SACN report ref	Comment	Name
General	While this report overall is acceptable, There is little or no comment with respect to pregnancy. I consider this to be an omission as advice to women in pregnancy is very varied. It should be made clearer from the outset of the report that pregnant women are excluded from consideration.	Doris M Campbell
General	1) The press release reflected the conclusions of the report and was generally clearly written. It was however disappointing that the emphasis on improved intake of fibre was rather sidelined in favour of the recommendation to reduce intake of sweetened drinks and fruit juices, rather than being given equal prominence. It would be interesting to understand why this occurred. In general people are more responsive to a positive message. 2) Much of the evidence supporting the recommendations is based on association between intake and outcome rather than effects seen from RCTs. The problems associated with conducting RCTs for disease prevention are well known and almost inevitably must use biomarkers of risk, the validity of which are also subject to debate. Is it acceptable that this level of uncertainty is not really conveyed in the overall recommendations? 3) The strength of the advice is reported to be upgraded by "Evidence from appropriately controlled experiments demonstrating one or more plausible and specific mechanisms in humans." (A2.21). It would be useful to add a bullet point to each table where a convincing mechanism has been identified as relevant in humans. This would help in the identification of areas where better mechanistic studies need to be undertaken. 4) All cohort studies are corrected for BMI except, I assume, those reporting on BMI as an end-point. Should this be made clearer in the summary document so that the message that being obese is detrimental to health is reinforced while at the same time highlighting that it is NOT a high proportion of carbohydrate in the diet that is a problem but just too much food. The evidence behind such a statement is supported and strengthened by the table at 5.121. This is key to counteracting so much misinformation available on-line.	Elizabeth Lund
General	**Pre-amble** The draft Report is welcome in its scope, though is lacking in execution. Overall, it appears the review activity has been too extensive for reviewers to attend to essential detail, and will largely be 3 (possibly 4) years out of date by the time of publication, which compares with 6 months—the accepted maximum for peer reviewed publications. Here this reviewer has limited his attention to two aspects which leave him surprised about the execution and analyses conducted (**Methodology**) and about an aspect of diet that is often handled particularly poorly, as is the case in the draft Report (**Glycaemic index and load**). Details of references	Geoffrey Livesey

	omitted from the draft Report are not provided here as it is not intended to perform the task allocated to SACN to undertake the literature searches necessary to bring both data and contemporary thinking up to date. In summary, the subject matter in the report is important to address, is impressive in breadth, though is neither comprehensive (in breadth or depth or update) nor duly accurate. Comments below are listed by paragraph numbers as used in the draft Report, and often are headed by quotations from the paragraph cited. Each paragraph may be addressed more than once. One additional set of comments is made at the end (headed **Last words on GI and GL**) which has great importance for aligning perspective with science. A conflict of interest statement appears towards the end of this document, and recent publications authored by Geoff Livesey that are relevant to the SACN draft Report and the comments made herein are listed.	
General	The report is based on self-reported intakes of carbohydrates and does indeed highlight the inaccurate method of self-reported measures of intake and mis reporting. So conclusions are being drown regarding intakes which may or may not be accurate. Of course this is the only available method open to us to measure carbohydrate intake but based on this fact shouldn't there be a push from research councils or the food standards agency (DoE) to develop biomarkers of carbohydrate intake that are indeed more accurate than self-reported intake measures and that can be used at a population level? Only when we know more accurately our intake of individual and total carbohydrate can we know at what level is beneficial and what is harmful.	John Lodge
General	1. The report represents a major achievement in reviewing the literature in this area and highlights gaps in the evidence as well as differences in terminology between studies. A grid summarising the findings for different outcomes for different carbohydrate components showing limited/moderate evidence and whether a beneficial/detrimental effect) would be useful. 2. A section on recommendations for future research would be valuable.	Geraldine Mcneill
General	It was of great relief to me that this report has been commissioned. As a newly qualified Dietitian I have repeatedly come up against conflicting information surrounding the quantities of carbohydrate an individual should consume. From my research it appears the answer to that question links with the individuals physiology and to some extent is unanswerable without careful monitoring of biological markers. I appreciate that Government recommendations have to be generalised to the general population and feel that it is my job as a Dietitian to ensure the outliers are given specific advice whether that be following a low fat or low carbohydrate diet. The overall overcome of this report suggests to me that weight loss is highly correlated with health improvements and that a one	Laura Cherry

	size fits all approach has not and continues not to solve the obesity and metabolic diseases so prevalent today. The report is by far from conclusive with its findings but I strongly believe that from this document the 'eat well plate' is outdated and no longer relevant for the general population. As most segments contain carbohydrate either in the form of starch, refined sugars, fructose or lactose. I worry that if this issue is not addressed by health professionals, the media and the internet destroy the reputation and faith that the general public have in the nutrition and dietetics associations.	
General	The scientific review seems thorough and far-reaching. A wide set of literature has been reviewed with a corresponding wide spectrum of outcomes (incident disease endpoints, biomarker endpoints and dietary effects). Much of the evidence reviewed was deemed as scientifically insufficient or conflicting. In particular the sugars target is based on limited effects on disease risk. The free sugars energy target is founded on reduction in risk of dental caries, and the dietary collinearity between sugars intake and energy intake. I would contend that setting targets on the latter relationship might be problematic in a public health context. The inference in the report is that a population reduction in sugars intake will help achieve reduction in energy intake. This emphasis on changing diet composition to effect lower energy intake is against a backdrop of no clear public health message about individual responsibility to harness overconsumption (reduce total intake of energy) and preventing obesity. In fact confusingly SACN increased energy intake requirements in 2012. Changes in food choice to limit dietary sugars intake may not be a magic bullet to curb overconsumption, and in fact the report confusingly advises that the sugars energy deficit should be made up from starchy foods and sugars from milk and milk products (12.26). Equally advice in relation to changing other macronutrient components (starch or fibre) without emphasis on total energy seems ill-founded. Secondly, the population average target for free sugars of 5% energy seems incongruent with an individual target of 10% energy. Are these two targets based on the current distribution of sugar energy in the population? Given current dietary patterns, behaviour change will have to be great to achieve such a stringent population target of 5% sugars energy. For fibre intake the targets are based on harder endpoints, namely lower colorectal cancer risk and cardiovascular disease. A diet pattern high in pulses, whole grains, vegtables and fruit is advised. Given the lack of dietary change in relation to fruit and vegetables consumption over the last 20 years alongside an intense campaign public health campaign around 5-a day, the target of a 33% increase in fibre intake in men (and more in women) seems extremely ambitious. From a public health standpoint how does achieving this target square with diets of 5-a-day?	Margo Barker
General	This is such a topic and I cannot possibly do it justice in the time. My view is that complex polysaccharides are important physiologically. They act as a sponge in the foregut and depending on their chemistry some are fermented. PS 1 modulate nutrient absorption in the foregut 2. Have a complex very minor effect in cholesterol metabolism Increase stool weight. A deficiency of Complex polysaccharides has no proven contribution to any disease, vascular, metabolic or neoplastic.	Martin Eastwood
General	This is a submission on the draft SACN report 'Carbohydrates and Health'. My background and experience, which have led me to	Mary Flynn

	make this submission, is as follows: I am a Public Health Nutritionist and have been a member of the Nutrition Society for over 20 years[1]. In addition I am a member of the British Dietetic Association, the Irish Nutrition and Dietetic Institute and, previously, of Dietitians of Canada. My work has involved formulating the most recent Food-Based Dietary Guidelines for Ireland and Canada (see references outlined below). I have led research teams in this area. Of direct relevance is the research I have conducted using dietary intake data with the specific objective of formulating healthy eating advice to prevent obesity and related chronic disease. This has involved exploring the reciprocal relationship between total fat and sugar in the diet to find ways of limiting both sugar and saturated fat within the energy requirements of people of different age, gender and activity levels. In addition I have over 20 years' experience of working as a Dietitian to help people with chronic disease risk factors adopt food intake patterns that maintain their weight within a healthy range and protect against diet-related causes of these conditions.	
General	Dear Chairman I would like to congratulate you and the members of your group for putting such a comprehensive draft report together. I have enjoyed reading the draft and appreciate having a reference point for updating my knowledge about nutrition, food, health and chemistry of foods to mention a few. Following a quick read, I would like to ask if adding a brief section on the relationship between cooking methods and domestic food processing and preservation and how they may affect the health outcomes considered by the report would add some value and be relevant to this report especially since dietary recommendations are also covered. Secondly, the report does not appear, from my very quick read, to cover or make brief reference to the effect of physical activity / exercise on health outcomes such as constipation, bowel cancer, insulin resistance. For example how does exercise/physical activity affect gut motility and absorption of non-digestible carbohydrates. I feel that the relationship between level of activity and the metabolic process and resulting changes leading to disease, cure, prevention & control where relevant could add more weight to a very good report.	Victoria Iyamide Nnatuanya
General	SACN's report on Carbohydrate and Health represents the outcome of one of the most rigorous systematic reviews of the scientific literature to date. In July this year, the report came to the same conclusion as the World Health Organisation (March 2014), in recommending that the intake of free sugar should be reduced to no more than 5% of total energy. This conclusion was based on convincing evidence that the overconsumption of dietary free sugars contributes to weight gain, obesity, and dental caries, most noticeably in children. I welcome and endorse this recommendation from SACN, primarily because it will serve to help reduce obesity-related disease and co-morbidity. Moreover, from a nutritional perspective, because free sugar is non-essential in our diet and tends to be associated	Bruce Griffin

[1] I am happy if the Nutrition Society wishes to include in their submission reference to the recent Public Health Nutrition Medal that they awarded me

	with energy-dense rather than nutrient-dense foods, the recommendation is very unlikely to have any adverse consequences for human health. It also has potentially greater health implications in protecting subgroups of the population who show increased sensitivity to the adverse metabolic effects of excessively high intakes of dietary sucrose and fructose (>20% total energy). These subgroups include children and teenagers over consuming sugar-rich foods (sugar sweetened beverages and confectionary), and overweight and obese adults with subclinical signs of cardio-metabolic risk and early liver disease.	
General	The evidence for a 5% energy intake (EI) population value is weak and based on two assumptions that have not been evidenced in the SACN report; (1) that a proportion of people eat no added sugars thus requiring a population target of 5% EI in order to achieve individual maximum targets of 10% EI; (2) that a 5% population added sugar intake would drive calorie reduction. The latter is based on a graph which correlates EI with % EI from added sugar. With energy on both sides of the equation, a correlation is unsurprising and you would probably see this with fat as well. The first assumption should be evidenced with an analysis of the NDNS which properly models the required population target to achieve individual maximum targets of 10% EI added sugars. This could easily be done and it may be that the answer is higher, or even lower, than 5%. The second assumption should be evidenced with results from RCT which have lowered sugar consumption and examined the resulting calorie reduction. Again, this can be done. At present, I feel that SACN have simply tried to match the evidence to the WHO 5% EI target rather than letting the evidence drive the UK target. The issue of unintended consequences should also be explored. Dr Mary Flynn made a good point at the Nut Soc meeting about sugar-containing foods being used in practice to make 'healthier' options more palatable, e.g. high fibre cereals, low fat yogurt, fruit products, wholegrain bread. SACN need to investigate whether a stricter population target of 5% energy would have an unintended impact on intakes of healthier options, fibre consumption or, indeed, fat intakes (e.g. due to the substitution of crisps for sweets). One way around this would be to recommend a food-based, rather than a nutrient-based, approach whereby the population are encouraged to eat less of certain foods (sugar-sweetened soft drinks, biscuits, cakes, confectionery, high sugar cereals, crisps) and to eat more of other foods. Again, this was suggested by Prof Susan Jebb at the Nut Soc meeting. Assuming that SACN are determined to go down the nutrient-based route, a final issue is what message we give to the public. Do we ask them to lower sugar intakes to 5% EI, e.g. maximum 25 teaspoons daily? Or 10% EI? Given that energy requirements vary considerably, would the % energy message be confusing, or inappropriate? At the end of the day, public health recommendations have to be communicated in some way to groups of people, and to individuals. This needs to be considered now before we end up with unwieldy targets that cannot be understood, or implemented, by those who need them most. We only have to remember the problems with non-milk extrinsic sugars (NMES) which could not be analysed, nor which bore any resemblance to what consumers saw on food labels. We were stuck with NMES for more than twenty years! Let's not make the same mistake again with this opportunity to review sugar recommendations.	Carrie Ruxton
General	Summary of studies used in SACN carbohydrate report	Nino Binns

Obesity/Overweight

	Sucrose	SSB
BMI Cohort: Adults	The NHS and the Pawtucket Heart Health Program provided data on sucrose intake and change in body weight (Colditz et al., 1990;Parker et al., 1997). Neither showed evidence of a statistically significant relationship	One publication: Black Women's Health Study (Palmer et al., 2008). Inconclusive
BMI Cohort: Children	None	Six publications from six cohort studies (Kvaavik et al., 2005;Nissinen et al., 2009;Libuda et al., 2008;Striegel-Moore et al., 2006;Phillips et al., 2004;Fiorito et al., 2009). Collectively, these studies provide conflicting evidence: US studies tending to find small but positive associations and the European studies tending to report no evidence of a statistical association.
BMI RCT Adults	None	None
BMI RCT Children	None	One trial (James et al., 2004): intervention was all carbonated beverages, not exclusively sugar- sweetened; low number of children consented to participate and drop-out ca. 10% of children Statistical significance not reported
Weight gain: Cohort Adults	One publication San Antonio Heart Study (Haffner et al., 1991). No statistically significant differences in sucrose intake between weight gainers and non-gainers.	One publication from the Framingham Heart Study (Dhingra et al., 2007) but presented results on both mixed sugar- and non-calorically sweetened beverages.
	Sucrose	SSB
Weight gain: Cohort children	No studies	Four publications, four cohort studies: Cardiovascular Risk in Young Finns Study, The Oslo Youth Study, The Pennsylvania Study of Health and Development of Young Girls study and the Nepean study (Nissinen

			et al., 2009;Kvaavik et al., 2005;Fiorito et al., 2009;Tam et al., 2006). Overall, the four studies provide inconsistent directions of association but generally do not support an association between sweetened beverages and weight gain or obesity when expressed as a categorical outcome.		
	Weight gain RCT adults	None	None		
	Weight gain RCT: children	No studies	Only one trial mentioned in Annex (James et al., 2004) (see above) Summary reports two additional studies: RCT (n=224) in USA overweight and obese adolescents either non-caloric beverages in place of sugars-sweetened drinks for a year or a control group who received no intervention (Ebbeling et al., 2012). At one year, intervention group had gained significantly less weight and had compared with the control group; % fat mass change also tended to be less (p=0.12) RCT (n= 641) normal-weight Dutch children 250 ml/day of a sugars-free, non-caloric sucralose-sweetened beverage or a similar sucrose-containing beverage that provided 104 kcal per serving (de Ruyter et al., 2012). After 18 months intervention children receiving the non-caloric beverage had lower BMI z-score, skinfold thickness, waist to hip ratio and less fat mass cf. children receiving sucrose beverage.		
		Foods high in added sugars	Sugar Sweetened Beverages		
	Body	Two publications, from two Danish cohort studies: the	Two publications: from the Danish Diet,		

	fatness cohort adult	Danish Diet, Cancer and Health Study and the MONICA study (Halkjaer et al., 2009;Halkjaer et al., 2004). No apparent trend between sugar consumption and waist circumference change in adults	Cancer and Health Study and the Framingham Heart Study (Halkjaer et al., 2009; Dhingra et al., 2007). Contradictory results with regard to body fat distribution and consumption of sweetened beverages. (USA study inc fatness; no effect in European study)		
	Body fatness cohort children	Two publications on foods rich in added sugars (candy, baked goods and sugar content of breakfast cereals). The Massachusetts Institute of Technology Growth and Development Study (Phillips et al., 2004) and the National Heart, Lung, and Blood Institute Growth and Health Study (Albertson et al., 2009). Both female-only. Data are not supportive of a relationship between sweet food consumption and body fatness measures in children	Five publications from five cohort studies: the DONALD Study, the Pennsylvania Study of Health and Development of Young Girls, the MIT growth and development study, the Paediatric Bone Mineral Accrual Study and ALSPAC (Libuda et al., 2008;Fiorito et al., 2009;Johnson et al., 2007;Mundt et al., 2006;Phillips et al., 2004) The cohort studies presented here do not provide consistent evidence of a change in body fat amount or distribution with sweetened beverage consumption assessed in childhood or adolescence.		
	Body fatness RCT adults	None	None		
	Body fatness RCT children	None	See weight gain above		
1.3 p7	***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				Geoffrey Livesey

	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. • 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. 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'.	
1.4 p7	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 the last 6th months of publications and aim to include later ones wherever possible. • A cumulative meta-analysis is essential to assess the stability of effects/associations, but there is no evidence of any having been performed.	Geoffrey Livesey
1.4 p7	With regard to inclusion and exclusion criteria, including a table within the main text may facilitate the translation and enhance the	Dr Farzad

	readability. Similarly in paragraph 2.4 and with regard to sugars, a table can help clarifying the concept and facilitating the translation.	Amirabdollahian,
1.5 p8	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 reviews 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." • The Report includes the nonsense statement quoted here. One is obliged to consider the data to be able to think whether the additional study could potentially impact on the conclusion drawn and so reconsider the conclusion. Moreover, without the attendant search critical publications might be missed. The procedure as has been adopted allows reporting bias 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. 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 seems outrageous. If it is not known how a new study affects the conclusion, then no conclusion can be reached.	Geoffrey Livesey
1.8 p8	1.8 Interpretation of interventional studies.	Geoffrey Livesey

	• 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 no strengths 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?	
1.8 p8 1.9 p9	Limitations of cohort studies and RCTs (Paras 1.8 and 1.9, pages 8 and 9). Other limitations of cohort studies e.g. failure to control for other foods and nutrients which may affect the outcomes (e.g. failure to control for SSB intake in studies of white rice intake and type 2 diabetes or for sodium in relation to blood pressure as an outcome) and of RCTs e.g. the fact that they may have narrow inclusion criteria (e.g. age, health stratus, BMI), may use extreme levels of intake and are often short term. Many of these points are made in the later text but a comprehensive list here would be useful.	Geraldine Mcneill
2.2 p16	Terminology for sugars should be more clear e.g. in table 2.2 (page 16) as well as the verbatim definition it would be useful to have a grid showing all possible categories of sugars e.g. in whole fruit or vegetables, in milk/milk products, in dried fruit, added by manufacturer, cook or consumer, in honey, in fruit or vegetable juices, to show exactly what is included in each definition. If the term 'free sugars' is to be adopted in the UK it needs to be very clear whether the definition includes sugars in fruit purees added as sweeteners (as the manufacturer is adding fruit not sugars and they are not fruit concentrates) or in starch hydrolysates e.g. HFCS (which are not 'naturally occurring' and could be seen as excluded by '*sugars* added by the manufacturer'). It is worth pointing out that none of the definitions in table 2.2 can be based on chemical analysis alone as in e.g. fruit yoghurt some sugars will be derived from whole fruit and/or milk and others from added sweeteners. The values for such foods have to be based on recipe calculations which are not always available. If research is to move forward, reliable values for added sugars will be needed: will manufacturers provide these on food labels? Will the new UK food tables provide values?	Geraldine Mcneill

2.22-2.26 p15	**1) The extrinsic sugar** I do not think that using free sugar instead of extrinsic will solve the confusion around this issue. I agree with authors in the fact that extrinsic sugar and non-milk extrinsic sugars are not the best terms to use in the future. It causes confusion among my students rather than a public member. However, this issue is still not resolved by replacing it by free sugar as defined by WHO. The term "added sugars" as used in the US seems to be the best option in terms of health and food labelling. I have the following reasons for that: - Consumers want to know if the sugar is added or it is inherently found in foods, and this is very relevant to health. There is no evidence that the form is of significance in unprocessed foods. - The term free sugars equalise the value of free sugars found in fruit juice or smoothie with the value of sugars added during the processing. Why free sugars found in fruit juices would be equalised with sugars that are adding during processing? This can be really a misleading food label. I suggested using added sugars vs. non-added sugar (or intrinsic, natural, native or innate). I do not why "intrinsic" was originally coined to mean those which are contained within the cell, while it could perfectly mean all sugars that naturally exist either free or within the cells. Why we need to worry about the form of the sugar if it exists naturally in food? People will never consume high amounts of naturally occurring free sugars if they use it in foods. If it is extracted/ concentrated and added to other foods, then it is added sugar. It is as simple as that.	Fandi Ibrahim
2.27 p16	**2)** I fully agree and support all changes related to fibre definitions, methodology, and new reference value. **3)** One issue that was not addressed in the report, and it is relevant to fibre is that the way how total carbohydrate is estimated. In the UK, it is measured as the total of individually measured carbohydrate, but in many other countries, it is measured by difference. The new definition of fibre will reduce the total carbohydrate and increases the total fibre, but that is a positive change after all.	Fandi Ibrahim
2.3 p17	**Terminology: Division according to gastrointestinal and metabolic fate of carbohydrates** It is important to have this functional division of carbohydrates based on gastrointestinal fate, not least due to its impact on energy calculations. It is also necessary to have a term to describe carbohydrates entering the colon that is distinct from the term Dietary Fibre for which a separate definition has been adopted. In the SACN report the terms 'digestible' and 'non-digestible' have been used: *2.3. …. Digestible carbohydrates are absorbed and digested in the small intestine; non-digestible carbohydrates are resistant to hydrolysis in the small intestine and reach the human large intestine where they are at least partially fermented by the commensal bacteria present in the colon…..* In essence the full terms are therefore: 'Digestible in the small intestine' 'Non-digestible in the small intestine' It is necessary to make this distinction as 'digestion' can encompass a number of mechanisms depending on the context in which it	Klaus Englyst

	is used. Digestion is often used to describe the whole process of energy/nutrient recovery occurring in the whole GI tract. The contribution of the products of colonic fermentation to energy is an example whereby the use of 'non-digestible' could be misleading if taken out of context. It is also for this reason that the term 'unavailable carbohydrates' is no longer in common usage. Furthermore, use of the pre-fix 'non-' represents an absolute which does not necessarily best reflect the variance that occurs within the normal physiological range with resistant starch defined as 'the sum of starch and starch degradation products that on average reach the small intestine'. Another approach to this division are the terms 'available carbohydrates' and 'resistant carbohydrates' (Englyst et al 2007. EJCN 61, S19-39): Available Carbohydrates: (Available as carbohydrates for metabolism): consist of the sum of carbohydrates that are digested and absorbed in the small intestine providing carbohydrates for metabolism. This is equivalent to the longstanding usage of 'available carbohydrates', the focus of which is 'available for metabolism as carbohydrate' and therefore links directly to the energy contributing property. Resistant Carbohydrates: (Digestion resistant carbohydrates): consist of the sum of carbohydrates that either resist hydrolysis by the endogenous enzymes of the small intestine, or are poorly absorbed and/or metabolised. In terms of concept, the prefix 'resistant' may be considered as less absolute than the prefix 'non'. This is of relevance not only in the context of resistant starch as noted above, but also within the broader definition of digestion encompassing nutrient release and absorption in the colon. Therefore the term 'resistant', which provides a better representation of a blurred boarder, may be considered as better reflecting that the digestion process does not necessarily stop on entry to the colon for those carbohydrates that have resisted digestion in the small intestine. It is proposed that this category include carbohydrates that are 'poorly absorbed and/or metabolised' to better encompass components such as the sugar alcohols which can have varied gastrointestinal/metabolic fate.	
p2.6 p11	Referring to the difference to the Oligosaccharides in human milk, in what way? difference in quality, quantity or type? Please clarify.	Dr Farzad Amirabdollahian
p2.21 p15	Paragraph 2.21 and also in sections referring to the DRVs such as Chapter 11, while there is reference to dietary recommendations, the definition of dietary reference values (as set in 1991 COMA report) has been neglected. It is unclear what is the approach to dietary reference value/s and what is the approach to the 'dietary recommendation' in this current report. Is this report looking at the body of knowledge within the framework of COMA 1991 LRNI, EAR and RNI? If use of the LRNI, EAR and RNI as yardsticks is not methodologically appropriate (e.g. due to variation in energy requirements); this needs to be clarified. Alternatively, if the body of knowledge required for setting these criteria for total	Dr Farzad Amirabdollahian

	carbohydrate, sugars and fibre is inconclusive, this needs to be stated in Chapter 11 to clarify the conceptual framework of producing reference values or recommendations.	
Table 2.4 p19	For UK, US, WHO and EU; the year of the reports should be added for the sake of clarification of the point of reference; esp. because the references are not included in this table.	Dr Farzad Amirabdollahian
Ch3 p21	In chapter 3 on the dietary sources and intakes of carbohydrates I do wonder why the NDNS was used as the major source of information on intakes. Given the level of underreporting in this survey it might be useful to report more results from the Family Food Surveys (from the Living Costs and Food Survey) which are carried out every year. However only the NDNS can give detail on the intakes of different age groups as Family Food is based on purchase data..	Wendy Wrieden
P3.3 p21	Paragraph 3.3 and with reference to broad category food level and detailed food group level, the explanation is not representative of the rigorous and strong methodological effort undertaken. Like all other nutrients considered in the NDNS and national surveys, the nutrient intake data have been presented in view of the nationally agreed 'Main and Subsidiary Food Groups' which are available as Appendix P at www.gov.uk. Thus, the wording of paragraph 3.3 is to be amended to refer to broad main food group levels and more detailed subsidiary food group levels as per NDNS methodology.	Dr Farzad Amirabdollahian
p3.4 & 3.5 p21	Paragraphs 3.4 and 3.5 need to be supported by reference.	Dr Farzad Amirabdollahian
3.37 p26	Para 3.37 page 26. Nationally representative surveys of the intake of sugars in children across Scotland commissioned by the Food Standards Agency Scotland[1,2] which contain detailed analyses of intake of sugars and sugar-containing foods according to age sex and socio-economic deprivation should be mentioned.	Geraldine Mcneill
Ch4 p28	In chapter 4 (Background on Health Outcomes) I was a bit confused as why lifestyle risk factors were not included in Table 4.1. Tobacco use, diet and physical activity are included in the comment on p 28 but not in the table.	Wendy Wrieden
Colo-	• The comments re biomarkers for colorectal cancer (CRC) are well made (see areas for future research).	Elizabeth

rectal Health (4.8-4.13):	• Studies on laxation are a good example of an end-point that can be measured following a short intervention and thus the data from RCTs in this area are some of the most trustworthy. There is the added advantage that this can be of immediate benefit to an individual. However, the point is well made that it is still uncertain whether this has any impact on more serious health outcomes such as CRC. • The role of gut bacteria in the maintenance of colo-rectal health is currently a very active area of research. This is well reported in relevant tables throughout the document (e.g. table 6.4 and 9.27) and will hopefully at least produce better markers of risk to long-term health.	Lund
Chs 5-10 p35 onwards	Chapters 5-10; boxes summarising the body of knowledge for each research question are quite informative. A statement to address the research question based on the information presented in tables can also be supplied to facilitate the translation.	Dr Farzad Amirabdollahian
5.114 p68 5.116 p69	Body weight or BMI as an outcome: it is not always clear whether it is a the *change* in weight or BMI or just the value at the follow up in a cohort study e.g. the heading above para 5.114 in 'body weight change' but the box under para 5.116 says 'body weight'	Geraldine Mcneill
5.135-6 p74	For studies with energy intake as an outcome it is important to know that these are studies where participants could chose most of their diet ad libitum, e.g. paras. 5.135-6 and particularly the studies used in fig 1 p.202.	Geraldine Mcneill
Chapter 6 p82	In chapter 6 most of the sections refer to 'sugars' not NMES or 'free sugars'. It is not clear whether the included studies could have analysed total sugars i.e. including those in whole fruit and milk: this seems likely since most food tables only report the chemical analyses so will be a limitation of the evidence in this area. The point that this term would capture sugars in HFCS (made later in para 11.7 page 200) should be highlighted here.	Geraldine Mcneill
Ch6 p82	Review of total sugars and sugar-sweetened beverage (SSB) obesity aspects • The SACN (and the Leeds reviewers) have conducted a very thorough detailed review that is commendable. • The overall data on sugars and SSB and body weight are surprisingly limited and inconclusive (see table below). • The conclusions of the report, as drafted, overstate this evidence.	Nino Binns

	• These data do not support a change in the population target. • I have not reviewed the data on energy intake – but this is an intermediate endpoint in terms of body weight changes. If these data were used to seek approval for a health claim, for example, it would not be acceptable. • The data on energy intake vs. % energy from sugar do not support a change in population target. **RCTs – level of evidence** Two recent and key RCT on children provide interesting new data but the conclusion in the summary report is overstated. The RCT de Ruyter et al (2012) is well conducted and is a valid comparison and points towards a potential role of SSB in the diet. The study by Ebbeling et al (2012) is similarly quite a good study; its primary objective was intended to reduce sugar intake form SSBs (intervention group) but is somewhat flawed because the control group had no intervention other than being recruited to the study and told to continue as normal. The conclusion of the section on page 96 is as follows: The overall conclusions (para 11.8 page 200 and elsewhere) use strong language compared with the assessment of the evidence (my emphasis): 'Randomised controlled trials conducted in children and adolescents indicate that consumption of sugars-sweetened beverages, as compared with non-calorically sweetened beverages, resulted in weight gain and an increase in body mass index. ' Current dietary advice universally cautions the use of SSBs. This is appropriate advice for many people – but SSBs are not unique in their potential to lead to inappropriate energy intakes. They are probably uniquely 'more studied' at present but this should not lead to too much focus on one product rather than on the whole diet. In addition, focusing on one macronutrient may lead to unintended consequences. For example, in habitual self-selected diets where a sugar-fat seesaw is usually evident (Sadler et al 2013). (See also Markey and Lovegrove abstract 122 from Glasgow showing the effect of sugar reformulation)	
Ch6 p82	**Sugars: Free sugars term** It is important to have a term that provides distinction from the sugars present in milk products and intrinsically in fruit and vegetables, as dietary advice should be targeted at reducing free sugars rather than total sugars, which could impact negatively on the milk, fruit and vegetable food groups.	Klaus Englyst

	The challenge of achieving measurements of free sugars has been sited as an obstacle for the use of this kind of term. The fact that labeling requirements are for total sugars, with no realistic expectation of including a separate free sugar category, should not prevent guidelines based on free sugars. In practice most of the sugars present in packaged products are free sugars, with less than 10% of the total sugar consumption from packaged products coming from intrinsic and milk sugars (NDNS data). The proposed DRV population average of 5% of energy from free sugars would suggest that the guideline daily amounts value adopted by industry for front of pack labeling purposes should be revisited.	
6.64 p98	Dental caries is still the biggest cause of hospital general anaesthetics for children in the UK, and is wholly preventable. Anything we can do to help usher in population level interventions, public policy, fiscal measures, advertising restrictions, etc the better. This is one step in the right direction.	Deborah Moore
Ch6 p82	In Section 6, 6.61 and 6.62 on the frequency of sugar intake. It was not clear if this is actual sugar or sugar recorded in food. We do not normally eat sugar on its own so I was a bit puzzled by this. The main comment on this report should really be in connection with the recommendations. In regard to sugars I think the use of the term "free sugars" rather than non-milk extrinsic sugars is an excellent idea and much easier to explain than NMES. The population DRV for free sugars of 5% of energy makes sense as does the figure of no more than 10% of energy at an individual level. However this may still cause confusion given that the European RIs for Food Labels are in terms of total sugars. There needs to be some way of distinguishing reference intakes (as stated on food labels) that are minimum amounts e.g. dietary fibre and those that are maximum as in the case of sugars.	Wendy Wrieden
Chapter - 8. Fibre	• The suggested change for the UK to use the more physiological definition of fibre is to be greatly welcomed. • Although the terms insoluble and soluble fibre are useful when considering mechanism of action they cause confusion to the general public and are probably better replaced by cereal fibre and fibre from fruit and vegetables should a more detailed description be needed. The use of just the term fibre for recommendations should make for a much clearer message. • As mentioned above, the impact of fibre in the colon and rectum in respect to gut bacterial populations and function including production of short chain fatty acids, mucosal defence and modification of luminal pH are key research areas in understanding mechanism of action and potential modifiable markers of risk. These effects may not be limited to colo-rectal health as there is the potential for systemic responses such as immunological changes and modification of liver function through SCFA production.	Elizabeth Lund
Ch8 p114	The move (or return) to the term dietary fibre is also welcomed as I believe that the DRV for NSP is now confusing given that food	Wendy

	labels were using the AOAC definition of dietary fibre. I understand that the amount of dietary fibre is no longer used with the new food labelling legislation but products that make a claim will still need to declare it. The new or perhaps not so new (the same dietary goal was proposed by NACNE and COMA pre 1991) DRV of 30g seems appropriate but will make current population intakes look even more unsatisfactory!	Wrieden
Ch8 p114	**Dietary Fibre: Focus on natural sources** The emphasis within the report that dietary fibre should mainly come from natural sources is appropriate. The majority of the evidence is based on prospective cohort studies where intakes essentially reflect dietary fibre coming from natural sources, as there is limited history of wide scale fibre fortification practices. **Dietary Fibre: DRV of 30g and potential implications of a perceived 'fibre gap'** This represents quite an increase from previous DRV, and is higher than current average intakes. In conjunction with the position that dietary fibre intake should be from natural sources the implied message is that the 'fibre gap' should be met by increased intakes of fruit, vegetables and wholegrain products. However in practice the suggestion that there is a 'fibre gap' is already being used to market products fortified with resistant oligosaccharides and resistant starch. This is not consistent with the position taken in the report where the focus is on naturally occurring sources. It may be appropriate to extend the last statement in 12.27 (and possibly also 12.33) to emphasise that in terms of meeting DRV for dietary fibre, a cautious approach should currently be adopted in order to avoid overconsumption of fortified products and the potential displacement of natural high fibre foods from the diet. **Dietary Fibre: Codex, EC and SACN definitions are not defined by methodology** The Codex, EC and SACN definitions of dietary fibre are similar in principle, describing the carbohydrate component escaping digestion in the small intestine but with the added provision that extracted or synthesized components must be demonstrated to have a physiological benefit. Although a departure from other nutrient classifications, these dietary fibre definitions do provide the broad criteria by which to include or exclude various components. The criteria set out in the definition are sufficient description and as outlined below attributing fibre to what is recovered by a single method may be counterproductive. In section 12.27 of the report it states: *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 have a degree of polymerisation of three or more monomeric units, plus lignin. Dietary fibre is to be chemically determined using the Association of Official Analytical Chemists (AOAC) method 2009.01 (McCleary et al., 2010; McCleary et al., 2012).* Several aspects of this statement would benefit from clarification to limit inconsistencies: - The definition here is focused on the naturally integrated components only, with the implication that when extracted and synthesised components are present they are considered by a separate set of criteria. This approach would seem to fit best with	Klaus Englyst

	the current evidence. - The naturally occurring components are: NSP and the small amounts of lignin associated with plant cell wall material; resistant starch, which in the context of most processed foods in the human diet is restricted to RS type 3 only; resistant oligosaccharides, which are limited to fructans (cereals onions etc) and alpha-galactosides (legumes) occurring naturally. These components can be measured specifically by chemical methodologies of which there are several available as recognized by Codex Alimentarious when this topic was considered. - In contrast, the AOAC 2009.01 is the only method specified in the statement. However, the phase 'chemically determined' used in the statement is not consistent with this methodology, which is instead empirical in nature rather than measuring components specifically. The implication is that the method does not provide information on what has been recovered and therefore no possibility of evaluating conformity with the actual definition. The AOAC 2009.01 will recover a wide range of extracted and synthesized components which may or may not conform with the definition. - In terms of measuring RS, for most processed foods that will only contain RS type 3, the AOAC 2009.01 method offers no advantage over the more commonly used AOAC gravimetric fibre methods that also recover RS3. - In terms of measuring the resistant oligosaccharides, the AOAC 2009.01 does this by a refractive index HPLC technique that provides no information on the components measured. Some laboratories have reported inflated values for this part of the method indicating that accuracy may sometimes be an issue for some product types. In practice fructans and alpha-galactosides can be measured specifically when present, representing viable analytical options for measurement of the naturally occurring oligosaccharides. In summary dietary fibre is now defined by a set of chemical/origin/functional criteria, rather than by what is recovered by a specific method. Any method or combination of methods that correctly identify the dietary fibre components present in foods would be appropriate. Sufficiently detailed analytical tools should be available to provide distinction between component types. Such distinctions are important both for correct labeling and also to ensure that sufficiently detailed information is available to assess the impact of various fibre types in future research studies.	
8.121 p146	Para 8.121 page 146 last sentence is not clear	Geraldine Mcneill
8.125 p147	Para 8.125 page 147 box states 'whole grains' but should be 'cereals'	Geraldine Mcneill
Chapter 10 p180	Chapter 10 Glycaemic index and load	Geoffrey Livesey

	*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), as 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 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? Do I recall incorrectly? *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" • The para gives a false impression of GI and GL and how trials can modify these quantities. • 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 often fail (Livesey et al published). • The finding that all trials induced some weight loss may be unduly critical. Reduction in GL can induce weight loss. 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 needs defining here, even if defined elsewhere not found. • 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? 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 cancelling out each other in a total cardiovascular disease all sexes combined analysis. Mixing no effects with effects 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 no effects with effects would be a sure way to get borderline significance/non-significance as reported in the Report. • The conclusion of "No association" is wrong for the data available in the literature, this SACN definitely activity 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 questionable whether the present meta-analysis has 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 data). 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 data). 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 met-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 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 correlation with incident disease, such studies are generally biased to the null. • One or more 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. If not they will be biased towards null. • For each one of the above reasons a meta-analysis result can be rejected. • 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 or 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 (Livesey et al an AJCN Supplement). • It is not surprising, therefore, that combing results from all such studies reveals little effect, as indicated in the presented meta-analysis. • 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 associated with 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 (Bingham). *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 regularly stated bias against prospective cohort studies in favour of intervention studies or whether it is stated whenever there is 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). *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. *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. • At least one epidemiological study has suggested an association. ****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 breaches (Livesey et al) threshold. • 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 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.
	• A comprehensive meta-analysis of more studies than in the SACN Report has already been published (Livesey et al, 2013a,b) 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. Significantly dependence on the FFQ correlation for carbohydrate, implying the studies 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)
	• Combing all studies together (<5 mmol/L and >5mmol/L) can be suspected to average out as no effect among healthy persons.
	• For some purposes, an analysis excluding pre-diabetes and diabetes from the analytical model has some limitation, for example, when fasting blood glucose can be considered as a continuum throughout the range. The exclusions are artificial cut points relevant to clinical issues rather than scientifically defined.
	• It can be considered that the no-effect 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 loose power compared with studies of moderate duration.
	• The reported “no-effect” 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 short term probably 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 presently presented in the 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.	
Ch11 p199	Paragraph 2.21 and also in sections referring to the DRVs such as Chapter 11, while there is reference to dietary recommendations, the definition of dietary reference values (as set in 1991 COMA report) has been neglected. It is unclear what is the approach to dietary reference value/s and what is the approach to the 'dietary recommendation' in this current report. Is this report looking at the body of knowledge within the framework of COMA 1991 LRNI, EAR and RNI? If use of the LRNI, EAR and RNI as yardsticks is not methodologically appropriate (e.g. due to variation in energy requirements); this needs to be clarified. Alternatively, if the body of knowledge required for setting these criteria for total carbohydrate, sugars and fibre is inconclusive, this needs to be stated in Chapter 11 to clarify the conceptual framework of producing reference values or recommendations.	Dr Farzad Amirabdollahian
Ch11 p199	This report provides an excellent review of the role of dietary carbohydrates in cardio-metabolic health, colo-rectal health and oral health. The clarification of the complexity of dietary carbohydrates from the perspective of terminology, classification and definitions is very welcome and will be very useful. The approaches used are largely commendable. This report provides a transparent and detailed outline of the research considered and the strength of evidence for conclusions and recommendations. The concerns I have relate to two areas of the	Mary Flynn

	report: 1. *Chapter 11 Dietary Reference Values* The formulation of Dietary Reference Values for carbohydrates is undertaken without considering other nutrients in the diet – particularly macronutrients. Therefore while the conclusions on ideal amounts of dietary carbohydrates for health may be well-founded, failure to consider the consequent effects of changing carbohydrate intakes on other nutrients, particularly fat and saturated fat, may result in dietary intakes that are more harmful. The Institute of Medicine (IOM) in their 2002-2005 report outline an approach to developing recommendations on macronutrient intakes that takes account of the inter-relationships that exist between macronutrients – particularly the reciprocal relationship between fats and carbohydrates (see Institute of Medicine 2002 – 2005 below).	
11.5 – 11.13 P199-203	DRVs for sugars (paras 11.5 – 11.13, pages 199-203). Changing the term 'non-milk extrinsic sugars' with a term more easily grasped by non-nutritionists and is used by other countries would be welcome: 'free sugars' is probably the best alternative term as long as what is included is made absolutely clear (see comment 6 above). A comparison of NDNS data using the two definitions would be helpful as there is likely to be very little difference in the % food energy so studies using NMES (now provided in UK diet analysis programmes as well as NDNS datasets) could be used in evidence reviews along with studies using 'free sugars'. It would be useful if total sugars from the NDNS could be given in this comparison so that older studies which use total sugars can be compared with newer studies using narrower definitions, at least for the range of intake.	Geraldine Mcneill
Figure 1 p202	Using Figure 1.1 (page 201-2) to justify setting a 5% target for free sugars raises a number of issues. I am assuming that the studies looked at ad libitum energy intake and were carried out for a reasonable length of time in diverse populations, though these points need to be made clear. Most importantly, do the studies report total sugars or free sugars? The exact category of sugars needs to be clearly stated on the x-axis label. If the studies use total sugars and free sugars are perhaps 60% of total sugars then the range of intakes becomes more representative of UK diets but there would be even less evidence for a steeper relationship between sugars intake and energy intake at lower sugars intake, as suggested in para 11.10. Second, to achieve a reduction of 100 kcal/day by reducing sugars intake by 4% energy would represent a reduction from the current values of 11-15% (according to age) to 7-11% not 5%: in older children reducing from 15% to 5% energy would lead to a reduction of approx. 250 kcal/d by these extrapolations which could be considered too great a reduction for health. Third, if individual intakes should be no higher than 10%, would the population average necessarily be 5% (i.e. around half of the population having less than 5%), or might it be closer to 7 or 8% with a narrower and possibly more skewed distribution than at present? Fourth, although it is recognised that this report is	Geraldine Mcneill

	designed for risk assessment not risk management, the recommendation for SSBs would be more useful if it could more quantitative, include examples and be linked to the sugars recommendation e.g. 5% energy would be 25g free sugar in an 8MJ/d diet i.e. approx. the amount of sugar in one 330 ml can of SSBs: one of the now widespread 500ml bottles of SSBs per day would provide approx. 7.5% energy. For public health, bringing down the intake of SSBs among those with highest intakes is important and a clearer upper limit e.g. no more than 1 litre (3x330 ml cans or 2x500 ml bottles) per adult or older child per week, with suggested replacement with water, low calorie drinks or milk for children (i.e. not fruit juice) would be valuable.	
Fig1 p202	My main comment is that I think they have mis-represented my data in Figure 1 on Page 202. It actually took me a while to work out how they arrived at the data in this graph from my papers! Drummond et al (2003) and Drummond & Kirk (1998) were two intervention studies where 2 groups of subjects received different dietary advice – one to reduce fat only and the other to reduce both fat and sugar. The SACN report have selected the end point data (% energy from sugar and total energy intake) from the 2 groups. They don't take into account differences in energy intake between these groups at baseline – which I think is significant particularly in the 2003 paper. In fact, if they had selected the baseline values for Group 1 & 2 (2003 paper) the relationship would have been the other way round – Group 1 NMES= 10.0% EI = 9.7MJ Group 2 NMES = 11.4% EI = 8.49MJ Also, in the 2003 paper, Group 2 significantly reduced % energy from NMES over 8 weeks with no significant impact on energy intake. In the 1998 paper Group 2 significantly increased %total sugar (and NMES) at 6 weeks had significantly reduced energy intake – largely due to a reduction in dietary fat – indicating a sugar-fat seesaw. So I think they are not presenting the data appropriately in terms of the study design. In addition the 1998 paper demonstrates that over time as Group 2 significantly reduced EI whilst increasing %energy from sugar (and decreasing %energy from fat) it lead to significant weight loss, which is important to acknowledge since weight control is the key issue after all. I also think it is misleading to join the data points with a line in Figure 1 in the report. It suggests that the 2 data points are from one group over time, whereas they are from two different groups at one single point.	Sandra Drummond
Ch11 & CH12	***Specific comments on the report*** ***FREE SUGARS*** Chapters 11 and 12 setting DRVs for Carbohydrate and free sugars specifically should be amended to consider the effects on	Mary Flynn

	other macronutrients – especially total and saturated fat. This would be in line with the approach used by IOM which accounts for the fact that macronutrients are sources of energy that can be used interchangeably (see Institute of Medicine 2002 -2005). The approach outlined in the SACN Draft Carbohydrates and Health Report in paragraph 11.10 to formulate the 5% mean Free Sugars intake goal is flawed in that it only considers energy and not the complex dietary inter-relationships between fat and sugar. Dietary intake studies in the UK and Ireland have consistently demonstrated the reciprocal relationship that exists between total fat (and saturated fat) and sugar intakes (see Flynn *et al* 1996 and Flynn & Kearney 1999 below). This is often referred to as the fat-sugar see-saw. We have explored this is in the detailed revision of Ireland food based dietary guidelines to protect against chronic diseases including obesity, and to improve health – including dental health (see Flynn *et al* 2011a, Flynn *et al* 2011b, FSAI 2011 and FSAI 2012 below). In an earlier study of dietary intakes in Ireland the inverse relationship between total fat and non-milk extrinsic sugars (NMES) sugars intake was found to extend to saturated fat (see Flynn *et al* 1996). Examination of the food sources of NMES sugars in this study categorised these foods into those that were 'fat-free high sugar foods' and 'fat-containing high sugar foods'. The 'fat-free high sugar foods' were found to be associated with lower fat and saturated fat intakes while the 'fat-containing high sugar foods' were not. Furthermore the low fat and low saturated fat diets that contained higher quantities of the 'fat-free high sugar foods' were associated with higher intakes of fibre and micronutrients with the exception of vitamin A (Flynn *et al* 1996). This demonstrates that 'fat-free high sugar foods' have a role - albeit in limited amounts, as part of a healthy diet. The role of 'fat-free high sugar foods' play in palatability of low fat, high fibre diets arises when formulating dietary guidelines. For this task 'fat-free high sugar foods' need further classification into those that become part of a low-fat high fibre or micronutrient-rich food (e.g. sparing use of sugar in porridge or preserves on wholemeal bread) *vs.* those that are simply additional (e.g. sugar-sweetened beverages or sugar added to tea). This is outlined in a report of our work revising the food-based dietary guidelines for Ireland (Flynn *et al* 2011b). An iterative approach to develop twenty-two 4 day food intake patterns until average intakes met a range of nutrient and energy goals that represented the variable nutritional requirements of all in the population aged 5 years and older. Achieving low saturated fat intakes (<10% energy) was difficult as was achieving the dietary fibre goal of 25g per day for those with energy needs below 9.2MJ/day. These healthy eating goals, including keeping intakes of non-milk extrinsic sugars (NMES) below 10% energy, were largely achieved – but the mean NMES intakes ranged between 6.2 – 11.5% energy. Notwithstanding the fact that Free Sugars will amount to slightly lower levels compared with NMES, the mean goal of 5% energy intake would not be achieved. In the published report (Flynn *et al* 1996) we outlined the advice on sugars as follows: *'This revision found that fibre goals were difficult to achieve, as previously reported in Ireland, particularly in food patterns that were based on lower energy requirements (<10MJ/ <2,400 kcals). Specific adjustments of food patterns involved the sparing use of two fat-free sources of sugar - namely, table sugar and preserves, to increase the acceptability and palatability of fibre-rich food sources, such as wholemeal cereals, breads and stewed fruit – an approach that has been previously used.* This approach is in agreement with the SACN Draft Carbohydrates and Health Report regarding the recommendation that 'consumption of sugar-sweetened beverages should be minimised in both children and adults' and frequent intake of sugars should	

be avoided for better dental health. The relevant section of this report (Flynn *et al* 2011b) is outlined below:

> *However this revision of healthy eating advice also includes advice to avoid frequent intakes of sugar for the promotion of good dental health. Previous work in Ireland has highlighted an inverse relationship between saturated fat and sugar and preserves and the inverse relationship between sugar and fat intakes is well established. Notwithstanding this, the inclusion of sugar and sugary foods for their own sake (i.e. independent of fibre-rich foods) was not supported in this revision. Consumption of sugary foods, such as soft drinks, or sugary foods that also contain fat, such as confectionery, increase intakes of energy and possibly fat, without providing essential nutrients, and high-fat sweet foods may be positively associated with obesity.*

In conclusion, therefore, this submission calls for the amendment of the approach used to set DRVs for Carbohydrate and free sugars so that the effects on other macronutrients – especially total and saturated fat can be considered. As part of this, the role of different food sources of sugar needs to be considered i.e. those that

a. are 'fat-free' *vs.* 'fat-containing' and
b. become 'part of a low-fat nutrient dense food' *vs.* 'those that are strictly additional'

DIETARY FIBRE

Achieving absolute dietary fibre goals 25+g per day is difficult for those with energy needs below 9.2MJ/day (Flynn *et al* 2011b). The DRV recommended for total dietary fibre intake is given in absolute terms as a goal of 30g per day and this is only be easily achievable by those with high energy intakes due to their larger food intakes. Consideration should be given to setting the requirement in terms of energy needs and providing the recommendation in terms of overall food intakes – i.e. a certain fibre density (g fibre/MJ). Such an approach will adjust fibre intakes for those with lower energy needs (small adult females) and protect against compromising micronutrient status.

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CH12 p210	*Chapter 12 Overall Summary and Conclusions – 12.26 and 12.27* The dietary reference value recommendations outlined in Paragraph 12.26 and 12.27 for free sugars and for dietary fibre, respectively, are not feasible. 1. FREE SUGARS - I am in total agreement with the conclusions that free sugars must be limited and that the previous upper limit of 30% needs to be revised considerably (Paragraph 11.5). However the recommended mean intake of 5% is set too low for palatability of low fat, low saturated fat diets. For similar reasons the maximum level of free sugars intakes needs to be increased to ~12%. Notwithstanding that reservation, it is clear from the draft report that free sugar intake in form of sugar sweetened beverages needs to be strictly curtailed. Therefore I am in total agreement with the recommendation that consumption of sugar sweetened beverages should be minimised in both children and adults given the evidence associating their intake with a greater risk of Type 2 Diabetes and the likelihood of incomplete appetite compensation for the calories they provide. 2. DIETARY FIBRE – The DRV recommended for total dietary fibre intake is given in absolute terms as a goal of 30g per day and this will only be achieved by those with high energy intakes. Consideration should be given to setting the requirement in terms of energy needs and providing the recommendation in terms of overall food intakes – i.e. a certain fibre density (g fibre/MJ). Such an approach will adjust fibre intakes for those with lower energy needs (small adult females) and protect against compromising micronutrient status.	
12.21 p215	12.21 "There is no evidence from prospective cohort studies to suggest an association between glycaemic index and cardiovascular disease or coronary heart disease." • Wronge. • The conclusion would be stunning if it were not for knowing the review is well out of date.	Geoffrey Livesey

	• 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. • 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 being associated with even low levels of perturbations in HbA1c compared with 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 or built into an appropriate model rather than ignored as was done in the SACN commissioned meta-analyses. 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 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 in the original studies and across the studies. Unfortunately there was no evidence of this in the draft Report.	
12.21 p215 General comments and publications references	Last words on GI and GL • 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. They 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 advise) has been devised to select an optimal diet since all food-based advice and compositional-based advise 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 a 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. • It is an embarrassment to consider the UK continues to not recognise that the quality of fatty foods and quality of carbohydrate foods matters more than is evident among its health messages, food labels and food tables, most especially that the quantity of fats and carbohydrate together has proved difficult to control. <u>Conflict of interest.</u> None here other than to seek for higher standards of meta-analyses and to see the science and appropriate application of	Geoffrey Livesey

	Glycaemic Index and Glycaemic Load. The author holds shares in Independent Nutrition Logic Ltd, Wymondham, Norfolk (glivesey@inlogic.co.uk). The views expressed are without prejudice the personal views of Geoffrey Livesey, BScHons., PhD. RNutr(Public Health). Publications with information relevant to SACN Report and to GI and GL, in part or in full. 1. Livesey G, Taylor R, Livesey H, Liu S. Is there a dose-response relation of dietary glycemic load to risk of type 2 diabetes? Meta-analysis of prospective cohort studies. Am J Clin Nutr 2013;97:584-96. (**With supplemental data and meta-analyses online accompanying the main article on line**) 2. Livesey G, Taylor R, Livesey H, Liu S. Is there a dose-response relation of dietary glycemic load to risk of type 2 diabetes? Meta-analysis of prospective cohort studies: . World Biomedical Frontiers, Diabetes http://biomedfrontiersorg/diabetes-2013-may-2-1/ Accessed: June 17 2013 2013. Internet: http://www.ncbi.nlm.nih.gov/pubmed/23364021 (**Focuses on significance of effects in men as well as women**) 3. Livesey G, Taylor R, Hulshof T, Howlett J. Glycemic response and health a systematic review and meta-analysis: the database, study characteristics, and macronutrient intakes. Am J Clin Nutr 2008;87:223S-36S. 4. Livesey G, Taylor R, Hulshof T, Howlett J. Glycemic response and health a systematic review and meta-analysis: relations between dietary glycemic properties and health outcomes. Am J Clin Nutr 2008;87:258S-68S.	
12.23 p215	12.23 "……." • What is stated might well be perceived as 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 implied), rather GI and GL is applied within the context 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 limit a consumer's ability to identify an optimum diet, prevent appropriate dietary choice (as well as blocking free choice), and unduly worry many type 2 diabetes patients and others world-wide who apply GI and GL to their own benefit.	Geoffrey Livesey
12.26 P216	As an academic in Dental Public Health with an MSc in Public Health Nutrition, I am very pleased to see the current focus on free sugars. I wholeheartedly agree that the current population level of no more that 10% of energy is too high, and that it should be lowered to 5%, thus recommending that at the individual level the majority of the population should be below 10%.	Deborah Moore

12.28 p217	I think we should all remind ourselves that the main issue with carbohydrate is not just quantity but its requirement for the right balance of Vitamins of the B group [some of which we may not yet know!] If we produce animals and crops that have been fed on artificially produced feed and fertilizers and then further refine purify and adulterate it no amount of guidelines on quantity will have an effect on our health.	Diana Sandy
12.4 p210	12.4 "carbohydrate, glycaemic index, and glycaemic load" General comment: • It is unclear why there is negative focus 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 relatively little association of such issues with these other measures, which can suggests author bias. • Not mentioned is that a major downfall of intervention studies is that long-term studies tend towards convergence of treatments and controls thought to be due to knowledge crossing between participants of treatment arms and via newspaper and magazine articles about possible heathy attributes of diets. • Not mentioned is another major problem with the consideration of intervention studies and their meta-analyses, that is 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, 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 the importance of larger differences worldwide, or between an optimal intake and a countrywide intake.	Geoffrey Livesey
12.10 p212	Para 12.10 page 212 implies that the studies were designed to look at the effects of increasing sugars intake from existing values whereas I suspect the opposite was true: the former might not be approved by an ethics committee.	Geraldine Mcneill
12.11 p212	This is a very comprehensive review of the evidence on carbohydrates and health taking which takes a somewhat cautious approach in its conclusions. It only considered evidence from prospective cohort studies and randomised controlled trials in contrast to the WHO draft guideline on sugars intake which I understand included non-randomised trials, population studies and cross-sectional studies (12.11) for its advice. This makes sense in view of the fact that the SACN report was looking at all carbohydrates and there was a need to limit the review and base it on the best evidence available – which in many cases was limited.	Wendy Wrieden

	The difficulty of disentangling carbohydrate intake and its effects from the rest of the diet is acknowledged but perhaps more of an issue is whether it is helpful to look at the effect of total carbohydrate intake given what we know of the variety and effects of the different compounds that are termed carbohydrates. The few effects reported for high carbohydrate diets per se cannot be taken in isolation from the other components of the diet but I suppose the chapter 5 (on total carbohydrates) is still required.	
12.26 P216	**5% energy from sugars target** Figure 1 of the SACN report seems to be pivotal in terms of driving the figure for a population target of 5 % energy derived from sugars in the diet These data are derived from the endpoints of the studies and might look different if data were based on the change in sugars intake. SACN comment but dismiss the fact that there is only study down at 5% energy (Raben 2002 not 2001). In this study subjects were given large amounts of sucrose foods and mainly drinks (152g sucrose) per day or equivalent non-caloric sweetened foods. The rest of their diet was their normal diet eaten ad lib. The product substitutes were not iso-caloric so the study simply shows that adding a large amount of sugar to the diet results in an increased energy intake and slight weight gain – i.e. compensation was not complete. This was perhaps not surprising when sucrose % energy was changed from average 11% to about 28%. The converse was true of the low sugar substitutions – compensation to increase calorie intake was not complete. The Brynes (2003) study was a 4 arm study of 24 days in which subjects were given large amounts of olive oil, sucrose, instant potato or rye bread to help achieve the interventions namely high fat, sucrose, high GI and low GI. Figure 1 of the SACN report compares the high GI (instant potato) diet with the sucrose diet. If the sucrose diet had been compared with the olive oil diet (see Table 2 of Brynes 2003) the slope in Figure 1 would be in the opposite direction because fat has a higher propensity than sugars to increase energy intake.	Nino Binns
Annex 2.10 p221	A2.10 Conversion of NSP to AOAC fibre and vice versa • It is good that this discrepancy is highlighted. Nevertheless, sensitivity analysis ought to have been undertaken—one was not found. The sensitivity analysis would explain the potential relative bias of three different modes of expressing the results (NSP, AOAC, and MIXED in a combined result). 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 a conversion factor, which might be inaccurate for the populations instant.	Geoffrey Livesey
Annex	A2.16 and A2.17	Geoffrey

2.16 p222	• The difference between the two paragraphs is not adequately drawn to the eye.	Livesey
Annex 2.21 p224	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? 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?	Geoffrey Livesey
Annex 2.21 p221	A21 to A2.23 …….. • All is written in the past tense. Likely, all would have been written in the future tense if agreed beforehand. This brings some concern the protocol was stitched together after results became known. • It is unclear whether data from the prospective cohort studies were appropriately transformed before meta-analysis. • 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 (as it is then hidden in the curvature). • 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.)	Geoffrey Livesey
Annex 2.23 p225	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.	Geoffrey Livesey
Annex 2.7 p 221	A2.7 "fixed effect model was used" "should" • The present reader is deeply shocked by speculation in A2.7. • Efforts to obtain smaller errors by using fixed effects is not called for and is abhorrent in the extreme in meta-analytical context. It is simply not science to speculate that a difference in results between subgroups within a research centre is not possible. One type of within laboratory subgrouping mention in A2.7 is for men and women, which can respond differently within each research centre. Indeed that a research centre considers to present results for subgroups separately is because they hypothesize differences exist. If a population mean is needed then random effects should be used to combine data. However, if meta-analysts really wish to achieve the correct reduction in heterogeneity, then the outcomes should be	Geoffrey Livesey

	modelled appropriately. In the case of gender differences this can be achieved using zeroed centred covariate that is the fraction of the population sample that is male (or female, centred on 0.5 reduced to 0, i.e. range -0.5 to +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), the results will have the correct reduction in heterogeneity, and obtain additional information about the size of difference between gender. Not having analysed the studies properly leaves draft SACN Report with unquantified biases (some of which definitely would be large) and so unquantified outcomes with unduly small confidence interval. This problem has a major implication for the levels of statistical significance obtained.	
Annex 2.8 p 221	A2.8 The criterion (I^2 >75%) • 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 inefficiency. • 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 75% and 76% the same, difference between the two would be entirely arbitrary and unwarranted.	Geoffrey Livesey
Annex3.3 p226	Para A3.3 page 226 5th line does not make sense.	Geraldine Mcneill
Publications references	[1] Sheehy C, McNeill G, Masson L, Craig L, Macdiarmid J, Holmes B, Nelson M. (2008) Survey of sugar intake among children in Scotland. Aberdeen, Food Standards Agency Scotland. http://www.foodase.org.uk/results.php?f_report_id=607 [2] Masson LF, Bromley C, Macdiarmid JI, Craig LCA, Wills W, Tipping S, McNeill G.(2012) Survey of diet among children in Scotland (2010). Aberdeen, Food Standards Agency Scotland. http://www.foodbase.org.uk/results.php?f_report_id=777	Geraldine Mcneill
Future Research Topics	Future research should focus around improving the credibility of nutritional advice in relation to long-term health outcomes. Understanding mechanism There should be a focus of effort toward understanding mechanism where cohort studies suggest benefit. Without mechanism association is meaningless. https://medweb.nch.org/INTERMED/Data/ComponentFiles/1099/14_ABOG_May%202013.pdf This work could include studies using animal models but using human relevant concentrations of dietary factors such as fibre.	Elizabeth Lund

	Many animal studies have already been conducted but using for example too high concentrations of fibre or too little fat and at an inappropriate age range etc. However, there should be increased effort to use currently available data such as gene expression patterns from animal studies to design human studies with relevant and practical markers of risk. Better markers of risk amenable to dietary modification As is pointed out in the report, many markers of risk are valid at the population level but less predictive for each individual. Biomarker based advice is more likely to be successful in getting people to change their diet if there is a more personalised and certain link between risk marker and disease outcome. The issue of identifying better markers of long-term disease risk is not specific to nutrition studies. The use of post genomic technologies is still in its infancy in nutrition. Across the world we have large numbers of bio-banks, mostly set up in the last decade or two. In the future these may provide better prospective data in relation to biomarkers in apparently healthy people and disease outcome. Assuming such biomarkers can be identified then research into nutritional interventions able to target beneficial changes in such biomarkers will carry considerably more credence and should allow better preventative approaches. Understanding mechanism can inform the identification of risk markers. The area of inflammatory markers is a good example but there are still issues around the validity of for example blood samples to identify inflammation at local sites or even to distinguish between chronic and acute inflammatory responses. Research into improving these risk markers should continue and will help to provide more convincing end-points for RCTs. Implementing advice As a member of a Health Research Authority ethics committee I see an increase in research effort as how to support people to lose weight and to understand socioeconomic factors limiting the uptake of advice. Much of this work is undertaken on a small scale and there should perhaps be a more concerted effort to bring together psychologists and nutritionists and medical professionals. (The need for education of doctors and nurses in this area has been highlighted to me anecdotally by the naivety of the content of their proposals, such as actually encouraging the use of low carbohydrate diets). The ethics of giving advice is an interesting research topic, again far wider than just related to carbohydrates or even nutrition. Should the government give advice when it is based on so much uncertain data? Should this level of uncertainty be conveyed to the general public or would this undermine the message? There may be research published in this area that we as nutritional scientists should be made aware of?	

Contributors:

- Dr Farzad Amirabdollahian, Principal Lecturer in Nutrition, Liverpool Hope University
- Dr Margo Barker, Senior Lecturer in Human Nutrition, University Of Sheffield
- Dr Nino Binns, Consultant in Nutrition and Food Regulation
- Doris M Campbell Honorary Reader, Obstetrics and Gynaecology, University of Aberdeen
- Laura Cherry, Dietitian
- Dr Sandra Drummond, Senior Lecturer in Nutrition, Queen Margaret University College
- Martin Eastwood, formerly consultant gastroenterologist, Western General Hospital, Edinburgh, and Reader in Medicine at the University of Edinburgh
- Klaus Englyst, Englyst Carbohydrates Ltd
- Mary Flynn, Chief Specialist Public Health Nutrition at Food Safety Authority of Ireland (FSAI)
- Professor Bruce Griffin, Professor of Nutritional Metabolism, University Of Surrey
- Dr Fandi Ibrahim, Lecturer in Nutrition and Human Health, University Campus Suffolk
- Geoffrey Livesey, Independent Nutrition Logic
- Dr John Lodge, Reader in Metabolic Nutrition and Head of the Food and Nutrition Research Group, Northumbria University
- Elizabeth Lund, Independent Consultant, Nutrition and Gastrointestinal Health
- Professor Geraldine Mcneill, Nutrition epidemiology and public health nutrition, University of Aberdeen

- Deborah Moore, School of Dentistry, University of Manchester
- Victoria Iyamide Nnatuanya
- Dr Carrie Ruxton, Nutrition Communications
- Diana Sandy, Nutritionist
- Dr Wendy Wrieden, Principal Research Associate, Newcastle University