There are many areas of the guidelines in which the evidence cited is either seriously deficient (in that it does not represent the current body of evidence) or poorly interpreted (in that it mechanistically rules studies in or out based on the presence or absence of keywords which appear to be arbitrarily selected).

A cursory look at many of the guidelines reveals serious deficiencies in critical thinking applied to the task. An example can be found in the section on healthy weight which appears to say we are overweight because we consume too many calories. That is perfectly true but it does not illuminate the problem at all. It is the equivalent of saying a person is an alcoholic because they consume more alcohol than others. Also true but it tells us nothing about why that might be the case. The fact that you have chosen not to look into what might be driving the consumption of more calories (it's not too hard, you set it out in the sugar and weight gain section), shows an extraordinary lack of enquiry in the construction of this document.

The guidelines are based on the assumption that the diet consists entirely of whole foods. Based on that, a mild suggestion (for example) to limit added sugar is perfectly acceptable as there are no significant quantities of sugar in whole foods. The reality is however that nobody in Australia eats a diet consisting of whole foods. We all shop in supermarkets where 99% of the products are processed foods and 98% of them have sugar added. The guidelines appear to be designed on the assumption of a society which simply does not exist. To be of any use whatsoever to Australians the Guidelines must be based on what Australians actually eat, not some utopian whole food fantasy scenario.

I have not examined each of you recommendations in detail, but below I have set out some of the difficulties I perceive in the ones with which I am most familiar.

2.2.4 Fruit

The evidence reviews have focused on population studies which show (a slight) benefit to fruit (when combined with vegetable) consumption. But the review ignored evidence from randomised controlled trials that show that increased fruit and vegetable consumption have a negligible impact on plasma cholesterol fractions[1] [2] or anti-oxidants[3] [4] (the two primary hypotheses for a mechanism for reduction in CVD risk).

Given this, it seems odd to assign a grade of B to the evidence. I could understand that if there not higher quality evidence which tended to contradict the population studies, but in cases (such as this) where such evidence exists it seems foolhardy to grade the evidence so highly. On this basis the evidence is Grade D (at best) and probably merely interesting (at least in regard to fruit consumption).

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[1] John JH, Ziebland S, Yudkin P, Roe LS, Neil HAW. Effects of fruit and vegetable consumption on plasma antioxidant concentrations and blood pressure: a randomised controlled trial. Lancet 2002;359:1969-1974.
[2] Zino S, Skeaff M, Williams S, Mann J. Randomised controlled trial of effect of fruit and vegetable consumption on plasma concentrations of lipids and antioxidants. BMJ 1997;314:1787-1791
[3] Clarke R, Armitage J. Antioxidant vitamins and risk of cardiovascular disease. Review of large-scale randomised trials. Cardiovasc Drugs Ther 2002;16:411-415
[4] Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of antioxidant vitamin supplementation in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet 2002;360:23-33.

Indeed, a recent (2009) review[5] of the evidence from the authors of the primary meta-study relied on by your evidence report says:

> *This Review summarizes the evidence for a relationship between fruit and vegetable consumption and the occurrence of coronary heart disease…Most of the evidence supporting a cardioprotective effect comes from observational epidemiological studies;* ***these studies have reported either weak or nonsignificant associations****. Controlled nutritional prevention trials are scarce and* ***the existing data do not show any clear protective effects of fruit and vegetables on coronary heart disease****. Under rigorously controlled experimental conditions, fruit and vegetable consumption is associated with a decrease in blood pressure, which is an important cardiovascular risk factor. However,* ***the effects of fruit and vegetable consumption on plasma lipid levels, diabetes, and body weight have not yet been thoroughly explored****. Finally,* ***the hypothesis that nutrients in fruit and vegetables have a protective role in reducing the formation of atherosclerotic plaques and preventing complications of atherosclerosis has not been tested in prevention trials****.* ***Evidence that fruit and vegetable consumption reduces the risk of cardiovascular disease remains scarce*** *thus far.*

That appears to say that there is no real evidence of a protective effect. How can you then proceed to make a Grade B recommendation that increased fruit consumption is protective of heart disease (or anything else)?

3.1 Fats

You make a recommendation to include foods which contain unsaturated fats. However the evidence provided in the evidence report relates only to the benefits attributable to Omega-3 Fats[6] (and then only in relation to dementia).

The dominant food source of unsaturated fats in the Australian diet is Omega-6 seed oils. But you appear to completely ignore this reality in the recommendation. Given this, it would appear prudent to either remove the guideline completely or ensure that you specifically highlight your evidence only supports a recommendation concerning omega-3 fats (and then, it would appear only for a reduced risk of dementia).

You make a recommendation to reduce consumption of saturated fats, however the evidence report does not include a single evidence statement suggesting they are harmful:

You found **no evidence** of an association between:

- Saturated fats (or total fat) and CVD
- fat intake and weight gain
- fat intake (or type of fat) and type II diabetes

[5] Dauchet L., Amouyel, P., and Dallongeville, J. (via MedScape). Fruits, vegetables and coronary heart disease. Nature Reviews Cardiology, 6 (2009): 599-608. doi: 1011038/nrcardio.2009.131

[6] 12.1 CVD – Neutral, 12.5 Cancer – Neutral, 12.8 Dementia – reduced risk

- fat intake and hypertension
- fat intake and cancer

I know you make it clear that the evidence report is only covering new evidence and that you rely on your previous research for your assertion, but even the most recent large scale meta-analysis[7] (performed by the Cochrane collaboration) arrives at the same conclusion as your researchers (but this time with regard to the totality of the evidence to 2011).

They say there was no significant effect of any dietary fat intervention on total mortality or CVD mortality. There was also no effect on myocardial infarction, stroke, cancer deaths (although there was evidence that some interventions involving substitution of saturates with polyunsaturates increased cancer deaths) or diabetes.

Given the total lack of evidence, how do you justify recommendations for lower total fat intake and lower SFA intake?

There is a significant line of studies[8] which suggest that increased Omega 6 polyunsaturate consumption increases cancer risk in humans. Your evidence report does not however address this question at all. This appears to be a major deficiency in the recommendations and should be directly addressed.

3.3.2 Sugar

By splitting your evidence analysis into sugar sweetened drinks, juices and other added sugar you effectively divide the evidence set into cohorts which are too small from which to draw a definitive statement. The only active ingredient in soft drink (and juice) is sugar. It is nonsensical to treat soft drink and juice studies as a separate niche.

Taken together, even the very limited selection of studies reviewed by your team raises very real concerns for sugar consumption (even after excluding naturally occurring sugars). Your evidence report for example finds:

- "three of the four cohort studies reviewed showed positive associations with **fructose and pancreatic and colo-rectal cancer**"[9] and
- "Regular consumption of soft drinks may play an **independent role in the development of pancreatic cancer**."[10]

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[7] Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore H, Davey Smith G. Reduced or modified dietary fat for preventing cardiovascular disease. Cochrane Database of Systematic Reviews 2011, Issue 7. Art. No.: CD002137. DOI: 10.1002/14651858.CD002137.pub2

[8] For a recent example see: Emily Sonestedt, Ulrika Ericson, Bo Gullberg, Kerstin Skog, Håkan Olsson, Elisabet Wirfält. Do both heterocyclic amines and omega-6 polyunsaturated fatty acids contribute to the incidence of breast cancer in postmenopausal women of the Malmö diet and cancer cohort? Int J Cancer. 2008 October 1; 123(7): 1637–1643. doi: 10.1002/ijc.23394

[9] 14.1

[10] 14.1 and Soft Drink and Juice Consumption and Risk of Pancreatic Cancer: The Singapore Chinese Health Study: Cancer Epidemiol Biomarkers Prev February 2010 19; 447

- "Total sweetened beverages (predominantly sucrose-based) were **strongly related to bladder cancers** in the one case-control study examined"[11]

- "After **24 days** and with no significant difference in total energy intake, the authors reported **a significant difference in weight change** in the group consuming the high carbohydrate/low GI diet (-0.27 kg, SE 0.3) compared to the group consuming the high sucrose diet (+0.84 kg, SE 0.3, P<0.02 between groups)."[12]

- "4/8 fruit juice studies found **increased risk of weight gain**"[13]

- "two cohorts and one clinical trial [of fruit juice consumption] found **increased risk of weight gain** in children"[14]

You have not attempted to specifically address the question of fructose (added or not). That is an oversight bordering on negligence given the overwhelming literature now being produced around the subject.

There was sufficient evidence for the American Heart Association to issue a precise warning[15] on sugar (and in particular fructose) in 2009 and even to prescribe a maximum daily allowance of 9 teaspoons of added sugar per day per adult male. The fact that your review has chosen to completely ignore this position statement (and the science it cites) as well as the significant body of human trials on fructose feeding between 2009 and 2012 is extraordinary.

Even on the basis of the evidence you do cite, the inclusion of a recommendation to 'limit consumption' of foods containing added sugars is appallingly weak. It should say 'sugar consumption should be avoided and in any event be no more than 9 teaspoons per day for an adult male.'

[11] 14.1
[12] 14.3
[13] 15.2
[14] 15.2
[15] Dietary Sugars Intake and Cardiovascular Health Circulation, Vol. 120, No. 11. (15 September 2009), pp. 1011-1020, doi:10.1161/CIRCULATIONAHA.109.192627 by Rachel K. Johnson, Lawrence J. Appel, Michael Brands, et al.