Scientific Advisory Committee on Nutrition

Consultation on the SACN draft report Saturated Fats and Health Report

Comments Form

Organisation:	MRC Epidemiology Unit, University of Cambridge
Name of commentator and contact details:	Professor Nita G. Forouhi

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Closing date: 5pm 3 July 2018

General comments	Comments Please insert each new comment in a new row
General overview	It is really helpful and a very positive contribution that the SACN committee have reviewed the evidence on saturated fats across a range of health outcomes, including but not limited to cardiovascular disease. The WG are to be congratulated on a comprehensive and clearly laid out review.
Evidence review	The draft report's review strategy is comprehensive and very clearly laid out. I have some suggestions for further evidence to consider for appraisal and for possible inclusion. RCT evidence: 1. Hamley S. The effect of replacing saturated fat with mostly n-6 polyunsaturated fat on coronary heart disease: a meta-analysis of randomised controlled trials. Nutr J. 2017 May 19;16(1):30. doi: 10.1186/s12937-017-0254-5. PMID:28526025. This was published since the cut-off date of the SACN draft report. 2. Schwingshackl L, Hoffmann G. Dietary fatty acids in the secondary prevention of coronary heart disease: a systematic review, meta-analysis and meta-regression. BMJ Open. 2014 Apr 19;4(4):e004487. doi: 10.1136/bmjopen-2013-004487. PMID:24747790 Prospective cohort study evidence from the PURE study with pooled data from 18 countries was published in 2017. Two linked publications and a commentary are relevant (see references below). The evidence from PURE is both for SFA and CVD mortality and morbidity (and by type – CHD or stroke), and also for lipid markers. Associations with and without substitution of nutrients were appraised. There are pros and cons of including this study in the evidence review, including substantial differences in levels of consumption of major macronutrients in some countries included in PURE (such as very high carbohydrate intakes in Asian countries). **1.** Dehghan M et al. Lancet, 2017. doi: 10.1016/S0140-6736(17)32252-3; PMID: 28864332 **2.** Mente A et al. Lancet Diabetes Endocrinol, 2017. doi: 10.1016/S2213-8587(17)30283-8; PMID: 28864143 3. Forouhi NG et al. Lancet Diabetes Endocrinol, 2017. DOI: 10.1016/S2213-8587(17)30285-1; PMID: 28864144
Lipid endpoints	As outlined in the draft report, the prespecified lipid markers were appraised, but these did not include non-HDL cholesterol or apolipoproteins (apo B and apo A1). It would be additionally helpful to include these lipid parameters, as they are predictors of CVD (e.g. Emerging Risk Factors Collaboration, Di Angelantonio E, Sarwar N, Perry P, Kaptoge S, Ray KK, Thompson A, Wood AM, Lewington S, Sattar N, Packard CJ, Collins R, Thompson SG, Danesh J. Major lipids, apolipoproteins, and risk of vascular disease. JAMA. 2009 Nov 11;302(18):1993-2000. doi:

	10.1001/jama.2009.1619. PMID:19903920), and are related to macronutrients intake (Mente A et al. Lancet Diabetes Endocrinol, 2017. doi: 10.1016/S2213-8587(17)30283-8; PMID: 28864143). Particularly for apolipoproteins, the review of RCTs by Mensink included the appraisal of dietary fatty acids and lipids and lipoproteins. Mensink RP. World Health Organization 2016. Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis. http://apps.who.int/iris/bitstream/handle/10665/246104/9789241565349-eng.pdf;jsessionid=AEC184FC30ED7F210DC40618B5D6AB00?sequence=1

Please add extra rows as needed

Comments by paragraph	**Comments** Please insert each new comment in a new row
Para 8.6	The outcome “CVD events” in this analysis by Hooper et al included a broad list of endpoints [cardiovascular deaths, cardiovascular morbidity (non-fatal myocardial infarction, angina, stroke, heart failure, peripheral vascular events, atrial fibrillation) and unplanned cardiovascular interventions (coronary artery bypass surgery or angioplasty)]. The comparability of this vs other definitions of CVD events in other systematic reviews would be valuable. It is difficult to reconcile the significant effect for the combined CVD events endpoint, vs. the null results for all individual endpoints considered (such as for CVD mortality, and for major CVD events including total MI, non-fatal MI or stroke). Some comment on this would be appropriate (this is linked also to the information in paragraphs 8.35 to 8.38, and paras 8.82 and 8.88). The pooled analysis for RCTs of CVD events (for an effect of reduced SFA intake) had an I^2 value of 65%. It would be helpful to critique this in terms of quality criteria and robustness of pooling, and place this in context of the lower I^2 values for other pooled analyses (e.g. within the Hooper review – e.g. a value of 30% for CVD mortality (stated in para 8.5), or other reviews).
Para 8.14	The pooled analysis for RCTs of CVD events (for an effect of SFA substitution with PUFA) had an I^2 value of 69%. It would be helpful to critique this (see related comment above).
Para 8.50	For the reported 24% risk reduction in CHD events in the Hooper et al study, it would be important to place the interpretation in context that the upper bound of the 95%CI is at 1.00 (this is mentioned later in para 8.59, but would be helpful at para 8.50 as well), and an I^2 value is not included.

Para 16.5 and 16.6	Should state beneficial for what (currently stated “would be beneficial) – should emphasise for cardiovascular health.
Para 16.7	This is a very important statement about the relevance of recommendations on macronutrients to dietary advice on foods, and it is well made. However, information on the evidence on the health effects of different types of fat-rich food sources is not appraised in the current report. While this is considered outside of the scope of the review, it would be highly relevant to give associated information, such as the evidence on the divergent health effects of foods such as processed red meats versus dairy products, both rich in SFA. A further nuance is on the accumulating evidence for different health effects within a food group – for instance, within dairy products there are beneficial effects of some dairy types, particularly fermented dairy products such as yoghurt (and cheese). More reference to the importance of foods, over and above nutrients, should be made, and could be highlighted as an area for further review and research need.
Para 17.1, 3rd bullet point	I am not convinced that this will be helpful. Looking back in time to some very old trials is likely to be unhelpful as the dietary landscape has changed substantially and it is better to plan new research than to look back at old data.
Para 17.2, 3rd bullet point	It seems vague to specify to “undertake an intervention study” – would be better to indicate some specific information that would fill existing gaps – e.g. specific to the UK? which population? include people of different ethnic groups? men and women? primary or secondary outcome? In people with and without statin use? which disease outcomes? Only cardiovascular or other endpoints too? The point about “novel study designs” deserves more information. There is only this single isolated mention in this paragraph of Mendelian randomisation (MR) approaches; information including some examples of where a Mendelian randomisation approach has been useful will help.

Please add extra rows as needed