Comments of the SACN draft report on Carbohydrates and Health.

From Geoff Livesey, Independent Nutrition Logic Ltd*

Preamble

I feel for others who have worked so hard to get so far.

It pains to bring them messages of discontent.

Hope is hope for a better report to come.

And for funding to match

the hard work that

remains to be

done.

The draft Report makes only a start towards understanding the relations between food carbohydrates and health, an activity that has wide ranging applications: it is highly important for the wellbeing of individuals, the economy of health provision, the effectiveness of the UK workforce, our gross domestic product, and the sustainability of our pensions. The draft Report content shows a wide coverage of effects and associations for food carbohydrates and health among persons ostensibly healthy or near healthy, with results considered to be relevant to the UK population. It is evident that a considerable amount of work has been undertaken to cover the broad range of carbohydrates about which questions are asked: On this the review team can be congratulated. The large effort, however, appears not to be without consequences for the accuracy and quality of the overall project and the draft Report. Inaccuracies, and quality of execution and reporting of the draft Report affect not only the highly important applications mentioned, but also reflect poorly on the quality of UK science, with potential to affect internal and external investments, the direction of future health-related research, and the confidence people have in its institutions and professions.

There are a number of weaknesses that are problematic for acceptance of the Report's results, conclusions and recommendations, and which strongly suggest inadequate funding and/or time allocation to enable satisfactory quality and completion. Thus there is a lack of necessary depth,

methodological knowhow and inquisitiveness, occurrences of unnecessary subjectivity among procedures adopted, undue speculation, and statements indicative of possible reporting bias. All these problems beset scientific manuscripts with a high rate of rejection. Moreover, the relevant health science literature covered is not up-to-date and methodology used is not desirably comprehensive. In addition, the draft Report is not presented in a useful, easy to read or helpful format, with too much information about literature search (which is repetitive, tedious to read and mostly unnecessary to state) and not enough about the context or conditions of peoples to which the results apply. Among all this it seems there is a lack of training and/or experience, and expertise.

Altogether, these issues make the conclusions drawn not only feel unreliable but at best inaccurate and preliminary with a high risk for misleading of the public, scientific, and health communities about the size of nutrient effects, associations, and health risks, statistical significances, and options available to support maintenance of health and health risk reduction among our population. If the activity, its results, and the Report were to be considered final, it would provide an example of a well-intended but inadequately resourced activity (perhaps too much bitten off to chew). Moreover, educationally, the Report would provide a poor example of how to conduct and complete what is a highly important activity. Furthermore, a push towards completion now without further necessary literature search and re-analysis would undermine the real potential of the review team.

Further, care needs to be taken when affecting nutrition guidelines yet there no evidence and no discussion in the draft Report of the impact of the recommendations on how people will respond to the changes suggested. For example, suggestions for using nutrient energy from starch to replace energy from sugars (from a guideline level of 10%, which is already below regular intakes, to the even lower value of 5%) could result in a higher incidence of type 2 diabetes unless the starchy food is moderate to low GI [1].

None of these problems are to be expected of a report from SACN for Public Health England or for the Department of Health. Clearly, something needs to be done. For the report to be completed the literature used needs to be brought up to date and meta-analytical methods used must be objective, rather than being intended to be objective, and brought up to date, too. Methods used need to be used confidently and not entertain undue and often erroneous assumptions or appear as though presented in the hope that they are seen to be satisfactory. This is an important area of research. It is important, too, that science in the UK is seen to be among the best. The review ought not to be without adequate current and future funding and insight to bring the content to a level appropriate for a national authority and the health and financial wellbeing of the people and the countries of the UK.

Conflict of interest:

Dr Geoffrey Livesey is a Director and shareholder of Independent Nutrition Logic Ltd, which employs him as a consultant to work with commercial, governmental, and educational establishments and to undertake research on commissioned works on matters regarding health and nutrition. He has received payment or other support covering a period during 1999–2014, and has an expectation of support for the future from commercial entities with an interest in the subject matter of the SACN draft Report, even if it does not benefit him personally, but benefits his position or administrative unit, such as a grant or fellowship or payment for the purpose of financing a post or consultancy. Dr Livesey has held commissions to advise WHO, FAO, and IOM on the science of carbohydrates and energy in nutrition and health. The views expressed herein are without prejudice the personal views of Geoffrey Livesey, BSc (Hons 1st)., PhD. RNutr(Public Health).

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Comments on the draft Report:

Comments below are listed by paragraph number as used in the draft Report, and often are headed by quotations from the corresponding paragraph. Each paragraph may be addressed more than once. For methodology, comments are listed in the order they appear in the Report consecutively for the main article, the appendix, and from the evidence base. To locate all comments for any particular carbohydrate or attribute search by carbohydrate or attribute (e.g. glycaemic load) because comments pertinent to each, as in the Draft Report, may be split between sections (e.g. between Methods, Results , Recommendations, Appendix, and Evidence base etc.). Note that absence of comment should not be taken to mean a statement in the draft Report or elsewhere is agreeable.

Comments may be prefixed with one or more asterisks, (e.g.*****A2.7 "fixed effect model was used" "should") to draw attention to one or more major issues.

Methodology

1.3 "Due to the wealth of data available and because of the concerns around their limitations, case-control, cross-sectional and ecological studies were not considered. Only prospective cohort studies and randomised controlled trials were considered for this report. ".

- The statement admits to not examining the totality of evidence. Scientific reasons for not doing so are not presented; administrative reasoning alone is of questionable acceptability.
- The opening statement appears in stark contrast with the conclusions, which often indicate there is limited evidence or insufficient data (which calls for updating of the literature search).
- A more appropriate rationale would simply be one that seeks the highest levels of evidence according to study design, a rationale that is widely accepted.
- Where limited evidence is found after systematic search (not older than 6 months), it is inaccurate to draw any conclusion yet there are several instances where such inaccuracy arises. The category of insufficient evidence as proposed would be appropriate but is too seldom used, downgrading of evidence would be appropriate.

1.3 "Evidence on adverse effects of very high intakes of specific carbohydrates, e.g. gastrointestinal symptoms, was not part of the remit of this report. "

- Consideration of "adverse effects" is an essential part of any assessment of benefits since at a national and individual level the risk of adverse effect can be persuasive of no overall benefit.
- The statement leaves open whether or not adverse effects other than gastrointestinal ones sometimes mentioned arise. If it is intended to not mention adverse effects, a rationale should be provided; the administrative "remit" is of questionable acceptability.
- If adverse effects are to be considered elsewhere, such as a committee on toxicology, this ought to be the rationale given for the non-considerations.

- Dietary fibre, which is recommended in the draft Report to be eaten in higher amounts than previously and currently, is not without adverse effects.

1.4 "These [reviews] were based on literature published through December 2009, November 2010 and January 2012, respectively." "

- This range of years is too out of date to be representative or even systematic. January 2012 is 2.5 years ago. It is well recognized that such reviews should include at least up to the last 6th months of publications and aim to include later ones wherever possible, eg. Update just prior to going to press.
- A cumulative meta-analysis is essential to assess the stability of effects and associations, but there is no evidence of any having been performed.

1.5 "[Last search dates of] January 2010…December 2010…February 2011…June 2012"

- Again, these are the last search dates and are insufficiently up to date to demonstrate the results are current or representative of the available literature.

1.5 "the update search was not a systematic review"

- What does this mean? Either the Report accepts systematic review or it doesn't.
- Systematic reviews should be described either as meta-analytical systematic reviews when meta-analyses are conducted or narrative systematic reviews when there is insufficient data for meta-analysis.

1.5 "After this cut-off date additional studies were considered only if they were thought potentially to impact on or inform the conclusions drawn in this report."

- Data from all relevant studies should be included before drawing a conclusion.
- The Report includes the nonsense statement quoted here above. One is obliged to consider the data to be able to think whether the additional study could impact on the conclusion drawn and so reconsider the conclusion. Moreover, without the attendant search, critical publications might be missed. The procedure as adopted allows reporting bias to be introduced into the Report's conclusions. In a systematic meta-analytical review one can only disregard the recent studies if a cumulative meta-analysis has found prior stability for the conclusion reached and I^2 is non-significant and near zero, even this is not ideal.

1.5 "This was particularly the case where there was limited evidence or when it was difficult to interpret how evidence from the update search affected the conclusion."

- The first part of the statement is ambiguous. Please be clear about what was limited, the data already considered, the data in total with the most recent study/ies, or the data in the most recent study/ies?

- The second part of statement appears bizar. If it is not known how a new study affects the conclusion, then no conclusion can be reached.

18.1 Interpretation of cohort studies.

- The Report does not provide an unbiased statement. Comments in the Report describe the weaknesses of cohort studies, yet few strengths are reported; one has to get to the subsequent para to find a strength, then it seems only one is given.

18.1 Interpretation of interventional studies.

- The opening statement leaves it unclear about what to do when the disease is defined by metabolic or physiological states, e.g. blood glucose and diabetes, hypertension and high blood pressure.
- The examples as given in this section of the Report are particularly poor. Variation in nutrient compositions which differ among studies might simply enable meta-regression to adjust for potential confounding . In this section of the Report there are few strengths and weaknesses considered attribute to RCTs. A key weakness of long-term RCTs is the convergence of regular and treatment diet interventions, which may arise when participants the treatment arm learn via the grape-vine that a treatment diet might have some benefits. It is never clear how soon that convergence might arise making a no-effect conclusion open to doubt.
- The last sentence in the para is hard to understand. Does “total carbohydrate” mean the total of available carbohydrate or does total carbohydrate include unavailable carbohydrates. Or is the author of the sentence trying to say that the definitions of carbohydrate are often unclear, and can sometimes be Available carbohydrate and sometimes Total carbohydrate (including dietary fibre) or other definition, or is there some implied reference to variation in carbohydrate intakes often being accompanied by variation in fat intake?

*****A2.7 “fixed effect model was used” “should”

- The present reader is surprised at speculation in A2.7 that assumes subgroups within a research centre would not differ.
- Indeed that a research centre present results for subgroups separately is because they hypothesize differences (might) exist. One type of within laboratory subgrouping mention in A2.7 is for men and women, who can (and often do) respond differently to diet within each research centre. If a population mean is needed then random effects ought to be used to combine data. However, if meta-analysts really wish to achieve the correct reduction in heterogeneity, then the outcomes should be modelled appropriately. In the case of gender differences this can be achieved using a zeroed centred ‘covariate’, that is the fraction of the population sample that is one sex or the other male zeroed for mixed sex (i.e. zeroed on 0.5). Then the meta-analysts will be able to combine observations for the different sex groupings (male population data, female population data, and mixed-sex population data of

varied sex ratio). The results will have the correct reduction in heterogeneity and obtain additional information about the size of difference between sexes.

A2.8 The criterion (I^2>75%) "It was agreed that if the result produced an I^2 of more than 75%, the pooled estimate would not be presented because it indicates that there is excessive heterogeneity and the result would have little meaning."

- The criterion would exclude outcomes with a large heterogeneity even if all results were in the same direction and have a large effect. In other words, could exclude important information about health with a size of effect/association that is conditional to subgroups and covariate domains (which could be hidden by the procedures in A2.8). The criterion would also include studies perceived originally as large (adequately powered) but in practice were imprecise due to error attributable to large-study inefficiencies.
- It is always better to present the result, and speak to the caveat. The alternative risks assertions of lack of transparency (such as made here) and prevents retrospective re-consideration of the result when/if appropriate. Being transparent also treats outcomes with I^2 of 74% and 76% the same, difference between the two would be entirely arbitrary and unwarranted.

A2.10 Conversion of NSP to AOAC fibre and vice versa

- It is good that this discrepancy is highlighted, and that it has been addressed in the evidence base. Perhaps better still would have been a dummy covariate centred on AOAC (AOAC=0, NSP=1), which would have informed about the size of difference between the two fibre analysis approaches and whether the results were significantly different; this without having to implicate any doubtful conversion factor, which might be inaccurate for a population instant.

A2.16 and A2.17

- The difference between the two paragraphs is not adequately drawn to the eye.

A2.21 "relative risk above 1.2 for greater risk or below 0.8 for decreased risk"

- Unclear, take RR=1.2, does this mean say 1.2 over 5 quantiles, 1.2 over one quantile or 1.2 over 1SD or 1.2 over a targetable range of intakes irrespective of habitual range of intake, or something esle?

A2.22 "No conclusion- insufficient evidence. & No conclusion- inconsistent evidence."

- The first of these categories is not applied sufficiently often (problem suspected is in the definitions developed for attribution).
- What is meant by inconsistency here, does this mean probable heterogeneity or something else?

A2.23 "normal diet"

- What is a normal diet?
- There is no specification here, but something possibly like it does appear in the main article.

A21 to A2.23 ……..

- All is written in the past tense. Likely, all would have been written in the future tense if written and agreed beforehand. This suggests some deviance in presentation, and for what purpose?
- It is unclear whether data from the prospective cohort studies were appropriately transformed before meta-analysis. At least some were it seems after rendering some of the evidence base.
- There was no identification of the cause of curvature in dose-response studies as reported. Such can arise because of inequality of the dose range among studies. In such case, further evidence of non-linearity is essential; otherwise there is a real possibility that the meta-analysis will have underestimated heterogeneity (hidden it in the curvature). Curves are shown for dietary fibre dose, yet the data considered by IOM was linear.
- There is no evidence that individual studies were assessed for significant or even visual nonlinearity. If linearity is indicated at the level of individual studies, then two-step meta-analysis would be appropriate (i.e. dose-response with linear trend within study, followed by meta-analysis with or without covariates to the combined trends).

*****Literature selection and Data analysis in the evidence base

- The body of the Report is unclear about methodology used, as often noted in the forgoing.
- Examination of data analysis details in the evidence-base reveals a number of issues that are surprising. It is essential that meta-analysis results are corrected to avoid finding an effect due to bias towards low combined errors.
- Studies are excluded when “less than one year in duration” or “have not prescribed ad libitum dietary regimen”. However:
    - there are several weight loss trials referred to in the main body of the text / appendix that are from 6 weeks to 6 months duration.
    - The same criteria might well apply to food intake /energy intake and satiety trials when these are used to make inferences about obesity, but this has not been adhered to.
- Excluded are interventions that use a dietary portfolio (combination diet) or mixed component regimen, e.g. the prescribed diet included plant sterols, soy protein, viscous fibres, and nuts etc. or studies that do not permit the effect of carbohydrate/carbohydrate type to be evaluated
    - This is not always adhered to with respect to sugars, some of which are accompanied by modified fat intake.
- Concern about inclusion of low quality prospective cohort studies has been expressed previously in this response.

- Scoring for study quality is not defined.
- Body weight can be in the causal pathway but is always considered in the Report as a confounder, it might not be.
- It is claimed that "Our aim is to undertake a meta-analytic approach where possible, but we will take into consideration the nature and magnitude of the evidence base and the extent of heterogeneity in the data".
    - However, while heterogeneity was evident in most analyses, there are numerous instances (if the protocol has been adhered to) where heterogeneity has been excluded from the overall error term (or 95%CI for effect/association) for combined effects/associations. These are when heterogeneity I^2>0 to 100 is assumed zero by protocol thus it is stated:
    - ******"Fixed effects meta-analysis will be used for randomised controlled trials". Unless there is zero heterogeneity, this is not the right thing to do, more especially in nutritional studies with many real and potential differences among RCTs: This is because the fixed effects then bias the combined error term towards zero and so can result in finding 'significant effects' when no significant effect has arisen.
- "I^2 >50%" is purely arbitrary, this whatever 'experts' have done this beforehand. They too need to consider what they have done. Nutritional studies cannot be considered like drug studies, and drug studies are known to have biases.
- "I^2 …more useful proportion of total variation" Sometimes tau^2 is more useful, I^2 is more useful for tests because it is more precise that tau^2.
- "lack of information" allowing individual studies to be excluded. It is customary to write to authors of original studies to acquire the missing data. To not do so is to not maintain necessary standards, and risks useful information being lost as this activity is deferred to the next (later group) undertaking a meta-analysis (when original authors might by then have lost access to information).

******Chapter 4 Diabetes.pdf Figure 4.8, Funnel plot:

"There was no evidence of any small-study effect such as publication bias, as is shown by the contour-enhanced funnel plot below:

Figure 4.8 Contour-enhanced funnel plot for publications presenting incident diabetes mellitus type 2 and dietary fibre"

- The plot is confusing—it doesn't appear to have been constructed correctly because the plot indicates (contrary to the Report's statement quoted) that there is massive publication bias but this is due to the pseudo-confidence bounds being plotted about zero association; this rather than correctly about the combined mean.
- Trim-and-fill analysis is preferable to Eggers plot, the hypothetical 'missing' or 'filled data' and 'filled mean' can be shown, too, together with the adjusted combined mean and test of significance of bias when residuals are analysed).

- It is seldom done, but the pseudo-confidence bounds ought also to be presented for the random effect analysis, too, whenever $I^2>0$ whether or not I^2 is significant.

*****Chapter 4 Diabetes.pdf Figure 4.10, Funnel plot:

- The plot has the same problem as mentioned above for Figure 4.7 in the same pdf.
- All funnel plots released need to be checked for correct construction and drawn to be correct and informative without confusing the reader.

*****Chapter 4 Diabetes.pdf "Please interpret observational data with caution: With observational studies there is substantial potential for biases."

- The Report's statement in itself is biased because nutritional intervention studies also have considerable potential for biases.
- The more general and well accepted caution is sufficient, that observational studies are not sufficient to prove causality.
- Observational studies provide what one hopes is the best estimate of risk. This is the best one can do also with RCTs when measurements are risk factors, but even then risk is often not estimated but is unquantified and only inferred as important. Quite possible the Observational studies are the more transparent of the two.
- RCTs to test diets for incident diabetes or incident colorectal cancer may well be impractical and unethical if diabetes type 2 and colorectal cancer take the generally accepted 10 or 20 years for development. All this leaves RCTs being ideal theoretically but doubtful practically when starting with heathy participants. Starting with diseased participants one can at least begin to monitor progress, such is the advantage when examining drugs.
- A less biased perspective towards favouring RCTS over Observational studies would be appropriate for the measures made among studies in this draft report.

*****Chapter 4 Diabetes.pdf Figure 4.10, Forest plot for glycaemic index and Diabetes type 2.

- The plot and conclusions are inaccurate—see next.

*****Chapter 4 Diabetes.pdf Figure 4.10, Funnel plot: "There was a little evidence of possible small-study effect from the contour-enhanced funnel plot, though half the studies did not suggest any evidence of a protective association."

- The statement strongly indicates the authors lack an appropriate ability to present and interpret funnel plots (see above comments).
- In addition, the claim made is contrary to the observations in Figure 4.19.
- There is also a failure to account for adequacy of the FFQs for carbohydrate among these studies. Meyer et al 2000, Stevens et al, 2002, Mosdol et al 2007 and Sahyoun et al 2008 all had inadequate FFQs (Barcley et al 2008, Livesey 2013[2, 3]). If an FFQ has poor correlation

for the amount of carbohydrate in food, its use will cause heavy biased towards the null. Interestingly these are the studies that found no significant effect.

- The last study reported is 2008, why are there no studies of later date? Remember too that there are problems in the EPIC studies, with FFQ needing to be validated within each region, and difficulty of small numbers in each region, which limits the number of adjustments that can be made.

*****Chapter 4 Diabetes.pdf Incident Diabetes Mellitus type 2 and glycaemic load.

- The section and analyses are out of date.
- The conclusions reached are inaccurate.
- A comprehensive meta-analysis of the relation between diabetes type 2 and glycaemic load is available (Livesey et al 2013 ab [3, 4]).

*****Chapter 4 Diabetes.pdf

- In view of the forgoing it is known that:
- Some of the combined outcome results for observational studies are inaccurate beyond the draft Report's meta-analyst expectations.
- None of the combined outcome results of observational studies take account of the adequacy of the FFQ for optimal reporting quantitatively and could have done so. Thus none of combined outcome results are likely to be accurate. This includes also the subgroup analyses.
- All meta-analyses of intervention studies should have weighted studies according to random effects, which becomes fixed as I^2 becomes zero. Instead it is indicated in the Data Analysis section that fixed effects were applied. Whenever $I^2>0$, even by a small amount, the use of fixed effects causes a bias in the combined mean and error size favouring towards a significant effect.
- Consequently, extreme caution should be taken when applying results from this Report, whether for intervention studies or for prospective cohort studies.
- These problems affect all chapters
- The only solution is to update an redo the analyses..

Chapter 2. Classification, biochemistry, absorption, metabolism and definitions of carbohydrates

****2.1, Table2.1 Sugars (DP1-2) and Sugar alcohols/polyols

- Table 2.1 of the draft report includes an error that was present in the 1998 WHO/FAO report and is reproduced (perpetuated) by commissioned authors Cummings & Stephens in the WHO/FAO scientific update in EJCN 2007 [5]. The error was corrected with the recommendation from FAO/WHO (2003) (page 76) [6]. in their report on Food energy and analytical methods Stating "The term 'sugar alcohol' should be phased out of food labelling and replaced with 'polyol'. Polyols should be recognized as carbohydrates, but not sugars". Subsequently, without reference to WHO/FAO (2003) [6], a WHO/FAO commissioned paper from Englyst, Liu & Englyst 2007 [7]. provided a correct tables of carbohydrate characteristics in regard polyols,
- This underlies why polyols are permitted in sugar-free products, because they are not sugars.
- Ploysols are not always DP 1-2 and can be DP>2, for example polyglycitol, and maltitol syrups.
- The correct classification places polyols apart from sugars as a 4th category among carbohydrates (FAO/WHO 2003)[6, 7]. (see Annex 1)
- To emphasize, none of the Sugars definitions provided by the UK, USA, WHO and EU include polyols (cf also Table 2.4 of the draft Report).
- Polyol(s) is the preferred term on food labels as used in the UK, USA, and Europe Australia, New Zealand, and elsewhere. Sugar alcohol though still used occasionally in the literature is becoming archaic.
- Limits for intake set for polyols differ from the limits set for sugars, emphasizing again the importance of not confusing polyols with sugars.
- An advisory is mandatory in labelling on excessive intake of polyols, but not for sugars.

**2.2 "In 2006/7, an FAO/ WHO update on some of the key issues relating to carbohydrates in human nutrition endorsed the primary classification recommended by the 1997 Expert Consultation,"

- It is incorrect to say FAO/WHO "endorsed" the classification. This was not a full consultation that allows attribution to FAO/WHO, rather indeed the perpetuated error is attributable to the commissioned authors and perhaps the manuscript referees. It is only the general concept of categorisation by size that remained acceptable to FAO/WHO 2003 [6], who recommended polyols be categorised correctly (see comments above at 2.1).

2.4/1 "Chemically combined"

- Chemically combining fructose and glucose does not always lead to sucrose. The sentence needs modification.

2.5 "which are three, four and five sugar polymers respectively"

- Potentially confusing when polymers in nutrition are defined DP>9

Chapter 2.

- Xanthan gum is now an appreciable part of 'gluten-free' carbohydrate foods and other foods. It is not considered in this chapter. Indeed, without reason, all nsp from a similar *eg bacterial) source are not considered in this sections of the draft Report.

2.12 [Soluble and insoluble dietary fibre].

It would be worth noting the FAO/WHO (2003) recommended discontinuation of this classification of dietary fibre. It is also not preferred in the USA but exists historically. Does this mean the ref should be (FAO/WHO 1998, 2003).

2.17"transported to the liver" "glycaemic carbohydrate". "immediately"

- Omits to say that liver is not absorbing most of the carbohydrate, as might be implied from the text at present.
- The term glycaemic carbohydrate is undefined and not recommended over available carbohydrate (FAO 2003).

2.18. Glucose energy value 15 kJ/g (3.6 kcal/g) etc.

- The energy values quoted from FAO (2003) are subject to biases towards the over generalisations of the Australian food labelling system and further errors arise in inter conversions between kJ/g and kcal/g that would have been unacceptable to the UK committee originally responsible for assigning kJ values for macronutrients. As a scientific document, scientifically valid values should be used by SACN.

2.19 Glycaemic index and glycaemic load

- The section is confused by unnecessary detail, lack of direction, and complicated sentences. I rewrote the paragraph to help minimise these problems, bring clarity, accuracy and direction.

Glycaemic index (GI) and glycaemic load (GL) are two measures of the glycaemic characteristic of foods. GI is a relative measure of the capillary blood glucose response to a specific ingredient, food or portion of a meal, as compared with the response to a reference food having the same amount of available carbohydrate (usually 50g). The reference food can be either pure glucose or another, alternative, carbohydrate food (e.g. white bread). When alternative foods are used as reference they are calibrated against glucose (Brouns *et al.*, 2005). A foods GL is the product of GI and its available carbohydrate content (Brouns *et al.*, 2005), so taking into account both the quality of the carbohydrate food and the amount of available carbohydrate it contains. GI (thus also GL) is influenced mostly by the types and structures of carbohydrates present in foods and to lesser extents by the types and amounts of protein, fat and non-starch polysaccharide present. External

influences on a foods GI include, milling, cooking, cooling and storage conditions (Brouns *et al.*, 2005; Venn & Green, 2007). Variation in GI among foods reflect mostly the variation in rates of carbohydrate digestion and absorption, and to lesser extents variation in the rate of glucose production other than from the digestive tract (e.g from liver) and disposal from the circulation into the tissues (Schenk *et al.*, 2003; Eelderink *et al.*, 2012a; Eelderink *et al.*, 2012b). The critical relations for GI and GL are the associated risks or benefits, and whether GI and GL values have greater predictive value for health than available carbohydrate alone, which is expected mechanistically where the risk/benefit reflects a carbohydrates foods influence on glycaemia, and excessive glycaemia has (or associates with) adverse effect on health.

2.26able 4. [Definitions for Sugars]

- To emphasize comment at 18.1. None of the sugars definitions provided by the UK, USA, WHO and EU include polyols.
- In Canada 'wholegrain' does not include the germ, thus making an important difference both nutritionally and by comparison with definitions from most if not all other counties mentioned in the SACN report.

Chapter 3. Dietary sources and intakes of carbohydrates

3.7 Mean intakes of total carbohydrate in the UK in adults and children aged 4 years and over were 200-240 grams/ day.

- At 200g/d total available carbohydrate, person consuming diets of GI>50 are at increased risk of Type-2-diabetes owing to the amount of the available carbohydrate, total. and higher GI of carbohydrate foods eaten (Livesey et al, 2013 [3, 4]), a comprehensive systematic meta-analysis of all the then published prospective cohort studies.

3.11 "Mean total sugar" "glucose and fructose"

- Please be specific from the beginning of the paragraph. Does this mean total Sugars: or total sucrose, or total non-milk extrinsic sugars or something else?
- Would this be free glucose and free fructose, or totals from all sugars?

Intrinsic and milk sugars intakes and dietary sources

Starch intakes and dietary sources

3.22. "……."

- If there is no data or data are irrelevant for adults please state the case, otherwise readers may consider the report to be incomplete.

Non-starch polysaccharides intakes and dietary sources

- It is impossible for consumers to compare data on the food label (as Total dietary fibre – as required in food regulations) with NSP in this report. There should be a separate heading for Total dietary fibre. If such information has not been collected then the reason for not collecting the information should be given and a recommendation to have this information collected be made. Whatever the food politics in the UK, absence of information is unhelpful to consumers funding this study.

Chapter 4.Background on health outcomes (disease prevention)

4.3 "……."

- The first sentence of this paragraph is too long and complex leading to ambiguity. Readers might be left wondering 'Does tobacco use result in obesity?'

4.4 "obesity associated with type 2 diabetes

- For the sake of helping the consumer and the journalist writing for them, please reword to make evident also that type 2 diabetes can occur in non-obese individuals, not meaning only those who are overweight but also those with little body fat. Ditto other metabolic diseases.

5.6 "Nearly all trials are conducted in overweight or obese individuals and the diets involve energy restriction goals and most trials result in weight loss."

- It is difficult to know at this stage of reading (or for persons dipping in and out) whether the parameters presented to describe the cohort and trial studies and questions asked are those selected by prior design of the review group or those that were variably identified in the original reports. There is little evidence that the review group tested hypotheses that might have been raised in the literature as possible explanations of heterogeneity.
- The clinical significance of a find can be the same whether the outcome or association arises directly or indirectly via body weight change. Only the mechanism of action differs; this providing that the studies have been analysed and interpreted appropriately. If the mechanism is shown to involved a difference between treatments in change in body weight, then the risks/benefits investigated potentially then also have a plausible mechanism— others not investigate might be predictable, too.

5.7 “consideration has been given to”

- What does this mean – an opinion was expressed or data were analysed to address the question? In some ways the review is telling us of the problem and not how the review group overcame it.

5.8 “2012”

- Search needs updating.

5.9 “No significant association”

- We are not informed with sufficient information, such as duration of intervention or follow-up, range of carbohydrate intake and difference in treatments, whether normal overweight, obese or mixed body weight groups, whether in male, female or mixed-sex population samples etc.
- At this stage of reading it is unclear whether cohort and intervention studies were combined in one meta-analysis (unwise) or examined separately. This uncertainty is present throughout the report and could have been avoided by an appropriately wise statement in the methods section.

5.10 “Three studies could not be included in the meta-analysis”

- Reason is lacking and should be stated.

5.1 to end chapter. “higher, lower,”

- There is no point of reference with respect to the average diet in UK adults or relative to guidelines. This makes the context difficult to appreciate and application of the conclusions impossible. Overall the conclusions are doubtfully agreeable but quantitation is problematic for a variety o f reasons mentioned elsewhere herein. In some groups of people a measure may rise in others fall, overall average effects for the combined groups tending to the null while in reality there may be significant effects.
- The data are concerned with studies reporting total carbohydrate. It should be mention early on that no consideration is given to carbohydrate quality among the data extracted for review. Thus carbohydras of high GI and Low GI may differ, as shown in some published studies.
- It is not clear in the draft report whether the conclusions reached apply to the UK population or whether population and ethnicity are important factors.

5.35 “Adequate evidence …A diet higher in carbohydrate and lower in fat may decrease fasting total cholesterol concentration, but it is not possible to exclude confounding”.

- If it is not possible to exclude confounding, how can the evidence be adequate? Explicit qualification of these contrasting notions would be helpful.

Post 5.43 "Higher carbohydrate, average protein diets and fasting total cholesterol concentration.

- If the mechanism of effect is via weight reduction, is it appropriate to call this confounding?
- If it is not possible to exclude weight loss, this may be because there is too little data or the meta-analyst has not analysed weight loss as a contributor/confounder. Please be informative.

Post 5.49 "Higher carbohydrate, average protein diets and fasting triacyleglycerol concentration.

- If the mechanism of effect is via weight reduction, is it appropriate to call this confounding?
- If it is not possible to exclude weight loss, this may be because there is too little data or the meta-analyst has not analysed weight loss as a contributor or confounder. Please be informative.

Post 5.54 and other boxed conclusions. "The effect is biologically relevant"

- Does this mean the effect size is biologically relevant or the direction of the effect is in the right direction to be beneficial? This is not easily establish from within the draft report.

Post 5.56 "Higher carbohydrate, lower fat, average protein diets and fasting LDL-cholesterol concentration."

- If for carbohydrate one is not being concerned about carbohydrate quality, why then is one concerned about the type of fat as a potential confounder? Might the conclusion be indicative of biased in the report?

Post 5.59 "Higher carbohydrate, lower fat, average protein diets and fasting triglacylglycerol concentration." Box.

- Given the nature of the intervention, is it appropriate to claim possible confounding by fat. Rather than apply a negative view to these studies, why not state it as it is (as seems): 'The effect may be due either to more carbohydrate or less fat or to both; to date, potentially confounding variables have not been explored'.

Pre 5.64, to pre 5.68, heading "Higher carbohydrate diets and …"

- The headings are insufficiently informative, ditto the box conclusions.
- As in other places there is an issue in some of these boxes: Carbohydrate quality is not considered while fat quality is considered.

5.84 “blood glucose level”.

- Please be specific, fasting or random sample etc.

*5.95 ditto 5.97, 5.98 “Due to different methodologies…not possible to conduct a meta-analysis.”

- If no meta-analysis is possible, how can evidence be considered “adequate”, surely it would be limited? Explain what the common finding is. This applies throughout the draft report.

5.114 “stratified”

- Is “stratified” meant to inform that subgroup analysis was undertaken or was something else done. Please be specific.

5.121 “Due to high heterogeneity between trials (I^2=80%), it is not possible to report the meta-analysis pooled estimate.”

- Of course it is possible, and desirable to do so alongside a scientific valid reasoning of the caveat.

*5.122, heading “Higher carbohydrate ….average…higher,, lower… average compared with lower…higher…lower average and higher”

- The headings is far too complex. The last half of the first sentence might be used instead.
- No effect? What about the heterogeneity of study treatments?
- Can a probability of 94% be considered ‘No effect’? In reality it is misleading to claim ‘No effect’ on limited evidence, rather ‘inconclusive would be appropriate. This is a problem encountered throughout the report.
- There appears to be considerable reuse of data in different meta-analyses. Doing so risks a chance significant result. This makes it difficult to accept conclusions of “Effect” as a real effect. How was this overcome?

5.134 “……”

- It would be appreciated if some information about restrictions on eating were presented. If all studies allowed ad libitum energy intakes it would help to say so.

5.138 “No significant association [for carbohydrate intake on colorectal cancer]”

- It is possible that low dose of carbohydrate (in association with fat) promotes colorectal cancer and high dose is protective (in association with fibre) or even vice versa if carbohydrate directly promoted higher insulin-like activities). Combining all doses, a conclusion of 'No effect' might then arise under the current system of data synthesis and would be misleading.
- Only by considering the cohort studies and the intervention studies together might a No effect conclusion arise, yet there are two conclusions, one of which is premature, and the other of which is only apparently substantiated. Adequate evidence?

Tables 5.1 to 5.3 [absent information]

- It would be useful to have tables of findings with adequate evidence and possible evidence. If not at this point in the Report, a reference at this point about where such tables can be found.

Summary and Conclusions

*5.151 "There is a lack of evidence on total carbohydrate intake in relation to oral health"

- Readers dipping into the report would be confused by this viewpoint. We know that caries is the result of acidogenic carbohydrate fermentation, including oral fermentation of starch in addition to sugars. Some qualification is needed if the summary statement is not to be abused.

*5.152 "No association is indicated between total carbohydrate intake and …glycaemia…"

- We know that there is a blood glucose response to carbohydrates. Some qualification is needed if the summary statement is not to be abused.

5.153 "Total carbohydrate is the sum of the sugars, starches and dietary fibre"

- It is unclear whether this definition is in keeping with the definition in all the papers reviewed or even in keeping with the intended definition for the purposes of this Report.
- If this definition is intended, a statement is needed making explicitly clear that studied reporting available carbohydrate rather than total carbohydrate have been excluded. This needs to be made both at the outset of the Chapter 5 and again in the summary.
- A reference within the Report to the intended definition for the purposes of the review is needed.
- If this definition is intended, the whole of chapter 5 is irrelevant to consumers in the UK which defines carbohydrates, total, differently.

*5.154 “Randomised controlled trials assess the effect of varying total carbohydrate intake, by reciprocally varying fat, type and quantity, and/or protein intake”.

- Is this statement really true. Reduced energy diets can be created by reducing intakes of any macronutrient without reciprocation.

*5.154 “These trials indicate no significant effect of varying total carbohydrate intake on vascular function, inflammatory markers and risk factors for type 2 diabetes mellitus.”

- The report carries too little information to know whether or not any of the Reports conclusion for Chapter 5 are valid.
    - Among the many meta-analyses, and reuse of data across meta-analyses, there has to be concern about the validity of p-values for combined outcomes.
    - Because numerous questions have been asked, there has been a lack of essential focus on any one question.
    - The Report states only that meta-analyses have been undertaken. However, nutrition is complex and studies are not fully controllable within and among them for equality, which makes meta-analysis without covariates a tool that lacks power to address questions of importance.
    - The report does not recognise the potential for an exchange of adverse effects of carbohydrate for adverse effects of fat which can obscure the knowledge that needs to be acquired, and leaves the reader with the impression that both are safe at any level.
    - There is no distinction between ‘confounding’ and ‘mechanism of action’ in studies where body weight complicates interpretation of the observations made. This is all the more important if the caveat raised in this paragraph is true “applies to all cardio-metabolic risk factors investigated”.

5.155 “The trials do provide evidence ”

- A reference to the para in the Report supporting the conclusion would be helpful to readers (i.e. stating para 5.??).

5.156 “Overall, prospective cohort studies indicate that total carbohydrate is neither detrimental nor beneficial to cardio-metabolic health.” and “risk of colo-rectal cancer. ”

- The report does not recognise the potential for an exchange of adverse effects of carbohydrate for adverse effects of fat which can obscure the knowledge that needs to be acquired, and leaves the reader with the impression that both are safe at any level.

Chapter 6.Sugars, sugar alcohols, sugars-sweetened foods and beverages

6.6 “update search”

- Date of update not stated. It is nevertheless out of date.

6.7 No association [for sugars and coronary events].

- This is consistent with the metabolic effects from intervention studies.
- It is hard to know what dose range the conclusion applies.

6.9 “No effect [for sugars and blood pressure]”

- It is hard to know what dose range the conclusion applies to.
- It is hard to know over what duration the no effect applies to.

6.11 “No effect [for fasting total cholesterol]”

- It is hard to know what dose range the conclusion applies.
- It is hard to know over what duration the no effect applies.

6.13 “No effect [for fasting LDL-cholesterol]”

- It is hard to know what dose range the conclusion applies.
- It is hard to know over what duration the no effect applies.

6.15 “No effect [for fasting HDL-cholesterol]”

- It is hard to know what dose range the conclusion applies.
- It is hard to know over what duration the no effect applies.

6.19 Effect” and “Adequate evidence” [for energy intake]”

- It is hard to know what dose range the conclusion applies.
- It is hard to know over what duration the no effect applies.
- Only one study investigated isoenergtic diets, it cannot be said therefor that there is Adequate evidence”
- Evidence on comparable studies with other energy sources has not been presented, it is hard therefore to particularize the conclusions to sugars.

6.21 “No association” [for sugars and Type-2 diabetes]

- It is hard to know what dose range the conclusion applies.
- Were all sub-cohorts included in the analysis?
- Were data appropriately transformed prior to analysis?
- It is questionable whether all studies excluded provided insufficient evidence for inclusion in a meta-analysis. It is customary to consult with the original authors to acquire the required information.

6.23 “Sugars and blood glucose concentration” “No effect”

- This would be a surprising conclusion. Perhaps “fasting blood glucose concentration” would not be surprising.

6.25 “Sugars and blood insulin concentration “ No effect”

- It is hard to know what dose range the conclusion applies.
- It is hard to know over what duration the no effect applies.
- The conclusion is surprising, perhaps fating insulin concentration is meant.

6.26 ‘Studies excluded’

- Please check the references are correct: Janket et al 2003 was excluded from an earlier meta-analysis, but not here?

6.27 “cases and controls”

- Clarification is need, case-control studies were said to be excluded?
- Perhaps cases and non-cases is meant? (Or is non-referent and referent cohorts meant)?

6.29 ‘Fructose, glucose, sucrose’

- It is unclear whether studies have been unduly excluded from the meta-analysis. It would be unusual for a food composition database to present sucrose as though fructose and glucose. It is incumbent on meta-analysts to correspond with original study author for clarifications of this type of issue. Failure to do so risks bias.
- Was all available data meta-analysed such as in dose-response meta-analysis?
- Was heterogeneity due to results from a single study?

6.30 ‘Janket et al excluded from meta-analysis for insufficient data’

- It is unclear whether the data applies to fructose from crystalline fructose or from high-fructose corn syrup, or from fruit eaten whole or juiced or is any source of free fructose.
- It is unclear what data is missing.

- Potentially inclusion/exclusion criteria adopted may not be appropriate or an inaccurate view of the published study has been taken?

6.34 “Sugars-sweetened beverages” and “Association”.

- It is unclear whether the association applies to data from carbonated sugar-sweetened beverages.
- No information is presented to compare with intensely sweetened carbonated beverages (lacking sugar), which also shows effect.
- Whether or not there is reality among the potential for confounding has not therefore been excluded in the presented analysis.

*6.38 “maltitol” and “isomalt”

- Evidence is claimed for the polyol maltitol. However, the study of Sinaud et 2002 did not study maltitol, it studied a mixture of hydrogenated malto-oligosaccharides and hydrogenated malto-polysaccharides.

*6.38 “One trial reports no significant effect of the sugar alcohol isomalt on faecal weight”

- The t-test was two sided for the study on isomalt. However an elevation in faecal weight was expected.
- Other studies report very high consumptions 50 to 100g/d, whereas isomalt was studied at realistic dose of 30g/d. Elevating the dose would elevate wet weight significantly. High wet weights are achieved due to the osmotic properties of polyols.

*6.38 “The direction of the effect demonstrates that greater consumption of sugar alcohols is of minor benefit to health because of the limited presence of sugar alcohols in the diet”

- The conclusion makes no sense because it states in other words “direction of effects predict benefits are minor’. Surely it’s the size and direction of effect that predict minor health benefits among habitual consumers.
- On the basis of this idea, polyols have potential benefit over sugars in respect of stool wet weight and constipation (and the putative relation between faecal wet weight and colorectal cancer).
- Polyols have benefit in respect of laxation.

*6.51 “No association [between sugars sweetened beverages and BMI]”

- The conclusion needs qualification for the following reasons:
- There is little doubt from intervention studies in adults that sugar-sweetened beverages contribute energy and can elevate BMI. The extent of the effect after consideration of energy compensatory mechanisms is small (2-3kg of body weight and gain appears limited in

duration 1-2 year). It is unclear whether other, non-sugar, energy sources taken in drinks have the same effect—maltodextrin for example or whether this effect is limited to energy in beverages and not solids (foods). To date the evidence on these questions has not been adequately reviewed.

- The review is incomplete in not identifying prior meta-analyses.
- The review is incomplete in not spotting all relevant studies (Shulze et al 2006 [8]for example who point out the temporal response to change in ssb intake).

*6.53 “No association [with body fatness]”

- No association and no significant association are not the same.
- It is hard to believe that 5 cohort studies provided insufficiently comparable data for meta-analysis to be performed.
- Further information and appropriate qualification is essential to improve on crediblity.

6.56 “experimental group” and “intervention group”

- There is a distinct lack of clarity.
- The experimental group is undefined.
- The intervention group is also undefined.
- The reader does not wish to stop to puzzle over what is meant and be left hanging in uncertainty.

6.57 “lower BMI z-score”

- Overall there is little contextual information.
- Significantly lower (P-value)?
- Lower to a biologically relevant extent?
- Sufficient to not have to worry about other energy sources and physical activity?
- “good retention”, what is good to the layperson, and does any scientist outside the review process know what the Report author considers as “good”?

6.58 “insufficient voluntary reduction in other energy sources”

- There is little contextual information.
- How big a difference in body weight throughout the study?
- How long was the study?
- Was weight gain progressive or at a new steady state by the end of the study?
- Considering the weight gain achieved, was the effect size sufficient to explain the ‘epidemic. of obesity?
- Does the boxed conclusion apply at all levels of sugar sweetened beverage intakes?
- Is there an effect of beverage energy or sugars in the beverage?

Oral health

*6.59 “Five cohort studies”

- This was four cohort studies, one of which appeared in two publications.
- If four studies, please report 4 not 5 studies.
- Is there a similar problem throughout the Report with reporting study numbers?

6.62 “frequency of bedtime sugars consumption from drinks”

- Did post drinks tooth brushing make a difference?

*6.64 “Association”

- It is frustrating to learn of an association and not be presented with the strength of association. This concern arises frequently throughout the proposed report.

*6.65 “adjusted for tooth brushing”

- What does this really mean? Was the adjustment towards the centre, towards tooth brushing or towards not brushing teeth or something else?
- Were the adjustment similar for each study?
- Ditto for all other section referring to adjustments.
- It would be useful to know whether those who brush had no increased incidence of caries in this and similar sections.
- There is no reference to fluoridation or use of fluoridated mouth washes. Are these practices insufficient to prevent the effect of sugars. It is hard to know the exposures of participants for which results are evident in this Report.

6.66 “3.5 years and 18 months”

- Harder to comprehend than needs be.
- Surely ‘1.5 and 3.5 years’. Why the confusing mixture of units and non-chronological order?

Sugar alcohols

See ‘Annex 1 Polyols’ for each page and word of the SACN draft report for which comments arise together with some proposed changes.

*Prior to 6.69. The heading “Sugar alcohols”

- The term that should be used, for the sake of the consumer, is ‘polyols’ as in food labelling.

- WHO/FAO 2003 explained why— because polyols are neither sugar nor alcohol. That is sugar alcohol is ambiguous and often misunderstood by lay persons. (See comment against 2.1).

*6.70 “In the trials that employ a ‘no gum’ control group, it is unclear whether it is specifically the sugar alcohol or the act of chewing and the concomitant increase in salivary flow that contributed to the effect.”

- Fails to recognise that all mechanisms of effect for reduced caries here mentioned are secondary to polyols use.
- Polyols are permissive of the act of chewing. Chewing tends to stop when the sweetness ceases due to the polyols eventually being released and swallowed..
- The increased level and duration of chewing brought about by the pleasantness of the polyols encourages chewing and in being pleasant encourages the flow of saliva.
- Among the population exposed to sugar containing polyols, polyols further reduce exposure to sugars (the no-gum group is only one of two relevant types of behaviour in the population, no gum and sugar containing gum exposures), so having further effect.
- Other mechanisms include polyols support of remineralisation, polyols are not a substrate for plaque formation, and negligible acidogenesis.

*Table 6.2.

- According to this table there may be sufficient evidence for polyols effect on constipation among RCTs. This would be in keeping with advice and known laxation on consuming polyols in excess of current regular low levels of polyols consumption.

*6.75 A paragraph among the Summary and conclusions

- Please keep a link to the evidence.
- For example, state the paragraphs at which these conclusions were synthesised.
- Ditto for the conclusions in other paras.

*6.76 “sugars-sweetened beverages”

- Please be more specific
- Fruit juices?
- Carbonated ssbs?
- The weight on sugars is likely too great since a link with carbonated diet drinks also shows a some link to type 2 diabetes. To be fair this should be mentioned.

*6.78 RCTs, caries, polyols.

- See comment aside para 6.70.

*6.79 Polyols and faecal bulking.

- The potential use of polyols in laxation is not well represented among the analyses and narratives presented.

*6.80 Polyols and faecal bulking

- Biological relevance is that polyols do not lower faecal bulk and may therefore be considered safe in regard this aspect and putative risk to health.
- Some evidence indicates that certain finely divided fibres can result in constipation [9].
- Ditto there is greater constipation among the obese and elderly.
- Co-ingestion of polyols as sugar replacers in cereal products would likely be advantageous. Ideally such should support the crispness of the cereal (have low hygroscopicity), be well tolerated, have low glycaemic and insulinaemic potential, produce low amounts of gas, and encourage laxation, especially among the growing population numbers of the obese and elderly at risk of constipation.
- The para really ought to be more circumspect about the potential biological relevance of polyols. The number of studies reviewed is low compared with the numbers of studies available. In regard fecal bulk and colorectal cancers it remains unclear whether fecal bulk or laxation or one more than the other are of most importance. The Report's conclusion too easily accepts what is or is not important.
- The biological relevance of Bifdobacteria spp remains to be 'firmly established'.

Chapter 7. Starch and starch-rich foods.

7.15 "Association with brown rice Intake".

- Check that the data are adjusted for white rice intake otherwise this association may be due to replacement of white rice with brown rice, with brown rice being more neutral or beneficial in its association.

7.20 "No association [for potatoes and total cardiovascular disease events]"

- It is unclear whether the data were adjusted for sex.
- It is unclear whether the studies have sufficient quality even to draw a tentative conclusion.

7.23 "A full description of the studies can be found in the relevant systematic reviews."

- Please cite the relevant paragraphs, SACN systematic reviews, and published or other grey systematic reviews.

7.25 “Prospective cohort studies indicate there is no association between starch intake and incidence of coronary events or type 2 diabetes mellitus. ”

- The conclusion is difficult to reconcile with similar observations for potatoes and white rice.
- It would be preferable to say “Prospective cohort studies indicate there is no association between intake starch from dietary sources (as opposed to any individual foods) in the populations studied and incidence of coronary events or type 2 diabetes mellitus.”
- At least this suggestion leaves open that the effect is diluted by co-ingestion of starchy foods that have a beneficial association with CHD and type 2 diabetes (such as low GI starchy foods).
- The suggestion might help to limit abuse of the Report’s conclusion.

7.27 “Overall, the available evidence in relation to cardio-metabolic outcomes indicates no association with dietary starch when consumed in the amounts eaten in the typical UK diet.”

- The observations ignore the type of starch and the GI of the starchy food.
- The conclusion is insecure.
- Insufficient evidence is presented to know whether the overall conclusion reported are stable.
- Advice is to report on stability of each conclusion (throughout the whole report).

Chapter 8. Dietary fibre

All dietary fibres in regular diets

- Dietary fibre and glycaemic index (and dietary fibre and glycaemic load) have been shown to have associations and effect that are independent, with each having approximately similar effect and association sizes [3, 10-12]. It is unclear from the draft Report that the effect and association sizes reported for dietary fibre in this chapter are not due or related to confounding by GI or GL. This independence cannot be established by simple meta-analyses (e.g. within a forest plot) or even dose-response analysis without modelling to a detail not evident in the draft Report.

8.7 “Coronary events”

- Events is undefined and may be limited to one type or a mixture of several. Please define.

8.13 to 8.15 “Body weight change”

- It is unclear whether body weight or body weight change is meant.
- The conclusion may not be representative of published studies.

8.17 “The majority of the studies did not make allowance for the metabolisable energy that is available in fibre due to fermentation therefore the majority of studies will have over-estimated the decrease in energy intake.”

- The assertion may not be correct.
- Some jurisdictions applied an energy value of 4 kcal/g to fibre, others assigned no energy value to dietary fibre. Unless this issue was examined specifically, no such comment as extracted above can be drawn.

8.17 Box “No effect”

- The conclusion is not secure.
- The mean fall in energy intake as reported is sufficiently large to affect WHO/FAO estimates of poverty when made on an energy intake basis.
- There is no analysis of bias or of stability of the outcome.
- The CI range is large and there is no mention of sensitivity analysis.
- The quality of studies is not described.

8.19 “ no significant association between dietary fibre intake and the incidence of type 2 diabetes (Sakurai *et al.*, 2012).” and “indicates”

- Claims of no-significant association are meaningless—it all depends on the size of effect and on the precision of the study.
- Statisticians often say there is too much emphasis on statistical significance and not enough on effect/association size.

8.21 “No effect”

- There are too few studies analysed to draw a conclusion.
- The direction of effect may depend on the metabolic state of the individual, it may be raised when values are low and lowered when too high —independently of regression the mean, as noted in Livesey et al [10] for glucose levels stretching from healthy people to diabetic patients.

8.24“Five randomised controlled trials”

- It is surprising how few studies exists that report wet weight as opposed to dry weight or fat content or energy content.
- There is a chance that not all studies have been captured.

8.27 “A pooling project”

- Please define “pooling” because authors use “pooling” to mean different things, and it is often used unwisely.
- Some consider it means pooling of data at the level of participation of individual persons (correct).
- Some consider it means ‘pooling’ [sic] aggregate data from individual studies (incorrect, this is combining).
- EPIC authrs are poor at reporting the validity of FFQs used in each of the contributing clinics/laboratories, which makes it hard to know whether combining they do should be proportionate to study precision in each lab/clinic, or even whether the study quality is adequate in each contributing lab/clinic, and whether combining of many small studies can ever be as good as combining fewer large studies. All of this leads to difficulty for confidently finding real associations whatever the explanation of association.

Insoluble fibre

Cardiovascular disease

8.33 Clarity.

- The paragraph is unclear.
- Better to say how many studies were found and the dates spanning the search.
- Why does the reader need to keep being informed an update search was performed—especially that the update search is now out of date?
- Please give information that provides context to the studies and not unnecessary detail.
- This problem (immediately above) applies to each and every question the report asks
- The para tells us of two studies and a further meta-analysis. This is awkward because a meta-analysis cannot be conducted with just two studies. The text is therefore unclear.
- There is no information about the number of studies in the meta-analysis finally used.
- There is no warning about potential confounding as in other conclusions, suggesting potential for reporting bias.
- It is unclear what is mean by insoluble fibre—whether it is restricted to that from foods or some from enriched fractions or some from isolated insoluble fibres.

8.35 “The results from the later meta-analysis were used”

- Which meta-analysis? Three meta-analyses are mentioned from which we get “later” rather than latest or last.
- “results” but only one result is presented.
- It is unclear how many studies are represented in the meta-analysis used.

8.39 ……..

- As for para 8.35.

*8.40 “No association [Soluble fibre and Coronary events]”

- Stability of no association has not been established.
- There is insufficient evidence to claim no association. A category of insufficient evidence ought to be used.
- It is unclear what attempts were made to assess potential sources of heterogeneity (which is still possible for I^2=0 because I^2 is still an imprecise and inaccurate estimate).
- The source type (food, enriched fraction, isolate) is not identified.
- It is unclear what type of meta-analysis was undertaken.
- It is unclear whether potential for a significant association is hidden by a small range of soluble fibre consumption.

*8.42 “No association [Soluble fibre and Type 2 diabetes]”

- Stability of no association has not been established.
- There is insufficient evidence to claim no association. A category of insufficient evidence ought to be used.
- It is unclear what attempts were made to assess potential sources of heterogeneity (which is still possible for I^2=0 because I^2 is a very imprecise and inaccua5trate estimate).
- The source type (food, enriched fraction, isolate) is not identified.
- It is unclear what type of meta-analysis was undertaken.
- It is unclear whether potential for a significant association is hidden by a small range of soluble fibre consumption.

*8.46 “No association [Fruit fibre and Cardiovascular events]”

- Stability of no association has not been established.
- There is insufficient evidence to claim no association. A category of insufficient evidence ought to be used.
- It is unclear what attempts were made to assess potential sources of heterogeneity (which is still possible for I^2=0 because I^2 is an imprecise and inaccurate estimate).
- The source type (food, enriched fraction, isolate) is not identified.
- It is unclear what type of meta-analysis was undertaken.
- It is unclear whether potential for a significant association is hidden by a small range of fruit consumption.

*8.48 "No association [Fruit fibre and Coronary events]"

- As *8.47

*8.56 "Association [for Vegetable fibre consumption and coronary events].

- Is p=0.03 non-significant?
- If non-significant why is there an association when in other instances when non-significance is indicated as no association?

*8.58 "men but not women" and "Adequate evidence"

- Why was there no meta-analysis with covariate for the fraction of the sample populations that were men or women?
- It is unclear whether the evidence is adequate:
- The meta-analysis took no account of gender.
- All studies appear to have been included without first having fully examined (I^2=0 or non-significant) or explained (I^2>0 or significant)
- It is unclear whether an adequate account has been made to verify the FFQ in each study and whether outcomes are dependent on suitability of the FFQs.
- The errors will tend the combined mean towards the null.

8.65 "No association [legume fibre and type-2 diabetes]"

- Some qualification is probably warranted.
- The three studies reported are likely problematic in respect of suitable validity of their FFQs.
- A conclusion of insufficient evidence could be appropriate; this if inadequate evidence would be seen as too nearly offensive.

8.73 "No association for Legumes intake(…) and cardiovascular disease events"

- Meta-analysis with covariate as gender fraction as male (or as female) should have been examined.
- Sex as a source of heterogeneity can result in no significant association for the combined population, even when there is an association in both sexes, unless sex faction is examined simultaneously as an explanatory variable.

8.88 "No effect" and "Adequate evidence" [for Oat bran and fasting blood glucose concentration]

- The conclusion may be premature.
- Results on healthy persons with blood glucose <5mmol/L and >5 mmol/L should be examined separately with exclusion of regression to the mean (cf Livesey et al 2008[10]).

*8.94 “95%CI”

- It is unclear whether the energy values of the foods were directly determined or taken from food tables.
- If food tables, it is not clear what values were attributed to the energy values of fibre or products ingested.
- It is also unclear what the outcome is: change in energy intake or difference in intake between groups of difference in change in energy intake between groups.
- It is also unclear what study designs were used, some of which could have explained the large 95% CI values.
- Oat bran is an energy dense product and like muesli is easy to overeat without appropriate knowledge (some parallel with SSBs). It is unclear what the treatments were and what messages accompanied the study. One would expect provision of such a food as oatbran to increase energy intake so confounding the role of fibre if there was no appropriate message of restraint or if the consumer was unaware of the energy density of the product.
- Oat bran and B-glucan should be examined separately since one is more energy dense than the other.
- Altogether, insufficient information is presented to be confident in the conclusion.

8.96 “[multiple products for cereal fibre excluding oat fibre and energy intake]”

- The products are divers, so the singular conclusion is meaningless and potentially misleading for individual products.
- Confounding by resistant starch is unclear both in respect of fibre dosage or the energy density of the products.
- It is unclear what efforts were made within experimental design to tighten up on study error.

8.99 “An investigation into the heterogeneity demonstrated that the amount of reduction in transit times in response to wheat fibre is greater when initial intestinal transit times are longer and vice versa.”

- Among small studies, this could be due to regression to the mean. This reflects a problem with small studies.
- No information is provided about the context of these studies.

8.105 “Butyrate”.

- A number of studies suggest some brans elevate faecal butyrate and lower faecal pH in some circumstances.
- There is clearly heterogeneity in these responses (as assessed from the narrative).
- The circumstances of the beneficial responses has not been identified in the draft Report.

- A “No effect” conclusion may therefore be premature, with some forms being effective and others not.
- The combining of heterogeneous products is problematic.

Post 8.109 “Effect [for Cereal fibre and constipation]”

- The heterogeneity in responses suggests variable or conditional effectiveness.
- The “Effect” needs qualifying to identify in who or when the cereal fibre would be effective.
- Measures are needed for products with reliable effectiveness.

Post 8.111 “Wheat fibre and intestinal transit time in patients with constipation”

- The studies showing effectiveness are those showing effect on constipation.
- Some studies show no significant effect on constipation, it is unclear whether transit time is unaffected or not affected significantly in this context.
- If this is correct the conclusion here is biased towards those studies in which there is effectiveness on constipation.
- The “Effect” needs qualifying to identify in who or when the cereal fibre would be effective.
- Measures are needed for products with reliable effectiveness.

Post 8.113 “No Effect [for wheat fibre and colorectal adenoma]

- The conclusion is evidently incorrect.
- A contextual conclusion is needed.
- Effective in men, moderate evidence
- No effect in women, moderate evidence.
- The OR and 95% CI ought to be presented for women too.
- It would be useful to explain what defines “wheat fibre” so consumers can choose for themselves.

8.115 “Association [for cereal fibre and colorectal cancer]”

- Meta-analysis with a centred covariate ‘gender fraction’ would have been useful if the studies adequately span men and women; this especially given the result at 8.113.

8.119 “ No effect [for Breakfast cereal and energy intake].”

- Impact of breakfast cereal on energy intake is likely to depend on several factors.
- Museli type cereal is energy dense and may increase energy intake as people overestimate portion size.

- Bran type cereals are energy dense and may elevate energy intake as people overestimate portion size.
- The problem may be a balance between energy density on the one hand and fibre content on the other.
- No consideration appears to have been given to understand the heterogeneity observed.
- Because of the possibility of contextual results, it would be premature to draw a conclusion of "No effect"
- Because there are limited data to assess contexts, possibly 'Moderate evidence' is too emphatic.

8.125 'RR for total cereal and cardiovascular disease'

- The RR and 95%CI are not presented, which is undesirable.

8.127 and 8.128 'Cardiovascular disease and whole grains'

- It is unclear whether the study found in the update search that reported no effect of whole grains was included in the meta-analysis that now reports an association.
- All studies found should be included in the meta-analysis otherwise the reader might consider that there is reporting bias.

*8.129 Stroke

- Please define the types of stroke and their representation among the available data.

*8.131 Hypertension

- Please define hypertension as defined in these studies and the range of blood pressures across the populations.

*8.133-8.136 Blood pressure

- Please define blood pressure as defined in these studies, and the range of blood pressures across which the "No effect" applies.

**8.142 "Effect" and "Limited evidence" for Whole grains and energy intake.

- 75% of the 'whole grain estimate[of effect]' is contaminated by intentions to treat with decreased fat, and increased vegetable, fruit, and grains in addition to just small amounts of whole grains.
- It is surprising that a conclusion of "Effect" was reached for whole grains given such evidence; this even given the caveat.
- Limited evidence does not adequately describe the evidence presented.
- The Report's conclusion provides possible evidence of reporting bias.

- The multiple nutrient treatment study of Tinker et al should be removed from the meta-analysis, leaving a narrative result; narrative unless a literature search uncovers further study.
- Standards of reporting differ here (8.142) from elsewhere (8.144).

*8.144 "I^2=82%... too high to report results"

- I^2 too high; this can only be helpful in reporting results if the ratio of the combined mean random ES/tau^2 is small and non-significant.
- Why not present data for the population of study values, ensuring that the CI is correctly calculated for the population. (This can be done in Stata, not sure of other packages.)
- Heterogeneity is likely due to studies with FFQs of low validity.

**8.146 "No effect and Adequate [for fasting blood glucose]"

- Data should be considered separately for <5mmol/L and >5mmol/l as results when sufficient in number can be opposing, a result that cannot be considered as "no effect" but would be normalising.
- Adequate evidence?
- Data are contaminated with the study of Tinker et al (see comment against 8.142).

*8.147 "Adequate evidence [for whole grain and fasting blood insulin]".

- Data are contaminated with the study of Tinker et al (see comment against 8.142).
- It is unclear whether outcomes are related to the treatment average fasting insulin obtained in these trials, which is a likely outcome if sufficient data were to be collected from individuals without diabetes.
- The standard of evidence differs from those presented elsewhere where meta-analyses are deemed adequate.

*8.148 "No effect" and "Adequate evidence [for whole grain intake and fasting blood insulin]"

- Data are contaminated with the study of Tinker et al (see comment against 8.142).
- Weight loss trials and unrestricted energy intake trials should be considered separately.
- There are too few studies of a heterogeneous analysis/design to draw conclusions about unrestricted diets.
- Data are contaminated with weight loss trials, which are important but inappropriate for the generation of guidelines for maintenance of health.

*8.150 "No effect" and "Adequate evidence [for whole grain intake and insulin sensitivity/resistance]"

- Data are contaminated with the study of Tinker et al (see comment against 8.142).
- Data are contaminated with weight loss trials, which are important but inappropriate for the generation of population wide guidelines for maintenance of health.

- There are too few studies of a heterogeneous analysis/design to draw conclusions about unrestricted diets.
- Weight loss trials and unrestricted energy intake trials should be considered separately.
- The conclusions reached of No effect and Adequate evidence are therefore premature.

*8.152 “No association [for whole grains and colorectal cancer]”

- With just three studies it is not possible to assess sensitivity of the outcome to individual studies.
- With just three studies it is not possible to assess stability of the outcome.
- With just three studies and borderline association, the conclusion reached is premature.

Children and adolescents

*8.156 “No association [for dietary fibre and body fatness]”

- With just three studies it is not possible to assess sensitivity of the outcome to individual studies.
- With just three studies it is not possible to assess stability of the outcome.
- With heterogeneity in measures it is doubtful whether a common metric can be achieved.
- With one of the three studies apparently not considering dietary fibre intake, it is doubtful that it should be considered aside the other two for assessment of dietary fibre.
- A non-significant association is not the same as no association.
- It is unclear whether adjustments were made for major factors that could affect body fat.
- It is unclear whether exposures were adjusted for energy intake.
- Doubts exist in the literature about whether cohort studies are able to assess impacts related to energy value of the diet, especially that energy values have variably accounted for energy in dietary fibre.
- With just two/three studies and borderline association, and the plethora of uncertainties, the conclusion reached is premature.

Tables 8.1 to 8.3 “[for] Insufficient evidence”

- Revisions are needed to include studies for which the Report has premature conclusions.
- There are many intervention studies of fibre and body weight that have not been considered in this review.
- It is an oversight to have excluded many studies of isolated dietary fibres.
- No consideration has been given to conditions for which dietary fibre intake affects or is expected to affect energy intake and fat distribution.
- Potential differences of effects for under eating and over eating are not considered.

8.160 "Randomised controlled trials indicate there is no effect of dietary fibre intake on cardiovascular or type 2 diabetes mellitus risk factors"

- The conclusion could only apply to hypothesized risk factors
- Even then the conclusion must be limited to the risk factors that have been adequately examined.
- Not all conclusions of adequate evidence are correct in the present report.
- Conditionality of response of risk factors has not been adequately investigated, such as impact on blood glucose <5mmol/L and >5mmol/L at rest and in the post-prandial state.
- No consideration has been given to non-linear associations.
- No consideration has been given to the problem of thresholds of association/effect.
- There is a high risk that this conclusion may be written onto effects in type 2 diabetes patients for which there is evidence of effect of dietary fibre on risk factors.

8.158 to 8.162 [….]

- The results are far from definitive even for the available evidence. Deeper consideration of the data is essential.
- It would be better to not report than to report potentially misleading information. A more cautious summary would be appropriate. Even where benefits are suggested, the evidence provides information on neither stability of outcomes nor applicability to population sub-groups nor information on the proportion of the population or sub-population likely to respond with a benefit.

9.6 "evidence on non-digestible oligosaccharide or inulin"

- Please be specific, does this mean FOS and inulin, or does it mean inulin and oligosaccharides unrelated to inulin?

***9.8 "No effect" and "Limited evidence" for "Non-digestible oligosaccharides and fasting total cholesterol, HDL-cholesterol and triacylglycerol concentration"

- Information on inulin should be presented separately from that for other poly/oligosaccharides.
- The information reported for inulin appears to be incomplete.
- Brighenti, F et al 1999 inulin and blood lipids is missing
- Brighenti, F 2007 inulin meta-analysis showing decreased triglycerides and blood lipids is missing.
- Tovar et al 2012 inulin and triglycerides is missing.
- Many of nine studies in Wu et al 2010 inlulin-type fructans and blood lipids are missing.
- It is possible some of these missing studies may have been excluded on grounds of study inclusion/exclusion criteria, possibly study duration . Nevertheless , meta-analysis with a covariate for study duration would have been useful and possibly avoid conflict with the literature were effects have been shown.
- The conclusion of "No-effect" consequently appears premature.

- The statement of “Limited evidence” perhaps describes only that found by the Report’s authors and not that available in the literature.
- The search and analysis needs to be updated .

*9.11 “No-effect” and “Moderate evidence” for effects of oligosaccharides on energy intake

- Based on the comments presented here (at ***9.8) it is uncertain whether all evidence available has been found.
- Data on inulin should be presented separately from other oligosaccharides.
- Modified dextrin is undefined.
- It is unclear whether the primary purpose of these trials was to assess effects on energy intake, which if it were not, may mean the studies were not designed to address this question.
- It is unclear how much heterogeneity exists for the energy values applied to oligosaccharides or whether correct values were applied for assessment of energy intake.
- The observation has borderline significance, a significant effect may have arisen from a dose response met-analysis. Effects at low and high dose levels can be in opposing directions.
- For reasons presented here, the conclusion of “No effect” is premature.

9.12 to 9.14 “Fasting blood glucose” and “inulin” and “other oligosaccharides”

- Only five RCTs were identified that presented evidence on fasting blood glucose.
- The meta-analysis did not take account of different expectations for different levels of fasting blood glucose.
- “Inulin and “other oligosaccharides” should have been analysed separately.
- Many other studies indicate significant effects are expected for some oligosaccharides, although these are for non-diabetic Japanese men and women.
- The conclusion of “No-effect” is premature and potentially misleading.
- The conclusion of “Adequate evidence” is premature and equally potentially misleading.

9.15 to 9.17 “No effect” and “Moderate evidence” for fasting blood insulin.

- Given the extent to which the prior analyses of inulin do not represent the available literature, it has to be considered that effects on fasting blood insulin might be too.
- The conclusions of “No effect” and “moderate evidence” needs re-examination.

9.18 to 9.23 Non-digestible oligosaccharides and faecal weight.

- All non-digestible substrate must elevate faecal weight, even those that are completely fermented. Variance arises due to dose and extent of fermentation. Studies failing to show

effect are likely confounded or simply underpowered for the diet consumed. Some diets have higher day-to-day or week-to-week variability on 'basal' faecal weight.

- Dose response meta-analysis was not conducted though should have been.

9.24 to 9.25 "No effect" and "limited evidence" for short-chain fatty acids.

- No significant effect is expected at low to zero dose.
- Effect is expected at a sufficiently high dose.
- A dose response meta-analysis should have been conducted (the artificial limit of 10 studies being needed for dose response analysis is trumped by expectation of dose responsiveness when there are as many as eight studies (at least eight studies).
- The conclusion of "No effect" is premature"
- It is unclear whether or not the evidence is unduly limited.

9.26 Fructo-oligosaccharides and bacterium.

- The report is unclear about whether lower doses of FOS show no effect or show a non-significant effect or that too few studies had been conducted to be sure about an effect or whether there were no satisfactory studies identified on FOS taken at below 10g/day.
- Given the lack of success on finding studies on inulin (above), the successfulness of the search for relevant studies on FOS remains unclear.

***9.31 Inulin and faecal bacteria

- Given the lack of success on finding studies on inulin (above), the successfulness of the search for relevant studies on inulin in this section is unclear.
- Given that there was no attempt to weight observations from the various trials, it is unclear whether the "No effect" is a valid conclusion.
- It is unclear whether the effect of inulin is dependent upon molecular size and processing, some studies may have used polymeric inulin rather the oligosaccharide inulin. Some process potentially may degrade inulin to be more like fructo-oligosaccharides.
- Critical examination of studies is needed following a search that is thorough.

**9.32 to 9.34 Inulin and other non-digestible oligosaccharides, and calcium absorption.

- Other non-digestible oligosaccharides are not defined.
- Observations on inulin should be presented separately from other Non-digestible oligosaccharides.
- No-effect is observed in adults, contrast children and adolescents. It is unclear whether or not results in adults may be conditional.
- It is unclear whether the methodology applied in adults is the same as that in children and adults.

9.35 to 9.37 “No effect [for Retrograde resistant starch and energy intake]”.

- Study precisions were far too poor to draw conclusions about clinically significant effect sizes for any substrate including resistant starch [studies were not adequately powered].
- The dose, duration of treatment, and energy values applied to the resistant starch are unclear.

9.38 to 9.62 “No effect [for Retrograde resistant starch and faecal weight]”.

- Dose-response meta-analysis should have been reported.
- The response may depend on the origin or structures of the resistant starches and caution should be given about generalisation, especially given apparently incomplete analysis.
- Stability of outcomes have not been assessed.

9.43 to 9.62 “Effect [for Retrograde resistant starch and Faecal pH and short-chain fatty acid content]”.

- Insufficient information is reported as the Report describes a comparison of positive effects of unreported amounts of RS with neutral effects of specified amounts of RS in both 9.44 and 9.45.
- It is unclear whether overall there is a significant effect, whether there is a dose-response effect, and whether claim of effect is generalizable (“in general”).
- Whether or not the results from these studies are truly insufficiently comparable is not clear, no reason has been given for insufficient comparability and there is no evidence of attempt to generate a common metric, and no attempt to explain why contrary outcomes have arisen.

Infants, children and adolescents

9.49-9.50 Calcium absorption and non-digestible oligosaccharides.

- Observations on inulin and GOS should have been presented separately from ‘other’ non-digestible oligosaccharides.
- “demonstrated” in 9.5 is too powerful a term because the evidence is limited to “Moderate evidence” and for reasons given above under the heading “**9.32 to 9.34 Inulin and other non-digestible oligosaccharides, and calcium absorption.”
- Saying ‘because no evidence was found after combining 4 studies in adults’ would be more accurate, and saying ‘reasons for not finding effect in adults’ would be better.

9.51-9.53 Non-digestible oligosaccharides and infant faecal bacteria up to 3-mo age

- It is unclear whether the update search results were related to inulin (having said there is only one study on inulin this is highly relevant).

- The range of types of non-digestible oligosaccharides is not made known in the report.
- It is insufficient to say that six trials reported no significant effect without demonstrating that the studies had sufficient power to detect an effect.
- Aside the problem immediately above, were these individual studies in agreement over the direction of the non-significant effects?
- Were reasons for non-significant effect in some studies identified, such as methodological or inadequate dose, duration of study?

9.56 to 9.57

- "Content" is undefined, mg/d or mg/100g stool or other unit?

Summary and Conclusions

**9.59

- "no evidence" is ambiguous. Suggest 'no trials' or 'evidence not supporting'.
- "relevant health/disease outcomes". Be specific, either 'health or disease' or 'factors risking health or disease'. "Relevance" is non-specific, apparently lazy, and suggests both unclear thinking and lack of systematic consideration.

**9.61

- There is evidence from within the report that the literature searches were not successful in capturing all available evidence. For example, there is one published meta-analysis on inulin that concludes a lowering of plasma triacylglycerols and another that suggests no-significant effect. The present study appears inferior to each of these, and really ought to have addressed the prior published meta-analyses and attempted conditional reasoning for discrepancies between studies.
- It is unclear whether information presented adequately fits the groupings "children and adolescents". References to earlier paragraphs would be helpful to create a link to the evidence and enable a check that what is stated is supportable.

**9.62

- Comments herein suggest the studies reported do not have the power to draw the conclusion reached concerning energy intake effects of resistant starch. Moreover, there is concern about whether the studies were sufficiently meticulous to address the question asked. The conclusion when reached is almost certain to be conditional depending on scope for caloric control via energy restriction or for unrestricted access to energy and for dose.

**9.63

- The summary concludes, amongst other things, that "The effects on faecal parameters demonstrate the colonic fermentation of these carbohydrates". However, the report presents no evidence on fermentability of these carbohydrates and therefor the summary

misrepresents the evidence. The same results could well be obtained by magnesium sulphate that is non-fermentable, or even the drug metformin, which we would not explain as due to its fermentability. Moreover non-fermentable carbohydrates are expected to increase faecal weight and acidification may be due to shorter transit time. Far from "demonstrating" the results provide no evidence on fermentation.

- The language used is unhelpful to either consumers or health professionals. "symptoms" to many ley persons usually implies a disease process, but there is no suggestion from the data that there is any such process or safety issue [13].
- The last two lines of para 9.63 ("; equally….") add nothing. Already it is questioned whether there would be health outcomes. It would be preferable to state that 'elevated bifido, faecal weight, and butyrate are potentially of benefit to health, but for the present such benefits are largely hypothetical'. (This applies also to many 'markers' of cardiometabolic risk.)
- The search criteria does not include bifidobacteria (or other micro-organisms) or short-chain fatty acids, it would be reasonable therefore to assume all caveats arising in the draft report related to these measures of outcomes might have only limited justification.

9.64

- "relevant" is lazy, undefined. Please state which health outcomes.
- The paragraph is long and states very little. Indeed it would add nothing that could not be achieved with minor modifications of the earlier paragraphs (as suggested herein).
- Loss of the last half of the paragraph would be no loss to the science and would be essential for having a punchy summary; it would also eliminate what might be reporting bias.

Chapter 10 Glycaemic index and load

*10.3 "measures of the glycaemic characteristics of the diet"

- Strictly, they are measures of the glycaemic characteristics of foods used to estimate the glycaemic characteristics of diets.

*10.3 "The GI is a relative measure of the plasma glucose response induced"

- Strictly, 'since standardisation GI is a relative measure of the capillary blood glucose response induced …'

*10.3 "quality and quantity of carbohydrate"

- This one may seem pedantic, however, it is the 'quality of the carbohydrate food/meal/ingredient and the quantity of carbohydrate in the food/meal/ingredient'. This recognises that GI is a measure for the food/meal/ingredient (not the carbohydrate) because

the GI is affected by non-carbohydrate in the food/meal/ingredient as well as the structures and composition of the carbohydrate in the food as eaten.

*10.4 GI and GL units

- Neither GI nor GL are unitless. Moreover, both are linked to a particular standard but the draft Report doesn't state which applies here (e.g. % of glucose or % white bread, and g/d or g/2000kcal etc).

*10.4 two GI unit increment….and…20 GL unit increase.

- Why 2 and 20? The SD's are reported in this para to be 5 for GI and 26 for GL. Isn't 1 SD the SACN standard for reporting for the project? Why is the data presentation biased in this way?

- Also, it ought to be recognised that adoption of SD values for the UK can give a false impression of the importance of GI and GL among and across other world regions because the SD value is greater worldwide and can be greater, too, in regions other than the UK.

*10.5 "The difference between these two types of trials is that the glycaemic index trials do not vary carbohydrate quantity, but change the quality to modify the GI. The GL trials reduce carbohydrate intake, resulting in a higher proportion of fat, often including saturated fatty acids, and/or protein intake, as well as changing the carbohydrate quality to modify the GI"

- Although the paragraph may appear clear, in the context in the Report's mention of effects on macronutrient intakes, weight loss and confounding of GI and GL trials by weight loss, the paragraph and immediate following sections give a false impression of GI and GL and how trials can modify these quantities, macronutrient intakes and body weight .

- GL trials can aim to modify GI, protein, fat, fibre, etc. etc. GI trials modify GL only by exchanges of foods of different GI and carbohydrate content. Such GI trials also aim to balance changes in protein, fat, fibre, etc., with the specific objective to balance differences in composition between foods of lower GI used in place of foods of higher GI.

- As a side issue; this balancing act might not take place among free-living persons when choosing lower GI in place of higher GI. Even in studies aiming to achieve such balances they can fail. Thus outcomes depend on the circumstances: (Livesey et al 2008 [10, 14]). Thus lower GI trials of ad libitum food intakes have been associated with lower energy intake (from available carbohydrate, protein, and fat) but not lower dietary fibre intake. Trials of lower GI under conditions of controlled energy intakes have shown only minor changes in diet macronutrient intakes. Trials of intermediate levels of control of food intake show intermediate effects [14].

- The Report's comment that trials on GI and GL induced some weight loss may be used in the Report unduly critically. Reduction in GL can induce weight loss as shown in randomised controlled trials [10]. Potentially this is a part of the mechanism (not a real confounder). Nearly all dietary trials result in weight loss – likely more so among persons in an overweight environment and especially as they regain 'food consciousness', but also because where food selection is concerned, aiming for a new goal limits food choices - at least until the new approach to eating is learned.

10.6 to 10.7 "No association" and total cardiovascular disease events"

- Total cardiovascular disease events need defining here, even if defined elsewhere not found in the draft Report.
- What events were included? What events were excluded? Were FFQ adequately validated in each included study? Was exclusion of studies undertaken when the correlation for the FFQ was 60 or less, for example? Were studies included that did not demonstrate their own validation of FFQ (for example most EPIC study centres do not report independent validations)? Were studies of low validity for carbohydrate also excluded?
- Men and women may differ. Women being more susceptible than men for a GI-CHD relation, and perhaps men more susceptible than women for a stroke event, these perhaps tending to cancel out each other in a total cardiovascular disease all sexes combined analysis (or causing apparent non-significance even when there is a real association). Mixing nearer no association with association would be a sure way to get borderline significance/non-significance as reported in the Report.
- Given the above it is questionable whether the borderline non-significance reported is interpretable. Mixing a near no association with association would be a sure way to get borderline significance/non-significance as reported in the draft Report.
- The conclusion of "No association" is wrong for the data available in the literature, this SACN activity definitely needs to be updated and executed correctly.

10.8 to 10.9 "Coronary events" and "No association"

- This needs to be defined even if defined elsewhere.
- The conclusion here differs from those in published meta-analyses!!!!
- It is certain that r the present meta-analysis has not been conducted adequately (cf above for total coronary events).

10.10-10.11 Stroke and GI.

- It is unclear whether the meta-analysis results includes the 2 studies in the update search and others since if published.
- It is unclear whether the literature is up to date (to within six months of the reports intended publication date).

10.12-10.13 Blood pressure and GI.

- It is unclear whether the meta-analysis results include the 2 studies in the update search.
- It is unclear whether the literature is up to date (to within six months of the reports intended publication date).

10.12-10.16 Fasting total-, LDL-, & HDL-cholesterol & triacylglycerol and GI.

- The discussion in 10.16 and in the boxed conclusions is somewhat lazy, it implies rejection of effects of GI on these blood lipids if secondary to effects of GI on bodyweight—without evidence. An unbiased approach would be to state that 'the effects may be primary to reduction in GI whether directly or indirectly via effects on body weight. Effects on body weight may be confounded by factors other than GI.' Etc.

10.21-10.22 "(C-reactive protein)".

- No meta-analysis is mention. It is unclear whether meta-analysis would reveal a significant effect, which is one objective of meta-analysis, to improve power of observation to a greater level than in small studies with non-significant results.

10.23-10.24 "Eating motivation" , "No effect" and "moderate evidence".

- So many factors affect eating motivation. To date all such studies (whether or not about GI or GL) appear to lack sufficient power to yield stable and clinically relevant effects.
- A claim to no effect needs to be qualified.
- A claim of moderate evidence fails to recognise that study protocols have not yet reached a suitable stage of development to address low but potentially meaningful differences in eating motivation.

****10.25-10.26 "Type-2 diabetes" and GI

- The analysis includes results from some studies rejected by other meta-analysts for inadequate FFQs.
- Inadequacy of FFQs relates to their validity (poor correlation between FFQ used and a better measure for the food component/factor under study).
- If there is a poor correlation during validation, there is a greater likelihood of poorer correlation, and higher risk of confounding, when attempting a correlation with incident disease, such studies are generally biased to the null.
- Several; of the studies also has FFQs that were not validated within the population studied, so has a doubtful FFQ.
- Most studies did not validate their FFQ for GI, though did validate the FFQ for carbohydrate, which is important in that diet GI is weighted by carbohydrate intake so requires FFQs to be adequately validated for carbohydrate at least.
- It is unclear whether GI values used in the meta-analysis are adjusted for energy intake according to Stampfer and Willetts method. If not they will be biased towards null.
- For any one of the above reasons a meta-analysis result can be rejected, thus thee are several reasons to reject the meta-analysis result in the present draft Report..

- From the forgoing, the Report result will be biased towards marked underestimation of the role of GI in prevention of type-2 diabetes.

****10.27-10.28 “Fasting blood glucose” and GI, and “No effect”

- It has been established (independently of regression to the mean) that lower GI and GL can elevate fasting blood glucose in those persons with rested and fasted morning plasma glucose <5mmol/L, but lower it in those with fasting blood glucose > 5mmol/L (online supplement to Livesey et al 2008 [10]) – thus it appears that low GI is normalising og blood glucose.
- It is not surprising, therefore, that combing results from all such studies reveals little effect, as indicated in the ‘meta-analysis’ conducted for the draft Report.
- The conclusion of no effect is therefore premature, and for the present can be rejected.

****10.31-10.32 “Insulin sensitivity” and GI, and “No effect”

- There is no mention of attempts to create a common metric for these studies.
- There is no mention of any temporal effects, since earlier studies of shorter duration generally indicate improvement.
- It is unclear whether duration of intervention is a significant factor in these studies.
- It is possible that increasing duration of study associates with lowering of power of these studies (error become larger over time).
- These considerations are significant because small effects in the right direction may mount-up over time.

*10.33-10.34 “Colorectal cancer” and “No-association”

- It remains to be established whether a no-association is due to inadequate FFQs (cf comments immediately above).
- The issue of adequacy of FFQs may also apply to studies of ‘total or available carbohydrate’ intake, and possibly other nutrients [2, 4, 15] .

Glycaemic Load

*10.35-10.36 “Total cardiovascular events”

- Total cardiovascular events is undefined.
- It is unclear which variable are confounding.
- No attempt is described to eliminate potential co-variables.
- It is unclear whether the comment that “it is not possible to exclude confounding variables” has specific information supporting it or whether the comment is just a repeatedly lazily stated bias against prospective cohort studies in favour of intervention studies or whether it is stated whenever there is a feeling of bias against the effectiveness of an intended nutrient of dietary factor. Because nutritional intervention studies are difficult to fully control, the issue of confounding arises there too but is often overlooked or

suggested is negligible, but upon detailed analysis can be found statistically significant and of importance (Livesey, et al 2008 [10]).

*10.39-10.40 “Fasting total-, LDL-, HDL-cholesterol and triacylglycerol” and GL

- The results are most pertinent to those striving for weight reduction. The majority of the population is ‘walking’ into weight gain.
- Thus Reports results are not relevant for the majority of the population until they start to take measures to lose weight.

*10.41-10.42 “(C-reactive protein)” and GL

- Too few studies were analysed to establish stability of this result.
- “No- significant effect” is meaningless if studies were underpowered or not representative, so more detail needs to be presented to be convincing.
- No meta-analysis appears to have been conducted.
- Studies may have been too short in duration. At least one epidemiological study in initially healthy persons (and one RCT in healthy persons and one intervention study in T2DM modifying GI) has indicated clinically significant association over the longer term.

****10.43-10.44 “(Body weight)” and GL

- Too little information is presented to claim no-effect.
- Studies of short to long (12 mo) duration have previously shown effect when meta-analysed with time as non-linear covariate, and when GL reduction is sufficient, and vice versa (Livesey et al 2008 [10]).
- It appears probable that collection of insufficiently data and performance of inadequate analysis could be the problem underlying the claimed no-effect.

****10.45-10.46 “Type-2 diabetes” and GL

- GL has units, a statements of unit/day is somewhat lazy reporting. Unit is not expressed.
- Moreover, original studies report g GL that are adjusted to a mean or median reported energy intake, which varies among studies. This expression (g GL reported/amount of energy reported) does not have the errors implicit in g/day for data collected from food frequency questionnaires.
- A comprehensive meta-analysis of more studies than in the SACN draft Report has already been published (Livesey et al, 2013 [3, 4]) but not referred to in the Report, finding:
    - Association, RR 1.08 per 20 gGL/2000kcal – average for men and women
    - Significant in both men and women.
    - Heterogeneity reduced to 2% by three out of four pre-published hypothesized factors:
        1. Significantly higher RR in women than in men.
        2. Significant dependence on the FFQ correlation for carbohydrate, implying the studies have markedly underestimate the importance of GL.

3. Ethnicity, significantly higher values in studies of European-Americans versus all other ethnicities combined.

- No significant effect of duration of follow-up due to instability about this factor (inadequate number of very long term studies, >15y).
- Significance of effect at all doses >95g GL/2000kcal.
- Stability of outcomes over increasing number of studies (except for duration of follow-up >15y).
- Stability against a wide range of potential confounders that were explored.
- Discussion that reduced GI could achieve sufficient GL reductions except at very high intakes of GL when carbohydrate reduction would also be required to meet an optimum target GL of 100g/2000kcal—chosen as a rounded value closely above a lowest point of significant effect on the dose-response curve.

****10.47-10.48 “Fasting blood glucose [and] No effect” and GL

- Really needs to consider <5mmol/L and >5mmol/L separately or in a meta-analysis with treatment average fasting blood glucose, fibre intake and GL dose modelled in.
- “No effect” is doubtful. Studies including shorter duration show significant effects of severity of abnormality of fasting glucose concentration (including <5 mmol/L), fibre intake and GL (or GI) as determinants in an appropriately structured meta-analytical model (Livesey et al [10])
- Combining all studies together (<5 mmol/L and >5mmol/L) can be suspected to average out as no effect among healthy persons (though sufficient observations are needed to show difference above and below 5mmol/L, such as in Livesey et al 2008 [10].
- For some purposes, analyses excluding pre-diabetes and diabetes from the analytical model has some limitation when fasting blood glucose can be considered as a continuum throughout the range. The exclusions of ‘pre-diabetes’ [term disliked] and diabetes make artificial cut points relevant to clinical issues rather than discontinuations in a continuous variable.
- It can be considered that the no-effect reported in the draft report could be due to insufficient detail and range of results in the analytical model.
- In addition, studies of too long duration (beyond achievement of steady state) may lose power compared with studies of moderate duration.
- In view of all the above, the reported “no-effect” in the draft Report might be misleading.

****10.51-10.52 “Insulin sensitivity/resistance [and] No effect” and GL

- Information is too limited to be convinced of a no effect.
- No effort seems to have been made to find a common metric.
- Modification of carbohydrate intake is expected to modify GL and in the short term influences insulin sensitivity/resistance.

10.55 Outcomes with insufficient evidence (tables 10.1, 10.2, 10.3)

- The criteria for deducing this is unclear, as sometimes no-effects are concluded when there is insufficient evidence, especially as currently presented in the draft Report.
- Being able to undertake a meta-analysis with sufficiently low I^2, and being able to establish stability of effect, each seem not to be among any criteria.
- The veracity of the lists is unclear. At least the searches performed are not up-to-date.
- The accuracy of Table 10.3 is doubtful. It may be more a matter of need for more evidence to apply appropriate analyses to rid the collection of studies of heterogeneity. Inconsistency may imply inaccuracy of studies, but it may be inaccuracy of the models used for analysis of the studies and/or having sufficient numbers of studies to reveal factors hypothesised as explanatory.
- Cumulative meta-analysis ought to have informed this table.

Chapter 11. Dietary reference values.

- The chapter is too long on sugars and includes too much unnecessary speculation.

11.3-11.4 Carbohydrate intake.

- There needs to be a further statement that there is some evidence that high intake may be detrimental to health. This arises from various sources, some of which are mentioned in the Report, others elsewhere (Livesey et al 2013 [3]) when examining glycaemic load), and still others in relation to plasma triacylglycerol, which has great relevance amongst advice for patients with type 2 diabetes when essentially all National diabetes association's include advice to cut by 10% carbohydrate intake and replace with MUFA.
- The analyses conducted fail to consider that too much carbohydrate and too much fat can each be detrimental to health, and that replacement of one by another then has no or diminished observable effect, whereas the real effect is to maintain detriment. Concern might be better focused on the quality of the carbohydrate food and quality of the fatty food, rather than the outdated fat versus carbohydrate debate.

11.7 high-fructose corn syrups (isoglucose).

- The report perpetuates a common mistake.
- Isoglucose and high-fructose corn syrups are not the same thing and should never be used interchangeable synonyms. The two should be listed separately.
- It would be appropriate to list product types which are essentially free sugars, such as free-sugar extracts and syrups (e.g. agave nectar, syrup etc.).

11.7 "it is proposed that the UK adopts the definition of 'free sugars'"

- Free sugar is sometimes used to mean monosaccharide (e.g. fructose not in sucrose).
- Free-sugars might also be confused with sugar-free, which already is in use in food labelling.

- The proposal is too strong, and omits the need for further consideration by regulatory authorities.
- It would be appropriate to recommend that the UK consider dropping “non-milk-extrinsic sugars” to be replaced by another term understandable to the consumer, that facilitates analysis, and that reflects recent knowledge about sugars and health.
- It would be further appropriate to recommend the UK considers adoption of other terms, such as added sugar as used in the EU.
- In selection of a term, it would be appropriate to consider a term that meets consumer understanding, such as ‘added sugar’.
- In selection of a term it would be appropriate to consider how the sugars, however called, would be analysed.
- It is not impossible that Added sugar could be used for consumer understanding, while being measureable as something else (e.g. some standardized approach to non-sedimentable sugars).
- Algorithms exist for the estimation of added sugars, those in use are not satisfactory which is breeding attempts for improved algorithms, but there are still problem foods.
- It is not a simple issue and it would be inappropriate to jump straight into change without further consideration: ’consider’ being the key term to use in any proposal here.

11.8-11.13 Advice on sugars.

- The advice makes no distinction between sugars in foods and sugars in drinks (only that in drinks, e.g. fruit juices and sugar sweetened beverages is associated with diabetes. It is unclear whether this outcome is linked to sugars or carbohydrate energy in drinks, artificially sweetened maltodextrin-based drinks might well do the same, ditto lipid-based drinks, and there is evidence that carbonated drinks without sugars are linked to diabetes type 2. It is also unclear whether the absence of milk drinks or the presence of sugary drinks that is linked to diabetes, especially that milk protein impact on glycaemia in both healthy persons and type-2 diabetes patients, while sugary drinks displace milk drinks.
- The advice regarding caries makes no distinction between the amount of sugars and the duration of contact of sugars in the mouth/pattern of consumption , the amount is not necessarily the issue.
- The presented rationale with respect to sugars is unclear in scope and includes suggestive and inferential statements (“suggests”, “implies”, “and thus”). Limiting the advice to only evidence-based narratives would be appropriate.
- There is little to put the data on sugars and energy balance into the context of obesity. There is no evidence that obesity is increased by more than 2-3 kg due to sugars consumption. There is no doubt that energy is involved but it is unclear in what way, and to what extent. Do sugary drinks increase energy intake when they are not prescribed to be taken daily? What is the evidence from sugar reduction strategies? Among populations does sugar reduction elevate fat intake?

Section 11.9 notes that "Although there is limited evidence relating to sugars intakes below 10% of energy intake, there is little reason to doubt that the relationship continues to be approximately linear at lower percentages of energy from sugars".

- Does this section need speculation to support the argument made. Reason (no evidence) has been given elsewhere herein.

Section 11.10 "This figure assumes no dietary compensation for the additional energy supplied in the higher sugars diets, which may not reflect true dietary behaviour and, therefore, the estimate should be treated with some caution."

- The speculation here is not warranted. The weight gains are well below expectations for no-caloric compensation. Indeed it is evident that >95% compensated occurs within 1 year (Livesey et al,2010[16]). Once again the draft Report's argument over relies on speculation. Speculation can never be a "useful guide".

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In 11.2: "Since free sugars intake is a dietary factor shown to increase energy intake [under conditions of prescription to increase sugars intake], decreasing the population intake of free sugars is a step that could be taken to help reduce the current UK over-consumption of energy".

- The question is: How can this be achieved effectively. Where is the evidence from practice? Where are the ≥12 month studies relevant to body weight, which is the primary need for compliance with the exclusion criteria.

- A clearer, focused, rationale would be helpful. A simple rationale is that added sugars are non-essential, are understood by the consumer, are an easily identifiable source of energy (on food labels when presented as added sugar), are consumed among the UK population which has a high prevalence of overweight and obesity and associated health issues, and can be reduced from the diet without known adverse effect. Governments should be doubtful about the extent to which this would yield valuable results nevertheless without being accompanied by negative views of fat (more specifically fat types), and these two messages alone would still allow negative effects of high glycaemic load from starchy foods. Also one study noted just below (Drummond et al 1998) indicates advice to lower fat and sugar intake together could be less effective for body weight reduction than advice to lower fat intake alone.

Under 11.12, Figure 1.

- The section speculates and the Figure includes inaccuracies.
- None of the studies demonstrated the development of obesity among normal weight persons consuming increased amounts of sugars.
- Studies demonstrate a potential to manipulate body weight by approx. 2 kg through manipulations of sugars intake, some studies showing elevation of body weight some shown a decrease in body weight, each compared with a reference group.

- The concern here is that overemphasising sugars as the cause of obesity will do little to help identify other causes so leaving the NHS exposed to unacceptable future costs aside the burden of overweight and obesity upon individuals.
- Figure 1 is raw, not presentable in the style shown. It gives the impression that the science might be similarly raw, which appears to be true in many places.
- Contains no evidence for the Report's claim that 5% sugars results in lower energy intake than 10% sugars.
- Saris 2000:
    - Conditions do not appear to be ad-libitum (Foods were provided. Intakes were limited between 75 to 125% of energy requirements. Non-compliers were excluded).
    - For simple versus complex carbohydrate diets, the difference in body-fat mass at the end of 6 months was about equal to the difference in body-fat mass at the start of the 6 months intervention. No effect.
    - Ditto for body fat-free mass. No effect.
- Poppit 2002:
    - Replacement of fat with sugar (and complex carbohydrate) was intended, but it seems the supervising nurse's encouragement to participants to eat more sugar resulted in dietary supplementation with sugar but still no effect on body weight. No effect.
- Raben 2001:
    - Raben 2002 only is listed in the reference list.
    - Raben 2002 provide a balanced and fair discussion.
    - Sugar intakes are excessive by any standard, approaching 30% energy.
    - There is no comparative control for seasons or for carbohydrate or fat supplementation.
    - Food intakes were assessed by questionnaire hence one has to be cautious about interpretation of food intakes quantitatively.
    - Sugar (energy) is compared with a sweetener (no energy). The prescription alone might elevate energy intake and leave energy balance positive until control of energy balance is re-established (approx. 1 year when weight gains remain marginal compared with the range of weights from normal to obese, as described for total fructose : Livesey 2010 [16]).
    - The study is too short in duration for individuals to fully adapt and to know the impact on energy balance in the longer term (including the giving up on other foods to which persons had habituated).
    - The results from Raben 2002 are said not to be in agreement with those of Saris 2001, but this is not apparent in the figure shown.
    - The authors claim that neither sucrose nor the artificial sweetener stimulated either sucrose or total carbohydrate intake as was suggested previously (author citations 7,10).
    - The conclusion in Raben 2002 is similar to that on wider evidence based reviewed narratively by Livesey 2014 [17]
- Drummond & Kirk 2003:
    - The apparent rise in energy intake with %E from sugars in the Figure is entirely due to differences in the energy intake at baseline!

  - Energy intake in the higher sugar group decreased from 9.70 to 0.39 (-0.31) whereas in the lower sugar group it deceased less, from 8.49 to 8.39 (-0.10). This would suggest the higher sugar diet decreased energy intake not increased it as claimed in the Reports Figure 1.
- Drummond 1998:
  - Food intakes were assessed by questionnaire hence one has to be cautious about interpretation of food intakes quantitatively.
  - Inference implied by Figure 1 of the draft Report for body weight differences due to difference in reported sugar intakes would not be supported by corresponding changes in body weight.
  - Differences in body weight between higher and lower sugars intakes at 6 months were less than differences in body weight at baseline.
  - The higher sugars (reduced fat) diet lowering body weight faster than the lower sugars (reduced fat) diet.
  - For lowering body weight, advice to reduce fat intake only was more effective than advice to reduce both fat and sugar intake.

- Overall, the evidence on sugars intake and obesity is suggestive, but evidentially it remains to be established that sugar really is more obesogenic than other major sources of energy—on average for either other carbohydrates and fats.

*****11.13 “It is recommended that for sugars]” “likely”. Applicable to carbohydrates, starches, and glycaemic index and load)

- Undue speculation exists even in the proposed recommendation.
- There appears to be a lack of understanding of the literature.
- The analyses undertaken have a very limited perspective on the context of carbohydrate consumption.
- No evidence was found in the report that showed 5% sugars intake if achievable would have the impact suggested. Clearly focused, zero speculation, and evidential conclusions are needed.
- There are three issues for which evidence exists: dental, obesity, type-2 diabetes. Primary mechanisms include acidogenesis, energy load, and glycaemic load respectively. Sugars are not unique in that starchy foods also contribute to all three. All three are conditional, efficacy or association being evident when too much carbohydrate is consumed over prolonged periods in the day when sugars (beverage and fruit juice drinks) or higher glycaemic starch (foods) are consumed.
- Efficacy or association due to displacement of fats by carbohydrate becomes evident when the glycaemic index of the carbohydrate is above or below average. When glycaemic index is average, no effect is seen, so carbohydrates appear inert when they are not.
- Also, replacement of detrimental fat with detrimental carbohydrate makes it appear that carbohydrates are without effect on (or associate with) health.
- There is greater need to limit the claims made both to “Effect/Association” and to No effect/No association.

Dietary fibre

*****11.15 Definition: “Therefore it is recommended that dietary fibre should be defined as all carbohydrates that are naturally integrated components of foods and that are neither digested nor absorbed in the small intestine and has a degree of polymerisation of three or more monomeric units, plus lignin.”

- The issues below (and no doubt additional issues of others) have not been considered. Consequently the definition as recommended may be premature i.e. not yet adequately thought through and can be rejected for the time present.
- All definitions of dietary fibre have problems, and the present one is no exception.
- The definition excludes “synthetic components” whereas SACN 2008 did not so exclude. However, would polydextrose be excluded?
- How would methods distinguish between inulin as an ingredient and inulin as naturally present in food.
- Should resistant starch be considered non-natural if generated by retrogradation?
- Should resistant starch exclude mono and disaccharides generated during transit of the upper GI tract from resistant starch as analysed in the laboratory, at present such are included in the resistant starch definition?
- While the draft recommended definition differs from those proposed elsewhere, which new method of analysis measures an amount limited by the recommended definition?
- Presumably, gums (e.g. guar) would be considered as dietary fibre?
- Would polysaccharides synthesised by bacteria (e.g xanthan) be excluded if the microorganism was not a traditional food.
- If increased faecal bulking is a criterion for defining dietary fibre, and this supported by a relation between faecal bulking and colorectal cancer, what relation has faecal weight to colorectal cancer independently of the laxative effect of fibre? Might some laxatives be more effective?
- What rationale exists in the draft Report for lignin being a component of fibre, no evidence is provided on relations to heart disease, stroke, type 2 diabetes or colorectal cancer? (Meanwhile perhaps lignin content is a better indicator for a better health outcome than dietary fibre itself.)

11.19 “[A] dietary reference value for fibre of 30g per day is proposed as it is an amount which was shown in the evidence reviewed to be associated with reduced health risk”.

- This recommendation is supported by the evidence, though it is unclear how this amount related to energy intake. Had the data been adjusted to a common level of energy intake for all studies a more precise and useful value would likely have arisen.

- Among these studies, do persons developing colorectal cancer experience increased degrees of constipation that they remedy by eating more dietary fibre in the years prior to diagnosis? What discussion in the report considers this element, which if true would not make fibre protective but indicative of higher risk only?
- Figures 2, 3, 4, the unnumbered figure after 4, and figures 5 and 6. It is a pity that non-of these figures includes the data as bubbles to show the relative positions and contributions from individual cohorts. Not taking time to present these is somewhat lazy even if it is difficult—remember this is of national and world-wide importance. The 'dose markers' above the dose axis fail to communicate this information.
- Interpretation of non-linearity is problematic. Is it due to uneven distribution of weights, an outcome of having first applied log transformation (important for these risk analysis) then transforming the data again as geometric means, or is it due to some interaction not identified. How much heterogeneity is removed by the cubic or polynomial that might be due to some unidentified interaction?
- When, earlier, data were first examined for the Institute of Medicine in about 2002 (USA), linear associations arose with minimal heterogeneity, so why now the curvature? Might it be that some studies cover a narrow range of fibre intake, so explaining curvature overall, but this may then be an artefact that hides heterogeneity.
- Figures 2, 3, 4, the unnumbered figure after 4, and figures 5 and 6. Presumably the confidence intervals are for the fitted regression and not for the population of cohort values. Why do the figures not have appropriate legends. Why do we see copy-and-paste jobs rather than pastes from original computerised drawings?

****11.20 "may have additional benefits"

- The claim is ambiguous and might be taken to mean the fibre-health relations are established as due to non-digestible carbohydrate in fibre. However evidence is unclear that it is due to non-digestible carbohydrate, since, for example, dietary fibre from fruit and vegetables may not be protective in some health cases.
- However reasonable the claim might seem it is speculation ("may") that ought not to be there.

11.21 "no laxation trials" , "as a proportion of the adult dietary reference value" and "multiple of 5"

- Please be specific, do you mean laxation trials or do you mean no trials of effect on faecal weight. If the latter then laxation is not meant. If laxation is meant, then please be specific about the measure, e.g. transit time or even effect on water content of faeces?
- Please be specific 'as a proportion of the adult dietary reference value for energy' if that is what is meant.
- Please be specific, numbers sometimes have units: 'multiple of 5 g/d'.

11.22 Recommendations: Definition and limited "method 2009.01"

- There was limited discussion on this in the forgoing paragraphs. The recommendation needs further consideration for reasons outline above and by wider range of stakeholders and regulators. The proposal can nevertheless be offered up for further consideration.

11.22 Recommendation "[Dietary fibre DRV at] 30g/d"

- There is no discussion of whether this is applicable to both men and women. As for children, it might be pragmatic to assess different values for men and women on the basis of the relative energy DRVs. It's a pity that we have no risk data for men and women separately. Why?

11.22 Recommendation Methods "2009.01" or "985.29" and "991.43"

- Unclear/confusing: The first bullet point implies method 2009.01 should be used for dietary fibre analysis, meanwhile the second bullet point implies methods 985.29 and 991.43 should be used.
- The draft report includes no substantive discussion on methods choice. Consequently it is inappropriate to even begin to include the analytical methods within the recommendations.
- Perhaps the shortest route through this is to tabulate the methods used by each publication for the cohort studies that yielded the evidence, which is what might have been done behind the scenes, but needs to be in the Report, then simply report which were used rather than making a recommendation.

11.22 "No specific recommendation is made for children aged 2.0 years and less, due to the absence of information, but a diet containing increasing amounts of whole grains, pulses, fruits and vegetables is encouraged in the context of a normal growth pattern."

- This is beyond the scope of the presented analyses.
- It should instead be recommended that an experts body address this issue.

11.22 "Dietary fibre intake should be largely achieved from a variety of foods, such as whole grains, pulses (e.g. kidney beans, haricot beans, lentils), potatoes, vegetables and fruits, where it is a naturally integrated component."

- There is limited analysis presented for a role of fibre from the different food groups.
- It is doubtful that emphasis should be placed on potato, which associated with greater levels of type 2 diabetes, and likely CHD in women.
- It is doubtful that fibre from fruits contributes to much of the fibre benefits for type-2 diabetes and CHD, and fibre from vegetables only a little more if any.
- The report ought to be stating some of such information and only including in recommendations the information derived from the reviewed science.

Chapter 12 Overall Summary and Conclusions

*****12.2 "In accord with the SACN *Framework for Evaluation of Evidence*, strict inclusion and exclusion criteria were applied in the systematic reviews to ensure the evidence considered was of sufficient quality to be able to draw sound conclusions (SACN, 2012).

- It is clearly evident that the criteria are neither strict nor adequate in regard the prospective cohort studies as is particularly evident for "carbohydrate". Since drawing up the guidelines there is now significant additional confirmatory evidence on the adequacy of food frequency questionnaires (FFQs) and hence prospective cohort studies using them. It is evident that the use of FFQs with low correlations between carbohydrate content of food intake assessed by FFQ compared with more accurate methods yield results strongly biased towards the null. This was identified by (Brunner et al [15]), shown to matter for carbohydrates by Barclay et al [2] and confirmed and established as highly significant cause of strong bias to the null during a comprehensive dose-response meta-analysis (Livesey et al 2013[3]). The Report includes meta-analysis of prospective cohort studies without examining the sensitivity of combined outcomes to the size of the FFQ correlation.
- Through not taking this issue into account, we can no longer consider the assurance given within the quoted statement extracted (above) that the draft Report has excluded studies low in quality.

12.4***"carbohydrate, glycaemic index, and glycaemic load"

- It is unclear why there is negative focus in the draft Report particularly on carbohydrate, glycaemic index, and glycaemic load. The same negatives apply to dietary fibre intake, whole grain intake and added sugars intake, all of which are imprecise 'measures' and reveal considerable conditionality in their associations with incident disease while can be accompanied by varied intakes of other micro- and macronutrients and phytochemicals. There is no review of this issue in the draft Report to justify the negative focus identified, so that reporting bias and speculation are not excluded. Its fine to have statements about the nature of problems, it is wrong to target individual examples without proper analysis of the issue. Among the Report's presented meta-analyses, none were encountered to have addressed this issue analytically.
- Not mentioned is that a major downfall of intervention studies is that long-term studies tend towards convergence of treatments and controls.
- Not mentioned another major problem with the consideration of intervention studies and their meta-analyses, is that it is often not stipulated whether the analysis is for a rate of change over a defined period or for a new steady state.
- Overall the draft Report reports negatively rather than on balance. There are few statements of advantages. For example, population based studies concern relevant doses, while intervention studies may not do so. By contrast, intakes of particular nutrients or nutritional aspects may be uniform across the population, making the range of intakes too small to see a significant association even when one actually exists.

Total dietary carbohydrate

12.6-12.8 “High heterogeneity” “carbohydrate […] neither detrimental”

- “Carbohydrate is not detrimental” is a statement that is very likely incorrect and can be easily abused . There is some evidence in the Report that high intakes associate with detriment. Just because these are not reached by 95% of the population in the UK does not mean the statement is true everywhere, or for everyone in the UK. Nor does the statement apply to total carbohydrate for all foods. Also it applies to diets only. Some fair caveat would be appropriate to avoid abuse of the statement. We have not to forget that replacing detrimental fat with detrimental carbohydrate can result in studies showing no effect. Evidence exists in the literature if the review team care to look for it.

Sugars and sugar-sweetened foods and beverages.

12.9 “beverages”

- Not all sugars are sufficiently acidogenic to be cariogenic, isomaltulose is an example [18].
- According to the classification of carbohydrates as presented in the draft Report, polyols are sugars, and these too are not sufficiently acidogenic to be cariogenic. The solution here though is to correctly classify polyols as a category apart from sugars (and to avoid the use of the term sugar alcohol, which for lay and other persons is a highly misleading name). (see comment against para 2.1)
-

****12.12 “higher intakes of sugars”

- Lazy speak? A higher percentage of dietary energy as sugars is in the figure that is reported. Please be clear about the science.
- It is further unclear whether the studies did not prescribe a higher energy diet in the first place (i.e. calories versus no calories, which would elevate the percentage of total energy as sugar too), thus it is unclear whether sugars per se are problematic as stated. Similar studies prescribing higher energy intakes of solid foods not sugars also lead to a higher energy intakes, body weight and BMI. Some consideration of the data is needed so that appropriate caveats can be made to reflect the science and inform appropriately.

12.15 Starch and starch-rich foods

- Potential exists for abuse of the statement. Starch contributes to higher glycaemic load diets which are detrimental to health in regard incident type 2 diabetes (Livesey et al, 2013[3]). An appropriate caveat is needed against the overall conclusion.

12.16 Dietary fibre

- A caveat is required because the associations found for whole grain can be due to greater amounts of dietary fibre; this especially that cereal fibre has a stronger association with incident disease than has dietary fibre from either vegetable or fruit.
- A claim to indicate no association for dietary fibre and body weight has a reasonable probably of being incorrect:
    - Body weight reduction due to high fibre is of similar magnitude of effect as is body weight gain due to sugar consumption, and perhaps of greater effect on a g/g basis. Firstly, there are many studies in total that find an association or effect, and these far outweigh those finding no effect—short, medium and long term studies show consistent results. Second, intervention studies can indicate no-significant effect when too little dietary fibre has been administered. Third, fibre expands on food structure to limit intake, and limited intakes result in apparent food energy values of fibre to be lower than when energy intake is unrestricted by higher fibre. Fourth, prospective cohort studies in regard energy intake are limited in precision by not being able to adjust for energy intake in the same way as for other nutrients, the results remain unadjusted and less precise and so are are biased to the null, which might partly explain the finds in this Report. Fifth, high fibre diets cannot be used in cystic fibrosis because of the difficult of administering sufficient energy for growth. Sixth, a high fibre food (gruel) is known to limit growth of the very young.
- It would be better to avoid conclusions which are in doubt.
- Perhaps what is meant is that body weight did not appear to confound benefits of dietary fibre found on incident disease.

12.19 “health/disease outcomes”

- The author of this phrase might know what is meant, but it is ambiguous to the reader. It would be understood if ‘incident disease’ was said (an option for disease prevalence does arise as relevant studies do not feature among the studies reviewed).
- A problem here is that the conclusions relate mainly to difficulty in the reviewers finding relevant papers.
- A further problem is that the conclusions relate to strict criteria excluding useful data. For example, the conclusions do not speak for studies of marginally shorter duration. Inevitably the choice of study duration used in the criteria is somewhat arbitrary, (not too short and not too long, while what is too short and what is too long are issues of judgement rather than strict science).

12.20 “…..”

- There appears to be undue overlap with 12.19.
- “unclear”, “but”, “unclear”, please clarify. Be specific about why the EU authorisation is of doubtful value in combined mines of the review team. If a coherent reason is not possible, it would be preferable to not raise an undefined issue.

Glycaemic index and load

**********12.21 “There is no evidence from prospective cohort studies to suggest an association between glycaemic index and cardiovascular disease or coronary heart disease.”

- Wrong, totally wrong.
- The conclusion would be stunning if it were not for knowing the review is well out of date, even then it is very highly surprising that the earlier studies were not found.
- Published meta-analysis of prospective cohort studies show a strong association for CHD and glycaemic index and load in women, with no significant association in men. The lack of effect in men might be attributed to several things, possible higher levels of alcohol consumption; possibly poorer reporting on FFQs when conducted on large numbers of participants compared with smaller numbers in FFQ validation studies.
- Combined studies for the mixed-sex population with dummies centred on 0 for gender has potential to retain a very significant relation between CHD and both GI and GL for the population as a whole.
- Combined studies for the mixed-sex population with a sex-fraction covariate is needed for examination of population average associations.
- One might in addition note that since there is a well-established association with type 2 diabetes, there is a high risk expected for CHD and GI and GL owing to CHD risk being more sensitive to perturbations in HbA1c than is the risk of type 2 diabetes.

*****12.21 “Glycaemic load is associated with a greater risk of cardiovascular disease”

- Even though there were a small number of studies captured in the search performed, there are more now. Consideration should be given to exclusion criteria, studies with inadequate FFQs should be excluded.

*****12.21 “The available evidence does not suggest an association between glycaemic index or load and colo-rectal cancer incidence.”

- The review is out of date. New meta-analyses are required.
- The review took no adequate account of adequacy of FFQs, a new meta-analysis should do so.
- The review for GI and GL should , where possible, ensure data entered is energy adjusted according to Willett’s / Stampfer and Willett’s method in the original studies, and importantly, too, across the studies towards a common energy intake, e.g. 2000kcal.

*****12.23 “……..”

- What is given is unnecessary reporting bias.
- For diets, GI and GL inform about a domain that associated with risk/benefits that are not accessed by the other carbohydrate components reviewed in the draft Report. It is not intended that GI or GL be used alone as the indicator of a healthful diet (as often seems to be wrongly implied elsewhere), rather GI and GL is applied within the context of what is deemed healthy food-based advice. It should be further recognised that healthy food-based

advice is not optimal for identifying higher versus low GI or GL foods, despite the occasional opinion claiming that it does so (analysis shows the contrary).

- The bias expressed in the report would limits a consumer's ability to identify an optimum diet, prevent appropriate dietary choice (as well as free choice), and unduly worry many type 2 diabetes patients and others world-wide who apply GI and GL by choice to benefit their own condition.

Dietary carbohydrate recommendations

*****12.24 "carbohydrate, glycaemic index, and glycaemic load"

- It is unclear why there is such negativity focused particularly on carbohydrate, glycaemic index, and glycaemic load. The same negatives apply to dietary fibre intake, whole grain intake and added sugars intake, all of which are imprecise measures and can be accompanied by varied intakes of other micro- and macronutrients and phytochemicals. There is little or no consideration of this problem in the draft Report. Thus there seems to be a possibility of author bias and speculation.
- Not mentioned is that a major downfall of intervention studies is that long-term studies tend towards convergence of treatments and controls.
- Not mentioned another major problem with the consideration of intervention studies and their meta-analyses, is that it is often not stipulated whether the analysis is for a rate of change over a defined period or for a new steady state.
- Overall the draft Report reports negatively rather than on balance. There are few statements of advantages. For example, population based studies concern relevant doses, while intervention studies may not do so. In contrast, intakes of particular nutrients (or nutritional attributes) may be uniform across the population, making the range of intakes too small to find a significant association.
- Further, the range of nutrient intakes (or attributes) in any particular country may be too low or too high compared with an optimal intake, so that 1SD change within a country might underrepresent importance of larger differences worldwide.

Glycaemic Index and Load

*****12.24-12.27

- The section repeats a great deal of what has already be stated in earlier sections. This section and perhaps the report as a whole should be cut in length to 50% its current size and restructured to give far better focus.
- The recommendation on dietary fibre definition needs wider consideration at a regulatory level.
- There is an absence of data and discussion on the gastrointestinal discomfort arising from the consumption of more dietary fibre.

- There is an absence of data and discussion on the prevention and relief from constipation with higher fibre food (friuts, vegetables and whole grains), and especially in pregnancy, post-partum, and older adults. Particular types of whole grain products may cause constipation. Optimum cereal forms for easing constipation may not be whole grain. Recommendation of potatoes as a good source of fibre is problematic because of the risk of type 2 diabetes.
- 12.32 The recommended dietary patterns were not investigated or not reported in the draft Report. It would be appropriate to set up an appropriate studies and review.
- 12.33 the definitions “low” and “high” have particular meanings in food regulations, such that the recommendations here would not make sense. What would seem acceptable is to say ‘lower’ and ‘higher’.
- Women in pregnancy deserve advice too, pregnancy is excluded but in due course there needs to be some explanation of where/what they should turn to for advice and some future commitment to review foods and diets in pregnancy.
- All too frequently, GI and GL attract negative comment because commentators think the scientific community present these concepts as primary health measures. They are wrong. GI and GL are one of several attributes of foods (and diets) that impact on health. In general, food-based advice has primary position, only within food groups is a GI or GL measure selected. No food-based advice (and no compositional based advice) has been devised to select an optimal diet since all food-based advice and compositional-based advice can result in food selections of only high or only low GI as well as only moderate GI overall and so also optimal and suboptimal GL. For the perceivable future, only when GI or GL is used with an appropriately healthy food-based selection process can optimal diets be obtained.
- Omitting GI and GL from choices of healthy nutritional advice is further suboptimal. Individuals choose their preferred approach to organising their diets. Unduly ignoring an important option limits the potential success of health measures in total.
- It is an uncomfortable to consider the UK continues to not recognise that the quality of fatty foods and quality of carbohydrate foods matter more than is evident among the UK’s health messages, food labels and food tables, most especially that the quantities of fat and carbohydrate together has proved difficult to control in the UK and other world regions

Commentary on Fructose

A3.1 “HFCS, also known as isoglucose”

- It might well be “also known”, but then HFCS would be wrongly known as isogucose. The two products are prepared differently. Moreover HFCS can have different proportions of glucose and fructose (from 45 to 90% fructose, though the latter is less common than 45 and 55% fructose versions) . By contrast, a G:F ratio of 1:1 is expected for isoglucose.
- The paragraph is overlong with inaccuracies.

A3.3 Fructose.

- The paragraph does not represent the wealth of published narrative and meta-analytical reviews on fructose (Livesey 2014 [17], Livesey 2010[16], Livesey 2009 [19], Livesey 2008 [20], Sievenpiper's group 2011-1012 [21-27]. Very long-term studies are needed, but need to account for seasonable variables, carbohydrate load, glycaemic index, energy intake (as appropriate).

A3.6 "Fructose" "There is only one trial" "men" only "The body absorbs free fructose and glucose, or the same sugars derived from sucrose and HFCS, in exactly the same way"

- Overall the review of the literature on fructose is weak and misses important narrative and meta-analytical reviews. There is a high risk of reporting bias.
- "Fructose" is also used in foods added as pure (solution, syrup or crystalline) fructose in drinks, these among appearances of sucrose, HFCS, isoglucose and variable mixtures in fruit juices.
- "in exactly the same way". This misses some important point!
- It can be recommended that the review is updated, and that the range of viewpoints on fructose is strictly kept in balance.

A3.8 "insufficient evidence"

- Because there is a larger literature than admitted in this review, it might be perceived that the review did not look sufficiently broadly at the available evidence. It would be improper to hide behind the criteria for study selection for substances with potential adverse effect.

Dental health

A4.35 & A4.38 etc: "15 days" study. "pH 3.1"

- There are many studies of 15 days and longer for other health effects and carbohydrates not mentioned in the review. Reason for including one at this point for a specific product type suggests inconsistency in the systematic approach to the review, albeit in the appendix.
- Some other studies mentioned may also not meet the inclusion/exclusion criteria. Too little information is provided in the draft report for the reader to know without checking the original publications. In other words, the review is not self-supporting, which raise an issue about transparency.

A4.1 to A4.45 all Dental Health sections.

- There is a great deal of published information on polyols and dental health. It would help to begin the Report with what is known and what is not known, and whether a not a not know needs to be known.

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ANNEX 1 Polyols

Find also polyols among the broader list of comments listed in paragraph order in the main article of comments).

Note than, for the present purpose, relevant sections of the SACN draft report are copied here, page by page, and into which modifications suggested and original text needing to be deleted are identified by background colour. Orginal page numbers in the SACN draft report can be found at the base of each page here (e.g. as **Page 3** at the bottom of the next page). **Notes** are given not as annotations for information to note and not for including in any final report.

Background colour key: *annotations or modifications suggested* and original text.

Chapter 6.

Sugars, ~~sugar alcohols~~ polyols, sugars-sweetened foods and beverages…….. 82

Note 1:

- Polyol is the preferred term to use (FAO 2003) and is used in food labelling in the UK and Europe. It avoids confusion for the consumer (and others c.f. EJCN 2007) who sometimes think sugar alcohols are either sugars or alcohols.
- The distinction is essential also for food regulations and other regulations, thus polyols are permitted in sugar-free products, i.e. they are not sugars nor are they alcohol incurring an excise duty.
- Limits for intake are set for polyols that differ from the limits (where stipulated) for sugars, emphasizing again the importance of not confusing polyols with sugars.
- An advisory is mandatory in labelling on excessive intake of polyols, but not for sugars.
- To emphasize, none of the Sugars definitions provided by the UK, USA, WHO and EU include polyols (cf also Table 2.4 of the draft Report).
- Thus also, polyols can be referred to as 'sugar replacers' because they are not sugars but have some useful physical properties in common with sugar (e.g. cooking, mouth-fee, and visual characteristics) and some better physical properties than sugars in foodstuffs according to application (e.g. some have greater and some lesser moisture uptake than sugars).

Table 2.1 Chemical classification of carbohydrates (FAO/WHO, 1998) with modification to acknowledge recommendations in FAO/WHO (2003) and to be compatible with food labelling in the UK and Europe.		
Class	**Sub-group**	**Components**
Sugars (DP 1-2)	Monosaccharides Disaccharides ~~Sugar alcohols/ polyols~~	Glucose, galactose, fructose Sucrose, lactose, maltose ~~Sorbitol, mannitol~~
Polyols (DP ≥1)	Monosaccharide alcohols Disaccharide alcohols Oligosaccharide alcohols	Erythritol, xylitol, sorbitol, Isomalt, lactitol, maltitol Polyglycitol
Oligosaccharides (DP3-9)	Malto-oligosaccharidess Non-digestible oligosaccharides	Maltodextrin Raffinose, stachyose, fructo-oligosaccharides
Polysaccharides (DP>9)	Starch Non-starch polysaccharides	Amylose, amylopectin, modified starches Cellulose, hemicellulose, pectins, storage polysaccharides (e.g. inulin, guar gum), microbial polysaccharides (e.g. xanthan gum), other hydrocolloids (gums)

Note 2: FAO/WHO, 1998 was not so expert on polyols (or sugars it seems) and not surprisingly made a mistake by thinking sugar alcohols were sugars. FAO/WHO (2003) corrected this error, but the mistake was perpetuated in FAO/WHO via EJCN (2006) presumably because the authors of the publication were not sufficiently clued up on FAO(2003) or about sugars and polyols. Fortunately, none of the arguments presented in the draft Report about sugars, as defined above, apply to sugar alcohols, which is not surprising as they differ in important ways including in food regulations in the UK, Europe, and elsewhere : see Note 1.

The amount of energy yielded from different carbohydrates in food, that are digested in the small intestine varies according to the molecular form i.e. glucose, disaccharides and starch, the actual available energy content per unit weight is 15kJ/g (3.6 kcal/g), 16kJ/g (3.8 kcal/g) and 17kJ/g (4.0 kcal/g), respectively (FAO, 2003).

Note 3: These are values written into FAO 2003, they are not accurate. They arrive from Australia where they have a number of inaccuracies. In the UK, for example the available energy is applied in labelling and food tables for glucose, disaccharides, malto-oligosaccharide, and starch is 15.7 kJ/ (3.75 kcal/g) *as mono-saccharide equivalent*. The value is to represent a mix of glucose, disaccharides and starch present in the average diet. Actual values are in kcal/g are 3.72, 4.1 and 4.2 kcal/g for glucose, disaccharide and starch respectively. The advantage of expression in the mode 'as monosaccharide equivalent' is that the molecular size affects the energy value per gram weight due to the difference amounts of 'water of hydrolysis', absent from starch but present in fully hydrolysed starch, i.e. in glucose.

2.18 "An available energy content per unit weight has been estimated as 8 kJ/g (1.9 kcal/g) for fermentable non-starch polysaccharide, 9 kJ/g (2.2 kcal/g) for resistant starch, 8-9 /g (1.9-2.2 kcal/g) for low and non-digestible oligosaccharides and 6-10 kJ/g (1.4-2.4 kcal/g) for low and non-digestible ~~sugar alcohols~~ polyols (Elia & Cummings, 2007)."

Note 4: Not all are 100% non-digested, upwards of 10% may be digested. This was an oversight in the Elia and Cummings paper in EJCN 2006, obviously one I should have spotted at the time.

~~Sugar alcohols~~ Polyol [See notes 1 and 2]

Colo-rectal health

6.37. Three randomised controlled trials were identified that presented evidence on ~~sugar alcohol (polyol)~~ polyol supplementation in relation to faecal wet weight (Van Es et al., 1986; Sinaud et al., 2002; Gostner et al., 2005). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p68).

6.38. Two trials report an effect of lactitol or ~~maltitol~~ *[wrong name]* supplementation on increasing faecal weight (Van Es et al., 1986; Sinaud et al., 2002). One trial reports no significant effect of the ~~sugar alcohol~~ polyol isomalt on faecal weight (Gostner et al., 2005). From these trials, ~~sugar alcohol~~ polyols intake is found to have a low faecal bulking effect with the resulting increase in faecal wet weight being 0.5-1g per gram of ~~sugar alcohol~~ polyol consumed. ~~Sugar alcohols~~ Polyols are likely to be consumed in small amounts in the diet.

Note 5: Since up to 50g polyol daily in divided doses is the max intake without watery stools , this might suggest faecal weight could increase by 25 to 50g or by approx. a quarter to a half more faeces. Because low- and non-digestible polyols also have potential to elevate water content of faeces, polyols can bring about rapid transit and laxation more effectively than sources of dietary fibre. Excessive intakes of polyols can relieve constipation, meanwhile some highly divided dietary fibres can block the large bowel causing constipations. Thus, it may be important to not consider effectiveness of low and non-digestible carbohydrates by their generation of faecal mass alone. Also not to forget that doubling dietary fibre intake will mean that we shall need to almost double the expenditure on sewage works, and there may be increased risk of ill-health attributed to such waste being processed or inadequately processed meantime.

~~Sugar alcohols~~ Polyols and faecal weight

- Effect
- Limited evidence
- The direction of the effect demonstrates that greater consumption of sugar alcohols is of ~~minor~~ potential benefit to health. bBecause of the limited presence of ~~sugar alcohols~~ polyols in the diet in the UK at the current time such potential benefits are limited . Some individuals may occasionally choose to consume higher amounts of polyols (e.g. as sugar-free boiled sweets) to promote laxation.
- The effect is potentially biologically relevant , especially for limiting the incidence and relief from constipation

6.38 “maltitol” and “isomalt”

- Evidence is claimed for the polyol called maltitol.
- However, the study of Sinaud et al 2002 did not study maltitol
- What the report claims as maltitol are hydrogenated malto-oligosaccharides and hydrogenated malto-polysaccharide.

6.39 Three randomised controlled trials were identified that presented evidence on ~~sugar alcohol~~ polyol supplementation in relation to faecal bacteria content (Ballongue et al., 1997; Gostner et al., 2005; Finney et al., 2007). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p86).

6.40 Two trials, report lactitol 20g/d and the ~~sugar alcohol~~ polyol isomalt at 30g/day to increase faecal Bifidobacterium content (Ballongue et al., 1997; Gostner et al., 2005) In the other trial supplementation of lactitol at 5 or 10g/day has no significant effect on faecal bacterial content in one trial (Finney et al., 2007).

~~Sugar alcohols~~ Polyols and faecal bacteria content

- Effect
- Limited evidence
- Whether the effect is beneficial or of biological relevance is currently unclear

6.41. Three randomised controlled trials were identified that presented evidence on ~~sugar alcohol~~ polyols supplementation in relation to faecal pH (Ballongue et al., 1997; Gostner et al., 2006; Finney et al., 2007). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p99).

6.42. Two trials report an effect of lactitol supplementation on decreasing faecal pH at doses of 10g/day or more. The other trial reports no significant effect of the ~~sugar alcohol~~ polyols isomalt on faecal pH (Gostner et al., 2006)

~~Sugar alcohol~~ Polyols and faecal pH

- No effect
- Limited evidence

6.43. Three randomised controlled trials were identified that presented evidence on ~~sugar alcohol~~ polyols supplementation in relation to faecal short chain fatty acid content (Ballongue et al., 1997; Gostner et al., 2006; Finney et al., 2007). The data were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Colo-rectal health review p99)

6.44. One trial reports an effect of lactitol supplementation on increasing faecal acetate and lowered faecal propionate content (Ballongue et al., 1997). One trial reports no significant effect of supplementation with the ~~sugar alcohol~~ polyols isomalt on faecal short chain fatty acid content (Gostner et al., 2006). It is not possible to determine the effect of the other trial as results are only reported from baseline and graphically (Finney et al., 2007).

~~sugar alcohol~~ Polyols and faecal short chain fatty acid content

- No effect
- Limited evidence

NB. It is unclear why the draft SACN report refers to faecal short-chain fatty acids when what matters is acidification in the colon and rectum, which is inevitable with lactate, acetate, propionate and butyrate production from fermentation. Acidification is inevitable and more readily indicated by the measurement of fermentation rather than by faecal pH reduction, a lack of which simply informs that the absorptive process is fasterthan the fermentation process so absorption is effectively complete or not. However, the authors of the report seem to suggest that fermentation is of no consequence.

~~**Sugar alcohols**~~ **Polyols**

6.69. Six randomised controlled trials were identified that presented evidence on ~~sugar alcohol~~ polyols containing gum and dental caries incidence in both the mixed and permanent dentition in children and adolescents (Finn *et al.*, 1978; Glass, 1983; Beiswanger *et al.*, 1998; Alanen *et al.*, 2000; Machiulskiene *et al.*, 2001; Szoke *et al.*, 2001). The data on measures of dietary exposure, caries incidence/prevalence and risk assessment methods were insufficiently comparable to enable a meta-analysis to be performed. No further trials were identified in the update search (Oral heath review p41-43).

6.70. Four trials report an effect of sugar-free chewing gum containing sorbitol, mannitol or xylitol in reducing caries incidence in comparison with not using a chewing gum (Beiswanger *et al.*, 1998; Alanen *et al.*, 2000; Machiulskiene *et al.*, 2001; Szoke *et al.*, 2001). Two trials report no significant effect of sugar-free chewing gum containing sorbitol and/or mannitol in reducing caries incidence in comparison to a no gum control group (Finn *et al.*, 1978; Glass, 1983). In the trials that employ a 'no gum' control group, it is unclear whether it is specifically the sugar alcohol or the act of chewing and the concomitant increase in salivary flow that contributed to the effect.

~~Sugar alcohol~~ Polyols and dental caries incidence in both the mixed and permanent dentition

- Effect
- Moderate evidence
- The direction of the effect demonstrates that use of chewing gum containing ~~sugar alcohol~~ polyols in comparison with not using a chewing gum is beneficial to oral health
- The effect is biologically relevant

Note 5: The need for long-term studies in assessing the risk of caries when using sugar replaces is especially doubtful. The role of acidogenesis and plaque formation in caries development particularly from sucrose is well established, yet the draft Report makes no reference to the use of polyols as sugar replacers to reduce the risk of caries development. Meanwhile there are multiple assessments of risk by way of metabolic markers for cardiovascular disease yet the predictive value of the vast majority of such markers is low and sometimes doubtful. Meanwhile markers of caries risk have very high predictive value and include the following, which are missing from the draft Report:

- Evidence of insufficient oral acidogenesis in humans studies from polyols alone compared with sucrose as a positive control.
- Reduced substrate for dental plaque formation in humans from polyols alone compared with sucrose as a positive control.

Note 6: The role of starch and starchy foods in acidogenesis is absent from the review.

Note 7: The absence of significant acidogenesis (pH ≤5.7) due to certain sugars is also not covered, for example isomaltulose.

Table 6.2 Insufficient evidence-randomised controlled trials

Risk factor/healthoutcome/measure	***Exposure***
Transit time	~~sugar alcohol~~ polyol

Note 6: There is little doubt that polyols will shorten transit time when sufficient is consumed. All low molecular weight substances must do this (irrespective on establishment of the kinetics), for example, magnesium sulphate, non-fermentable low-molecular-weight soluble polymers, fermentable polyols, each to the point of causing laxation when sufficient doses are consumed Livesey 2001 [28, 29]. Consensus is that "Each individual may experiment with intake amounts and make adjustments based on their own experience – as they may do routinely with everyday foods having the same effects when eaten to excess" as quoted from Bär, Barlow et al 2001 [13]. Studies outlining the use of polyols for improving transit (laxation) are reviewed by Livesey 2003 [29], which extends the recommendations from The Royal College of General Practitioners [30] who as medics focus on the polyols sorbitol (as medics have often done).

Annex 1 References that may be helpful in understanding polyols

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2. Livesey G: Health potential of polyols as sugar replacers, with emphasis on low glycaemic properties. Nutrition Research Reviews 2003, 16:163-191 http://www.polyols-eu.com/files/Publication.pdf.

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