Author’s response to reviews

Title: Association between diet quality, dietary patterns and cardiometabolic health in Australian adults: a cross-sectional study

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Thank you for the opportunity to respond to the reviewer comments. Please find below detailed responses to the Reviewers’ comments.

Reviewer reports:

Reviewer #1: This is an interesting paper reporting on the association between diet and markers of cardiometabolic health. Strengths of this study pertains to the use of several methodological approaches to characterize the "whole diet" of participants including a rather novel RRR approach to derive a posteriori dietary patterns and an a priori approach using the Dietary Guideline index (DGI), both approaches being used in a same representative population.

Please find my comments as follows:

Abstract

-L3: please provide a short explanation of the differences between diet quality and dietary pattern when first mentioned in the abstract (improve understanding for readers)

Response: Additional text has been added to line 3 as follows: “Diet quality indices score dietary intakes against recommendations, whereas dietary patterns consider the pattern and combination of dietary intakes.”

-L10: " 'the' Dietary Guideline Index (DGI)"?
Response: ‘A’ has been replaced with “the”

-L11-12: "using fiber density, SFA: PUFA and total sugars intake as intermediate markers." => not clear, seems to refer to DGI too

Response: This has been revised in lines 10-13 as follows: “Diet quality was estimated using the Dietary Guideline Index (DGI). Dietary patterns (DPs), derived using reduced rank regression, were estimated using fiber density, SFA: PUFA and total sugars intake as intermediate markers.”

-L13: define DP abbreviation

Response: Dietary patterns are now defined at first mention in line 11.

-L31: "casual" should be "causal"? (same goes L454)

Response: Thank you. This has been revised accordingly.

Introduction

-L37-38: "The underlying biological pathways through which these diseases are mediated include markers of poor cardiometabolic health" => this sentence is not clear.

Response: This sentence has been revised as follows in lines 38-40: “The development of these conditions is mediated through multiple biological pathways, such as elevated blood pressure and levels of triglycerides (TAG), total and LDL cholesterol and glucose [2].”

-L41: references [3,4] provided to justify the need for studies on diet and references [3,5] associated with the statement "research to date" are from early 2000s. More recent references would be appreciated

Response: The following references have been revised:

-Line 42 ref 3-5:


-Line 44 ref 3, 6, 7:


-L45: references [7-9]. Please also provide references from another group

Response: The following references have been revised in line 46 ref 9-11:


-L46: by "cluster analysis", did you mean dietary patterns derived from factor analysis or principal component analysis? Then cluster is not the appropriate term here

Response: Thank you, “cluster” has now been replaced with “factor” in line 47.
L62-63: "RRR may better predict risk of disease than purely data-driven DP methodologies" => do you have a reference for that? Is it still true when the dependent variables in the RRR are nutrients and not biomarkers or outcomes as in your study?

Response: The following two references have been added to line 64 which demonstrate this using either nutrients of biomarkers as the intermediate markers. The first study includes RRR derived dietary patterns using nutrients and the second study uses biomarkers. In both examples, RRR-derived dietary patterns, but not PCA-derived dietary patterns, were associated with the outcomes of interest.


Methods

-The method section is very long with a lot of details on the setting of the NHS, NNPAS and NHMS studies or the procedures used to collect data. Please select what is really relevant to the present manuscript. Some details may not be necessary in this manuscript and should be either replaced by a reference to a previous publication or put to some extent in supplementary material.

Response: This section has been revised to remove details on the number and percentage of fully or adequate responders in the NNPAS and NHMS as this information is described elsewhere (ref 27).

-L80: were these 14,363 private dwellings included in the 15,565 adequate respondents of the NHS? Overall it is not clear how the samples from the NHS, NNPAS and NHMS relate to one another.

Response: The NHS and NNPAS are separate surveys and do not contain the same participants. The third survey, the NHMS invited participants to participate from both the NHS and NNPAS. Additional text has been added to lines 76-79 to add clarification: “The AHS consists of two separate surveys (the National Health Survey [NHS] and the National Nutrition and Physical Activity Survey [NNPAS]) and the National Health Measures Survey (NHMS), a third component in which participants from both surveys were invited to participate [27].”

-L91: there was an important selection of your sample (from 12,153 to 2121). How representative of the Australian population was your analytical sample?
Response: As shown in Figure 1, 2812 are excluded as they are <19 years of age (i.e. children and adolescents). Of the 9,341 adults, 5,570 were excluded as they did not provide biochemical data. Of the 3,771 adults who provided biochemical data, 921 were excluded as they are pregnant or breastfeeding or missing a second day of dietary data. Of the 2,850 eligible adult participants, 729 were excluded for missing outcomes or covariates.

The representativeness of our study estimates was maximised by using survey weightings that were calibrated by the Australian Bureau of Statistics to account for probability of selection (due to the random nature of sampling) or non-response associated with the biochemical data (see 4363.0.55.001 - Australian Health Survey: Users’ Guide, 2011-13 www.abs.gov.au/ausstats/abs@.nsf/mf/4363.0.55.001). In addition, the authors have added text to lines 436-440 to acknowledge missing data as a limitation to the generalisability of our findings.

-L106: was there a correction applied for weight if people did not take out their shoes and heavy clothing?

Response: Interviewers encouraged respondents to remove their shoes and any heavy clothing, e.g. jumpers, before they took measurements, however, this was voluntary, and may not have occurred in some cases. Interviewers were not required to record if they thought clothing may have impacted significantly on measurements. The following text was added to line 103-106: “Subjects were encouraged to remove their shoes and any heavy clothing prior to having measurements taken, although this was not compulsory, and no correction was applied if they did not.”

-L112: please define ABS

Response: ABS has now been replaced with “Australian Bureau of Statistics”.

-L112-113: how was LDL cholesterol estimated for people with TAG>4.5mmol/L then?

Response: If a participant had a TAG level of ≥ 4.5 mmol/L then LDL cholesterol was not estimated in these individuals and instead they were coded as having missing data by the Australian Bureau of Statistics. These individuals were thus not included in our analytical sample as we included only individuals with complete data for all exposures, outcomes and covariates (lines 227 and lines 232-233).

-L114: "without the need for fasting" => only participants that were fasting were included in this study to have data on LDL, TAG and glucose

Response: Fasting was not required for the testing of Apolipoprotein B (g/L), HbA1c (mmol/mol) and total and HDL-cholesterol. When testing for LDL, TAG and glucose fasting was required and only fasted data were used. This has been added to lines 232-233.

-L118-119: "Impaired fasting plasma glucose was defined as > 6.0mmol/L and <7.0 mmol/L" => what about participants with fasting plasma glucose >7.0 mmol/L?
Response: Participants with fasting plasma glucose >7.0 mmol/L were not included in this definition of impaired fasting glucose as levels >7.0 mmol/L indicated diabetes. This variable was used for the descriptive statistics on the prevalence of impaired fasting plasma glucose in the sample population and thus participants were classified as either having impaired fasting glucose (> 6.0mmol/L and <7.0 mmol/L) or not (< 6.0mmol/L or >7.0 mmol/L). For the purposes of this study, we estimated diabetes prevalence using the definition of self-reported diabetes diagnosed by a doctor.

- L125-126: “Data on anti-hypertensive and lipid lowering medication were not recorded” => this was mentioned as a limitation in the discussion but how do you think this could have affected your results?

Response: The following sentence has been added to line 453-455: “Thus, we cannot discount bias associated with incongruences between blood pressure data before and after medication use and any associated behavioural changes.”

- L129-132: the method to derive the cardiometabolic risk score indicates that this score is actually dependent on your population since you use the mean and sd to normalize your values, then the way people are classified may depend on the overall health status of your population and not on absolute criteria. Why not use absolute thresholds to define "abnormal levels" and derive a risk score. Why not use the metabolic syndrome outcome which gather multiple cardiometabolic risk factors?

Response: While scores have been normalised, we did not classify individuals using this score – the aim was to develop a continuous risk score. The purpose of normalising was to put all risk factors on a same scales rather than adding up raw data, which would result in a non-weighted score.

We acknowledge that a categorical score based on absolute criteria would be of interest. However, the authors were primarily interested in understanding the magnitude of the effect of diet on cardiometabolic outcomes. We aimed to take into account the fact that cardiometabolic risk increase risk across the spectrum of any given marker (e.g. glucose), not just at high levels. Moreover, using a continuous score increased the statistical power to investigate our aim.

Using this risk score allowed us to combine multiple, related risk factors into one measure of risk. Given that we might expect diet to marginally impact each of these factors, combining these outcomes may be a useful way to examine the impact of diet on cardiometabolic health.

- L151-154: the description of the DGI score could be more detailed for some items that may appear as surprising or unclear (e.g. cereals without distinction between refined/whole grains or meat in "recommended" components and unsaturated fat in "discouraged", "extra sugar").

Response: The following text in lines 152-158 has been revised: “Dietary intakes of individuals, based on an average of two 24-hour recalls and brief questionnaire items, were scored according to ten recommended dietary components (food variety, fruit, vegetables, cereals [total cereals and proportion that is wholegrains], meat and alternatives [total lean meat and alternatives and
proportion that is lean], dairy and alternatives and fluid intake [total beverage and proportion that is water] and six dietary components that should be limited (discretionary foods, SFA, unsaturated fat, added salt, added sugars and alcohol).”

A list of items in the DGI and how they were scored is provided as supplementary material (Supplementary Table 2). References to two more detailed manuscripts are provided (ref 35 and 36). These manuscripts focused entirely on diet quality and thus had more space in the manuscript to elaborate on the detailed methods of the DGI. Given these additional resources, we believe that the revised text above is sufficient.

-L191: what do you mean by "standardised food group intakes"?
Response: Z-scores were generate for intakes of all food group. This has been added to line 195.

-L196: no description of DP3?
Response: As detailed in the results in line 268, DP explained less than 10% of the variation in response variables and so it was not used to evaluate associations with health outcomes. The following text has been added to lines 200-202: “As the third DP explained less than 10% of the variation in response variables it was not further investigated and no sensitivity analysis was conducted.”

-L206: "currently on a diet to lose weight" is duplicate
Response: Thank you for pointing this out. “lose” has been changed to “gain”.

-L228-229: "dieting" and "atypical dietary intake on day of reporting" => how were these variables distributed among normal weight and overweight/obese people? Among healthy/unhealthy people? There may be a social desirability bias in these variables especially for the "atypical dietary intake" and these variables may act as a proxy for your outcomes. How were your results without including these variables? Or without including participants that reported an atypical day?
Response: The authors agree that these variables are important in our associations with our outcomes of interest and was why they were chosen as covariates in the regression models. Regarding distribution of the dieting variable, in obese individuals compared with non-obese individuals, the proportion of individuals currently on a diet for weight loss reasons was slightly higher (8% vs 3%), while the proportion of individuals who reported not currently being on a diet was lower (82% vs 91%). Regarding distribution of the atypical intake variable, in obese individuals compared with non-obese individuals, the proportion of individuals reporting that their intake was typical of their usual intake was higher (79% vs 71%).

Omission of the variables on dieting and atypical diet from the regression model did not change the pattern of significant results. For example, the association between DGI and BMI was: coef -0.017 SE 0.007, P-trend=0.020.
L234-235: were the weightings applied to correct for the differences between the distribution of sociodemographic characteristics in your sample compared to the Australian population?

Response: The following text has been added to line 244-246: “Survey weightings that were calibrated against population benchmarks (i.e. age, sex and area of usual residence) were used to account for the complex survey design.”

Did you test models in which your outcome variable would be "abnormal value, yes/no" for the studied biomarkers instead of the continuous value?

Response: No, we did not include binary or categorical outcomes in our investigation of associations with dietary patterns or diet quality. This clarification has been added to lines 233-235: “Markers of cardiometabolic health markers were treated as categorical or binary outcome variables for the purpose of descriptive statistics and as continuous variables when evaluating associations with dietary intake.”

As described on page 5 in this rebuttal, we made this decision based on research that suggests that dichotomising variables limits the statistical power to detect associations. In addition, dichotomising variables increases the risk of a positive result being a false positive, underestimates the extent of variation between groups (where individuals close to but on opposite sides of the cut point are characterised as being very different rather than very similar) and conceals any non-linearity in the relation between the variable and outcome. (Altman & Royston BMJ 2006; 332:1080).

Results

L242-244: the comparison should be made between the analytical sample and the excluded participants

Response: This has been revised accordingly in lines 254-256: “Characteristics of the omitted sample with the analytical sample were broadly similar, although slightly more adults who were middle aged, highly educated and living in major cities were included in the analytical sample (Supplemental Table 4).”

L246: should be high LDL-cholesterol

Response: Thank you, this has been revised accordingly.

L246: how many participants had a plasma glucose above 7?

Response: 86 individuals (4%) had a plasma glucose above 7 mmol/L.

L254-256: even if significant, some correlations were very small and don't seem very relevant to characterize your DP (e.g. DP1 and inverse correlation with total sugars, DP2 and positive correlation with fiber density or SFA: PUFA)
Response: The authors acknowledge that total sugars can be challenging to interpret as it includes sugars from natural sources (e.g. pome fruits) and added sugars (e.g. fruit drinks and chocolate). However, an inverse correlation of DP1 with total sugars is consistent with intakes of fruit drinks and chocolate, as shown in Table 2. In addition, DP2’s positive correlation with fiber density is consistent with direction associations with high intakes of fruits, such as pome fruit, tropical fruit and stone fruit.

Table 1: the percentage explained in food intakes were very small and could deserve some comments (normal for RR derived DP?). How do the explained variation in responses (total) and in each individual response relate to one another? For example DP3 explains only 7.99% in total but have higher explanatory power for each component compared to DP1 and 2.

Response: The explained variation of total responses is on a different relative scale compared to its components and hence cannot be compared. The comparison should be made between DP1 and DP2, not within DPs. The explained variation is similar to what would be expected from RRR. For example, the explained variation in food intakes was 3-5% in a study by Jacobs et al. (Curr Dev Nutr 2017, 1 (5) e000620).

Table 2: is 448 for whole grain bread in T2 (DP1) the correct value?

Response: Thank you, this has been revised to “45”.

-L279: "were more physically active"

Response: This has been revised accordingly.

-I would like to see a comparison between DGI scores and DP1 DP2 scores (for example, scores for DP1 and DP2 in tertiles of DGI and maybe some information about the correlation/% explanation of the DGI to the response variables used in RRR (Fibre density, SFA: PUFA, Sugar))

Response: The authors agree that more comparison between DGI and DPs would be beneficial. The following information has been added to Table 3:

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All</th>
<th>Diet quality</th>
<th>P-trend2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
<td>T3</td>
</tr>
<tr>
<td>Dietary pattern 1</td>
<td>0.07 ± 0.04</td>
<td>-0.58 ± 0.06</td>
<td>0.07 ± 0.07</td>
</tr>
<tr>
<td>Dietary pattern 2</td>
<td>0.01 ± 0.04</td>
<td>-0.31 ± 0.06</td>
<td>0.10 ± 0.05</td>
</tr>
<tr>
<td>Fibre density, g/MJ</td>
<td>2.87 ± 0.04</td>
<td>2.23 ± 0.04</td>
<td>2.85 ± 0.06</td>
</tr>
<tr>
<td>SFA: PUFA</td>
<td>2.69 ± 0.05</td>
<td>2.89 ± 0.09</td>
<td>2.72 ± 0.08</td>
</tr>
<tr>
<td>Sugar, %E</td>
<td>19.0 ± 0.24</td>
<td>18.2 ± 0.40</td>
<td>19.5 ± 0.37</td>
</tr>
</tbody>
</table>

The following text has been added to the results:
Lines 291-292: “Higher DGI scores were associated with higher DP-1 and DP-2 scores, greater fiber density and lower SFA: PUFA ratios.”

Discussion of the comparability of both DGI and DP-1 is included in the discussion in lines 365-368 and 482-484.

Discussion

-L352: "healthier" DPs => do you consider that DP2 is a healthy DP? DP2 is positively correlated with sugars and seem to have contradictory effects on health with lower HDL-C (less healthy) and lower DBP (healthier). Please discuss these results as well.

Response: We have revised this sentence to include reference to DP1 only, as we do not consider DP2 a healthy dietary pattern given its complex pattern of food group intakes and associations with outcomes.

-L358: why "other"?

Response: This has been revised to “non-anthropometric”

-L357-358: "Our findings also suggest that there may be differing mechanisms through which dietary components influence other markers of cardiometabolic health." => please elaborate on this. It is not very clear how your findings allow to conclude this (also found in the abstract, l403-404 and l449-450)

Response: This has been removed and revised as the mechanisms of the association between diet and cardiometabolic health were outside of the scope of this study.

-L369-370: "The inconsistency of associations with markers of cardiometabolic health may be partly attributable to differences in the ethnicity of the sample population" => what do you mean by that? What is the ethnic diversity in your sample? Was the ethnicity of your sample different from previous studies? (of note that you observed similar results with a study performed on Hispanic adults). Other hypotheses for the inconsistencies may also be confounding due to the cross-sectional design with differences between countries in metabolic health surveillance and thus awareness of metabolic abnormalities, diet recommendations following detection of abnormalities etc.

Response: We chose to highlight the potential role of ethnicity in our results given that Ford et al. (ref 49) identified differences in prevalence of poor cardiometabolic health according to ethnic groups (e.g. 31% of Caucasians had hypertriglyceridemia compared with only 18% of African Americans). Given that our sample was predominantly (80%) born in Australia or another English language speaking country, our ethnic diversity is likely to lower (information on ethnicity was not available).
These data have been added to Table 3 and the following text has been revised in lines 380-386: “In line with a recent cross-sectional study of multiple diet quality scores in US women (predominantly Caucasian) [17], but in contrast with findings for the 2005 HEI (50% Caucasian) [48], diet quality was not associated with total or HDL-cholesterol. Similar inconsistencies are evident for other markers, such as TAG and HbA1c [48]. The inconsistency of associations with markers of cardiometabolic health may be partly attributable to differences in the ethnicity of the sample population [49], given that 80% of our sample were born in Australia or another English speaking country. However, it is likely to largely be due to methodological differences, such as reverse causation given the cross-sectional design, the method of assessing diet quality, choice of covariates and variation in sample sizes and resulting statistical power.”

-L376-377: I agree that few studies have used RRR methodology but you can still compare the DP you derived from RRR to other DP obtained with unconstrained methods, especially since DP1 can be compared to "healthy" pattern observed in most studies

Response: This discussion section has been expanded to include further examples and comparisons.

-Lines 393-414: “Of these studies, some have used biochemical response variables, such as cholesterol, to derive associations between DPs and markers of cardiovascular health [25] and risk [13]. Associations observed between DP-1, consistent with a ‘healthy’ DP, and anthropometric outcomes are comparable to other studies [20, 25, 54-56]. In a recent analysis of 10,008 individuals from the Multiethnic Cohort, a RRR-derived DP (using four biomarkers as response variables) and low in fruit drinks and white rice and high in whole grains and fruits was inversely associated with BMI [13]. Few studies have use nutrient intakes as response variables. In a longitudinal study of 2,037 Swedish adults, an ‘unhealthy’ RRR-derived DP characterized by some similar response variables to our study (high DED, SFA and low fiber-density) was associated with greater adiposity, cholesterol, TAG, SBP and DBP but not with CVD endpoints [54]. However, comparability of results was limited as this study was conducted in obese individuals only, who may be more metabolically sensitive to an ‘unhealthy’ diet [57]. Recent data from a AHS study showed that a ‘healthy’ DP, derived using factor analysis and characterized by high intakes of wholegrains and fresh fruit and low intakes of take-away foods and soft drinks, was associated with higher odds of having a healthy metabolic profile [20]. Recent data from a AHS study showed that a ‘healthy’ DP, derived using factor analysis and characterized by high intakes of wholegrains and fresh fruit and low intakes of take-away foods and soft drinks, was associated with higher odds of having a healthy metabolic profile [20]. Similar findings for a ‘healthy’ DP were observed in a national longitudinal study in Chinese adults [55] and a representative community sample of Lebanese adults [56] but were mixed in a prospective study of UK males [58]. Our DPs were derived to explain the maximum variation in DED, SFA: PUFA, and total sugars; it is likely that a DP that explained other nutrient intakes, as well as a DP derived using other methodologies, may show different associations with cardiometabolic health.”

-L377-380: this is not clear. What was used in other RRR studies? If other studies used cardiometabolic markers as response variables then an association between their RRR-derived DP and the same cardiometabolic markers would just be a confirmation. In addition, what really matters is the resulting DPs and the food characterizing them.
Response: As detailed in the previous response, this section of the discussion has been revised to include more examples (see lines 393-414).

-L391: even if DP2 was positively correlated with all response variable, the correlation was stronger with sugars

Response: While we agree that the correlations in DP2 appeared strongest for total sugars, statistically there was no difference and therefore we chose not to comment on this in the manuscript.

-L396: not clear what is counterintuitive here

Response: The word “counterintuitive” has been removed.

-L403-407: this paragraph is unclear. As already mentioned, in my opinion your results does not really allow to conclude about "differing mechanisms" as there was not really an evidence for association of the same type of foods with completely different outcomes in your study. I agree that food contains nutrients that may have differential associations with cardiometabolic health but that does not really justify a whole diet approach. The whole diet approach is rather justified by the fact that foods are not consumed individually and thus some interaction between nutrients consumed together may occur.

Response: All mentions of mechanisms have been removed and revised. Specifically:

-Line 30-32: “Findings support the need for comparison of whole-diet based methodologies that account for interactions between foods and nutrients.”

-Line 493-495: “Findings support the need to compare across multiple whole-diet based methodologies, which take into consideration the interaction between foods and nutrients consumed together.”

-L406-407: this sentence is not clear + ref 51 is about cardiovascular outcomes and not CVD risk markers

Response: This sentence has been removed.

-L413: is your sample still representative after the selection you made?

Response: This paragraph has been revised to acknowledge that the generalisability of the sample may have been limited by non-response bias. Lines 435-440 now reads as follows: “This study was conducted in a large, nationally representative survey of Australian adults. Although the generalisability of our sample may have been limited by non-response bias associated with those who volunteered to provide biological samples, our analyses used survey weightings that were specifically designed to account for such bias. Moreover, there was minimal difference in characteristics between the omitted sample and the analytical sample (Supplementary Table 4).”
L417: "data not shown"=> these data are found in supplementary table 4

Response: Thank you, this has been revised accordingly.

L418: you used 2 methodologies on the same population which allow comparison but I would have liked to see more direct comparison between the DGI and the RRR DPs

Response: The authors addressed this point on page 6-7 of this rebuttal by adding additional data to Table 3. The following text has been also been added to the results:

Lines 291-292: “Higher DGI scores were associated with higher DP-1 and DP-2 scores, greater fiber density and lower SFA: PUFA ratios.”

L425-426: do you know if people were aware of their cardiometabolic health?

Response: The following text has been added to lines 449-451: “Given that some measures of cardiometabolic health were self-reported, some individuals may have been aware of their poor cardiometabolic health and may have changed their diet as a result.”

L431: did all participants provide their 24h recall at the same period of the year? Would a "season" effect be relevant for food type intakes in the Australian population?

Response: Dietary intake data were collected throughout the year between 2011 and 2012. Detailed analysis of the impact of season on dietary intakes in the NNPAS is not available. Although a seasonal adjustment was also incorporated into the person weights in the NNPAS, we acknowledge that the timing of 24-hour recalls may limit their ability to capture usual intakes and variations by season, so this has been added as a limitation in lines 458-465: “Although the timing of 24-hour dietary recalls may limit their ability to capture usual intake and seasonal variations in dietary intakes, our use of two 24-hour recalls offers an advantage over previous studies based on one day of dietary recall [20] and our research has demonstrated that RRR DPs derived from the average of two days are comparable to those derived using usual intakes [14]. Any seasonal impact on DPs may have influenced smaller food groups, such as stone fruits, but is likely to be minimal in larger food groups, such as brassica vegetables, and in the DGI. Moreover, a seasonal adjustment was incorporated into the survey weighting in the NNPAS [27].”

L436-438: This sentence gives the impression that no prospective studies already investigated diet quality in relation to cardiometabolic health or diabetes incidence, which is not accurate. Please revise this sentence.

Response: This has been revised as follows: “Prospective studies that consider type 2 diabetes and cardiovascular disease incidence, and that compare both diet quality and DP methodologies within the same population are needed.”

L440-445: Overall, the use of RRR methodology is original and provide additional insights compared to classic unconstrained approaches. However, I am not sure how generalizable the
results are in a context of policy development since the DP derived from RRR are constrained with specific objectives (in your case specific nutrients) which makes the interpretation difficult in a broader public health context.

Response: We have added text to the discussion to acknowledge the limitations of RRR:

-Line 466-474: “Limitations of RRR should also be acknowledged. First, although the food groups are based on AUSNUT 2011–13, the number and definitions of the food groups used in this study may have affected the derived DP. Second, although our choice of response variables was based on published literature the use of different response variables may have resulted in a different DPs. Third, we generated and fitted RRR DP in the same data set. To rule out any effect of over-fitting and to show generalizability of RRR DP, future studies should consider deriving and applying DP in independent data sets.”

In addition, the interpretation of the DPs is not easy: DP don't really look like "normal" diets people can identify to. Maybe some discussion about the interpretation of the DPs should be added

Response: We agree that DP-2 is not easily interpretable and have discussed this in lines 415-421. DP-1 was characterised by being high in fruits, vegetables, wholegrains and nuts and seeds and low in high-added sugar and high-fat foods and beverages (i.e. fruit drinks and high-fat milk and cream). We believe that DP-1 is comparable to many ‘healthy’ DPs identified in the literature, including the Mediterranean diet and the DASH diet. We have also added discussion on the interpretation of DPs derived using RRR:

-Lines 411-414: “Our DPs were derived to explain the maximum variation in DED, SFA: PUFA, and total sugars; it is likely that a DP that explained other nutrient intakes, as well as a DP derived using other methodologies, may show different associations with cardiometabolic health.”

-Lines 470-472: “Moreover, RRR derives DPs that closely reflect nutrient intakes or intermediate markers, which may result in DPs less consistent with behavioural patterns compared to factor or cluster analysis.”

-L444: "evaluate"

Response: Thank you, this has been revised accordingly.

-L448: "healthier DPs" => DP1 may be considered as a healthy DP but DP2 is less interpretable

Response: The authors agree that DP2 is not “healthy” as so this sentence has been revised to only refer to DP1 only.

Reviewer #2: This is a well written paper. The RRR is a novel method to derive dietary patterns and the findings could interest readers who are exploring this method. However, the interpretation of the RRR derived dietary pattern remains a challenge.
1. It is not clear whether DP2 is healthy or unhealthy (e.g. high in fibre density and fruits, but also high in sugar and non-wholegrain cereals), which may explain why the associations with outcomes are conflicting. Although the authors have briefly explained; the Discussion could benefit from more in-depth discussion on what could cause the conflicting findings, with possible mention of the limitation of the RRR method.

Response: Limitations of the RRR method have been added to the discussion:

-Lines 411-414: “Our DPs were derived to explain the maximum variation in DED, SFA: PUFA, and total sugars; it is likely that a DP that explained other nutrient intakes, as well as a DP derived using other methodologies, may show different associations with cardiometabolic health.”

-Line 466-474: “Limitations of RRR should also be acknowledged. First, although the food groups are based on AUSNUT 2011–13, the number and definitions of the food groups used in this study may have affected the derived DP. Second, although our choice of response variables was based on published literature the use of different response variables may have resulted in a different DPs. Third, we generated and fitted RRR DP in the same data set. To rule out any effect of over-fitting and to show generalizability of RRR DP, future studies should consider deriving and applying DP in independent data sets.”

2. The RRR derived DP is rather specific e.g. DP1 suggest higher intake of apples, pears and wholegrain bread, which makes translation to practical dietary recommendations rather difficult. Could the authors elaborate on the public health relevance of the derived DPs?

Response: The authors have now included a full list of factor loadings for both DP-1 and DP-2 as a Supplementary table (Supplementary Table 5). This provides a more comprehensive overview of the DPs and highlights that while certain food groups characterised the DPs more than others, the resulting associations with cardiometabolic outcomes was due the combined effect of all included food groups in the DP. The overall pattern of DP-1 is consistent with a ‘healthy’ dietary pattern, which translates to public health messages that promote consuming fruits, vegetables, nuts and seeds, wholegrains, beans and legumes and limiting processed and high added sugars, salt and fat foods and alcohol. Moreover, dietary guidelines are developed using the totality of DP literature, rather than just one study, thus requiring a multitude of studies to confirm which DPs are important.

The following text has been added to the discussion to elaborate on the public health implications of our findings:

-Lines 482-484: “Both diet quality and DP methodologies (DP-1) support healthy eating initiates to improve cardiometabolic health that centre on diets rich in fruits, vegetables, wholegrains and lean meats and/or alternatives and low in processed foods and alcohol.”
3. While greater adherence to DGI and DP1 were associated with lower BMI and WC, the association between DGI and plasma glucose was not replicated with DP1. Detailed discussion on the differences between DGI and DP1 would be helpful.

Response: The authors are reluctant to draw conclusions from the results for a significant association between DGI and plasma glucose. This result may be due to chance given that the strength of the association is considerably weaker following adjustment for covariates and the second tertile of DGI was positively associated with glucose levels. The following text has been added to the discussion:

-Lines 378-379: “However, given the strength of the association between DGI and glucose levels observed in our study, we cannot discount the possibility of this being a chance finding.”