importance of healthy eating, and linking patients to evidence-based, reputable sources of further support, including referral to a registered or accredited dietitian, which is shown to be clinically effective for reducing blood lipid levels (48, 49).

**Implications for practice**

All dietary interventions reviewed showed promising but modest effects on direct or indirect CVD outcomes and may be recommended by general practitioners for cardiovascular risk reduction, though as discussed, there are some caveats to consider. The choice of dietary strategy for patients will depend on preferences and circumstances that may enable sustained behaviour change. Assessments of current weight (for energy deficit strategy), sodium intake (for salt reduction or substitution in hypertensives), and alcohol intake (for high alcohol intake) should be conducted, and Mediterranean diet may be useful whatever the background factors. Crucially, dietary strategies are not mutually exclusive; multiple compatible strategies (for example, sodium substitution and alcohol
reduction) may be applied simultaneously to potentially maximize the benefit to cardiovascular health and absolute risk reduction.
Additional information

Funding Statement

PG and HG received financial support to undertake this review is part of a project commissioned by the Australian National Heart Foundation to summarise evidence for assessing and managing absolute cardiovascular disease risk across a range of risk factors, including diet and its impact on direct cardiovascular outcomes. National Heart Foundation provided feedback on study design, including selection of relevant dietary strategies, but did not contribute to the data collection, analysis, interpretation of data, or writing/submission of this manuscript in any way. LB and KB did not receive any financial or material support for this work. LB is supported by an NHMRC fellowship and is KB supported via LB’s NHMRC fellowship direct research cost support.

Ethical Approval
Not applicable.

Declaration of Competing Interests
The SpiderCite automation tool used to conduct the citation analysis was conceived and developed by PG and other researchers at the Institute of Evidence-Based Healthcare. This is an open-access online tool, and no authors receive any financial gain from this product. PG is funded by an Australian National Health and Medical Research Council - NHMRC Investigator Fellowship APP1080042. LB is funded by an Australian National Health and Medical Research Council - NHMRC Investigator Fellowship APP117346. HG and KB are supported indirectly through these fellowships.

No other potential or actual conflicts of interest to declare.

Acknowledgements
We wish to acknowledge the National Heart Foundation for supporting this work. Thanks to Institute for Evidence-Based Healthcare team who worked on the broader evidence synthesis and especially to Justin Clark for advising the search approach and Melanie Vermeulen for developing the main findings table.
References


