

# Results of the public consultation on the WHO draft guidelines on saturated fatty acid and *trans*-fatty acid intake for adults and children

Comments were received from the following individuals and organizations

## Government agencies

Judith Benedics	Austrian Federal Ministry of Labour, Social Affairs, Health and Consumer Protection
Mary Flynn	Food safety Authority of Ireland
Laura González Céspedes	Instituto Nacional de Alimentación y Nutrición, Paraguay
Kanga Rani Selvaduray	Malaysian Palm Oil Board
Trinidad Trinidad	Food and Nutrition Research Institute, Department of Science and Technology, University of Santo Tomas Graduate School, Philippines

## Nongovernmental and consumer organizations and associations

Carol Dombrow	Heart & Stroke Foundation of Canada
Seema Gulati	National Diabetes, Obesity and Cholesterol Foundation, India
Intazamul Haque*	Diabetes Foundation India
Joseph Jutile Loiseau*	JSI, Ghana
Susanne Logstrup	European Heart Network, Belgium
Claudio Schuftan	PHM, Viet Nam

## Private sector (including industry organizations and associations)

Sebastian Aurich	BASF SE, Germany
Athanasia Baka*	IMACE, Belgium (duplicate submission)
Karyn Barry*	(no organization provided), US
Adam Cole*	(no organization provided), US
Peder Fode	Danish Drink and Food Federation, Denmark
Kalila Hajjar	FEDIOL, Belgium
Chacanne Hanson	Nestlé S.A., Switzerland
James Griffiths	Council for Responsible Nutrition (CRN), US
Megan Hellner*	Potentia Family Therapy, US
Maria Kalergis	Dairy Farmers of Canada
Anita Lawrence	Dairy Australia
Katie Rose McCullough*	North American Meat Institute, US
Shalene McNeill	The Beef Checkoff, US
Petr Mensik	EU Specialty Food Ingredients, Belgium
Gregory Miller	National Dairy Council & Global Dairy Platform, US
Laura Miranda	ConMéxico
Siska Pottie	IMACE, Belgium (duplicate submission)
Rocco Renaldi	International Food & Beverage Alliance, Belgium
Lawrence Rycken	International Dairy Federation, Belgium
Ashley Rosales	Dairy Council of California, US
Sushum Sharma	HTK Medical Center, Dubai, U.A.E., United Arab Emirates
Tracy Strilich	Stepan Company, US
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Samantha Wong	Food Industry Asia, Singapore

**Academic/research**

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Arne Astrup†	University of Copenhagen, Denmark
Salmeh Bahmanpour	Shiraz University of Medical Sciences, Islamic Republic of Iran
Pratima Borade*	Grant government medical college Mumbai, India
Grażyna Cichosz	University of Warmia and Mazury, Poland
VG Kumar Das	Academician Emeritus, Malaysia
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Sigal Eilat-Adar	The Academic College at Wingate, Israel
Nita Farouhi	MRC Epidemiology Unit, University of Cambridge School of Clinical Medicine
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Tony Kock Wai Ng	University Tunku Abdul Rahman, Malaysia
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Bernhard Watzl*	Max Rubner-Institut, Federal Research Institute of Nutrition and Food, Germany
Walter Willett	Harvard T.H. Chan School of Public Health, US

**Other**

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Amanda Atkins	Retired, Canada
Jean-Michel Lecerf	Institut Pasteur de Lille (private foundation), France
Catherine Price*	Science journalist, US
Permjit Singh	Private citizen, UK

\* Comments submitted, but completed declaration of interest forms not received

† Comments were submitted on behalf of a group. See Annex for a full list of signatories

UK, United Kingdom of Great Britain and Northern Ireland; US, United States of America

## Summary comments and WHO responses

Comments were compiled and summarized (and/or paraphrased), and brief responses prepared. (Comments received without completed DOI forms were not included in this process).

### WHO guideline development process

Summary comment	Response
Recommendations should only be made on strong, high quality evidence. Making recommendations that may lead to policy change, without sound underlying evidence is dangerous. Some of the evidence supporting this recommendation is not of high quality, including the evidence for an effect of lower saturated fatty acid intake on risk of cardiovascular diseases assessed in randomized controlled trials (RCTs), which is moderate quality.	<p>WHO develops recommendations and guidance on matters of public health importance even when the certainty in (i.e. quality of) the evidence is <i>low</i> or <i>very low</i>.</p> <p>The certainty in the evidence as assessed by GRADE is relative to the high certainty benchmark of well-conducted, double-blind, RCTs. Although several relevant outcomes for which associations were observed were assessed as being of low to very low certainty, several key outcomes were also assessed as <i>moderate</i> to <i>high</i> certainty. The NUGAG Subgroup on Diet and Health considered the overall evidence to be very robust.</p> <p>In addition, the certainty in the evidence is only one factor considered when formulating recommendations; other factors include: desirable and undesirable effects of the intervention; priority of the problem that the recommendation addresses; values and preferences related to the recommendation in different settings; the cost of the options available to public health officials and programme managers in different settings; feasibility and acceptability of implementing the recommendation in different settings; and the potential impact on equity and human rights. In situations where consideration of these other factors leads to a conclusion that is supportive of a recommendation, recommendations can be appropriate – and necessary – even when based on <i>low</i> to <i>very low</i> certainty evidence.</p>
Policy decisions shouldn't be made on conditional/weak recommendations – strong recommendations are necessary. Therefore WHO should not issue conditional recommendations.	Within the GRADE framework as utilized by the WHO and many other organizations, there are options for making recommendations that take into consideration the certainty in the evidence as well as a number of additional factors which allow for recommendations to be made when there is less confidence in the evidence and/or that the other factors considered strongly support a recommendation. Such <i>conditional</i>

	(or <i>weak</i> ) recommendations acknowledge the possibility that all may not benefit from a recommended intervention regardless of any particular circumstance, and provide end users with more flexibility in translating the recommendations given their particular situation. Policy decisions can therefore be made on <i>conditional</i> recommendations, but may require substantial debate and involvement of various stakeholders.
Further clarity on how a conditional recommendation should be interpreted and implemented is needed. End-users may not be able to differentiate between strong and conditional recommendations.	Multiple footnotes have been provided describing in detail what a <i>conditional</i> recommendation means in terms of policy-making, however, we will try to further highlight this, possibly by including a visible text box containing an explanation.
WHO should base its dietary guidelines on a wider base of information and not limit itself to western diets and lifestyle conditions.	WHO makes every effort to compile and review evidence from all settings, however there is a paucity of published epidemiological data on healthy diets from LMICs, particularly with respect to macronutrient intakes. A small number of studies from LMICs were included in the systematic review, including the multi-country PURE study, but the urgent need for more data from LMICS is noted in the <i>Research gaps and future initiatives</i> section with the following text: “further assess the health effects of replacing saturated fatty acids with different macronutrients in populations from different geographical regions, particularly low- and middle-income countries.

### The recommendations

Summary comment	Response
The inclusion of ‘if needed’ in the recommendation is confusing (i.e. WHO suggests using polyunsaturated fatty acids as a source of replacement energy, <u>if needed</u> , when reducing saturated fatty acid intake. Either it should be removed, or the statement should be aligned with the text in the Remarks which states “when a replacement is needed’.	Noted. This wording has been removed from the recommendations.
The way the recommendations are currently worded is very confusing, i.e.:  1. In adults and children whose saturated fatty acid intake is greater than 10% of total energy	Noted. The recommendations have been reworded to improve clarity as follows:

<p>intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake</p> <p>2. In adults and children, WHO suggests reducing the intake of saturated fatty acids to less than 10% of total energy intake</p>	<p>1. WHO recommends that adults and children reduce saturated fatty acid intake to 10% of total energy intake (<i>strong</i>)</p> <p>2. WHO suggests further reducing saturated fatty acid intake to less than 10% of total energy intake (<i>conditional</i>)</p> <p>This acknowledges that the evidence for reducing saturated fatty acid intake to less than 10% is less robust than for reducing to 10%.</p> <p>(Analogous changes have been made to the trans-fatty acid recommendations).</p>
<p>Proposal to translate the recommendations into practical portions, or type of food and not only in percentage of calories.</p>	<p>The recommendations are formulated in terms of percentage of energy intake such that policymakers can translate the recommendations at the country-level into culturally and contextually specific policies and actions that take into account locally available foods and dietary customs among other factors.</p>
<p>The guideline should include more information/details on carbohydrates as a replacement for saturated fatty acids.</p>	<p>Originally there was limited information available on the nature of carbohydrates as replacements in the evidence reviewed. The systematic review of observational studies commissioned by WHO was updated (in 2023) after the public consultation was held. In this review, additional information was available on carbohydrates in the replacement analyses such that it was possible to look at effects for different types of carbohydrates (e.g. whole-grains, low/high glycaemic, sugars) for some outcomes.</p>
<p>There is no clear basis for setting the recommendation at 10% energy or 1% energy.</p>	<p>There is limited evidence on which to base very specific thresholds and consequently the evidence reviewed does not provide specific thresholds with high certainty. However, in considering the totality of the evidence alongside practical considerations in terms of what can be achieved by individuals, the WHO Subgroup on Diet and Health formulated the recommendations with the indicated thresholds.</p>
<p>It appears that for both saturated fatty acids and <i>trans</i>-fatty acids, the first recommendations are intended as individual targets (not a population goal), while the second recommendations seem to be population goals. This needs to be clarified. If the first</p>	<p>Because much of the evidence that NUGAG reviewed came from assessment of individuals and dose-response relationships were observed for many outcomes the decision was made to formulate the recommendations such that the recommended levels of intake for saturated</p>

<p>recommendations are for individuals, information should be provided on how this might be assessed in practice.</p>	<p>fatty acids and <i>trans</i>-fatty acids are targets for individuals to achieve, not population goals. NUGAG further concluded that individual targets would be easier to implement, particularly in terms of updating food-based dietary guidelines, education/awareness campaigns, and other interventions aimed at eliciting desired behavioural change at the individual level. The recommendations were reworded, clarifying that the dietary targets are for individuals, not populations.</p>
<p>An additional recommendation to reduce industrially produced trans-fatty acids from the food supply by removing partially hydrogenated oils in foods products should be added.</p>	<p>Formulating a recommendation to remove partially hydrogenated oils from the food supply is beyond the scope of this guideline, however a remark has been included stating that: These recommendations do not preclude consumption of particular foods, however, foods containing high levels of industrially produced trans-fatty acids should largely be avoided.</p> <p>Additionally, text covering the elimination of industrially produced trans-fatty acids has been added to the <i>Translation and implementation</i> section as follows: “Elimination of industrially produced trans-fatty acids is among the priority actions identified by WHO in its 13th General Programme of Work which will guide the work of the Organization in 2019 – 2023. Industrially produced trans-fatty acids are the predominant source of dietary trans-fatty acids in many populations and can be found in baked and fried foods (e.g. doughnuts, cookies, crackers and pies), pre-packaged snacks and food, and partially-hydrogenated cooking oils and fats which are often used at home, in restaurants or in the informal sector, such as street vendors. Therefore, removing industrially produced trans-fatty acids from the food supply through legislation or regulatory action represents a well-defined mechanism for translating the recommendations into action and achieving significant reductions in trans-fatty intake at the population level.</p> <p>In 2018, WHO released the REPLACE action framework, which provides support in implementing the WHO recommendations on trans-fatty acids and is a roadmap for countries to implement the prompt, complete and sustained elimination of industrially produced trans-fatty acids from the food supply. In 2019,</p>

	WHO released six REPLACE modules, which provide practical, step-by-step implementation guidance to support governments. WHO recommends that countries adopt and implement one of two best-practice policy options for eliminating industrially produced T trans-fatty acids from the food supply. Prior to the release of REPLACE, industrially produced trans-fatty acids had already largely been removed or were in the process of being removed from the food supply at the national and subnational level in many countries. As of 2020, 32 countries have implemented mandatory trans-fatty acid limits, and of these, 14 countries have implemented a best-practice trans-fatty acids policy that either virtually eliminates industrially produced trans-fatty acids or bans partially hydrogenated oils, demonstrating that global reduction in trans-fatty acid intake may be an achievable goal.”
It is surprising that <i>trans</i> -fatty acid recommendations are being proposed for children and yet no studies were identified that met the inclusion criteria established for the systematic review of studies conducted in children.	The NUGAG Subgroup on Diet and Health concluded that although adverse clinical cardiovascular outcomes in children are rare, there is no evidence to indicate that the physiological response to a change in <i>trans</i> -fatty acid intake would be significantly different between adults and children. Therefore the Subgroup decided to extrapolate the results for <i>trans</i> -fatty acid intake in adults to children without downgrading for indirectness.

## Evidence

Summary comment	Response
<b>Evidence: evidence used in the formulation of recommendations</b>	
WHO commissioned a systematic review of observational studies which found no association between saturated fatty acid intake and cardiovascular diseases, but it was not considered when formulating the recommendations.	WHO commissioned a systematic review of prospective cohort studies for saturated fatty acids (and <i>trans</i> -fatty acids) after the scoping review failed to identify a recent systematic review that adequately answered the PICO questions as established by the NUGAG Subgroup on Diet and Health. The commissioned systematic review of prospective cohort studies was reviewed and discussed extensively by the NUGAG Subgroup on Diet and Health. However, the primary concern about this systematic review was that the studies included in the review did not allow for an assessment of replacement nutrients for

	<p>saturated fatty acids. Although the studies generally did not provide information on what was being consumed in place of saturated fatty acids, it was suspected that refined carbohydrates made up a significant portion of the replacement nutrients in many of the studies. The authors of the review note “Our a priori research question was to examine the effect on the health outcomes of higher compared with lower saturated fat, which we did by comparing highest and lowest intake estimates. Such a comparison, however, obscures the importance of reciprocal and possibly heterogeneous decreases in other macronutrients that accompany high saturated or trans fat intakes. Thus, an overarching consideration is that the effect estimates of higher intakes of saturated or trans fats on health outcomes is linked to the nutrient that it replaces. Most studies in the present review did not explicitly model the effects of nutrient substitution, but when total energy, protein, and alcohol are covariates in the multivariable model, coefficients for fat reflect substitution of saturated or trans fat for carbohydrate. Indeed, carbohydrate energy was typically lowest in those in the highest intakes of saturated and trans fat. Common sources of carbohydrate in typically studied populations were highly processed high glycaemic load foods which can increase risk of [coronary heart disease] independently of saturated and trans fats through different metabolic pathways; likely attenuating the observed associations between these fats and outcomes.”</p> <p>The systematic review of observational studies commissioned by WHO was updated (in 2021) after the public consultation was held. Results of this systematic review were reviewed by the NUGAG Subgroup on Diet and Health and reaffirmed the recommendations.</p>
<p>WHO did not consider other systematic reviews of RCTs which have arrived at different conclusions than those in the Hooper et al. review. Consideration should be given to other well conducted systematic reviews that were not commissioned by WHO in order to ensure other perspectives and reduce "perception of bias".</p>	<p>As per the WHO guideline development process, evidence in the form of systematic reviews is required to inform the development of recommendations. The systematic reviews must answer specific PICO questions developed during the scoping phase of the guideline development work, in which the literature is surveyed to identify critical issues related to the topic of interest. Part of the scoping work thus</p>



	<p>involves identifying recent systematic reviews which are then assessed to see if any are capable of providing answers to all PICO questions. The scoping review for saturated fatty acids and trans-fatty acids did not identify suitable, existing reviews and therefore several were commissioned by WHO to specifically answer the PICO questions as developed by the WHO NUGAG Subgroup on Diet and Health.</p> <p>Several systematic reviews were published in the same time period that the review by Hooper et al. as being finalized and published. Differences in the results and conclusions of several of the systematic reviews of RCTs largely stem from differences in the trials that were included, which in turn result from different inclusion and exclusion criteria (though many of the included trials across the various reviews are the same). The inclusion and exclusion criteria used in the Hooper et al. review were very carefully established taking into consideration many factors relevant to dietary interventions and development of disease. NUGAG felt these inclusion and exclusion criteria to be the most well-suited to answer the specific PICO questions regarding the effects of saturated fatty acid intake on risk of cardiovascular diseases.</p>
The Global Burden of Disease study 2016 is cited to make the case that cardiovascular diseases were leading causes of mortality in 2016 and several modifiable risk factors were identified including an unhealthy diet. However, this same study did not include saturated fat as a dietary risk factor amongst those listed.	This type of evidence is not formally reviewed as part of the WHO guideline development process as it does not directly measure the effects of consuming more or less of the nutrient on health effects. With that said, there have been many GBD studies published and some of them do note saturated fatty acid intake as a risk factor.
The results from The PURE cohort study of 18 countries (Dehghan et al 2017) should also be considered. This study highlighted that a “high carbohydrate intake was associated with higher risk of total mortality, whereas total fat and individual types of fat were related to lower total mortality.	The systematic review of observational studies commissioned by WHO was updated (in 2021) after the public consultation was held. The PURE study was included in the updated systematic review.
<b>Evidence: interpreting the evidence</b>	
The evidence to show that saturated fatty acids are associated with increased risk of cardiovascular disease and that replacement with polyunsaturated fatty acids reduces risk	Claims that saturated fatty acid intake is not linked to cardiovascular diseases are based largely on the results of meta-analyses of observational studies, as well as some meta-

<p>has weakened in the last several years after being challenged by several recent systematic reviews and meta-analyses of observational studies and RCTs that have found no link between saturated fatty acid intake and increased risk of cardiovascular disease. WHO did not consider these other systematic reviews and instead used outdated evidence from RCTs only to develop the guidelines (i.e. excluded observational studies from consideration). The currently available evidence for saturated fatty acid intake does not support the recommendations.</p>	<p>analyses of RCTs that show no link between SFA intake and relevant outcomes. The primary reason why most observational studies and some RCTs do not show a link as described below, however, is that replacement nutrients for saturated fatty acids have not been considered when analysing the results of these studies.</p> <p>It is well-established that changes in macronutrient intakes do not operate independently of one another and unless energy intake is being restricted, changes in one macronutrient must be accompanied by compensatory changes in other macronutrients. Therefore, when individuals in energy balance reduce their intake of saturated fatty acids, they must consume something in their place, and what they consume as replacement for the saturated fatty acids can independently impact the effect on risk of cardiovascular diseases. In the many prospective cohort studies that have reported no effect of lower saturated fatty acid intake, the replacement was not factored into the analysis and therefore it is unknown what was being consumed in place of saturated fatty acids.</p> <p>When the effects of replacement nutrients are assessed, there is consistent and robust evidence from both observational studies and RCTs that replacing saturated fatty acids with polyunsaturated fatty acids reduces risk of cardiovascular diseases.</p> <p>The systematic review of observational studies commissioned by WHO was updated (in 2021) after the public consultation was held. In this review, replacement analyses were assessed and showed that in addition to replacement of saturated fatty acids with polyunsaturated fatty acids, replacement with whole grains and fibre-containing carbohydrates and plant-based monounsaturated fatty acids also reduces risk of cardiovascular diseases and mortality. The recommendations were reviewed by the NUGAG Subgroup on Diet and Health in the context of this updated review and were reaffirmed. The recommendations are thus based on the most recent data from RCTs and observational studies.</p>
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<p>Data were selectively chosen, i.e. “cherry-picked” to support the draft recommendations on saturated fatty acid intake. For example, of the several outcomes that were assessed in RCTs, only cardiovascular events was considered in formulating the recommendations even though other major outcomes showed no effect.</p>	<p>Data was not selectively chosen in terms of assessing the effects on priority health outcomes and the results observed do not conflict, i.e. there is not an increase in any of the critical outcomes that would contradict what is observed for cardiovascular events. Other outcomes show a neutral effect or a non-significant reduction in risk. In formulating recommendations, it is not necessary to observe an increase in risk across all outcomes that have been prioritized for assessment. However, evidence suggesting increased cardiovascular disease risk with increased saturated fatty acid intake was coherent across several different study types (i.e. RCTs and prospective cohort studies) and outcomes (i.e. disease incidence, mortality and blood lipids).</p>
<p>Results are not generalizable to the general population because the majority of studies included in the systematic review of RCTs and disease outcomes (Hooper et al.) were in sick participants (e.g. with cardiovascular diseases, diabetes) and these results were extrapolated from adults to children. The results for children are also not generalizable because some studies were conducted in children with elevated cholesterol.</p>	<p>The Hooper et al. systematic review includes both at risk individuals and healthy individuals and is therefore more representative of the general population than if studies were restricted to healthy individuals. The updated systematic review of cohort studies included individuals free from diagnosed cardiovascular diseases or type 2 diabetes at baseline.</p> <p>The systematic review in children included both children with and without elevated cholesterol and of different body weights. It therefore was more representative of the general child population than if all studies restricted to only healthy or only with elevated cholesterol or body weight.</p> <p>Regarding the extrapolation of results from adults to children, the NUGAG Subgroup on Diet and Health reviewed the evidence for adults and children and concluded that disease progression can begin early in life. Although cardiovascular diseases typically present later in life, lesions in the aorta and coronary arteries that can signal the beginning of artery narrowing or ‘clogging’ (i.e. atherosclerosis) can begin to appear in childhood and are positively associated with dyslipidaemia (i.e. unhealthy changes in blood lipids such as cholesterol and triglycerides) and other cardiovascular disease risk factors. Elevated total and LDL cholesterol in childhood are in turn associated with an increase in cardiovascular disease risk factors in adulthood, including thickening of the inner</p>

	layers of the carotid artery which is an indication of subclinical atherosclerosis and predictor of future cardiovascular disease.
There is little to no evidence supporting the grouping of ruminant <i>trans</i> -fatty acids with industrially produced <i>trans</i> -fatty acids as has been done in the guidelines. The evidence provided in the guideline is insufficient. The quality of evidence regarding total <i>trans</i> -fatty acid intake and health outcomes for each of the draft DWG recommendations ranges in quality from very low to moderate, indicating that further research is “likely” to “very likely” to have an important impact on the confidence in the estimate of effect and may likely change the estimate (WHO, 2012). In the case of “very low” evidence quality, “any estimate of effect is very uncertain.” (WHO, 2012). In cases where evidence is insufficient the decision to not make a recommendation can be made as per the WHO Handbook. A recommendation to reduce <i>trans</i> -fatty acid intake that includes ruminant <i>trans</i> -fat is unprecedented among world-wide interventions to limit <i>trans</i> -fat intake and could result in difficulties with labelling regulations between countries that have adopted legislation based on industrial <i>trans</i> -fatty acids only. A recommendation to reduce <i>trans</i> -fatty acid intake that includes ruminant <i>trans</i> -fat is unprecedented among world-wide interventions to limit <i>trans</i> -fat intake and lacks strong scientific evidence to support the recommendation. Question making no distinction between ruminant and industrially produced <i>trans</i> -fatty acids in the recommendations given the low intake of ruminant <i>trans</i> -fatty acids.	The rationale for including ruminant and industrially produced <i>trans</i> -fatty acids together in the context of the recommendations is provided in the guideline as follow: “As per the original PICO questions, results were generated for total <i>trans</i> -fatty acid intake <sup>1</sup> , and separately for industrially produced and ruminant <i>trans</i> -fatty acid intake for both the meta-analyses of observational studies and regression analyses of RCTs and blood lipids. In the meta-analyses of prospective observational studies, results for total and industrially produced <i>trans</i> -fatty acid intake were similar in their effects on risk of coronary heart disease, but not for all-cause mortality or cardiovascular diseases for which only total <i>trans</i> -fatty acid intake demonstrated a significant association between reduced intake and reduced risk. No associations were observed for the analysis of studies reporting effects of ruminant <i>trans</i> -fatty acid intake. <sup>2</sup> In the regression analysis of RCTs, reduced intake of total <i>trans</i> -fatty acids or industrially-produced <i>trans</i> -fatty acids was associated with a beneficial effect on the blood lipid profile, regardless of which nutrient was used as a replacement. A significant effect of reducing ruminant <i>trans</i> -fatty acid intake on lowering LDL cholesterol was only observed when ruminant <i>trans</i> -fatty acids were replaced with polyunsaturated fatty acids. For all other blood lipid outcomes, results were not statistically significant, however, they were similar to those

<sup>1</sup> For the meta-analysis of prospective cohort studies, separate analyses were performed for total, industrially produced and ruminant *trans*-fatty acids, because most studies did not differentiate between industrially produced and ruminant *trans*-fatty acids and only reported results for total *trans*-fatty acid intake. For the regression analysis of total *trans*-fatty acids, all trials that assessed either total, industrially produced, or ruminant *trans*-fatty acid intake were included in a single analysis.

<sup>2</sup> Results for effects of industrially produced and ruminant *trans*-fatty acids on all-cause mortality and cardiovascular diseases come from studies in which dietary intake was assessed by tissue measurements. Although the NUGAG Subgroup on Diet and Health expressed concerns with the assessment of dietary intake of saturated fatty acids via tissue measurements, the correlation between *trans*-fatty acids measured in tissue and dietary intake has generally been shown to be stronger. Results for effects of industrially produced and ruminant *trans*-fatty acids on coronary heart disease come from studies in which dietary intake was self-reported.

	<p>for total and industrially produced <i>trans</i>-fatty acids, both in direction and magnitude.<sup>3</sup></p> <p>Intake of ruminant <i>trans</i>-fatty acids in the studies included in the analyses of both prospective observational studies and RCTs was very low relative to intakes of industrially produced <i>trans</i>-fatty acids and the difference between lower and higher intakes was very small. The available evidence suggested that differences in effects on health outcomes between ruminant, industrially produced, and total <i>trans</i>-fatty acids observed in many studies may likely be due to differences in the amount of <i>trans</i>-fatty acids being consumed rather than differences between type of <i>trans</i>-fatty acids. In order to further assess the nature of the observed differences, post-hoc analyses were conducted in which the intakes observed in the studies of ruminant <i>trans</i>-fatty acids were approximated in the studies of total <i>trans</i>-fatty acids, such that the highest intakes of total <i>trans</i>-fatty acids were limited to 0.7-1.3% of total energy intake and then compared to the lowest intakes. When total <i>trans</i>-fatty acid intake was assessed in this manner, the associations and dose-response relationships originally observed between lower <i>trans</i>-fatty acid intake and reduced risk of all-cause mortality remained, but those for cardiovascular diseases and coronary heart disease were no longer present. Based on these observations, the NUGAG Subgroup on Diet and Health concluded that at the low levels of ruminant <i>trans</i>-fatty acid intake in the small number of studies, the difference between the lowest and highest intakes wasn't large enough to obtain reliable results when compared to one another. It was further noted that in the very few studies assessing LDL cholesterol in which the highest levels of ruminant <i>trans</i>-fatty acid intake were closer to that observed for industrially produced and total <i>trans</i>-fatty acids, the effects of ruminant <i>trans</i>-fatty acid intake were similar to or more pronounced than that observed for industrially produced and total <i>trans</i>-fatty acids.</p>
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<sup>3</sup> The two trials that reported ruminant *trans*-fatty acid intakes at levels more similar to the intakes reported for industrially produced *trans*-fatty acids (i.e. >2% of total energy intake), reported greater reductions in LDL cholesterol.

	<p>It was therefore determined that the available evidence did not support making a distinction between industrial and ruminant <i>trans</i>-fatty acids, and because the vast majority of studies included in the systematic reviews reported results for total <i>trans</i>-fatty acid intake which includes intake from industrially produced and ruminant sources and is thus the total cumulative intake of <i>trans</i>-fatty acids, it was considered appropriate to assess/consider only evidence from total <i>trans</i>-fatty acids when formulating the recommendations on <i>trans</i>-fatty acid intake.</p>
<p>The recommendations on <i>trans</i>-fatty acids should not include conjugated linoleic acid (CLA). The current global regulatory framework overall clearly excludes conjugated fatty acids from the definition of <i>trans</i>-fatty acids. The rationale for inclusion of CLA in the definition of total <i>trans</i>-fatty acids as provided in the guideline, is not convincing and is based on weak evidence. While the evidence for adverse health effects with regard to cardiovascular disease risk is strong for industrially produced <i>trans</i>-fatty acids, it is unlikely that CLA has such a negative effect.</p>	<p>WHO performs its own independent assessment of the evidence and develops guidelines based on the WHO guideline development process.</p> <p>As CLA contains both <i>cis</i> and <i>trans</i> configurations, RCTs assessing CLA as it naturally occurs in foods (i.e. not from supplements) were included in the systematic review and regression analyses of blood lipids<sup>4</sup>, although the number of such trials were limited and intakes of CLA were very low. Nevertheless, results of these trials provided no indication that they had an effect on blood lipids that was significantly different from other <i>trans</i>-fatty acids when consumed at similar levels. The NUGAG Subgroup on Diet and Health therefore concluded that because CLA contributes to total <i>trans</i>-fatty acid intake it should be included in the definition of <i>trans</i>-fatty acids as used in the recommendations on <i>trans</i>-fatty acids.</p>
<p><b>Evidence: food-based evidence</b></p>	
<p>The recommendations do not take into consideration recent evidence showing that the health effects of saturated fatty acids depend largely on the specific food source of saturated fatty acids. There is increasing evidence to suggest that the matrix of a food is more important than simply which fatty acids it contains when predicting the effect of a food on cardiovascular disease risk and some saturated fatty acid-containing foods (e.g. milk, cheese, yogurt, dark chocolate, eggs) have been shown</p>	<p>WHO recognizes the interest in, and utility of, dietary guidance based on foods and dietary patterns (WHO is in the planning stages of guidance on elements of dietary patterns), and acknowledges the body of literature assessing health effects of a relatively small number of specific saturated-fatty acid containing foods, including work on attempting to understand the possible role of the food matrix in the context of disease risk. However, WHO nutrition guidelines and recommendations therein</p>

<sup>4</sup> Although a number of studies on the health effects of CLA supplements have been published, the amounts provide in supplements are generally well above what is consumed naturally in foods and assessment of this separate body of evidence was considered to be beyond the scope of this guideline.

<p>to have a neutral or even beneficial effect on cardiovascular diseases and type 2 diabetes. Therefore a purely macronutrient approach to formulating recommendations on saturated fatty acids, without considering different effects of various saturated fatty-acid containing foods, does not reflect the currently available evidence, and may lead to reduced intake or exclusion of nutrient-dense foods that may reduce the risk of cardiovascular diseases, type 2 diabetes and other serious noncommunicable diseases. The WHO evidence review should focus on foods and dietary patterns (rather than isolated nutrients) and a more food-based translation of the recommendations on how to achieve the reduction in saturated fatty acid intake is strongly recommended.</p>	<p>are intended for a global audience and therefore must be relevant globally. Macronutrient-based recommendations are therefore valuable because they are globally relevant and allow governments and national decision-making bodies to translate the recommendations as needed at the country level into culturally and contextually specific food-based dietary guidelines that take into account locally available foods and dietary customs. It must also be noted, that while the recommendations themselves do not explicitly address specific foods, they also do not prohibit consumption of any foods, including the foods provided as examples in the comments. However the recommendations on saturated fatty acids (and <i>trans</i>-fatty acids) must also be considered alongside other WHO recommendations on healthy diets.</p> <p>From a process perspective, the NUGAG Subgroup on Diet and Health was specifically tasked with updating the population macronutrient intake goals as originally established by the 1989 WHO Study Group on Diet, Nutrition and Prevention of Noncommunicable Diseases, and later updated by the 2002 Joint WHO/FAO Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases. This work includes not only updating guidance on saturated fatty acids and <i>trans</i>-fatty acids, but also sodium, potassium and free sugars (which has been completed), and total fat, polyunsaturated fatty acids, and carbohydrates, which is currently being finalized.</p>
<p>Food based meta-analyses (including food exposures and objective in vivo measurements of individual saturated fatty acids linked to dairy intake) have failed to find that intake of dairy as a group is associated with increased risk of cardiovascular disease. Current evidence does not support a positive association between intake of dairy products and risk of cardiovascular disease and type 2 diabetes. By contrast, fermented dairy products such as cheese and yogurt generally show inverse associations. There is also some evidence suggesting higher fat/full fat dairy is more beneficial for health than low-fat dairy for body weight and possibly other outcomes.</p>	<p>As noted, the recommendations do not prohibit consumption of any foods. The currently available evidence indicates that replacing saturated fatty acids regardless of source with polyunsaturated fatty acids, whole grains and fibre-containing carbohydrates, and plant-based monounsaturated fatty acids reduces risk of cardiovascular diseases and mortality.</p>

Coconut oil is a healthy MCT vegetable oil. There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity. Coconut has been consumed for millennia in the tropics and the Pacific with no indication of poor health outcomes. Coconut oil, which makes up about 20% of coconut meat, is part of this healthy tradition. Close to one billion people today consume coconut and coconut oil on a regular basis. If coconut oil caused heart disease, then there should be an epidemic of heart disease in all coconut consuming countries. Clearly, the evidence against coconut oil is indirect and invalid. Coconut oil is not purely a long chain SFA and should not be compared to the SFA present in pork, beef and butter.	As noted, the recommendations do not preclude the consumption of any foods. WHO guidance on tropical oils is currently being developed and will address coconut oil.
<b>Evidence: individual saturated fatty acids</b>	
In addition to studies assessing various whole foods containing saturated fatty acids, there is evidence that different, individual saturated fatty acids have differential effects on cardiovascular disease risk and risk factors. Medium-chain saturated fatty acids, odd-chain saturated fatty acids, and very long-chain saturated fatty acids, each appear to be metabolically beneficial.	The evidence coming from the systematic review of observational studies assessed saturated fatty acid intake as commonly consumed in different populations, which would include saturated fatty acids of different lengths, although medium-chain and very long-chain saturated fatty acids are less common in most diets. Therefore the NUGAG Subgroup on Diet and Health considered that recommendations on saturated fatty acids as a class were most relevant. The NUGAG Subgroup on Diet and Health further noted that while differential effects are possible across saturated fatty acids of different length, further research is needed before firm conclusions can be drawn.
Medium chain triglycerides (MCTs) are beneficial for health and must be recognized separately from long-chain saturated fat.	MCTs are short saturated fatty acids and are much less common in the food supply than other saturated fatty acids. To the extent that they contribute to diets in free-living populations as assessed in the prospective cohort studies, they contribute to saturated fatty intakes and therefore are covered by the recommendations.
In addition to the data for individual saturated fatty acids from the systematic review of RCTs assessing blood lipids, there are a number of observational studies that have assessed individual saturated fatty acids. It is not clear why these were not included as this supplements the evidence from the RCTs in a meaningful way.	Because the updated systematic review of observational studies included prospective cohort studies that assessed individual saturated fatty acids (including odd chain saturated fatty acids) the NUGAG Subgroup on Diet and Health reviewed evidence for individual saturated fatty acids from both RCTs and prospective cohort studies. In the



	<p>prospective cohort studies assessing the intake of individual saturated fatty acids takes, intakes were assessed by tissue measurements of individual saturated fatty acids. In reviewing these results the NUGAG Subgroup on Diet and Health noted that although assessment of saturated fatty acids in tissues can be a relatively reliable indicator of dietary intake, the potential contribution of endogenous synthesis cannot be consistently estimated.</p> <p>Although significant associations were observed between certain individual saturated fatty acids (as assessed by tissue levels) and type 2 diabetes, and differences were observed between individual saturated fatty acids with respect to their effects on blood lipids, with the exception of stearic acid (which showed little effect on blood lipids), results observed for associations between individual saturated fatty acids and disease outcomes were consistent with the results observed for total saturated fatty acids in that none of the statistically nonsignificant effects observed for individual saturated fatty acids suggested benefit with increased intake, but some suggested harm. In addition, the NUGAG Subgroup on Diet and Health had concerns with tissue measurements as described in the preceding paragraph (regarding uncertainty in the ability to ensure consistent measurement of endogenous synthesis of saturated fatty acids) as well as with the low reported intakes of lauric and myristic acids in the blood lipids analyses. Finally, there was no evidence available from RCTs assessing the effects of consuming individual saturated fatty acids on disease outcomes.</p> <p>In reviewing the evidence, the NUGAG Subgroup on Diet and Health further noted that saturated fatty acids as they are found naturally in foods are generally mixtures, and accordingly, intakes of individual saturated fatty acids tend to be highly correlated with one another. Therefore, recommendations for individual fatty acids may be of limited utility to end-users and difficult to implement; for example, in developing food-based dietary guidelines and related activities. The NUGAG Subgroup on Diet and Health Although therefore concluded that</p>
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	further research into the health effects of individual saturated fatty acids as well as how recommendations for individual saturated fatty acids might be effectively used is needed before recommendations for individual saturated fatty acids can be made.
<b>Evidence: polyunsaturated and monounsaturated fatty acids</b>	
High intakes of omega-6 polyunsaturated fatty acids are detrimental for health, and have been linked to the rise in obesity and inflammatory disease. Therefore unlimited replacement of saturated fatty acids with polyunsaturated fatty acids should not be recommended.	The recommendations in this guideline do not indicate a level of intake for omega-6 (i.e. n-6) polyunsaturated fatty acids. WHO guidance on polyunsaturated fatty acids is currently being updated. The recommendations in this guideline should be considered in the context of the current WHO (and forthcoming updates to the recommendations) on polyunsaturated fatty acids, as well as other guidance on healthy diets.
The recommendation to replace saturated fatty acids with polyunsaturated fatty acids neglects the fact that polyunsaturated fatty acids (primarily in the form of seed oils in many populations) are bad for ones' health.	Reviewing the evidence for health effects of polyunsaturated fatty acids on their own is beyond the scope of this guideline. Polyunsaturated fatty acids are only considered in the context of replacing saturated fatty acids or <i>trans</i> -fatty acids, and in both cases, improvements in health are observed. Evidence for the health effects of polyunsaturated fatty acids on their own is being reviewed by the NUGAG Subgroup on Diet and Health in the context of work they are doing to update WHO guidance polyunsaturated fatty acid intake.
The guideline doesn't address the n3:n6 polyunsaturated fatty acid ratio. Having a desirable n3:n6 ratio is important for health.	The n3:n6 ratio is not directly relevant to the evidence for saturated fatty acids or <i>trans</i> -fatty acids. The NUGAG Subgroup on Diet and Health is discussing the n3:n6 ratio in the context of work they are doing to update WHO guidance on polyunsaturated fatty acid intake.
The guideline does not provide guidance or information on optimal monounsaturated fatty acid:polyunsaturated fatty acid ratios.	Reviewing the evidence for health effects of monounsaturated fatty acid:polyunsaturated fatty acid ratios is beyond the scope of this guideline. Monounsaturated fatty acids and polyunsaturated fatty acids are only considered in the context of replacing saturated fatty acids or <i>trans</i> -fatty acids. Evidence for the health effects of polyunsaturated fatty acids on their own is being reviewed by the NUGAG Subgroup on Diet and Health in the context of work they are doing to update WHO guidance polyunsaturated fatty acid intake.

<p>It needs to be recognised that only a limited amount of saturated fatty acids can be replaced by polyunsaturated fatty acids. There is evidence to indicate that the use of vegetable oils high in monounsaturated fatty acids as part of a Mediterranean diet is associated with lower cardiovascular disease events.</p>	<p>As noted in the recommendations, saturated fatty acids can be replaced by polyunsaturated fatty acids, monounsaturated fatty acids from plant-sources, and carbohydrates from foods containing naturally-occurring dietary fibre such as whole grains, vegetables, fruits and pulses.</p>
<p><b>Evidence: blood lipids as an outcome</b></p>	
<p>There seems to be equal weight given to blood lipid markers (especially LDL cholesterol concentration) and the disease outcomes included in the evidence review. This is not appropriate as blood lipid markers are only risk factors and only LDL cholesterol concentration is currently considered as a “surrogate marker” for cardiovascular disease risk. The rationale for including LDL as a ‘critical outcome’ is somewhat questionable and the consideration of LDL cholesterol levels in isolation of other blood lipid risk factors is a poor indicator of risk.</p>	<p>Blood lipids, including LDL cholesterol, were not given the same weight by the NUGAG Subgroup on Diet and Health as disease outcomes (including incidence and mortality) when formulating the recommendations. While relevant disease outcomes and LDL-cholesterol were considered critical outcomes (i.e. outcomes that are critical for decision-making when formulating recommendations), other blood lipid outcomes were assessed as being important (i.e. outcomes that are important, but not critical for decision-making when formulating recommendations). And even though LDL cholesterol was considered a critical outcome and the certainty in the evidence for it from the blood lipids analysis was assessed as <i>high</i>, it was not given the same weight as the disease outcomes in determining the strength of the recommendations. For example, Recommendation 3 for <i>trans</i>-fatty acids was considered to be <i>conditional</i> because, as noted in the guideline: “evidence for disease outcomes comes only from a limited number of observational studies; most of the evidence comes from RCTs with LDL cholesterol as an outcome. Although the evidence for LDL cholesterol is of high certainty, LDL cholesterol is a well-established biomarker for measuring the effects of interventions on cardiovascular disease risk, and LDL particles are considered by many to be a causal factor for atherosclerosis and coronary heart disease, it is not a physical manifestation or confirmation of disease. Therefore, a conservative approach was taken, and the recommendation was considered to be <i>conditional</i>.”</p> <p>Furthermore, although other blood lipid measures were considered less important than LDL cholesterol, effects on these blood lipids was also consistent with cardiovascular benefit.</p>

LDL cholesterol concentration is being questioned as a reliable surrogate endpoint for assessing the impact of diet on cardiovascular diseases as outlined in the 2010 report by the Institute of Medicine.	As noted, based on currently available evidence, the NUGAG Subgroup on Diet and Health considered LDL cholesterol to be a robust a well-established biomarker for measuring the effects of interventions on cardiovascular disease risk including nutrition interventions. Robust evidence from studies assessing both biomarkers for disease and disease incidence are consistent with improvement in cardiovascular health.
There is increasing recognition of the limitation of using LDL-cholesterol concentration as a marker of diet effects on cardiovascular disease risk, as atherogenicity of the LDL-particle is also determined by resistance against oxidation, size, composition and cytotoxicity. There is evidence that SAT increases the LDL-particle size, and thus potentially reduces cardiovascular disease risk.	Studies assessing LDL particle size in relation to saturated fatty acid intake and cardiovascular disease risk were acknowledged by the NUGAG Subgroup on Diet and Health. However, robust evidence suggests that reducing saturated fatty acid intake – particularly via replacement with preferred nutrients – is associated with decreased risk of disease and death. The evidence reviewed for LDL cholesterol and other blood lipids was consistent with the improvement observed in cardiovascular health in studies assessing disease outcomes, and therefore suggests that changes in LDL cholesterol resulting from changes in saturated fatty acid intake are relevant to cardiometabolic health.
The weakness of serum LDL-cholesterol concentration and ratio to reflect changes in cardiovascular disease risk following dietary interventions have been highlighted by RCTs showing that replacement of saturated fatty acids with polyunsaturated fatty acids in the diet effectively lowers serum cholesterol but does not translate into lower risk of death from coronary heart disease or all causes.	In the systematic review of RCTs that informed the formulation of recommendations on saturated fatty acids, the effect observed on cardiovascular disease was shown to be dependent on lowering of total cholesterol. From the guideline: “The effect observed on cardiovascular disease persisted in various sensitivity analyses which included only trials that aimed to reduce saturated fat, that statistically significantly reduced saturated fat intake, that achieved a reduction in total or LDL cholesterol, or excluded the largest trial (65). Subgroup and meta-regression analyses suggested that the degree of reduction in risk of cardiovascular disease was positively correlated with the degree of reduction in serum total cholesterol, with greater reductions in total cholesterol being associated with greater reduction in risk. Subgroup analysis further suggested a greater reduction in risk of cardiovascular disease with greater reductions in saturated fatty acid intake.”
Saturated fatty acids have also been shown to raise HDL and not increase the LDL:HDL ratio.	In the systematic review of RCTs assessing blood lipids that informed the formulation of

Because higher HDL is associated with reduced risk of cardiovascular diseases, saturated fatty acids may have a favourable effect on cardiovascular disease risk.	recommendations on saturated fatty acids, only replacing saturated fatty acids with carbohydrates of unknown composition was associated with an appreciable increase in HDL cholesterol, which was still much smaller than the reductions observed in LDL cholesterol. Replacing with monounsaturated fatty acids resulted in no impact on HDL and replacing with polyunsaturated fatty acids resulted in a very small increase in HDL cholesterol. Replacing saturated fatty acids with monounsaturated or polyunsaturated fatty acids resulted in substantial decreases in the LDL:HDL ratio resulting from the large reductions in LDL cholesterol. As noted, in addition to the blood lipid evidence, there is robust evidence for impact on disease outcomes.
Evidence mostly comes from blood lipids. There is some evidence to suggest that cholesterol is not the main driver of atherosclerosis, inflammation is.	Evidence suggests that LDL-cholesterol is a valid biomarker for cardiovascular disease and is likely causal. Robust evidence from studies assessing both biomarkers for disease (blood lipids) and disease incidence are consistent with improvement in cardiovascular health.
The evidence supporting the guideline is entirely human data. However, there is an overwhelming body of evidence (including non-human studies) to show that hyperlipidaemia induced by diets high in saturated fats and cholesterol promote atherosclerosis.	In developing WHO guidelines, evidence assessed directly in humans is the most preferred form of evidence as all other evidence requires confidence in extrapolating the results to humans. The relationship between saturated fatty acid intake, hyperlipidaemia, and atherosclerosis is addressed implicitly in the evidence that the NUGAG Subgroup on Diet and Health reviewed, and there is consistent and coherent evidence from human studies assessing blood lipid and cardiovascular disease outcomes.
<b>Evidence: GRADE assessments and certainty in the evidence</b>	
It is not clear why data from adults is included in the GRADE evidence profiles for children.	Noted. It was originally included because the adult data was extrapolated to children in formulating the recommendations. However it is redundant and can be confusing given the large number of profiles and separate profiles for children in some cases. The adult data has been removed from the profiles for children.
There is data for total cholesterol – why wasn't this include in the GRADE evidence profile?	There was a very large amount of data generated by the various systematic reviews and not all of it is included in the guideline as the purpose of the guideline is not to reproduce all of the data, but to summarize the key evidence used in the formulation of

	recommendations. Total cholesterol was considered an important, not critical, outcome for decision-making and therefore was not included in the GRADE evidence profiles. GRADE assessment for all outcomes can be found in the respective systematic reviews.
<b>Evidence: Methodological considerations</b>	
The guideline states that there are “non-significant reductions in risk” for several outcomes for which the 95%CI crosses the null. It needs to be clearly stated in the guideline that the only significant finding observed for saturated fatty acids was for the endpoint of combined cardiovascular events, and that the results were null (non-significant) for all other disease endpoints including coronary heart disease and stroke.	There are different schools of thought on how to consider and report effect sizes. The NUGAG Subgroup on Diet and Health did not find it appropriate to dismiss outright observed effects when one of the bounds of the 95% CI sat on the null or slightly crossed it. Rather they reported effects objectively as either statically significant or not-statistically significant. Additionally, the certainty in the evidence for an outcome with a pooled effect for which the 95% CI touches or crosses the null is downgraded for imprecision, indicating that there is less confidence in these effects compared with those effects where this doesn't happen.
Regarding the subgroup analyses conducted by replacement nutrient in the Hoper et al. systematic review, a discussion of the pros and cons of the decision to group trials based on statistical significance of the difference in SFA intake and replacement nutrient between the intervention and control groups regardless of whether or not that macronutrient was the main replacement for SFAs is warranted. This decision seems post-hoc.	This decision was not made post-hoc. It was made so that the NUGAG Subgroup on Diet and Health could be confident that a statistically significant change in the replacement nutrient occurred, and was representative of likely real-world changes in which replacements may not be 100% of a particular nutrient.
<b>Evidence: general</b>	
There is repetition of text in the <i>Summary of evidence</i> section that describes the results of RCTs assessing blood lipids for both saturated fatty acids and <i>trans</i> -fatty acids.	Noted. The text in the Summary of evidence section has been streamlined such that the results of RCTs assessing blood lipids for both saturated fatty acids and <i>trans</i> -fatty acids is now only presented once.
Several comments received requested that discussion of various, very specific details of the systematic reviews and evidence to be included in the guideline.	The guideline already contains a very detailed summary of the evidence and is not intended to provide an in-depth scientific analysis of the evidence. Such information can be found in the respective systematic reviews.
The wording of the statement “The key research questions guiding the systematic reviews undertaken...”; may give the impression that systematic reviews were specifically conducted for this evidence review by the WHO. For the WHO guidelines on free sugars (2015) it was	The systematic reviews were commissioned specifically for the development of the guidelines and were guided by PICO questions developed by the WHO Subgroup on Diet and Health. Where it is indicated that systematic reviews were “conducted” (and it isn't clear that

clearly stated that two systematic reviews were commissioned, to assess the effects of increasing or decreasing intake of free sugars on excess weight gain and dental caries.	they were commissioned) the wording has been changed to “commissioned”.
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### Implementation of the recommendations

Summary comment	Response
<p>Regarding ways of implementing the recommendations, including taxation and restriction of marketing, translating a recommendation on nutrients to a particular food, dish or product may not be precise and may even have effects other than those intended in the formation of a healthy diet.</p> <p>In line with the above, suggesting the restriction of the sale and marketing of foods with saturated fats, as well as the use of fiscal policy with the same objective, could affect the variety, availability and affordability of important sources of nutrients for populations.</p>	Macronutrient-based recommendations allow governments and national decision-making bodies to translate the recommendations into culturally and contextually specific policies and actions as needed at the country level; policies and actions which take into account locally available foods and dietary customs.

### Implications of the recommendations

Summary comment	Response
There is concern that the recommendations will adversely limit dairy and/or meat intake and thus restrict important and often underconsumed nutrients that are important for bone health and accrual, and adequate diet. This is especially true in vulnerable populations such as the elderly and young children, particularly in developing countries where problems with dietary diversity and consumption of animal-source foods may be limited. 10% may be difficult to achieve in young children	The recommendations do not preclude the consumption of any foods, including dairy foods.

### Research gaps and future initiatives

Summary comment	Response
Owing to the difficult logistics in conducting large scale, long-term RCTs (including substantial costs) to assess effects of dietary interventions on disease incidence and	Noted. The text in the <i>Research gaps and future initiatives</i> section has been modified so that it indicates more research is needed without specifying study designs, as follows: “The above [recommendations for specific areas of

mortality, it is unlikely that these will be conducted in the future.	research] should be carried out using the most rigorous, but logistically feasible study designs.”
Research on lipid endpoints should be expanded to include non-HDL cholesterol and apolipoproteins, in addition to LDL cholesterol, HDL cholesterol, ratios and triglycerides.	Noted. Relevant text in the list of <i>Research gaps and future initiatives</i> section for both saturated fatty acids and trans-fatty acids has been added as follows: “when assessing lipid endpoints, inclusion of non-HDL cholesterol and apolipoproteins (in addition to LDL cholesterol, HDL cholesterol, ratios, triglycerides etc.)”
The role of combining information from dietary self-report and fatty acid biomarkers should be assessed.	Noted. Relevant text in the list of <i>Research gaps and future initiatives</i> section for both saturated fatty acids and trans-fatty acids has been added as follows: “explore ways of combining information from self-reported dietary intakes and fatty acid biomarkers for more robust dietary exposure assessments”.
Well-conducted observational studies should be included in research needs.	Noted. The text in the <i>Research gaps and future initiatives</i> section has been modified so that it indicates more research is needed for both saturated fatty acids and trans-fatty acids without specifying study designs, as follows: “The above [recommendations for specific areas of research] should be carried out using the most rigorous, but logistically feasible study designs.”
Greater clarity and consistency (and standardisation) is needed on clinical endpoints that are studied and reported, as currently some research focuses on total cardiovascular diseases (with varying definitions and conditions included), others only on coronary heart disease or coronary artery disease or stroke individually, and some more specifically report myocardial infarction or heart failure. This makes comparability of the results between studies very challenging.	Noted. Relevant text in the list of <i>Research gaps and future initiatives</i> section for both saturated fatty acids and trans-fatty acids has been added as follows: “efforts should be made to harmonize reporting of cardiovascular disease endpoints in order to improve ability to compare across studies and synthesize data”
Further research is needed on the health effects of different types of polyunsaturated fatty acids (total n-3, ALA, EPA+DHA, n-6/LA), monounsaturated fatty acids (plant/animal), carbohydrates (total, whole-grain, refined, high/low GI, fibre-rich or poor) and protein (plant/animal) when used as replacements for saturated fatty acids.	Noted. Long chain polyunsaturated fatty acids are generally not consumed in large enough quantities to serve as replacement nutrients alone. The following text has been added to the list in the <i>Research gaps and future initiatives</i> section: “further assess the health effects of different types and or different sources (i.e. plant, animal) of polyunsaturated fatty acids, monounsaturated fatty acids, carbohydrates, and protein when used as replacements for saturated fatty acids”.



Further research is needed on ways to differentiate between intake of industrially produced and ruminant <i>trans</i> -fatty acids.	Noted. The following text has been added to the list in the Research gaps and future initiatives section: “research on methods to more accurately differentiate between intake of industrially produced and ruminant <i>trans</i> -fatty acids” as well as “further research to better understand potential differential effects on health of industrially produced and ruminant <i>trans</i> -fatty acids”.
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## General comments

Summary comment	Response
It is indicated in the guideline that providing recommendations on the intake of saturated fatty acids and <i>trans</i> -fatty acids are to reduce the risk of NCDs in adults and children, particularly cardiovascular diseases which are a leading cause of NCD mortality. Because for saturated fatty acids the only NCD assessed are cardiovascular diseases (in adults), it should be clearly stated in the guideline that the focus of the recommendations for saturated fatty acids are on cardiovascular diseases only.	The priority outcomes are clearly stated in the guideline to include cardiovascular diseases and type 2 diabetes for both saturated fatty acids and <i>trans</i> -fatty acids. Therefore references to NCDs are appropriately used.
In the guideline it is indicated that the outcomes of interest include “incidence, mortality and morbidity” of NCDs, however it is unclear how “morbidity” was assessed in the evidence review.	“Morbidity” was intended to mean incidence of disease so use of the word morbidity was redundant. References to morbidity in the context of health outcomes have been removed.
Whether sub-types of stroke were included in the systematic review of RCTs should be clarified.	Because data on stroke were limited, it was not possible to tease out differential effects on ischaemic or haemorrhagic strokes, or whether a stroke was fatal. In the updated systematic review of observational studies, only ischaemic stroke was reported on.
Evidence suggests that refined or high glycaemic carbohydrates are much worse for health than saturated fatty acids. The evidence in the WHO systematic review on blood lipids supports this as replacement of saturated fatty acids with carbohydrates reduces HDL and increases triglycerides.	The recommendations in this guideline don't suggest that saturated fatty acids or <i>trans</i> -fatty acids alone are the only macronutrient of concern in the context of health. Accordingly, recommendations in this guideline should be considered in the context of other WHO guidelines on healthy diets, including those on total fat, polyunsaturated fatty acids, carbohydrates, sugars, sodium and potassium.
The most appropriate recommendation for both saturated fatty acids and <i>trans</i> -fatty acids -- considering neither are essential in the human	Noted.

diet and appear harmful to human health--should be to consume as little as possible.	
Most studies and other publications use the terms “saturated fat” and “animal fat” interchangeably but saturated fat is not the same as animal fat. Furthermore, when the term “saturated fat” is used, these papers usually mean long-chain saturated fat from animal sources. One should differentiate between saturated fatty acids from animal and plant sources.	Saturated fatty acid exposures as assessed in the prospective cohort studies included in the systematic review that informed the development of this guideline, included all sources of saturated fatty acids – plant-based and animal based.
[A number of references to additional studies covering various topics were suggested to be included].	Most studies that were suggested for addition to the guideline were either beyond the scope of the systematic review or were excluded based on inclusion/exclusion criteria.

### **Annex. Original comments as received during the call for comments**

Comments are listed in the order in which they were received.

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# Public consultation on the draft WHO *Guidelines: Saturated fatty acid intake and trans-fatty acid intake for adults and children*

## Survey response 1

### General information

Family/last name
Severi
Given/first name
Cecilia
Organization/affiliation
Medicina Preventiva, Fcaultad de Medicina, Universidadde la República
Sector
Academic/research
Sector [Other]
Country
Uruguay

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
I aggre all the guidelines. I propose to translate in practical portions, or type of food and only in percentage of calories.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
I aggre all the guidelines. I propose to translate in practcla porcions, or type of food and only in percentage of calories.
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Excelent
Summary of evidence (for saturated fatty acids only)
excelent
Recommendations (for saturated fatty acids only)
Translate in practical potcions, or type of food and only in percentage of calories.
Remarks (for saturated fatty acids only)
Translate in practical potcions, or type of food and only in percentage of calories.
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
Translate in practical portions, or type of food and only in percentage of calories.

## Survey response 3

### General information

Family/last name	LOISEAU
Given/first name	JOSEPH JUTILE
Organization/affiliation	JSI
Sector	Non-governmental agency
Sector [Other]	
Country	Ghana

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)	
Scope and purpose (same for saturated fatty acids and trans-fatty acids)	
Background (same for saturated fatty acids and trans-fatty acids)	
Guideline development process (same for saturated fatty acids and trans-fatty acids)	
Summary of evidence (for saturated fatty acids only)	
Recommendations (for saturated fatty acids only)	
Remarks (for saturated fatty acids only)	
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)	
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)	
Annexes 1, 6, 7 (for saturated fatty acids only)	

Additional comments
I would like to recommend to also focus on sedentary lifestyle as one factors of NCD alongside with saturated fatty and trans-fatty acids

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 8

### General information

Family/last name
Gulati
Given/first name
Seema
Organization/affiliation
National Diabetes, Obesity and Cholesterol Foundation
Sector
Non-governmental agency
Sector [Other]
Country
India

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
saturated fatty acid intake should be less than 10% of total calorie intake. Total fat intake from visible and non-visible sources should be less than 30% of total calorie intake. Tran fatty acid intake should be less than 1% of total calories.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Adequate dietary guidelines for Indians.
Background (same for saturated fatty acids and trans-fatty acids)
In the light of so many fad diets becoming popular, it is really misleading for general population to choose the adequate food articles. Indians have high propensity for obesity, diabetes, cardiovascular diseases and other lifestyle disorders. It is extremely important to have clear cut dietary guidelines for good health.
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
< 10 % for general population. People with dyslipidemia
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)



Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
< 1% since we cant completely avoid. distinction can be made between industrial and ruminant trans fat
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
saturated fats: < 19% of total calories. Trans fats

## Survey response 10

### General information

Family/last name
Atkins
Given/first name
Amanda
Organization/affiliation
None
Sector
Other
Sector [Other]
Retired
Country
Ireland

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>Current guidelines which discourage the consumption of meat and other natural protein and fat products because of the supposed dangers of saturated fats and trans- fatty acids included therein have resulted in a public health disaster. I , and many other private individuals have suffered failing health and diabetes from following food guidelines which encourage a high carbohydrate intake and preference for man made oils over naturally occurring oils which appear in natural foodstuffs including both saturated fats and naturally occurring trans-fatty acids..</p> <p>The focus on grain and other carbohydrates as the source of the majority of calories coupled with the encouragement in the growth of " low fat" products - has dramatically increased the consumption of sugars and supposedly healthy "whole grain" products. The demonisation of natural meat proteins and fats has led to the explosion of junk foods, high carbohydrate and inflammatory seed oil diets. These diets create metabolic eating disorders through changes in the way ones body understands and craves foods and this in turn then leads to the rapid rise in obesity seen everywhere when processed foods are introduced in preference to " real" foodstuffs - coming from animals.</p> <p>I, and thousands of other private individuals have now recovered there metabolic health, and put their diabetes into remission by deliberately ignoring the food guidelines and in particular those that demonise natural products - red meat, cheese, fatty fish, eggs, nuts through the supposed " dangers" of saturated fat.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
<p><b>Saturated Fats</b></p> <p>In my view ( as supported by my own health profile monitoring over 2 years) saturated fats should form a significant part of the human diet through consumption of meat, fish, diary, eggs. To the extent that Trans- fatty acids happen to form a small part of those products then it is not dangerous and takes a different form from that which is created by industrial processes.</p> <p>Seed oils - History will show that the increase in obesity, metabolic diseases, diabetes, CVD, alzheimers, PCOS, fatty liver disease and many cancers etc etc, has been a direct result of the vilification of saturated fats and the consequent explosion of seed oils which now infect all parts of the man-made product food supply. The growth in these diseases can be directly correlated to the growth in consumption of seed oils - in the western world through the development of a never ending supply of refined food products, all created with seed oils, and in Asia through the explosion of " dried noodle " products where the drying process takes ramen noodles and fries them in seed oils to remove the water content.</p> <p>The vilification of saturated fats also led to the explosion in "low fat" products - these products are all high in sugar to replace the taste. The result has been a perfect storm of carbohydrate and sugar consumption well past mans natural frontier and a completely skewed N3 to N6 ratio which is causing inflammation globally and directly leading to the current health crisis. Obesity is not a cause of this crisis, instead it is one of the many symptoms produced by a diet which is too high in refined carbohydrates and seed oils and too low in natural products including saturated fats.</p>

## Background (same for saturated fatty acids and trans-fatty acids)

I am 60 years old. As a child I was a classic product of a working class family, my mother made ends meet by enrolling the family as Heinz food testers my diet was a classic 70s diet of trying out all the new foods entering the food supply ( e.g. pot noodles mid 70s) . I had developed weight problems as a teenager and embarked on my first diet aged 15. I then spent 40+ years on a roller coaster of increasing weight, and primarily carbohydrate driven low fat diets. I stopped eating sugar in 1973, I drank only " diet" drinks since that time, I also gave up butter in 1973 instead opting for "heart healthy" spreads. I spent my 20s eating calorie restricted " healthy - Boil-in a bag" products designed to "help" me lose weight. I spent 40+ years faithfully trying to follow food guidelines whilst finding it increasingly difficult. In 2016 I was diagnosed with diabetes. At the point of diagnosis I had already lost 6kg (again) through yet another attempt at the recommended low fat diet. My hba1C was 10.3% and fasting blood sugars on that day 18.4. When told yet again to lose more weight through a low fat diet, I realised that this was just not the answer and began my own research.

I found this paper <https://nutritionandmetabolism.biomedcentral.com/articles/10.1186/1743-7075-6-21> and in particular read this paragraph "One female patient had an increased physical activity level during the study period in spite of our instructions. However, her increase in physical activity was no more than one hour of walking per day, four days a week. She had implemented an 11%-carbohydrate diet without any antidiabetic drug, and her HbA1c level decreased from 14.4% at baseline to 6.1% after 3 months and had been maintained at 5.5% after 6 months."

That same day I decided "if she can - so can I" and immediately adopted a low carbohydrate diet. By definition such a diet has the natural consequence of removing seed oils as these only appear in refined products which cannot meet the required 11% test.

Within 4 days my fasting blood sugar had reduced to 9.5 and my weight had started to drop. I had a comprehensive set of blood tests done at that time.

I converted to a low carb, high fat diet though still trying to avoid red meats and saturated fats due to their bad health reputation. I also commenced reading hundreds of scientific papers ( I was finance director of a publicly listed non -life reinsurance group before my retirement in 1985). At my first follow up at 6 weeks , my Hba1C had improved from 10.3% to 8.0% and I knew I was on the right track. My HDL cholesterol was still too low and my doctor, who knew nothing at all about nutrition, simply told me to increase my exercise levels in a bid to improve it. By that stage my reading had told me that actually consuming red meat and saturated fats would improve my HDL and LDL/ HDL ratios. So I embarked on a strategy of eating increasing quantities of saturated fat, cream, red meat, eggs, ensuring I ate no industrial seed oils at all, keeping my N6: N3 ratio to as close to 1:1 as possible ( I found 2-3:1 reasonably easy without refined foods). Sure enough over the next 18 months my health markers improved. As of today I have an Hba1C of 5.3%, ALL of the standard blood tests are now normal for ALL categories of 52 tests. Some of these tests have not been normal since my very first medical in my early 20s. As importantly numerous minor ailments had disappeared, I can now happily jog 5km, walk for 4 hrs, swim a mile, walk up 600 steps with no particular difficulty. None of these have been possible - ever - even as a child - my early memories are of falling behind on school trips with the " brownies" ( girl guides association for 7-11 year olds) . I AM STILL OVERWEIGHT . My BMI is still 32. I have taken no medication at all for diabetes, I have reduced my blood pressure medication and discontinued allopurinol ( gout prevention) . For the very first time in my life my weight is no longer a " roller coaster" . A consequence of the LCHF way of life is that hunger disappears. I know I damaged my metabolism through years of low fat dieting, so I am aware that it will be a long road to actually get down to a lower BMI however I also know that I have already achieved metabolic health and that translates through into all parts of my life. I now know that "snacking" kills - it drives blood sugars up, it creates hunger. I now know that both "low fat" foods and "seed oils" are toxic. Eating a low carb, healthy natural fat diet has improved every aspect of my health.

It has become clear to me that currently the state of our nutritional understanding is a complete mess. This has distorted all epidemiological research and almost all pharmaceutical research. The " standard American diet" with its emphasis on " heart healthy " whole grains, skimmed milks and polyunsaturated fats is killing people. Those who eat it also find that their desire for junk food shoots up because of the carbohydrate volume and the fact that N6 oils promote hunger. When ANY study is compared to " control" EVERY intervention will produce a benefit. This is because " control" is dire, not because that particular intervention is itself justified in terms of its own cost benefit. The result of this has been to hand extraordinary power to the pharmaceutical industry whilst at the same time promoting food policies which have given food companies free reign to produce cheap, addictive processed foods that are killing people.

Around the world, there are pockets of individuals that have understood this. Many of those individuals, like me , have formed their own conclusions and now implement an LCHF diet suited to their own tastes - it is possible to do this as a vegan, vegetarian, omnivore or carnivore - the key in all cases - avoid refined foods, avoid starchy vegetables, avoid seed oils, embrace saturated fats - from either vegetable or animal sources. ALL of them experience health benefits.

dietdoctor.com is showing individuals worldwide, how to adopt LCHF diet in order to lose weight and improve health - it has 300,000 subscribers

diabetes.co.uk is showing individuals with diabetes how to adopt LCHF in order to reduce/ remove diabetes medications and prevent complications, it has more than 270,000 members who have been through the "Low carb. program with more than 40% of participants eliminating diabetes medication at one year . There are more than 1,000 personal testimonials on the site ( including my own) documenting health improvements.

virtahealth is making the LCHF formula into a successful business model, showing patients how to adopt an LCHF( no seed oil)

diet with hands on support from a doctor via the internet. The studies it has produced so far show results exactly as I would expect them to do across all health markers tested - all exactly in line with my own N=1 and that of other N=1 diabetics. <https://blog.virtahealth.com/category/science-research/> I have no financial interest whatsoever in Virta Health. Nor do I think it is particularly necessary to pay for the service. Instead what needs to happen is a clear statement from WHO and each dietary guideline provider worldwide making some simple points :

- 1) the cause of metabolic disease is - refined foods, sugar, seed oils it can primarily be identified as a state of hyperinsulinaemia
- 2) metabolic disease is the underlying cause of most chronic illnesses from diabetes,, to CVD, PCOS , alzheimers and cancer
- 2) obesity is not the cause of ill health it is one of many symptoms brought on by hyperinsulinaemia
- 3) " refined foods" must be reformulated to remove industrial seed oils and replace with naturally occurring animal and vegetable fats.
- 4) we must increase dramatically the farm lands and pastured animal products reducing the grains in animal foodstuffs - this in turn will assist in stopping the erosion of world soils.
- 5) the continued emphasis on LDL lowering as a key measure of heart health needs to be discontinued- it patently does not work instead the focus should be switched to evaluating human insulin levels.
- 6) the standard " testing" protocols should include fasting insulin as a matter of course where insulin over 5 iul/ml ( current normal range 2-25) indicates the beginnings of hyperinsulinaemia treatable by reducing refined foods and seed oils in diets. Oral Glucose and insulin tolerance tests ( as developed By Dr Jospheh Kraft) should become the norm amongst those developing obesity.

As a result of the erroneous food guidelines of the last 30 years, WHO and other organisations have an enormous task ahead to put right the damage that has been done.

Practically all research conducted today is conducted with some kind of commercial bias. Practically all health advisory organisations and charities are funded by the very organisations that stand to gain the most from our ill health - processed food organisations feeding off our addiction, pharmaceutical companies profiting from chronic conditions and medical professionals performing thousands of needless interventions if only we had got the diet right in the first place. It is no accident that the medical professions receive practically no nutritional training and the nutrition professions are primarily funded by the food industry and thus promulgate their refined food agenda. ALL of these industries BENEFIT from the " limit saturated fat" guidelines - is it any wonder that you yourselves have been guided down this path?

WHO MUST rise above these commercial interests - it is time to listen to the people who have carried out their own N=1 experiments and successfully regained their health as a result. There are 1,000s of us. Our data is dismissed - not because it is incorrect but because it is commercially inconvenient. WHO needs to LISTEN to us. We have no commercial interests, our only desire is to pass on the message to others - that they can regain their health. We should NOT have to tell them that the key to doing so is to IGNORE current guidelines coming from any official source.

#### Guideline development process (same for saturated fatty acids and trans-fatty acids)

WHO needs to make a proper evaluation of the data available. It needs to make a call for all N=1 individuals who have blood tests pre and post switching their diet from a standard diet to one which has removed refined carbohydrates and industrial seed oils . These individuals should be asked to submit pre and post blood tests and to identify the prime elements of their diet - ie vegan, vegetarian, pescatarian, omnivore, carnivore. They should be asked to outline their dietary and health strategy.

This data needs to be evaluated against the inevitable avalanche of vested commercial interest submissions.

If WHO does this it will find that the key to good health has little to do with the proportion of meat/ non meat and everything to do with the proportion of refined processed foods and seed oils. As such the arguments concerning " plant based" versus " meat based" diets are likely to be of little consequence.

#### Summary of evidence (for saturated fatty acids only)

I am happy to submit my own N=1 blood profiles and dietary records showing the progress made.

#### Recommendations (for saturated fatty acids only)

Remove the current caps on saturated fats, instead replace with a cap on refined foods or any sort and industrial seed oils

#### Remarks (for saturated fatty acids only)

Stop the continued confusion that arises every time junk food is mentioned - it is nearly always stated to be " full of saturated fats ". This is simply not true, nearly all processed foods are made with seed oils not natural animal and vegetable products with a higher proportion of saturated fats.

#### Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

WHO needs to sponsor a proper study of the principles of a low carb, high fat diet excluding seed oils and refined carbohydrates in all arms and comparing long term outcomes between vegan, vegetarian , carnivore and omnivore options including the relevant saturated fats so that mankind can properly determine whether any of these options have a material impact on all cause mortality compared with each other.

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
There is plenty of research showing the benefits of an LCHF diet including saturated fats. It is constantly drowned in the cacophony of commercial interests. A fully independent trial needs to be carried out excluding ANY commercial principles based on the principles espoused by the outcome of the N=1 data
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments
Mankind is at a cross roads. It can admit it made some mistakes in the promulgation of food guidelines and the flight to carbohydrates and polyunsaturated fats, or it can continue to put its head in the sand and watch more and more of the population lose limbs and health.
Is WHO brave enough to put the wrong done to millions and continuing every day ?

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Trans fatty acids
WHO needs to dispel the notion that because animal products contain a little trans fatty acids, it is as unhealthy as trans fatty acids created by industrial seed oils. The two are completely different things.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

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Please provide any final thoughts or comments below.

i appreciate that I am only a single voice in a vast sea of commercial interests. However as a complete novice, I have come to understand just how distorted science has become and to be able to read between the lines of studies - as much for what is not being said as what is.

I now know exactly how my human body reacts to certain foods. I have successfully helped many lose weight myself and reduce medications . The results coming out of the various trials now focusing on LCHF diets are not news to me or the 1,000s of others N=1 who have already achieved the same results.

Saturated fats are not " unhealthy" they are a core fuel source for the human body. They plus cholesterol form a huge part in the bodies natural defence mechanisms. By disrupting our natural mechanisms, those seeking to improve global health have inadvertently put mankind through a vast uncontrolled dietary experiment. The result of that is everywhere. WHO can help put this right.

## Survey response 11

### General information

Family/last name	Sharma
Given/first name	Sushum
Organization/affiliation	HTK Medical Center, Dubai, U.A.E.
Sector	Private sector
Sector [Other]	
Country	United Arab Emirates

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)	1 I agree that Saturated fatty acids should be restricted to less than 10 %. 2 Saturated fats from Dairy sources ( eg. Butter, Desi Ghee-India) to be preferred over Tallow and Lard.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)	same
Background (same for saturated fatty acids and trans-fatty acids)	Same
Guideline development process (same for saturated fatty acids and trans-fatty acids)	Same
Summary of evidence (for saturated fatty acids only)	Same
Recommendations (for saturated fatty acids only)	1 Saturated fatty acids should be restricted to less than 10 %. 2 Saturated fats from Dairy sources ( eg. Butter, Desi Ghee-India) to be preferred over Tallow and lard.
Remarks (for saturated fatty acids only)	1 While reducing SFA, we need to have more CLARITY on the Replacement Ratios by Proteins, Complex Carbohydrates, PUFA, MUFA. 2 Replacement with Proteins: I feel it should be more ethnic/region specific e.g. For the same Body Mass index , Indians (as compared to a white people) have less muscles and more fat. So it makes sense to replace these calories by adding more proteins in diet. 3 While replacing with Carbohydrates, recommendations should clarify, it should be Complex Carbohydrates with Low Glycemic Index. 4 For replacement with Unsaturated fatty acids, THE ENTIRE DOCUMENT DOES NOT RECOMMENDED RATIOS of MUFA/PUFA, at most places it is only PUFA or MUFA. Message should be clear. 5 I feel keeping a ratio between MUFA to PUFA 1.5-2: 1 ( 1.5 to 2 parts MUFA and 1 part PUFA) is reasonable. 6 Ratio of Unsaturated(MUFA+PUFA) to Saturated Fatty acid also needs to be clarified. It should preferably be 2 : 1

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Same
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
No mention of Benefits of use of COLD PRESS OILS vs NON COLD PRESSED OILS( both available in market today).
Annexes 1, 6, 7 (for saturated fatty acids only)
None
Additional comments
None

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Trans-fatty acid from Industrialized Sources should be Completely Stopped. This will leave TFA from natural sources (ruminants) which is mostly less than one percent.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Same
Background (same for saturated fatty acids and trans-fatty acids)
Same
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Same
Summary of evidence (for trans-fatty acids only)
Same
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Trans-fatty acid from Industrialized Sources should be Completely Stopped. This will leave TFA from natural sources (ruminants) which is mostly less than one percent.
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
1 Complete Metabolism of TFA in human body needs further workup. 2 Human Lipase either does not or does incompletely, metabolise TFA. What happens to the remaining TFA in Long run. 3 For the isocaloric intake, TFAs cause more Obesity, DM, Insulin resistance in monkeys. What about humans ?
Annexes 1, 6, 7 (for trans-fatty acids only)
same
Additional comments
none

## Final comments



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Please provide any final thoughts or comments below.

Dear Sir,

I am honored to give my remarks on this more than praiseworthy document. I am practicing for over 35 years, and one of the most common question my patients ask, Doctor which Oils should I use and in what ratio ? To be very honest, in the absence of clarity on this issue, most of First Line Clinicians and Nutrition expert give a very vague remarks which is usually driven by unauthorized Websites.

I am sure once this document is out we as clinician will have greater clarity on this complex yet very important subject.

With best wishes.

Dr Sushum Sharma

MD, FACP, FRCP(Edin)

## Survey response 15

### General information

Family/last name
Barry
Given/first name
Karyn
Organization/affiliation
none
Sector
Private sector
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
These guidelines should also include sodium content.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
Sodium is a huge contributor to high blood pressure and other health issues.

## Survey response 16

### General information

Family/last name
Borade
Given/first name
Pratima
Organization/affiliation
Grant government medical college Mumbai
Sector
Academic/research
Sector [Other]
Country
India

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
I think not only saturated fatty acids and trans fatty acids responsible for cardiovascular disease but excess intake of gluten is also responsible for the damage to the intima of coronary arteries... So further studies and research is needed in this field
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments
I think not only saturated fatty acids and trans fatty acids responsible for cardiovascular disease but excess intake of gluten is also responsible for the damage to the intima of coronary arteries... So further studies and research is needed in this field

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
Excess intake of gluten in the wheat leads to increased inflammation in the body and can lead to cardiovascular disease... It need further scientific research.

## Survey response 17

### General information

Family/last name
Kehar
Given/first name
Sugandha
Organization/affiliation
National Diabetes & Cholesterol Foundation
Sector
Academic/research
Sector [Other]
Country
India

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Recommendation for Saturated Fatty Acids should be RDA 50 years) - 9.2 (in Males), 9.7 (in Females)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Recommendation for Tran fatty acids should be RDA 50 years) - 0.3 ( for Males), 0.4 (for Females)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 21

### General information

Family/last name
schuftan
Given/first name
claudio
Organization/affiliation
PHM
Sector
Non-governmental agency
Sector [Other]
Country
Viet Nam

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)



Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
agree with the guidelines

## Survey response 27

### General information

Family/last name
Haque
Given/first name
Intazamul
Organization/affiliation
Diabetes Foundation India
Sector
Non-governmental agency
Sector [Other]
Country
India

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
recommendation for saturated fatty acid should be RDA
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Recommendations for trans-fatty acid should be RDA
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 30

### General information

Family/last name
Hadidi
Given/first name
Daniel
Organization/affiliation
University of Colorado Anschutz Medical Campus
Sector
Academic/research
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Evidence for the reduction of saturated fat intake is currently insufficient; rather, stronger evidence exists for the reduction of refined carbohydrates. Evidence cited in support of reducing saturated fat intake below 10% of total energy intake is both outdated and of lower quality than the most current evidence, which suggests that saturated fat intake is not associated with cardiovascular disease, coronary heart disease, or stroke risk, the three most commonly cited diseases for which saturated fat intake has been cited as being a risk factor. While some trials have demonstrated reduced cardiovascular risk factors through replacement of saturated fat with polyunsaturated fat, other trials demonstrate both no change and in some cases increased risk.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
This comment is intended to warn against guidelines based on outdated or insufficient evidence regarding saturated fatty acid intake.
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
The strongest evidence should be relied upon when formulating guidelines, furthermore the pendulum-like history of nutrition guidelines in the U.S. is a cautionary tale against providing guidelines when inappropriate, as doing so may result in both negative public health consequences as well as reduced trust of the public in governmental guidelines.
Summary of evidence (for saturated fatty acids only)
<ul style="list-style-type: none"><li>- The largest meta-analysis, including nearly 350,000 individuals across 21 prospective cohort studies, demonstrated no association between saturated fat intake and coronary heart disease, cardiovascular disease, or stroke (PMID: 20071648).</li><li>- Ketogenic diets extremely high in saturated fat favorably affect fasting blood lipids, thus likely reducing risk for cardiovascular disease (PMID 12097663).</li><li>- Some studies indicate an inverse association between saturated fat intake and stroke mortality (PMID 20685950).</li><li>- Reduction of high glycemic carbohydrate consumption is more effective in prevention of cardiovascular disease than saturated fat intake reduction (PMID 21978979).</li><li>- Saturated fat replacement with carbohydrates has adverse effects on blood lipids (PMID 28864143).</li><li>- Some studies suggest an increase in atrial fibrillation risk when replacing saturated fat with omega 3 polyunsaturated fat (PMID 28803653).</li><li>- Certain saturated fats are more satiating than other foods and result in reduced food intake, which may aid in reducing cardiovascular disease risk via weight loss (PMID 28689741).</li></ul>

Recommendations (for saturated fatty acids only)
Saturated fatty acids are healthy sources of fat-soluble nutrients in addition to uniquely inducing satiety and reducing food intake, and should not be reduced. Instead, further exploration of the role of refined carbohydrates in cardiovascular disease risk should occur.
Remarks (for saturated fatty acids only)
Epidemiological studies demonstrating adverse effects of saturated fat may not take into account frequent consumption of saturated fat in combination with refined carbohydrate, which appears to be a more likely candidate for increasing risk of cardiovascular and other chronic disease. Caution is warranted in evaluating the results of such studies, as well as others which are funded by industries favoring replacement of saturated fat intake with increased refined carbohydrate.
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Increased exploration of the relationship between high saturated fat in conjunction with high refined carbohydrate consumption and chronic disease risk is warranted.
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments
This comment includes only a very small portion of the available evidence that precludes a recommendation to reduce saturated fat intake.

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

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## Final comments

Please provide any final thoughts or comments below.

## Survey response 31

### General information

Family/last name
Price
Given/first name
Catherine
Organization/affiliation
science journalist
Sector
Private sector
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
The evidence linking saturated fat to cardiovascular events (and health issues in general) is far weaker than the public knows and than most public health experts are aware of/ are willing to admit. The best summary that I've found is The Big Fat Surprise, a book by award-winning investigative journalist Nina Teicholtz. As a fellow investigative journalist myself, I believe it should be required reading for anyone on the committee; if you would like a summary of its conclusions (with supporting materials) you can visit <a href="https://www.nutritioncoalition.us/">https://www.nutritioncoalition.us/</a> . The bottom line is that saturated fat has never definitively been proven to be linked to adverse cardiovascular events. However, carbohydrates, which raise both insulin and triglyceride levels, have been linked to cardiovascular events. Point being, saturated fat is not the problem. And in fact, Teicholz convincingly argues that the polyunsaturated fatty acids of the sorts found in corn, safflower and canola oil, may actually be worse for health than even transfats. Remember: those oils were not part of our food supply until the turn of the 20th century. Today, they account for 8 percent of our total calories.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Again, I recommend that anyone on the committee be required to read <i>The Big Fat Surprise</i> or to visit <a href="https://www.nutritioncoalition.us/">https://www.nutritioncoalition.us/</a> . Transfat does indeed seem to pose serious health risks; however, polyunsaturated fatty acids may be as harmful, if not worse, and should not be recommended as alternatives. Instead, monounsaturated or saturated fats appear to be the best for heart health. The bottom line is that all of these recommendations should be based on solid science -- which in turn means EXCLUDING observational/epidemiological studies and relying on our knowledge of biochemistry, and actual randomized controlled trials.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
To reiterate my comment in the transfat field, we need to advocate for dietary guidelines based on actual science—meaning taking out strong/bullying personalities (Ancel Keyes, for example) and excluding—or at very least dramatically reducing the weight of—epidemiological studies. Nutrition has come a long way in the past 50 years thanks to advances in technology and endocrinology. Guidelines should reflect that.



## Survey response 33

### General information

Family/last name
Hellner
Given/first name
Megan
Organization/affiliation
Potentia Family Therapy
Sector
Private sector
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Id suggest naming the particular foods (i.e. meat and dairy fats) versus naming only the specific nutrients, as this is more recognizable for consumers.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Suggest naming foods, and not just individual nutrients to avoid (foods that tend to be higher in trans fatty acids) for ease of understanding on behalf of consumers.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 34

### General information

Family/last name
Adam
Given/first name
Cole
Organization/affiliation
None
Sector
Private sector
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Given that saturated fat is not essential in the human diet, and appears to increase LDL cholesterol and cardiovascular disease risk, the most logical recommendation would be for people to consume as little as possible.
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

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Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)

Summary of evidence (for trans-fatty acids only)

Recommendations (for trans-fatty acids only)
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Given that trans fat is not essential in the human diet, and appears to increase LDL cholesterol and cardiovascular disease risk, the most logical recommendation would be for people to consume as little as possible.
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Remarks (for trans-fatty acids only)
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The naturally occurring trans fat found in meat and dairy products should also be considered in this recommendation, as animal-based trans fat have been shown to be just as detrimental to human health as the added forms.
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Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for trans-fatty acids only)

Additional comments

## Final comments

Please provide any final thoughts or comments below.
--

The most appropriate recommendation for both saturated and trans fat--considering neither are essential in the human diet and appear harmful to human health--should be to consume as little as possible.
---

## Survey response 36

### General information

Family/last name
Aurich
Given/first name
Sebastian
Organization/affiliation
BASF SE
Sector
Private sector
Sector [Other]
Country
Germany

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
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We refer to p.11, footnote to inclusion of CLA to the definition of TFA. We do not agree to this.

Trans fatty acids (TFA) as the subject of discussion about adverse health effects are those fatty acids with trans double bonds that originate from industrial partial oil hydrogenation processes. The current global regulatory framework overall clearly excludes conjugated fatty acids from the definition of trans fatty acids.

We realize that in the draft WHO guideline, the argumentation for inclusion of CLA to the TFA Definition does not provide convincing scientific evidence that would allow for such inclusion.

While the evidence for adverse health effects with regard to CVD risk factors is strong for industrially produced TFA originating from partial oil hydrogenation, it is unlikely that CLA has such negative effect.

Overall, we conclude that CLA has to be excluded from definition of TFA in this guideline. Neither is CLA a TFA according to relevant international definitions, nor does the available data justify conclusion on adverse effects on CVD system.

Please see our comments file for full details.

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)

Summary of evidence (for trans-fatty acids only)

Recommendations (for trans-fatty acids only)

Remarks (for trans-fatty acids only)
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We refer to p.40, footnote to inclusion of CLA to the Definition of TFA. We do not agree to this.

Trans fatty acids (TFA) as the subject of discussion about adverse health effects are those fatty acids with trans double bonds that originate from industrial partial oil hydrogenation processes. The current global regulatory framework overall clearly excludes conjugated fatty acids from the definition of trans fatty acids.

We realize that in the draft WHO guideline, the argumentation for inclusion of CLA to the TFA Definition does not provide convincing scientific evidence that would allow for such inclusion.

While the evidence for adverse health effects with regard to CVD risk factors is strong for industrially produced TFA originating from partial oil hydrogenation, it is unlikely that CLA has such negative effect.

Overall, we conclude that CLA has to be excluded from definition of TFA in this guideline. Neither is CLA a TFA according to relevant international definitions, nor does the available data justify conclusion on adverse effects on CVD system.

Please see our comments file for full details.

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for trans-fatty acids only)

Additional comments
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Please see our comments file for the details, why we do not agree to the Definition of TFA including CLA.

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## Final comments

Please provide any final thoughts or comments below.
Please see our comments file for the details, why we do not agree to the Definition of TFA including CLA.



We create chemistry

BASF SE, 67056 Ludwigshafen, Deutschland

World Health Organization (WHO)  
Department of Nutrition for Health and Development  
(NHD)

5/29/2018  
Human Nutrition, Global Regulatory &  
External Affairs, G-ENH/MR – F31/230  
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**WHO Call for public comments on the draft WHO Guidelines:  
“Saturated fatty acid and trans-fatty intake for adults and children”  
Online public consultation open: 04 May to 1 June 2018**

**BASF’s comments on the draft WHO guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”**

Dear Sir or Madam,

With reference to the subject named above, please find the following information for your attention.

Primary note:

WHO has published draft WHO guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”, and has called for public consultation via its website for a period 04 May to 1 June 2018.

BASF SE is a manufacturer and marketer of high-quality specified conjugated linoleic acid (CLA) products, obtained from vegetable oils by using a distinct manufacturing process.

### **BASF would like to make the following comments**

We support the discussion and activity of WHO to the subject of trans fatty acids (TFA).

We also support the recommendations of WHO to the reduction of TFA intake in adults and children as well as the prevention of increasing the TFA intake in adults and children.

However, we oppose to the inclusion of conjugated linoleic acid (CLA) to the definition of TFA for the following reasons:

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**Chairman of the Supervisory Board:**  
Juergen Hambrecht

**Board of Executive Directors:**  
Martin Brudermueller, Chairman;  
Hans-Ulrich Engel, Vice Chairman;  
Saori Dubourg, Sanjeev Gandhi, Michael Heinz,  
Markus Kamieth, Wayne T. Smith



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**WHO Call for public comments on the draft WHO Guidelines:  
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Online public consultation open: 04 May to 1 June 2018**

**BASF’s comments on the draft WHO guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”**

1. Current global rules and legislations to the TFA definition shall be taken into account.

Following the current CODEX definition for TFA conjugated forms are clearly excluded: *“For the purpose of the Codex Guidelines on Nutrition Labelling and other related Codex Standards and Guidelines, trans fatty acids are defined as all the geometrical isomers of monounsaturated and polyunsaturated fatty acids having non-conjugated interrupted by at least one methylene group (-CH<sub>2</sub>-CH<sub>2</sub>-) carbon-carbon double bonds in the trans configuration.”* (Codex 2017)

In the European Union, the TFA definition from Regulation (EU) No. 1169/2011 on Food Information to Consumers, Annex I, *Specific Definitions* expressively only includes those forms of polyunsaturated fatty acids where double bonds are interrupted by at least one methylene group: *“4. ‘trans fat’ means fatty acids with at least one non-conjugated (namely interrupted by at least one methylene group) carbon-carbon double bond in the trans configuration”.* (EU 2011)

While several countries do not explicitly rule conjugated or non-conjugated forms in their legislation (i.e. Australia, Argentina, India), most countries in Europe with provisions to TFA labelling and reducing strategies to industrially produced TFA, but also countries Africa, Asia and South-America follow exactly this definition – clearly excluding conjugated forms, i.e. Austria, Cooperation Council for the Arab States of the Gulf, Colombia, Denmark, Hungary, Iceland, Norway, the Philippines, Singapore, Slovenia, South Africa, South-Korea, Sweden, Switzerland.

In North America, the United States and Canada repeatedly differentiated conjugated forms from the definition of TFA, especially when setting legal implementations to the reduction of industrially produced TFA originating from partial fat hydrogenation:

US FDA expressively excluded conjugated forms of fatty acids with a trans double bond from their definition of trans fatty acids for food labelling (US FDA 2003). In addition, while the latest ban of partially hydrogenated oils, which is the main source for industrially produced TFA, there was again no inclusion of CLA into the definition, simply as the evidence for adverse health effects goes back to TFA coming from partial hydrogenated process: *“This order does not apply to the use of conjugated linoleic acid (CLA) as a food ingredient.”* (USA FDA 2015)

In Canada, Food and Drug Regulation B.01.001(1) by Minister of Justice gives the definition to trans fats also restricting to isolated/non-conjugated forms: *“trans fatty acids [...] means unsaturated fatty acids that contain one or more isolated or non-conjugated double bonds in a trans-configuration.”* (Canada 2018). In addition, Health Canada expressively excluded CLA as food ingredient from the scope of prohibition of partially hydrogenated oils in 2017 (Canada 2017).

World Health Organization (WHO)  
Department of Nutrition for Health and Development  
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5/29/2018

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**WHO Call for public comments on the draft WHO Guidelines:  
“Saturated fatty acid and trans-fatty intake for adults and children”  
Online public consultation open: 04 May to 1 June 2018**

**BASF’s comments on the draft WHO guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”**

2. Current scientific evidence to CLA and adverse health effects related to industrially produced TFA shall be taken into account.

In general, adverse health effects that have been ascribed to trans fatty acids originating from partial hydrogenation process are not scientifically proven to be a general feature of CLA. There is a chemical difference between those fatty acids with isolated and those with conjugated double bonds. Differences in chemical structure usually lead to different reactivities. To our knowledge, there is a clear clinical and epidemiological evidence to the adverse health effects of industrially produced TFA from partial fat hydrogenation, which is yet not the case for the chemically differing molecule of CLA. Taking today’s scientific data into account, we realize, that there is yet no convincing evidence from clinical studies that CLA would behave like TFA in the human body.

Already in 2004, EFSA stated that *“In conclusion, while there is some evidence of adverse effects of supplemental CLA in humans for the trans-10,cis-12 isomer, no such effects were observed for CLA supplements containing mixtures of the trans-10,cis-12 and cis-9,trans-11 isomers. Furthermore, the adverse effects of the trans-10,cis-12 isomer were observed only at intake levels one or two orders of magnitude higher than those corresponding to intake from foods. Few studies have investigated the health effects in humans of naturally occurring CLA from foods and evidence is weak and conflicting with respect any health effects at current levels of intake.”* (EFSA 2004)

Then, in its 2010 „Scientific Opinion on Dietary Reference Values for fats, including saturated fatty acids, polyunsaturated fatty acids, monosaturated fatty acids, trans fatty acids, and cholesterol“, EFSA confirmed a clear differentiation of CLA from the adverse effects caused by industrially produced trans-fatty acids: *“There is no convincing evidence that any of the conjugated linoleic acids isomers in the diet play a role in prevention or promotion of diet-related diseases. The Panel therefore proposes not to set any Dietary Reference Value for conjugated linoleic acids.”* (EFSA 2010 c)

Specifically, for high-quality specified CLA products on the market, a relative 1:1 mixture of *cis9*, *trans11* and *trans10*, *cis12* conjugated linoleic acid isomers obtained from vegetable oils by a distinct manufacturing process is used mainly in the form of food supplements and similar applications for use as an adjunct in body fat reduction regimes. As stated by EFSA in 2010 to these products, there is no evidence of adverse effects on blood lipids, i.e. biomarkers of CVD risk by the consumption of this specified form of CLA: *“The Panel considers that consumption of the 1:1 CLA mixture under the proposed conditions of use has no significant effect on LDL-cholesterol concentrations, and that the magnitude of the changes observed in HDL- and triglyceride concentrations is unlikely to have an impact on coronary heart disease risk.”* (EFSA 2010 a and b)

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Department of Nutrition for Health and Development  
(NHD)

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Online public consultation open: 04 May to 1 June 2018**

**BASF’s comments on the draft WHO guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”**

In 2012, after reviewing the data as available from the studies Wanders et al. 2010 (*Effect of a high intake of conjugated linoleic acid on lipoprotein levels in healthy human subjects*) and Brouwer et al. 2010 (*Effect of animal and industrial trans fatty acids on HDL and LDL cholesterol levels in humans – a quantitative review*), which are referred to in the draft WHO guideline to be the justification for inclusion of CLA to the TFA definition, EFSA confirmed its prior position that “*the additional information provided does not contain evidence that would modify the previous conclusions reached by the Panel regarding the effects of CLA on blood lipids and lipoproteins*”. (EFSA 2012)

In addition, one of the most recent reviews referred to in the draft WHO guideline, the reference (73) by Brouwer published 2016 under WHO, Geneva: *Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis*, to our understanding does not provide new evidence to CLA:

Studies referred to in this review in context with CLA have either already been part of EFSA’s 2012 opinion (Brouwer et al 2010. PLoS One 2010;5(3):e9434, Desroches et al 2005. Am J Clin Nutr 2005;82(2):309-3019, and Brown et al 2011. Nutr Res 2011;31(1):33-41) or do not deal with CLA particularly (Zock Katan 1992. J Lipid Res 1992;33(3):399-410).

We also realize that neither in the review by Brouwer (WHO) 2016 nor in the draft WHO guideline itself the recent review article by Wang/Proctor 2013 „*Current issues surrounding the definition of trans-fatty acids: implications for health, industry and food labels*“ was mentioned. Although the underlying studies used in the 2013 review article might be considered in the draft WHO guidelines or the Brouwer (WHO) 2016 review, it is questionable whether the findings of this review article to differentiation of CLA and TFA’s were taken into account.

Finally, the review by Brouwer/WHO 2016 has already been part of Health Canada’s evaluation to prohibition of partially hydrogenated oils in 2017 with the result of excluding CLA from the TFA prohibition (see above).

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**WHO Call for public comments on the draft WHO Guidelines:  
"Saturated fatty acid and trans-fatty intake for adults and children"  
Online public consultation open: 04 May to 1 June 2018**

**BASF's comments on the draft WHO guidelines on "Saturated fatty acid and trans-fatty intake for adults and children"**

## Summary

Trans fatty acids (TFA) as the subject of discussion about adverse health effects are those fatty acids with trans double bonds that originate from industrial partial oil hydrogenation processes. The current global regulatory framework overall clearly excludes conjugated fatty acids from the definition of trans fatty acids.

We realize that in the draft WHO guideline, the argumentation for inclusion of CLA to the TFA definition does not provide convincing scientific evidence that would allow for such inclusion.

While the evidence for adverse health effects with regard to CVD risk factors is strong for industrially produced TFA originating from partial oil hydrogenation, it is unlikely that CLA has such negative effect.

Overall, we conclude that CLA has to be excluded from definition of TFA in this guideline. Neither is CLA a TFA according to relevant international definitions, nor does the available data justify conclusion on adverse effects on CVD system.

Kind regards,

BASF SE

Human Nutrition

A handwritten signature in blue ink, appearing to read "S. Aurich", written over a horizontal line.

Dr. Sebastian Aurich  
Manager Global Regulatory & External Affairs

World Health Organization (WHO)  
Department of Nutrition for Health and Development  
(NHD)

5/29/2018

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**WHO Call for public comments on the draft WHO Guidelines:  
“Saturated fatty acid and trans-fatty intake for adults and children”  
Online public consultation open: 04 May to 1 June 2018**

**BASF’s comments on the draft WHO guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”**

### References

Canada 2017, Health Canada, September 15, 2017, Notice of Modification - Prohibiting the Use of Partially Hydrogenated Oils (PHOs) in Foods, Reference Number: [NOM/ADM-C-2017-3]

Canada 2018, Minister of Justice, Food and Drug regulation, C.R.C., c. 870, Part B Foods, Division 1 General, B.01.001.(1), current to April 24, 2018

Codex 2017. GUIDELINES ON NUTRITION LABELLING - CAC/GL 2-1985, Adopted in 1985. Revised in 1993 and 2011. Amended in 2003, 2006, 2009, 2010, 2012, 2013, 2015, 2016 and 2017. ANNEX adopted in 2011. Revised in 2013, 2015, 2016 and 2017.

EFSA 2004. Opinion of the scientific panel on dietetic products, nutrition and allergies on a request from the Commission related to the presence of trans fatty acids in foods and the effect on human health of the consumption of trans fatty acids. EFSA Journal 81: 1-49.

EFSA 2010 a. Scientific Opinion on the safety of "conjugated linoleic acid (CLA)-rich oil" (Tonalin® TG 80) as a Novel Food ingredient. EFSA Journal 2010 8 (5): 1600

EFSA 2010 b. Scientific Opinion on the safety of "conjugated linoleic acid (CLA)-rich oil" (Clarinol®) as a Novel Food ingredient. EFSA Journal 2010 8 (5): 1601

EFSA 2010c. Scientific Opinion on Dietary Reference Values for fats, including saturated fatty acids, polyunsaturated fatty acids, monounsaturated fatty acids, trans fatty acids, and cholesterol. EFSA Journal 2010; 8(3):1461

EFSA 2012. Statement on the safety of the "conjugated linoleic acid (CLA)-rich oils" Clarinol® and Tonalin® TG 80 as Novel Food ingredients. The EFSA Journal 2012 10 (5): 2700

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US FDA 2003. Federal Register, Vol. 68, Issue 133, July 11, 2003, Food Labeling: Trans Fatty Acids in Nutrition Labeling, Nutrient Content Claims, and Health Claims

US FDA 2015. Federal Register Vol. 80 Issue 116, June 17, 2015, Final Determination Regarding Partially Hydrogenated Oils

Wang, Proctor 2013. Current issues surrounding the definition of trans-fatty acids: implications for health, industry and food labels. British Journal of Nutrition (2013), 110, 1369–1383

## Survey response 38

### General information

Family/last name
Khor
Given/first name
Geok Lin
Organization/affiliation
Universiti Putra Malaysia
Sector
Academic/research
Sector [Other]
Country
Malaysia

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>Asia Pacific Coconut Community (APCC) Comments on the draft WHO Guidelines on SFA and TFA</p> <p>Dear Sir/Madam:</p> <p>We are submitting these comments on the draft WHO guidelines: "Saturated fatty acid intake and trans-fatty acid intake for adults and children" on behalf of the Asian and Pacific Coconut Community (APCC). The APCC is an intergovernmental organization of 18 coconut producing countries which was organized in 1969 under the aegis of the United Nations Economic and Social Commission for Asia and the Pacific (UN-ESCAP). (<a href="https://www.apccsec.org/apccsec/apccsec-home.html">https://www.apccsec.org/apccsec/apccsec-home.html</a>)</p> <p>We wish to express our concern regarding specific recommendations and statements in the draft WHO guidelines, in particular:</p> <ol style="list-style-type: none"><li>1.❑The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.</li><li>2.❑"Saturated fat" is not the same as "animal fat."</li><li>3.❑Coconut oil is a healthy medium-chain vegetable oil. There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity. The evidence against coconut oil is indirect and invalid.</li><li>4.❑The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.</li></ol>

## Scope and purpose (same for saturated fatty acids and trans-fatty acids)

### 1. The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.

Currently, saturated fatty acids (SFA) are considered as only one group. However, to properly account for the different physiological properties of the individual fatty acids, SFA should be further divided into two groups: medium-chain fatty acids (MCFA, C6 to C12) and saturated long-chain fatty acids (LCFA, C14:0 to C18:0). Although FAO recognized the nomenclature of MCFA and LCFA as different sub-classes of saturated fatty acids in its report entitled "Fats and fatty acids in nutrition", the FAO dietary recommendations still treated them as a single group, ignoring its own nomenclature.

In 1980, Bach and Babayan wrote a review where they concluded that MCFA "must be treated separately and differently in our understanding of fats and oils." There is much evidence for the molecular and cellular differences between MCFA and LCFA. For example, MCFAs do not require carnitine to cross the mitochondrial membrane unlike LCFAs which do. , This results in faster uptake of MCFAs into mitochondria. Once inside the mitochondria, MCFAs are more rapidly oxidized than LCFAs. , Unlike LCFAs, MCFAs are not incorporated into membrane phospholipids and are not stored in liver fat , and adipose tissue. These differentiating properties justify the recognition of a separate category of medium-chain fatty acids (C6 to C12) and long-chain saturated fatty acids (C14:0 to C18:0).

Some authors have contested the classification of lauric acid (C12) as a MCFA based on its incomplete absorption to the portal vein. This phenomenon may be explained by the position of C12 on the triglyceride – that is, whether it is in the sn-1/3 or sn-2 position – as this influences the rate at which C12 is released by lipases in the body. , Trilaurin is a triglyceride wherein all of the attached fatty acids are C12. Upon ingestion, C12 in the sn-1 and sn-3 positions are hydrolyzed most rapidly, leaving a C12 in the sn-2 position. This may explain why C12 is not completely channeled towards the portal vein since some C12 may remain bound to the glyceryl group and is brought into the lymphatic system. This distribution effect, however, does not change the cellular properties of C12 as a MCFA. Further, the distribution of MCFAs between the portal vein and lymph depends on the amount and type of fat that is being consumed by the subject in proportion to the total diet. , In coconut oil, over 10% of the triglycerides is trilaurin and in the oil, C12 is equally distributed between the sn-1/3 and sn-2 positions.

Unfortunately, most publications do not distinguish MCFA and LCFA and simply lump all saturated fat into one group. This is erroneous and must be corrected.

### 2. "Saturated fat" is not the same as "animal fat."

The confusion regarding the use of the categories of "saturated fat" and "animal fat" may have originated from Ancel Keys when he used butter, margarine, and hydrogenated coconut oil to represent "saturated fat" in his early feeding studies. , Keys carried over this confusion in his 1986 Seven Countries Study (SCS) paper where he used the terms "saturated fat" and "animal fat" interchangeably. Although it was clear that it was animal fat that was being consumed, Keys mentioned "saturated fat" repeatedly and "animal fat" only once in the SCS paper. In fact, Keys is mistaken: almost half of the fatty acid composition of animal fat, in particular, beef and pork fat and butter, is unsaturated (see Table). Later, it was determined that the fats that were being consumed in the SCS actually included butter, lard and margarine which are all animal fats; margarine contains industrial trans fats, which are significant predictors of CHD mortality. Therefore, Keys's conclusion that "Death rates were related positively to average percentage of dietary energy from saturated fatty acids" is erroneous: in fact, the fats consumed in the SCS contained considerable amounts of unsaturated fat and trans fat. Thus, research papers which assume that animal fat and saturated fat are similar are also erroneous.

Unfortunately, most publications, including epidemiological surveys and feeding studies, use the terms "saturated fat" and "animal fat" interchangeably without presenting actual fatty acid profiles. Further, when the term "saturated fat" is used, these papers usually mean long-chain saturated fat from animal sources. Therefore, papers that refer to "saturated fat" and/or "animal fat" without an explicit definition of fatty acid profile should not be used in WHO guidelines.

### 3. Coconut oil is a healthy medium-chain vegetable oil

Coconut oil is unique among the major vegetable oils in that it is made up of about 65% medium-chain fatty acids (MCFA) and 92% total saturated fatty acids, with negligible amounts of cholesterol (0 to 3 ppm). In comparison, animal fats (in particular beef and pork fat) are predominantly long-chain (C14 and longer), with significant amounts of unsaturated fatty acids and cholesterol (from 1-2%) (see Table). Thus, coconut oil and animal fats are very different and the basis for the claim that coconut oil is unhealthy is erroneous.

There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity.

There is no direct evidence that coconut oil increases the risk of heart disease. In fact, all of the available evidence, from epidemiological, animal and human studies show that coconut- and coconut oil-based diets are healthy and superior to the western diet. Coconut milk, which contains 20% coconut oil, has been consumed by people in the tropics for thousands of years with no evidence of ill effects until the introduction of the western diet. Before the western diet was introduced, all of the inhabitants of remote Pacific islands consumed a lot of coconut and there was no evidence of heart disease and obesity. In the 1970s, Tokelauans and Pukapukans consumed coconut as the chief source of energy at 63% and 34% of their diet, respectively. Despite

this high coconut intake, vascular disease was uncommon in both populations. Similarly, the inhabitants of Kitava Island who maintained their traditional coconut diet uninfluenced by the western diet did not suffer from a high incidence of stroke and heart disease. Samoa is divided into two regions and provides a clear example of the contrasting effects of the western diet and traditional coconut-based diet. American Samoans who shifted to a western diet showed greater obesity and higher risk for heart disease as compared with Western Samoans who retained their traditional island diet. WHO reported that Pacific islanders were “2.2 times more likely to be obese and 2.4 times more likely to be diabetic if they ate imported fats than if they ate traditional fat sources.” The imported fats included vegetable oils and margarine while the traditional fats included coconut oil.

A number of human studies have shown that coconut and coconut oil-diet are not risk factors for heart disease, and in fact, are associated with healthy indicators. A study of regional diets in the Philippines in the 1980s showed that people in the Bicol region, which had the highest consumption of coconut, showed low incidence of atherosclerosis. The authors concluded that coconut oil does not increase the risk of CVD. In another Philippine study, coconut oil consumption and lipid profiles in a cohort of 1,839 Filipino women, aged 35–69 years, was positively associated with HDL levels. In a study conducted in Brazil, coconut oil showed favorable effects compared with soybean oil on the biochemical and anthropometric profiles of 40 obese women, aged 20 to 40 years. Observational studies in Indonesia likewise showed that consumption of coconut does not lead to adverse cardiovascular outcomes. In reviewing the effect of coconut consumption on cardiovascular risk factors and outcomes in humans, Eyres and co-workers concluded that: “Observational evidence suggests that consumption of coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes.”

A number of controlled studies on humans have shown that coconut oil does not raise risk factors for heart disease. For example, a randomized controlled study in Thailand showed that diets containing coconut milk of one to two meals per day with the fat content of no more than 30% of total energy did not increase either LDL level or cardiovascular risk factors. A 4-week randomised feeding study conducted at Cambridge University, UK, that compared coconut oil, olive oil and butter concluded that coconut oil did not significantly raise LDL concentrations compared with olive oil while butter significantly raised LDL concentrations compared with both coconut oil and olive oil. In addition, coconut oil significantly raised HDL concentrations compared with both butter and olive oil.

The evidence against coconut oil is indirect and invalid

While studies in coconut-consuming countries have consistently shown that coconut oil does not raise the risk of heart disease, the opposite conclusion is reported in studies done in the US and Europe where coconut oil consumption is low. Coconut oil was not part of the diet in Keys’ Seven Country Study. Recent evidence against coconut oil is based on correlation studies on lauric acid (C12) consumption in the US and not on coconut oil itself. In two influential studies, the amount of C12 consumed was less than 0.5% of total energy intake but was consumed from hydrogenated coconut oil and palm kernel oil; , therefore, these conclusions are invalidated by the presence of trans fat. In fact, the vast majority of the studies that have been used to support the recommendations against coconut oil have been done in the US and Europe where consumption of (unhydrogenated) coconut oil is low.

Coconut has been consumed for millennia in the tropics and the Pacific with no indication of poor health outcomes. Coconut oil, which makes up about 20% of coconut meat, is part of this healthy tradition. Close to one billion people today consume coconut and coconut oil on a regular basis. If coconut oil caused heart disease, then there should be an epidemic of heart disease in all coconut consuming countries. Clearly, the evidence against coconut oil is indirect and invalid.

4. The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.

From 1968 to 1973, Ancel Keys and Ivan Frantz, Jr., undertook a controlled human feeding study – called the Minnesota Coronary Survey (MCS) – which was meant to definitively prove the Keys paradigm. This study was the largest (n=9570), longest (5 years), and most rigorously implemented randomized controlled dietary study of cholesterol lowering by replacement of saturated fat with a vegetable oil rich in linoleic acid. It should be noted that the high saturated fat diet provided 18.5% of energy as saturated fat and 3.8% as unsaturated fat, while the high PUFA diet provided 9.2% of energy as saturated fat and 13.2% as omega-6 fat. This experiment showed that although the high omega-6 fat diet lowered serum cholesterol as predicted by Keys, the risk of death increased by 22% compared to the saturated fat diet. The results of this carefully designed and implemented experiment shows that the recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% will lead to higher risk of death from heart disease. Keys himself never published the results, although Frantz published partial results in 1989 which revealed the failure of the Keys hypothesis. These conclusions – that a high omega-6 fat diet reduces serum cholesterol but raises the incidence of heart disease – have been reproduced by a number of other independent studies, such as the Sydney Heart Study.

Although omega-6 and omega-3 polyunsaturated fatty acids are recognized as essential fatty acids, various international agencies have recommended intake levels ranging from 2-4% for omega-6 PUFA and 0.5-2.0% for omega-3, and a omega-6 to omega-3 ratio of about 4:1. However, the draft WHO guidelines do not recommend an upper limit and desirable ratio. As shown by the US experience, the recommendation to limit saturated fat to less than 10% of total energy and to replace saturated fat with PUFA with no guidance on PUFA intake may result in over-consumption of omega-6 fats which has been linked to the rise in obesity and inflammatory disease. This observation is supported mechanistically by a rat study that compared coconut oil with soybean oil, a high omega-6 oil, which showed that soybean oil which upregulated genes for obesity, diabetes, inflammation, mitochondrial



function and cancer while coconut oil gave favorable metabolic indicators.

After reviewing 25 years of the Dietary Guidelines for Americans, Cohen and co-workers (2015) observed that Americans have been religiously following the Dietary Guidelines and this has been coincident with a rise in obesity. This observation is supported by data from the US National Institutes of Health which show that the rise in obesity in the US starting in 1980 is coincident with the introduction of the American dietary guidelines.

Table. Fatty acid composition of selected vegetable oils and animal fat. (uploaded file)

Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Recommendations  This letter has presented several scientific arguments to justify a revision of the draft WHO dietary recommendations, specifically:  1. To recognize the category of medium-chain fatty acids (MCFA, C6-C12); 2. To correct the long-standing errors that saturated fat is inherently unhealthy and that animal fat is a saturated fat; and 3. To delete the recommendations that saturated fat should be kept below 10% and should be replaced – without limit – with a high PUFA diet.  Thank you for your attention. Sincerely yours,  Prof. Fabian M. Dayrit, PhD (Ateneo de Manila University, Philippines) Trinidad P. Trinidad, PhD (Food and Nutrition Research Institute, Department of Science and Technology, Philippines) Prof. Emeritus Khor Geok Lin, PhD (Universiti Putra Malaysia, Malaysia) Assoc. Prof. Wantanee Kriengsinos, PhD (Mahidol University, Thailand)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
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Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 42

### General information

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### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>Despite all the evidence that is available, most publications do not distinguish medium-chain fatty acids (MCFA) and long-chain fatty acids (LCFA) and simply lump all saturated fat into one group. This is erroneous and must be corrected. Most epidemiological surveys and feeding studies use the terms “saturated fat” and “animal fat” interchangeably without presenting actual fatty acid profiles. Further, when the term “saturated fat” is used, these papers usually mean long-chain saturated fat from animal sources. Therefore, papers that refer to “saturated fat” and/or “animal fat” without an explicit definition of fatty acid profile should not be used in WHO guidelines. Coconut has been consumed for millennia in the tropics and the Pacific with no indication of poor health outcomes. Coconut oil, which makes up about 20% of coconut meat, is part of this healthy tradition. Close to one billion people today consume coconut and coconut oil on a regular basis. If coconut oil caused heart disease, then there should be an epidemic of heart disease in all coconut consuming countries. Clearly, the evidence against coconut oil is indirect and invalid. The western diet, which seeks to replace saturated fat with polyunsaturated fat, has been shown to cause obesity and inflammatory diseases.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
<p>We are submitting these comments on the draft WHO guidelines: “Saturated fatty acid intake and trans-fatty acid intake for adults and children” on behalf of the Asian and Pacific Coconut Community (APCC). The APCC is an intergovernmental organization of 18 coconut producing countries which was organized in 1969 under the aegis of the United Nations Economic and Social Commission for Asia and the Pacific (UN-ESCAP). (<a href="https://www.apccsec.org/apccsec/apccsec-home.html">https://www.apccsec.org/apccsec/apccsec-home.html</a>)</p> <p>We wish to express our concern regarding specific recommendations and statements in the draft WHO guidelines, in particular:</p> <ol style="list-style-type: none"><li>1. The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.</li><li>2. “Saturated fat” is not the same as “animal fat.”</li><li>3. Coconut oil is a healthy medium-chain vegetable oil. There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity. The evidence against coconut oil is indirect and invalid.</li><li>4. The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.</li></ol>
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high omega-6 oil, which showed that soybean oil which upregulated genes for obesity, diabetes, inflammation, mitochondrial function and cancer while coconut oil gave favorable metabolic indicators.

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#### Recommendations (for saturated fatty acids only)

1. To recognize the category of medium-chain fatty acids (MCFA, C6-C12);
2. To correct the long-standing errors that saturated fat is inherently unhealthy and that animal fat is a saturated fat; and
3. To delete the recommendations that saturated fat should be kept below 10% and should be replaced – without limit – with a high PUFA diet.

#### Remarks (for saturated fatty acids only)

#### Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

#### Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for saturated fatty acids only)

- [FAO] Food and Agriculture Organisation. Fats and fatty acids in nutrition. Food and Nutrition Paper 91. 2010, p. 19.
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Additional comments

## Trans-fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)

Summary of evidence (for trans-fatty acids only)

Recommendations (for trans-fatty acids only)

Remarks (for trans-fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)



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Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

### Final comments

Please provide any final thoughts or comments below.
WHO should base its dietary guidelines on a wider base of information and not limit itself to western diets and lifestyle conditions.

## Survey response 43

### General information

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Sector [Other]	
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### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)	We are submitting these comments on the draft WHO guidelines: "Saturated fatty acid intake and trans-fatty acid intake for adults and children" on behalf of the Asian and Pacific Coconut Community (APCC). The APCC is an intergovernmental organization of 18 coconut producing countries which was organized in 1969 under the aegis of the United Nations Economic and Social Commission for Asia and the Pacific (UN-ESCAP). ( <a href="https://www.apccsec.org/apccsec/apccsec-home.html">https://www.apccsec.org/apccsec/apccsec-home.html</a> )
Scope and purpose (same for saturated fatty acids and trans-fatty acids)	We wish to express our concern regarding specific recommendations and statements in the draft WHO guidelines, in particular: 1.☐The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat. 2.☐"Saturated fat" is not the same as "animal fat." 3.☐Coconut oil is a healthy medium-chain vegetable oil. There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity. The evidence against coconut oil is indirect and invalid. 4.☐The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.
Background (same for saturated fatty acids and trans-fatty acids)	
Guideline development process (same for saturated fatty acids and trans-fatty acids)	

#### Summary of evidence (for saturated fatty acids only)

##### 1. The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.

Currently, saturated fatty acids (SFA) are considered as only one group. However, to properly account for the different physiological properties of the individual fatty acids, SFA should be further divided into two groups: medium-chain fatty acids (MCFA, C6 to C12) and saturated long-chain fatty acids (LCFA, C14:0 to C18:0). Although FAO recognized the nomenclature of MCFA and LCFA as different sub-classes of saturated fatty acids in its report entitled "Fats and fatty acids in nutrition", the FAO dietary recommendations still treated them as a single group, ignoring its own nomenclature.

In 1980, Bach and Babayan wrote a review where they concluded that MCFA "must be treated separately and differently in our understanding of fats and oils." There is much evidence for the molecular and cellular differences between MCFA and LCFA. For example, MCFAs do not require carnitine to cross the mitochondrial membrane unlike LCFAs which do. , This results in faster uptake of MCFAs into mitochondria. Once inside the mitochondria, MCFAs are more rapidly oxidized than LCFAs. , Unlike LCFAs, MCFAs are not incorporated into membrane phospholipids and are not stored in liver fat , and adipose tissue. These differentiating properties justify the recognition of a separate category of medium-chain fatty acids (C6 to C12) and long-chain saturated fatty acids (C14:0 to C18:0).

Some authors have contested the classification of lauric acid (C12) as a MCFA based on its incomplete absorption to the portal vein. This phenomenon may be explained by the position of C12 on the triglyceride – that is, whether it is in the sn-1/3 or sn-2 position – as this influences the rate at which C12 is released by lipases in the body. , Trilaurin is a triglyceride wherein all of the attached fatty acids are C12. Upon ingestion, C12 in the sn-1 and sn-3 positions are hydrolyzed most rapidly, leaving a C12 in the sn-2 position. This may explain why C12 is not completely channeled towards the portal vein since some C12 may remain bound to the glyceryl group and is brought into the lymphatic system. This distribution effect, however, does not change the cellular properties of C12 as a MCFA. Further, the distribution of MCFAs between the portal vein and lymph depends on the amount and type of fat that is being consumed by the subject in proportion to the total diet. , In coconut oil, over 10% of the triglycerides is trilaurin and in the oil, C12 is equally distributed between the sn-1/3 and sn-2 positions.

##### 2. "Saturated fat" is not the same as "animal fat."

The confusion regarding the use of the categories of "saturated fat" and "animal fat" may have originated from Ancel Keys when he used butter, margarine, and hydrogenated coconut oil to represent "saturated fat" in his early feeding studies. , Keys carried over this confusion in his 1986 Seven Countries Study (SCS) paper where he used the terms "saturated fat" and "animal fat" interchangeably. Although it was clear that it was animal fat that was being consumed, Keys mentioned "saturated fat" repeatedly and "animal fat" only once in the SCS paper. In fact, Keys is mistaken: almost half of the fatty acid composition of animal fat, in particular, beef and pork fat and butter, is unsaturated (see Table). Later, it was determined that the fats that were being consumed in the SCS actually included butter, lard and margarine which are all animal fats; margarine contains industrial trans fats, which are significant predictors of CHD mortality. Therefore, Keys's conclusion that "Death rates were related positively to average percentage of dietary energy from saturated fatty acids" is erroneous: in fact, the fats consumed in the SCS contained considerable amounts of unsaturated fat and trans fat. Thus, research papers which assume that animal fat and saturated fat are similar are also erroneous.

##### 3. Coconut oil is a healthy medium-chain vegetable oil

Coconut oil is unique among the major vegetable oils in that it is made up of about 65% medium-chain fatty acids (MCFA) and 92% total saturated fatty acids, with negligible amounts of cholesterol (0 to 3 ppm). In comparison, animal fats (in particular beef and pork fat) are predominantly long-chain (C14 and longer), with significant amounts of unsaturated fatty acids and cholesterol (from 1-2%) (see Table). Thus, coconut oil and animal fats are very different and the basis for the claim that coconut oil is unhealthy is erroneous.

There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity.

There is no direct evidence that coconut oil increases the risk of heart disease. In fact, all of the available evidence, from epidemiological, animal and human studies show that coconut- and coconut oil-based diets are healthy and superior to the western diet. Coconut milk, which contains 20% coconut oil, has been consumed by people in the tropics for thousands of years with no evidence of ill effects until the introduction of the western diet. Before the western diet was introduced, all of the inhabitants of remote Pacific islands consumed a lot of coconut and there was no evidence of heart disease and obesity. In the 1970s, Tokelauans and Pukapukans consumed coconut as the chief source of energy at 63% and 34% of their diet, respectively. Despite this high coconut intake, vascular disease was uncommon in both populations. Similarly, the inhabitants of Kitava Island who maintained their traditional coconut diet uninfluenced by the western diet did not suffer from a high incidence of stroke and heart disease. Samoa is divided into two regions and provides a clear example of the contrasting effects of the western diet and traditional coconut-based diet. American Samoans who shifted to a western diet showed greater obesity and higher risk for heart disease as compared with Western Samoans who retained their traditional island diet. WHO reported that Pacific islanders were "2.2 times more likely to be obese and 2.4 times more likely to be diabetic if they ate imported fats than if they ate traditional fat sources." The imported fats included vegetable oils and margarine while the traditional fats included coconut oil.

A number of human studies have shown that coconut and coconut oil-diet are not risk factors for heart disease, and in fact, are associated with healthy indicators. A study of regional diets in the Philippines in the 1980s showed that people in the Bicol region, which had the highest consumption of coconut, showed low incidence of atherosclerosis. The authors concluded that coconut oil does not increase the risk of CVD. In another Philippine study, coconut oil consumption and lipid profiles in a cohort of 1,839 Filipino women, aged 35–69 years, was positively associated with HDL levels. In a study conducted in Brazil, coconut oil showed favorable effects compared with soybean oil on the biochemical and anthropometric profiles of 40 obese women, aged 20 to 40 years. Observational studies in Indonesia likewise showed that consumption of coconut does not lead to adverse cardiovascular outcomes. In reviewing the effect of coconut consumption on cardiovascular risk factors and outcomes in humans, Eyres and co-workers concluded that: “Observational evidence suggests that consumption of coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes.”

A number of controlled studies on humans have shown that coconut oil does not raise risk factors for heart disease. For example, a randomized controlled study in Thailand showed that diets containing coconut milk of one to two meals per day with the fat content of no more than 30% of total energy did not increase either LDL level or cardiovascular risk factors. A 4-week randomised feeding study conducted at Cambridge University, UK, that compared coconut oil, olive oil and butter concluded that coconut oil did not significantly raise LDL concentrations compared with olive oil while butter significantly raised LDL concentrations compared with both coconut oil and olive oil. In addition, coconut oil significantly raised HDL concentrations compared with both butter and olive oil.

The evidence against coconut oil is indirect and invalid

While studies in coconut-consuming countries have consistently shown that coconut oil does not raise the risk of heart disease, the opposite conclusion is reported in studies done in the US and Europe where coconut oil consumption is low. Coconut oil was not part of the diet in Keys’ Seven Country Study. Recent evidence against coconut oil is based on correlation studies on lauric acid (C12) consumption in the US and not on coconut oil itself. In two influential studies, the amount of C12 consumed was less than 0.5% of total energy intake but was consumed from hydrogenated coconut oil and palm kernel oil; , therefore, these conclusions are invalidated by the presence of trans fat. In fact, the vast majority of the studies that have been used to support the recommendations against coconut oil have been done in the US and Europe where consumption of (unhydrogenated) coconut oil is low.

4. The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.

From 1968 to 1973, Ancel Keys and Ivan Frantz, Jr., undertook a controlled human feeding study – called the Minnesota Coronary Survey (MCS) – which was meant to definitively prove the Keys paradigm. This study was the largest (n=9570), longest (5 years), and most rigorously implemented randomized controlled dietary study of cholesterol lowering by replacement of saturated fat with a vegetable oil rich in linoleic acid. It should be noted that the high saturated fat diet provided 18.5% of energy as saturated fat and 3.8% as unsaturated fat, while the high PUFA diet provided 9.2% of energy as saturated fat and 13.2% as omega-6 fat. This experiment showed that although the high omega-6 fat diet lowered serum cholesterol as predicted by Keys, the risk of death increased by 22% compared to the saturated fat diet. The results of this carefully designed and implemented experiment shows that the recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% will lead to higher risk of death from heart disease. Keys himself never published the results, although Frantz published partial results in 1989 which revealed the failure of the Keys hypothesis. These conclusions – that a high omega-6 fat diet reduces serum cholesterol but raises the incidence of heart disease – have been reproduced by a number of other independent studies, such as the Sydney Heart Study.

Although omega-6 and omega-3 polyunsaturated fatty acids are recognized as essential fatty acids, various international agencies have recommended intake levels ranging from 2-4% for omega-6 PUFA and 0.5-2.0% for omega-3, and a omega-6 to omega-3 ratio of about 4:1. However, the draft WHO guidelines do not recommend an upper limit and desirable ratio. As shown by the US experience, the recommendation to limit saturated fat to less than 10% of total energy and to replace saturated fat with PUFA with no guidance on PUFA intake may result in over-consumption of omega-6 fats which has been linked to the rise in obesity and inflammatory disease. This observation is supported mechanistically by a rat study that compared coconut oil with soybean oil, a high omega-6 oil, which showed that soybean oil upregulated genes for obesity, diabetes, inflammation, mitochondrial function and cancer while coconut oil gave favorable metabolic indicators.

#### Recommendations (for saturated fatty acids only)

This letter has presented several scientific arguments to justify a revision of the draft WHO dietary recommendations, specifically:

1. To recognize the category of medium-chain fatty acids (MCFA, C6-C12);
2. To correct the long-standing errors that saturated fat is inherently unhealthy and that animal fat is a saturated fat; and
3. To delete the recommendations that saturated fat should be kept below 10% and should be replaced – without limit – with a high PUFA diet.

Remarks (for saturated fatty acids only)
<p>Most publications do not distinguish MCFA and LCFA and simply lump all saturated fat into one group. This is erroneous and must be corrected.</p> <p>Unfortunately, most publications, including epidemiological surveys and feeding studies, use the terms “saturated fat” and “animal fat” interchangeably without presenting actual fatty acid profiles. Further, when the term “saturated fat” is used, these papers usually mean long-chain saturated fat from animal sources. Therefore, papers that refer to “saturated fat” and/or “animal fat” without an explicit definition of fatty acid profile should not be used in WHO guidelines.</p> <p>Coconut has been consumed for millennia in the tropics and the Pacific with no indication of poor health outcomes. Coconut oil, which makes up about 20% of coconut meat, is part of this healthy tradition. Close to one billion people today consume coconut and coconut oil on a regular basis. If coconut oil caused heart disease, then there should be an epidemic of heart disease in all coconut consuming countries. Clearly, the evidence against coconut oil is indirect and invalid.</p> <p>After reviewing 25 years of the Dietary Guidelines for Americans, Cohen and co-workers (2015) observed that Americans have been religiously following the Dietary Guidelines and this has been coincident with a rise in obesity. This observation is supported by data from the US National Institutes of Health which show that the rise in obesity in the US starting in 1980 is coincident with the introduction of the American dietary guidelines.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

## Annexes 1, 6, 7 (for saturated fatty acids only)

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Additional comments

## *Trans*-fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
<p>From results of the above studies, there is no exact scientific evidence that coconut oil is harmful to health or has an adverse effect on CVD risk. Coconut oil is not purely a long chain SFA and should not be compared to the SFA present in pork, beef and butter. Considering that coconut oil is not purely long chain fatty acid (65% medium chain fatty acid), more studies are needed in order to conclude if it is not safe for consumption relative to CVD risk. Studies on permissible level of coconut oil consumption should be considered. Many gaps are still to be answered to conclude that coconut oil is not safe for consumption specifically in the prevention for risk of CVD.</p>



## **APCC Comments on the draft WHO Guidelines on SFA and TFA**

Dear Sir/Madam:

We are submitting these comments on the draft WHO guidelines: “Saturated fatty acid intake and trans-fatty acid intake for adults and children” on behalf of the Asian and Pacific Coconut Community (APCC). The APCC is an intergovernmental organization of 18 coconut producing countries which was organized in 1969 under the aegis of the United Nations Economic and Social Commission for Asia and the Pacific (UN-ESCAP). (<https://www.apccsec.org/apccsec/apccsec-home.html>)

We wish to express our concern regarding specific recommendations and statements in the draft WHO guidelines, in particular:

1. The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.
2. “Saturated fat” is not the same as “animal fat.”
3. Coconut oil is a healthy medium-chain vegetable oil. There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity. The evidence against coconut oil is indirect and invalid.
4. The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.

### **1. The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.**

Currently, saturated fatty acids (SFA) are considered as only one group. However, to properly account for the different physiological properties of the individual fatty acids, SFA should be further divided into two groups: medium-chain fatty acids (MCFA, C6 to C12) and saturated long-chain fatty acids (LCFA, C14:0 to C18:0). Although FAO recognized the nomenclature of MCFA and LCFA as different sub-classes of saturated fatty acids in its report entitled “Fats and fatty acids in nutrition”,<sup>1</sup> the FAO dietary recommendations still treated them as a single group, ignoring its own nomenclature.

In 1980, Bach and Babayan wrote a review where they concluded that MCFA “must be treated separately and differently in our understanding of fats and oils.”<sup>2</sup> There is much evidence for the molecular and cellular differences between MCFA and LCFA. For example, MCFAs do not require carnitine to cross the mitochondrial membrane unlike LCFAs which do.<sup>3,4</sup> This results in faster uptake of MCFAs into mitochondria. Once inside the

mitochondria, MCFAs are more rapidly oxidized than LCFAs.<sup>5,6</sup> Unlike LCFAs, MCFAs are not incorporated into membrane phospholipids<sup>7</sup> and are not stored in liver fat<sup>8,9</sup> and adipose tissue.<sup>10</sup> These differentiating properties justify the recognition of a separate category of medium-chain fatty acids (C6 to C12) and long-chain saturated fatty acids (C14:0 to C18:0).

Some authors have contested the classification of lauric acid (C12) as a MCFA based on its incomplete absorption to the portal vein.<sup>11</sup> This phenomenon may be explained by the position of C12 on the triglyceride – that is, whether it is in the sn-1/3 or sn-2 position – as this influences the rate at which C12 is released by lipases in the body.<sup>12,13</sup> Trilaurin is a triglyceride wherein all of the attached fatty acids are C12. Upon ingestion, C12 in the sn-1 and sn-3 positions are hydrolyzed most rapidly, leaving a C12 in the sn-2 position. This may explain why C12 is not completely channeled towards the portal vein since some C12 may remain bound to the glyceryl group and is brought into the lymphatic system. This distribution effect, however, does not change the cellular properties of C12 as a MCFA. Further, the distribution of MCFAs between the portal vein and lymph depends on the amount and type of fat that is being consumed by the subject in proportion to the total diet.<sup>14,15</sup> In coconut oil, over 10% of the triglycerides is trilaurin<sup>16</sup> and in the oil, C12 is equally distributed between the sn-1/3 and sn-2 positions.<sup>17</sup>

Unfortunately, most publications do not distinguish MCFA and LCFA and simply lump all saturated fat into one group. This is erroneous and must be corrected.

## **2. “Saturated fat” is not the same as “animal fat.”**

The confusion regarding the use of the categories of “saturated fat” and “animal fat” may have originated from Ancel Keys when he used butter, margarine, and *hydrogenated* coconut oil to represent “saturated fat” in his early feeding studies.<sup>18,19</sup> Keys carried over this confusion in his 1986 Seven Countries Study (SCS) paper<sup>20</sup> where he used the terms “saturated fat” and “animal fat” interchangeably. Although it was clear that it was animal fat that was being consumed, Keys mentioned “saturated fat” repeatedly and “animal fat” only once in the SCS paper. In fact, Keys is mistaken: almost half of the fatty acid composition of animal fat, in particular, beef and pork fat and butter, is *unsaturated*<sup>21</sup> (see Table). Later, it was determined that the fats that were being consumed in the SCS actually included butter, lard and margarine which are all animal fats; margarine contains industrial trans fats, which are significant predictors of CHD mortality.<sup>22</sup> Therefore, Keys’s conclusion that “Death rates were related positively to average percentage of dietary energy from saturated fatty acids” is *erroneous*: in fact, the fats consumed in the SCS contained considerable amounts of unsaturated fat and trans fat. Thus, research papers which assume that animal fat and saturated fat are similar are also *erroneous*.

Unfortunately, most publications, including epidemiological surveys and feeding studies, use the terms “saturated fat” and “animal fat” interchangeably without presenting actual fatty acid profiles. Further, when the term “saturated fat” is used, these papers usually mean long-chain saturated fat from animal sources. Therefore, papers that refer to “saturated fat”

and/or “animal fat” without an explicit definition of fatty acid profile should not be used in WHO guidelines.

### **3. Coconut oil is a healthy medium-chain vegetable oil**

Coconut oil is unique among the major vegetable oils in that it is made up of about 65% medium-chain fatty acids (MCFA) and 92% total saturated fatty acids, with negligible amounts of cholesterol (0 to 3 ppm). In comparison, animal fats (in particular beef and pork fat) are predominantly long-chain (C14 and longer), with significant amounts of unsaturated fatty acids and cholesterol (from 1-2%) (see Table). Thus, coconut oil and animal fats are very different and the basis for the claim that coconut oil is unhealthy is *erroneous*.

There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity.

There is no direct evidence that coconut oil increases the risk of heart disease. In fact, all of the available evidence, from epidemiological, animal and human studies show that coconut- and coconut oil-based diets are healthy and superior to the western diet. Coconut milk, which contains 20% coconut oil,<sup>23</sup> has been consumed by people in the tropics for thousands of years with no evidence of ill effects until the introduction of the western diet. Before the western diet was introduced, all of the inhabitants of remote Pacific islands consumed a lot of coconut and there was no evidence of heart disease and obesity. In the 1970s, Tokelauans and Pukapukans consumed coconut as the chief source of energy at 63% and 34% of their diet, respectively. Despite this high coconut intake, vascular disease was uncommon in both populations.<sup>24</sup> Similarly, the inhabitants of Kitava Island who maintained their traditional coconut diet uninfluenced by the western diet did not suffer from a high incidence of stroke and heart disease.<sup>25</sup> Samoa is divided into two regions and provides a clear example of the contrasting effects of the western diet and traditional coconut-based diet. American Samoans who shifted to a western diet showed greater obesity and higher risk for heart disease as compared with Western Samoans who retained their traditional island diet.<sup>26</sup> WHO reported that Pacific islanders were “2.2 times more likely to be obese and 2.4 times more likely to be diabetic if they ate imported fats than if they ate traditional fat sources.”<sup>27</sup> The imported fats included vegetable oils and margarine while the traditional fats included coconut oil.

A number of human studies have shown that coconut and coconut oil-diet are not risk factors for heart disease, and in fact, are associated with healthy indicators. A study of regional diets in the Philippines in the 1980s showed that people in the Bicol region, which had the highest consumption of coconut, showed low incidence of atherosclerosis. The authors concluded that coconut oil does not increase the risk of CVD.<sup>28</sup> In another Philippine study, coconut oil consumption and lipid profiles in a cohort of 1,839 Filipino women, aged 35–69 years, was positively associated with HDL levels.<sup>29</sup> In a study conducted in Brazil, coconut oil showed favorable effects compared with soybean oil on the biochemical and anthropometric profiles of 40 obese women, aged 20 to 40 years.<sup>30</sup>

Observational studies in Indonesia likewise showed that consumption of coconut does not lead to adverse cardiovascular outcomes.<sup>31</sup> In reviewing the effect of coconut consumption on cardiovascular risk factors and outcomes in humans, Eyres and co-workers concluded that: "Observational evidence suggests that consumption of coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes."<sup>32</sup>

A number of controlled studies on humans have shown that coconut oil does not raise risk factors for heart disease. For example, a randomized controlled study in Thailand showed that diets containing coconut milk of one to two meals per day with the fat content of no more than 30% of total energy did not increase either LDL level or cardiovascular risk factors.<sup>33</sup> A 4-week randomised feeding study conducted at Cambridge University, UK, that compared coconut oil, olive oil and butter concluded that coconut oil did not significantly raise LDL concentrations compared with olive oil while butter significantly raised LDL concentrations compared with both coconut oil and olive oil. In addition, coconut oil significantly raised HDL concentrations compared with both butter and olive oil.<sup>34</sup>

#### The evidence against coconut oil is indirect and invalid

While studies in coconut-consuming countries have consistently shown that coconut oil does not raise the risk of heart disease, the opposite conclusion is reported in studies done in the US and Europe where coconut oil consumption is low. Coconut oil was not part of the diet in Keys' Seven Country Study. Recent evidence against coconut oil is based on correlation studies on lauric acid (C12) consumption in the US and not on coconut oil itself. In two influential studies, the amount of C12 consumed was less than 0.5% of total energy intake but was consumed from hydrogenated coconut oil and palm kernel oil;<sup>35,36</sup> therefore, these conclusions are invalidated by the presence of trans fat. In fact, the vast majority of the studies that have been used to support the recommendations against coconut oil have been done in the US and Europe where consumption of (unhydrogenated) coconut oil is low.<sup>37</sup>

Coconut has been consumed for millennia in the tropics and the Pacific with no indication of poor health outcomes. Coconut oil, which makes up about 20% of coconut meat, is part of this healthy tradition. Close to one billion people today consume coconut and coconut oil on a regular basis. If coconut oil caused heart disease, then there should be an epidemic of heart disease in all coconut consuming countries. Clearly, the evidence against coconut oil is indirect and invalid.

**4. The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.**

From 1968 to 1973, Ancel Keys and Ivan Frantz, Jr., undertook a controlled human feeding study – called the Minnesota Coronary Survey (MCS) – which was meant to definitively prove the Keys paradigm.<sup>38</sup> This study was the largest (n=9570), longest (5 years), and most rigorously implemented randomized controlled dietary study of cholesterol lowering by replacement of saturated fat with a vegetable oil rich in linoleic acid. It should be noted that the high saturated fat diet provided 18.5% of energy as saturated fat and 3.8% as unsaturated fat, while the high PUFA diet provided 9.2% of energy as saturated fat and 13.2% as omega-6 fat. This experiment showed that although the high omega-6 fat diet lowered serum cholesterol as predicted by Keys, the risk of death increased by 22% compared to the saturated fat diet.<sup>39</sup> *The results of this carefully designed and implemented experiment shows that the recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% will lead to higher risk of death from heart disease.* Keys himself never published the results, although Frantz published partial results in 1989 which revealed the failure of the Keys hypothesis.<sup>40</sup> These conclusions – that a high omega-6 fat diet reduces serum cholesterol but raises the incidence of heart disease – have been reproduced by a number of other independent studies, such as the Sydney Heart Study.<sup>41</sup>

Although omega-6 and omega-3 polyunsaturated fatty acids are recognized as essential fatty acids, various international agencies have recommended intake levels ranging from 2-4% for omega-6 PUFA and 0.5-2.0% for omega-3, and a omega-6 to omega-3 ratio of about 4:1.<sup>42</sup> However, the draft WHO guidelines do not recommend an upper limit and desirable ratio. As shown by the US experience, the recommendation to limit saturated fat to less than 10% of total energy and to replace saturated fat with PUFA with no guidance on PUFA intake may result in over-consumption of omega-6 fats<sup>43</sup> which has been linked to the rise in obesity<sup>44</sup> and inflammatory disease.<sup>45</sup> This observation is supported mechanistically by a rat study that compared coconut oil with soybean oil, a high omega-6 oil, which showed that soybean oil which upregulated genes for obesity, diabetes, inflammation, mitochondrial function and cancer while coconut oil gave favorable metabolic indicators.<sup>46</sup>

After reviewing 25 years of the Dietary Guidelines for Americans, Cohen and co-workers (2015)<sup>47</sup> observed that Americans have been religiously following the Dietary Guidelines and this has been coincident with a rise in obesity. This observation is supported by data from the US National Institutes of Health<sup>48</sup> which show that the rise in obesity in the US starting in 1980 is coincident with the introduction of the American dietary guidelines.<sup>49</sup>

## **Recommendations**

This letter has presented several scientific arguments to justify a revision of the draft WHO dietary recommendations, specifically:

1. To recognize the category of medium-chain fatty acids (MCFA, C6-C12);
2. To correct the long-standing errors that saturated fat is inherently unhealthy and that animal fat is a saturated fat; and

3. To delete the recommendations that saturated fat should be kept below 10% and should be replaced – without limit – with a high PUFA diet.

Thank you for your attention.

Sincerely yours,

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**Table.** Fatty acid composition of selected vegetable oils and animal fat.

Median values	Vegetable oils (% of total fatty acids)					Animal fat (g/100g edible portion)		
Fatty acid	Coconut oil <sup>1</sup>	Maize oil <sup>1</sup>	Olive oil <sup>2</sup>	Palm olein <sup>1</sup>	Soybean oil <sup>1</sup>	Lard, Pork fat <sup>3</sup>	Tallow, Beef fat <sup>3</sup>	Butter <sup>3</sup>
C4:0								2.6
C6:0	0.4	0.0		0.0	0.0			1.6
C8:0	7.3	0.0		0.0	0.0			0.9
C10:0	6.5	0.0		0.0	0.0	0.1		2.0
C12:0	49.2	0.2	0.0	0.3	0.1	0.2	0.9	2.3
C14:0	18.9	0.2	0.1	1.0	0.1	1.3	3.7	8.2
C16:0	8.9	12.6	13.8	40.8	10.8	23.8	24.9	21.3
C16:1	0.0	0.3	1.9	0.3	0.1	2.7	4.2	1.8
C17:0	0.0	0.1	0.5	0.1	0.1			
C17:1	0.0	0.1	0.6	0.1	0.1			
C18:0	3.0	1.7	2.8	4.3	3.7	13.5	18.9	9.8
C18:1	7.5	31.1	69.0	42.9	23.5	41.2	36.0	20.4
C18:2	1.8	49.8	12.3	11.8	53.5	10.2	3.1	1.8
C18:3	0.1	1.0	1.5	0.3	7.8	1.0	0.6	1.2
C20:0	0.1	0.7	0.8	0.3	0.4			
C20:1	0.1	0.4		0.2	0.3	1.0	0.3	
C20:2	0.0	0.1		0.0	0.1			
C22:0	0.0	0.3	0.3	0.1	0.4			
C22:1	0.0	0.2	0.0	0.0	0.2			
C22:2	0.0	0.0		0.0	0.0			
C24:0	0.0	0.3	1.0	0.0	0.3			
C24:1	0.0	0.0		0.0	0.0			
<b>Total satd fat</b>	<b>94</b>	<b>16</b>	<b>19</b>	<b>47</b>	<b>16</b>	<b>39</b>	<b>48</b>	<b>46</b>
MCFA (satd)	63	0	0	0	0	0	1	7
LCFA (satd)	31	16	19	47	16	39	48	39
<b>Total unsatd fat</b>	<b>9</b>	<b>83</b>	<b>85</b>	<b>56</b>	<b>85</b>	<b>56</b>	<b>44</b>	<b>25</b>
MUFA	8	32	72	43	24	45	41	22
PUFA	2	51	14	12	61	11	4	3
n-6/n-3	18	50	8	39	7	10	5	2
<b>Cholesterol, mg/100 g</b>	0.0 - 0.3	0.02 - 0.06	<0.05	0.3 - 0.7	0.02 - 0.14	95	109	219
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## Survey response 44

### General information

Family/last name
Singh
Given/first name
Permjit
Organization/affiliation
individual
Sector
Private sector
Sector [Other]
Country
United Kingdom

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments
<p>I have not read the research provided so base my comments on my knowledge of saturated fats and trans-fats. I have no doubt that</p> <p>a) saturated fat ingestion must be limited</p> <p>b) trans-fats should be ingested with even greater caution and in my opinion, they should not be ingested at all (I know of no reason why they should be ingested or why they should be an added ingredient in foods).</p> <p>Of course, some food processors will resist using alternatives to trans-fats if alternatives mean added cost (and so reduced profits) but their concerns should be resisted if health is of primary concern.</p>

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments
See previous comments

## Final comments

Please provide any final thoughts or comments below.
<p>I read that trans-fats were banned in the US in 2006, so am amazed it has taken 11 years for the UK to get to this consultation. To have delayed correction of what is (to me) an obvious health hazard, is scandalous.</p> <p>Permjit Singh 18 May 2018</p>

## Survey response 46

### General information

Family/last name
Watzl
Given/first name
Bernhard
Organization/affiliation
Max Rubner-Institut, Federal Research Institute of Nutrition and Food
Sector
Academic/research
Sector [Other]
Country
Germany

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Recommendations should be given for foods and not for energy percentages of macronutrients. Further, replacing SFA with PUFA is easily achievable at the level of macronutrients, but not at the level of foods which are the sources of these fatty acids. The same is true for replacing industrial trans-FA with PUFA. As another example, the type of carbohydrate which is used to replace SFA is critical for the overall outcome of such replacement.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

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Please provide any final thoughts or comments below.

Overall the WHO Guidelines on Saturated Fatty Acids (SFA) are thoroughly designed and developed, and I am sure that all relevant studies are included and have been analyzed. However, developing dietary guidelines at the level of nutrients without addressing the food matrix seems to be outdated. Various European nutrition societies have already or are in the process of developing food-based dietary guidelines. People consume foods and not nutrients, and today consumers expect recommendations at the food level. The food matrix has a well-documented impact on the physiological effect of its nutrients. SFA are a good example for this issue.

SFA are a heterogeneous group of chemicals with different physiological effects. Depending on the food source short-, medium- or long-chain SFA are taken up with these foods. Dairy is known to contain odd-chain fatty acids, prospective studies suggest that these SFA are inversely associated with diabetes incidence. The WHO Guideline does not address any of these subtypes of SFA. Dairy intake is further related to a reduced risk of hypertension as well as of colon cancer. There is only a brief paragraph (page 17) explaining why SFA from food sources such as dairy have not been evaluated in more detail, in spite of several consistent meta-analyses suggesting a lower risk for several diseases in people consuming a moderate amount of dairy.

"Efforts to understand the effects of saturated fatty acid intake in greater detail have shown that individual saturated fatty acids may have differing effects on blood lipids (11), and emerging evidence has led to the suggestion that different saturated fatty acid-containing foods, such as dairy foods, may have different effects on risk of cardiovascular diseases and type 2 diabetes, either as a result of differing composition of saturated fatty acids across foods, other constituents of the foods, or a combination of the two (38-42). However, many questions remain to be answered before a clear understanding can be reached and firm conclusions drawn" (page 17).

Obviously WHO identifies the need for more studies relating SFA to their food sources (page 46): "RCTs comparing the effects of saturated fatty acids from different food sources (e.g. plant, animal and dairy) on cardiovascular diseases and mortality". Without evidence from such studies there is no need to publish an updated WHO Guideline on SFA, because the new recommendations do not differ from the previous recommendations.

## Survey response 47

### General information

Family/last name
McNeill
Given/first name
Shalene
Organization/affiliation
The Beef Checkoff
Sector
Other
Sector [Other]
Food Industry
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)



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Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
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It is noted that there is a distinction between intake levels of industrial and ruminant trans-fatty acids and there may be a difference in their impact on health outcomes, between the two types of trans-fatty acids. It is quoted in the Executive summary that, "Few studies have identified an association between intake of ruminant trans-fatty acids and CVDs; however, to date, ruminant trans-fatty acid intake in most study populations has been very low (Brouwer et al 2013)." However, the executive summary addresses trans-fatty acid as a combination of those that are industrially-produced and those that are naturally present in products from ruminant animals as seen in the remarks section, "Trans-fatty acids include all fatty acids with a double bond in the trans configuration regardless of whether they come from ruminant sources or are produced industrially."

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)

## Summary of evidence (for trans-fatty acids only)

### Prospective Cohort Data:

The draft summary of evidence regarding trans-fatty acid intake and health outcomes notes that in the meta-analyses of prospective cohort studies (de Souza et al., 2015), total and industrially-produced trans-fatty acid intakes were similar in their effects on risk of CHD events and there were no significant associations observed for the effects of ruminant trans-fatty acid intake on CHD events and/or mortality. It is noted that, "The evidence reviewed suggests that differences in effect on health outcomes between ruminant and industrially-produced trans-fatty acids observed in many studies are most likely to be due to differences in dose of trans-fatty acids rather than differences in type of trans-fatty acids," (WHO Draft Saturated and Trans Fatty Acid Guidelines [DWG], Pg. 32). This conclusion is supported by a footnote indicating that "a post-hoc meta-analysis of total trans-fatty acids showed that intake was no longer associated with CHD mortality when limited only to studies in which total trans-fatty acid intake was at similar levels to those reported in studies included in the analysis of ruminant trans-fatty acid intake, although a significant association remained for CHD events" (DWG footnote 5, Pg. 32), the publication by de Souza et al. 2015 is cited. The summary of evidence does not, however, provide the actual results obtained from this post-hoc analysis.

The objective for the cited post-hoc analysis, as stated by the authors (de Souza et al., 2015), was to determine if "...the generally low exposure levels to ruminant trans fatty acids were driving the lack of association observed for these outcomes..." i.e. CHD mortality and total CHD. To do so, the authors used total CHD event and mortality data to determine the association between a 0.8% of energy intake increase in total trans-fatty acid and CHD mortality, and 1.2% of energy increase and total CHD (intake estimates were selected to represent the highest ruminant trans-fatty acid intakes). The authors report a statistically weak (1.02), non-statistically significant (CI 0.90-1.16) risk ratio for total trans-fatty acids when using published, as well as unpublished data (1.03; 0.95-1.12), for CHD mortality. For total trans-fatty acid and total CHD, the results remain weak (1.17) but statistically significant (CI 1.07-1.29). Importantly, however, the 0.8% of energy intake for ruminant trans-fatty acids is estimated from one Norwegian study (Laake et al., 2012), and the 1.2% of energy intake increase is estimated from three studies, with one study (Jakobsen et al., 2008) deemed by the authors to be an "influential outlier" (de Souza et al., 2015, Appendix 2 eTable 20). Accordingly, the original study authors frame their penultimate conclusion, i.e. "based on currently available data from prospective cohort studies, ruminant derived trans fats are not associated with risk of CHD..." by posing "unanswered questions" and suggesting "future research" including, "...do threshold levels of ruminant trans fatty acid intakes exist, above which cardiovascular risk increases in a similar fashion to that seen with industrial trans fatty acids?" (de Souza et al., 2015) The WHO Draft Guidelines use this data to conclude that, "It was therefore determined that the available evidence did not support making a distinction between industrial and ruminant trans-fatty acids, and data solely from analyses of total trans-fatty acids were considered when formulating the recommendations on trans-fatty acid intake." [DWG, Pg. 33]

### Randomized Controlled Data:

The summary of evidence regarding trans-fatty acid intakes and health outcomes is missing one of two current syntheses of randomized controlled trial (RCT) data and thus does not represent the totality of available evidence. The publication by de Souza and colleagues (2015) discusses the available data from RCTs noting that "two quantitative syntheses of randomized controlled trials of ruminant derived trans-fats and biomarkers of cardiovascular risk arrived at opposite conclusions." WHO largely predicates their evidence summary of RCTs from one study (Brouwer et al., 2016). Brouwer et al. includes 16 RCTs, but excludes 18. The included studies contain only 3 RCTs investigating ruminant trans-fatty acids (n=133 subjects), all conducted in one region (i.e., North America). The duration of included studies ranged from 14 days to 8 weeks which, according to Willett and Mozaffarian (2008), may limit the interpretation of results from controlled feeding trials of trans-fatty acids due to the brevity of intervention. Regarding their evidence, Brouwer and co-authors recognize "...a limited number of high-quality ruminant TFA studies", and suggest that the low intakes of ruminant trans-fat world-wide "...correspond to a small resulting risk of negative health effects." They caveat this conclusion by saying, "...intake of ruminant TFA is now exceeding intake of industrial TFA in many populations." The significance of this observation is unclear, as estimated intake of ruminant trans-fat world-wide remains well below the 1.0% of total energy recommended by WHO, and well below the levels typically provided in RCTs (i.e. up to 5.0% of total energy). In fact, Wanders and colleagues (2017) reports the mean 90-95th percentile of ruminant trans-fat intake is 0.98% world-wide, indicating that most the world's population consumes far less. Additionally, estimates suggest that if a person were to consume the entire maximum saturated fat intake (10% of energy) from ruminant sources, total trans-fat would still remain less than 1% of total energy (Willett and Mozaffarian, 2008).

In contrast, the second quantitative syntheses of RCTs acknowledged in de Souza et al., 2015 was conducted by Gayet-Boyer and colleagues (2014) who pooled 13 RCTs investigating ruminant trans-fatty acids, including the 3 reviewed by Brouwer et al., and reported no linear relationship between ruminant trans-fatty acid intake levels of up to 4.19% of total energy and total cholesterol, HDL-C and LDL-C:HDL-C ratios. The authors conclude, "These data suggest that TFA from natural sources, at least at the current levels of intake and up to 4.19% of EI, have no adverse effects on these key CVD risk markers in healthy people" (Gayet-Boyer et al., 2014).

### References:

- Brouwer, IA. 2016. Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis. Geneva: World Health Organization.
- de Souza RJ, et al. 2015. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 2015;351:h3978 | doi: 10.1136/bmj.h3978.
- Gayet-Boyer C, et al. 2014. Is there a linear relationship between the dose of ruminant trans-fatty acids and cardiovascular risk markers in healthy subjects: results from a systematic review and meta-regression of randomised clinical trials. *British Journal of*

Nutrition 112:1914–1922.

Jakobsen MU, et al. 2008. Intake of ruminant trans fatty acids and risk of coronary heart disease. *International Journal of Epidemiology* 37:173–182.

Laake I, et al. 2012. A prospective study of intake of trans-fatty acids from ruminant fat, partially hydrogenated vegetable oils, and marine oils and mortality from CVD. *British Journal of Nutrition* 108:743–754.

Wanders, AJ et al. 2017. Trans Fat Intake and Its Dietary Sources in General Populations Worldwide: A Systematic Review. *Nutrients* 2017, 9, 840; doi:10.3390/nu90808.

Willett W and Mozaffarian D. 2008. Ruminant or industrial sources of trans fatty acids: public health issue or food label skirmish? *Am J Clin Nutr* 87:515-6.

World Health Organization, 2018. Draft guidelines on saturated fatty acid and trans-fatty acid intake for adults and children.

#### Recommendations (for trans-fatty acids only)

The WHO's Draft Guidelines indicates that they recommend the following:

"In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake, WHO recommends reducing trans-fatty acid intake (strong recommendation).

In adults and children, WHO suggests reducing the intake of trans-fatty acids to less than 1% of total energy intake (conditional recommendation)."

The WHO's recommendation to limit total trans-fat (i.e. collective ruminant and industrial) intake, rather than industrial trans-fat only, conflicts with the evidence WHO provides. The quality of evidence regarding total trans-fatty acid intake and health outcomes for each of the draft DWG recommendations ranges in quality from very low to moderate, indicating that further research is "likely" to "very likely" to have an important impact on the confidence in the estimate of effect and may likely change the estimate (WHO, 2012). In the case of "very low" evidence quality, "any estimate of effect is very uncertain." (WHO, 2012) The current draft recommendations do not discuss the evidence quality for ruminant trans-fat. Regardless, in situations where evidence is insufficient, as indicated is the case for ruminant trans-fatty acids based on the conclusions of de Souza et al. (2015) and Brouwer et al. (2016) [see Evidence Summary comments above], the WHO Handbook for Guideline Development (further referred to as The Handbook, 2012, Pg 47) indicates that the option not to make a recommendation is viable. The Handbook specifies that when there is a lack of evidence, their conclusive recommendation can be highlighted by stating: "No recommendation can be made because of insufficient evidence". While The Handbook recognizes that some situations may necessitate WHO guidance despite limited or no evidence, it is unclear if this is the situation for total trans-fatty acids and, in particular, ruminant trans-fatty acids.

In fact, it has been indicated that advice to limit saturated fat intake is likely sufficient to curtail ruminant trans-fat intake without a specific recommendation that collectively recommends a limitation of all trans-fat, both ruminant and industrial. Specifically, Brouwer et al. (2013) note that "The proportion of ruminant trans fatty acids in foods is low and will further decrease if consumers follow the advice to decreased intake of saturated fatty acids. Therefore, ruminant trans fatty acids are not an urgent research topic either." Similarly, Wanders et al. (2017) conclude, "The intake of ruminant trans-fat is therefore not seen as a major dietary problem for public health. The most important sources of animal trans-fat are full-fat dairy products and high fat meats. Current food-based dietary guidelines advise to reduce saturated fat intake by limiting the intake of full-fat dairy products and high-fat meats. Adhering to these guidelines will also reduce the intake of animal trans-fat."

A recommendation to reduce trans-fatty acid intake that includes ruminant trans-fat is unprecedented among world-wide interventions to limit trans-fat intake and could result in difficulties with labeling regulations between countries that have adopted legislation based on industrial trans-fatty acids only, versus those that adopt regulations based on the current draft Guidance, or those that strictly prohibit trans-fat labelling, such as the European Union (WHO EU Report, 2015). Similarly, food-based dietary guidelines also tend to exclude ruminant trans fats from recommendations to limit trans-fat intake. For example, the current U.S. Dietary Guidelines (USDA and HHS, 2015), recommend limiting industrial but not ruminant trans-fatty acids, due to the small levels of ruminant trans-fatty acids in the diet.

#### References:

Brouwer, IA. 2016. Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis. Geneva: World Health Organization.

Brouwer, IA, et al. 2013. Trans fatty acids and cardiovascular health: research completed? *European Journal of Clinical Nutrition* 67:541–547.

de Souza RJ, et al. 2015. Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 2015;351:h3978 | doi: 10.1136/bmj.h3978.

U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015–2020 Dietary Guidelines for Americans. 8th Edition. December 2015. Available at <http://health.gov/dietaryguidelines/2015/guidelines>.

Wanders, AJ et al. 2017. Trans Fat Intake and Its Dietary Sources in General Populations Worldwide: A Systematic Review. *Nutrients* 2017, 9, 840; doi:10.3390/nu90808.

World Health Organization, Regional Office for Europe, 2015. Eliminating trans fats in Europe: A policy brief. Copenhagen: World Health Organization.

World Health Organization, 2012. WHO Handbook for Guideline Development. Geneva: World Health Organization.

Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
<p>Annex 7 of the WHO Draft Guidelines provides a summary of factors considered for determining the strength of the draft trans-fatty acid recommendations. One factor considered is the “trade-off between benefits and harms” for which the draft Guidelines indicate there are “no known adverse effects on health have been documented.” It is unclear, however, based on this brief description if the unintended nutritional consequences of a recommendation to limit all trans-fats (both ruminant and industrial) coupled with a recommendation to limit all saturated fatty acids, was considered. U.S. data indicates that while cheese, beef and milk provide approximately one-third of the saturated fat in the American diet, they also contribute 50% of vitamin D, 46% of calcium, 42% of vitamin B12 and 12% of the heart healthy nutrient, potassium, as well as many other nutrients to the diets of Americans. More specifically, an evaluation of NHANES data on beef intake indicates that total beef consumption (not just lean beef) contributes 10% or less of total and saturated fat in the U.S. diet, yet provides significantly to intake of protein (including all of the essential amino acids) and other key nutrients such as vitamins B6 and B12, niacin, zinc, phosphorus, potassium and iron by U.S. adults. It would be helpful for WHO to clarify in the final Guidelines if consideration of nutrient insufficiencies and deficiencies resulting from the draft recommendations will be considered.</p> <p>References:  Huth PJ, et al. Major food sources of calories, added sugars, and saturated fat and their contribution to essential nutrient intakes in the U.S. diet: data from the National Health and Nutrition Examination Survey (2003-2006). <i>Nutr J</i> 2013;12:116.  Zanovec M, et al. Lean beef contributes significant amounts of key nutrients to the diets of US adults: National Health and Nutrition Examination Survey 1999-2004. <i>Nutr Res</i> 2010;30:375-81.</p>
Additional comments

## Final comments

Please provide any final thoughts or comments below.
A recommendation to reduce trans-fatty acid intake that includes ruminant trans-fat is unprecedented among world-wide interventions to limit trans-fat intake and lacks strong scientific evidence to support the recommendation.

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## Survey response 48

### General information

Family/last name
Astrup
Given/first name
Arne
Organization/affiliation
University of Copenhagen
Sector
Academic/research
Sector [Other]
Country
Denmark

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)

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#### Summary of evidence (for saturated fatty acids only)

The recommendation to reduce the intake of saturated fatty acids (SAT), and replace them with polyunsaturated fat (PUFA) and monounsaturated fat (MUFA) in order to reduce the incidence and mortality of cardiovascular disease (CVD) needs to take recent evidence showing that the health effects of SAT depend to a large extent on the specific food source of SAT into consideration. The WHO draft report does acknowledge that "...emerging evidence has led to the suggestion that different saturated fatty acid-containing foods, such as dairy foods, may have different effects on risk of cardiovascular diseases and type 2 diabetes, either as a result of differing composition of saturated fatty acids across foods, other constituents of the foods, or a combination of the two (38-42). However, many questions remain to be answered before a clear understanding can be reached and firm conclusions drawn." and that "... there is a need for more studies comparing the effects of saturated fatty acids from different food sources (e.g. plant, animal and dairy) on CVD and mortality".

Recommending policy without sound evidence is dangerous. We draw attention to the substantial existing research on this topic. The WHO recommendations cannot and must not ignore the meaningful evidence that suggests neutral or even beneficial effects of various foods containing SAT in relation to CVD risk as well as other health outcomes. Crude recommendations to reduce the intake of total SAT without considering specific fatty acids and food sources may cause member states, industry, and the consumer to make harmful decisions that reduce the intake of nutrient-dense foods that are actually beneficial for reducing the risk of CVD, type 2 diabetes and other serious non-communicable diseases. Such a recommendation will work against the intention of the WHO and weaken the impact of the recommendations on the incidence and mortality of important diseases.

Today there is substantial evidence to show that individual SAT fatty acids have differential effects on blood lipids and lipoproteins and relationships with CVD, and the food matrix in which SAT exists is a stronger determinant of health effects than the total SAT content. Among different fatty acids, medium-chain SAT, odd-chain SAT (predominantly from dairy fat), and very long-chain SAT, each appear to be metabolically beneficial, whereas different long-chain SAT (e.g. containing 14 to 18 carbons) have very different relationships with blood lipids and lipoproteins. Furthermore, many of the most important food sources of SAT, such as cheese, butter, whole fat milk, eggs, and unprocessed red meats, are neutral with respect to CVD events, whereas cheese is also linked to lower risk of type 2 diabetes. Other SAT food sources, such as yogurt and perhaps cocoa, are linked to lower risk of cardiovascular disease, weight gain, and diabetes. For these foods, it makes no more sense to recommend their reduction and replacement with unsaturated fat than to recommend total carbohydrate from all sources be reduced and replaced with unsaturated fat. Among major SAT food sources, only processed meats (preserved by salt, curing, or other preservatives) are consistently linked to higher risk of CVD. Because SAT content is generally similar in unprocessed vs. processed meats, this suggests harms of preservatives and cooking methods of processed meats, not harms of SAT. The major focus and recommendations of the WHO should be on reducing processed meats, which are strongly linked to CVD and diabetes.

As yet, the forms of evidence provided are food-based meta-analyses of prospective observational studies, randomized controlled trials with well-established surrogate end-points, and experimental human mechanistic studies. This totality of evidence has demonstrated that the food matrix is more important for health effects than single ingredients. The general advice to reduce the intake of SAT, without pointing at specific foods sources, is very likely to be counterproductive as consumers who aim at reducing SAT in order to reduce CVD risk may well replace some of these nutrient-dense foods and choose less healthy alternatives such as low-fat but otherwise sugar-rich foods. We strongly recommend a more food based translation of the recommendations on how to achieve the reduction in SAT intake. This will avoid the unnecessary reduction or exclusion of foods such as eggs, dark chocolate, cheese, and other fermented dairy, which are key sources of important nutrients such as high quality protein, calcium etc., and also have known health benefits if not consumed in unreasonable amounts. This has been recently extensively reviewed with regard to dairy foods (1).

We acknowledge that there is a lack of RCT's to show the effect these foods have on hard CVD end-points and mortality. However, in the absence of such trials WHO needs to consider the existing comprehensive evidence. To recommend that the general population reduce intake of specific foods without evidence to substantiate that this will improve health is inconsistent with the principle of "do no harm". Governments are responsible for delivering population-directed dietary advice, which must generate benefit for most, and minimal collateral hazard for even very small subgroups, unless they can be identified and guided separately.

#### Recommendations (for saturated fatty acids only)

Remarks (for saturated fatty acids only)
<p>How robust is the evidence linking saturated fat to cardiovascular disease?</p> <p>1. Evidence from randomized controlled trials with hard end-points</p> <p>The evidence to show that SAT is more harmful than PUFA and MUFA has weakened over recent years, and has been challenged by several meta-analyses of observational studies and RCT's. Even the most positive Cochrane analysis of RCT's from 2015 (the key WHO reference, Hooper et al. (2)) could not find any significant effect of high vs low SAT on 1) total mortality, 2) Cardiovascular mortality 3) Myocardial infarction 4) Non-fatal myocardial Infarctions 5) Stroke 6) Coronary Heart Disease events, 7) Coronary Heart Disease mortality. Only 8) The composite end-point "Combined cardiovascular events", provided a significant result [RR 0.83 (0.72 to 0.96)].</p> <p>A 2017 meta-analysis of RCT's by Hamley assessed the effect of replacement of SAT with mostly n-6 PUFA with an aim to reducing the risk of coronary heart disease (3). They found, on the basis of results from the adequately controlled trials, that there was no effect for major CHD events (RR = 1.06, CI = 0.86–1.31), total CHD events (RR = 1.02, CI = 0.84–1.23), CHD mortality (RR = 1.13, CI = 0.91–1.40) and total mortality (RR = 1.07, CI = 0.90–1.26). The scientists concluded: "Available evidence from adequately controlled randomised controlled trials suggests replacing SAT with mostly n-6 PUFA is unlikely to reduce CHD events, CHD mortality or total mortality. The suggestion of benefits reported in earlier meta-analyses is due to the inclusion of inadequately controlled trials. These findings have implications for current dietary recommendations."</p> <p>This analysis is consistent with the meta-analyses by Schwingshackl et al. (2013) and Ramsden et al. (2016), both of which found that replacement of saturated fat with linoleic acid did not lower risk of death from coronary heart disease or all causes (4,5).</p> <p>2. Evidence from randomized controlled trials with surrogate end-points</p> <p>The WHO report excludes observational studies, and puts weight on a meta-analysis of RCT's that have assessed surrogate end-points such as serum lipids and lipoproteins, including total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglyceride, LDL cholesterol to HDL cholesterol ratio, total cholesterol to HDL cholesterol ratio, triglyceride to HDL cholesterol ratio, apolipoprotein A-I (ApoA-I) and apolipoprotein B (ApoB)total-cholesterol (6). The main findings are that replacement of SAT with PUFA results in small but significant reductions in total cholesterol, LDL cholesterol, in HDL cholesterol, triglycerides, and in total cholesterol to HDL cholesterol ratio, and LDL cholesterol to HDL cholesterol ratio of 0.034.</p> <p>However, it is unclear if these changes in serum lipoproteins translate into a reduction in cardiovascular end-points and mortality. There is increasing recognition of the limitation of using LDL-cholesterol concentration as a marker of diet effects on CVD risk, as atherogenicity of the LDL-particle is also determined by resistance against oxidation, size, composition and cytotoxicity. There is evidence that SAT increases the LDL-particle size, and thus potentially reduces CVD risk (7, 8).</p> <p>The weakness of serum LDL-cholesterol concentration and ratio to reflect changes in CVD risk following dietary interventions have been highlighted by RCT's showing that replacement of SAT with PUFA in the diet effectively lowers serum cholesterol but does not translate into lower risk of death from coronary heart disease or all causes(5). Therefore, caution should be applied when interpreting the findings of the Mensink meta-analysis as supporting evidence for a beneficial effect of replacement of SAT with PUFA (6). In 2010 a consensus panel concluded that "Single risk factors have limitations when considered on their own because the effects of diet on CVD risk are mediated by many pathways, with blood lipids being only one. Although elevated LDL cholesterol is one of the major risk factors known, there is still a need for clinical endpoints for assessing the effects of diet on CVD risk." (9).</p> <p>3. Evidence from observational studies</p> <p>WHO does not assess the evidence derived from meta-analyses of prospective cohort studies, arguing that the quality of evidence for relevant outcomes is lower than in the analyses of RCTs, and that it was not possible to assess potential differential effects of replacing SAT with different nutrients in the meta-analysis of the cohort studies. However, in the assessment of totality of evidence all types of evidence need to be considered, and to exclude high-quality observational studies is a breach of the traditions of evidence-based medicine.</p> <p>Indeed, observational studies are considered very valuable for assessing the effect of SAT on CVD end-points, and particularly for the food-based analyses (see below). To summarize the outcome of the meta-analysis mentioned in the WHO report (10), they found that SAT intake is not associated with all cause mortality, CVD, CHD, ischemic stroke, or type 2 diabetes, and concluded that "dietary guidelines must carefully consider the health effects of recommendations for alternative macronutrients to replace trans fats and saturated fats". This is in line with the analysis by Siri-Tarino et al. (2010) and Harcombe Z et al. (2017) that showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD (11,12).</p> <p>4. Evidence from food-based analyses of cardiovascular disease risks</p> <p>In 2010 a consensus panel of experts concluded that: "There is increasing evidence to support that the total matrix of a food is more important than just its fatty acid content when predicting the effect of a food on CHD risk, e.g., the effect of SFAs from cheese on blood lipids and CHD may be counterbalanced by the content of protein, calcium, or other components in cheese. In addition, the special fatty acid profile (rumenic acid, trans vaccenic acid, and short-chain fatty acids) may modify the effect on CHD risk. Another example is dark chocolate, which has a high content of stearic acid, oleic acid, and polyphenols, and observational studies, mechanistic studies, and RCTs show that dark chocolate reduces risk factors of CVD." (9). Since then several food-based studies have been conducted, and today a number of meta-analyses have assessed the health effects of various foods containing significant amounts of SAT and concluded that future recommendations should be food based (1).</p>

#### 4.1 Butter

Butter is the most SAT dense food and a meta-analysis by Pimpin et al. (2016) assessed observational studies linking butter intake to major disease outcomes and mortality (13). Based on 9 publications, together reporting on 636,151 unique participants with 6.5 million person-years of follow-up, and including 28,271 total deaths, 9,783 cases of incident cardiovascular disease, and 23,954 cases of incident diabetes, they found that butter consumption was weakly associated with all-cause mortality (N = 9 country-specific cohorts; per 14g (1 tablespoon)/day: RR = 1.01, 95%CI = 1.00, 1.03, P = 0.045), but was not associated with cardiovascular disease (N = 4; RR = 1.00, 95%CI = 0.98, 1.02; P = 0.704), coronary heart disease (N = 3; RR = 0.99, 95%CI = 0.96, 1.03; P = 0.537), or stroke (N = 3; RR = 1.01, 95%CI = 0.98, 1.03; P = 0.737), and was inversely associated with incidence of diabetes (N = 11; RR = 0.96, 95%CI = 0.93, 0.99; P = 0.021).

#### 4.2 Eggs

Egg contains ~2.6 grams SAT/100 grams, and can easily be a significant contributor to daily SAT intake. However, eggs are also nutrient-dense foods that provide a wide range of important nutrients that are limitedly available in many other foods. High quality prospective, population based studies and a number of meta-analyses of prospective studies have found that egg consumption is not associated with risk of coronary heart disease, but with a lower risk of stroke, though subgroup analyses found an increase of CHD risk in diabetic populations (14-16). More recent studies have shown that the apparent increased risk of CVD among patients with type 2 diabetes is most likely due to lack of adjustment for confounding (17). Evidence from RCT's has shown neutral or beneficial effects on markers of diabetes and cardiovascular disease. For example, Fuller et al. compared the effects of an high-egg diet (12 eggs/wk) with a low-egg diet (

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for saturated fatty acids only)



Additional comments
<p>This is responsive is a collaborative work. Alle participants have registered on the WHO site and all have uploaded declaration of conflicts of interest forms.</p> <p>The authors:</p> <p>Arne Astrup, MD, DMSc Head of Department, Professor University of Copenhagen, Denmark</p> <p>Jean-Philippe Bonjour, M.D. Honorary Professor of Medicine Former Head Division of Bone Diseases Geneva University Hospitals &amp; Faculty of Medicine Switzerland</p> <p>Lisette CPGM de Groot, PhD Professor Nutrition and Ageing Wageningen University, The Netherlands</p> <p>Marcia C. de Oliveira Otto, PhD, FAHA Assistant Professor University of Texas, Houston, USA</p> <p>Emma Feeney, PhD Postdoctoral Research Fellow UCD Institute of Food and Health, Dublin, Ireland</p> <p>Manohar Garg, PhD, MND, MSc (Biochem), BSc (Hons), RNutr Professor, Director, Nutraceuticals Research Program University of Newcastle, Callaghan, Australia</p> <p>Ian Givens, PhD Professor of Food Chain Nutrition University of Reading, UK</p> <p>Ronald M. Krauss, MD Senior Scientist and Director Childrens Hospital Oakland Research Institute &amp; UCSF Benioff Childrens Hospital Oakland, USA</p> <p>Benoît Lamarche, PhD ,FAHA Chair of Nutrition Institute of Nutrition and Functional Foods Université Laval, Québec, Canada</p> <p>Jean-Michel Lecerf, MD Chef du Service de Nutrition &amp; Activité Physique Institut Pasteur de Lille, France</p> <p>Philippe Legrand Professor Laboratoire de Biochimie-Nutrition, Agrocampus-INRA, Rennes, France</p> <p>Michelle McKinley R.Nutr. Public Health Reader Queens University Belfast, UK</p> <p>Marie-Caroline Michalski, PhD Research Director CarMeN laboratory, INRA UMR1397/INSERM U1060, Villeurbanne, France</p> <p>Dariusz Mozaffarian, MD, DrPH Dean, Jean Mayer Professor of Nutrition and Medicine Friedman School of Nutrition Science &amp; Policy, Tufts University, USA</p> <p>Sabita Soedamah-Muthu, PhD</p>

## *Trans*-fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 49

### General information

Family/last name	Sanders
Given/first name	Thomas
Organization/affiliation	Kings College London
Sector	Academic/research
Sector [Other]	
Country	United Kingdom

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>The executive summary states that the intake of CVD is correlated with saturated fatty acid intake. This is not supported by the prospective cohort studies whereas it is for trans fatty acids. Historical data suggest that diets with a high in saturated fatty acids and low in polyunsaturated fatty acids were associated with elevated serum cholesterol and increased risk of CHD. Stroke has never been associated with saturated fatty acid. Stroke is more prevalent than CHD in Asia.</p> <p>The conclusions regarding replacement of saturated fatty acids by wholegrain as reducing risk is questionable when generalised to the global population especially Asia. Rice, maize and wheat are the major cereals but the wholegrain versions do not affect serum cholesterol. However, wholegrain oats, barley and rye do lower cholesterol because of their soluble fibre content. The meta-regression study that show a favourable effect of replacing SFA with wholegrain are confounded by other changes in diet that may be linked to a healthier life-style e.g. adherence to a "prudent diet" or vegetarian diet.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
<p>The guidelines focus entirely on human data. However, there is an overwhelming body of evidence to show that hyperlipidemia induced by diets high in saturated fats and cholesterol promote atherosclerosis -surely this should be considered.</p>
Summary of evidence (for saturated fatty acids only)
<p>The inclusion of trials using extremely high intakes of PUFA (e.g. &gt;15% energy) in the meta-analysis is not relevant as the intake are above recommended intakes. The data from the RCTs are not convincing if these studies are excluded. It needs to be recognised that the existing data is heterogeneous and the studies conducted were too small to detect effects. More weight should be placed on evidence from cohort studies.</p>

Recommendations (for saturated fatty acids only)
There is no clear basis for setting the recommendation at 10% energy. CHD has fallen sharply in North America, Australia and West Europe but intakes are closer to 11-12% energy. While the early studies in the 1960s -70s indicated that intakes were in the region of 20% energy, this level of intake is uncommon and the range is between 9-15% in Europe.
The proposal to replace saturated fatty acids with polyunsaturated fatty acids in many European countries would result in further increasing the intake of polyunsaturated fatty acids from on average 6% energy which may not be desirable. It needs to be recognised that only a limited amount of saturated fatty acids can be replaced by polyunsaturated fatty acids. There is evidence to indicate that the use of vegetable oils high in monounsaturated fatty acids as part of a Mediterranean diet is associated with lower CVD events.
Remarks (for saturated fatty acids only)
The recommendation does not address the issue whether all saturated fatty acids should be treated similarly especially as short and medium chain (C2-C10) fatty acids have no effect on cholesterol and stearic acid appears neutral. Some guidelines have suggest these non-cholesterol raising fatty acids be excluded from the target. Depending on the diet, the contribution of these cholesterol neutral to saturated fatty acid intake may account for 25% of the saturated fatty acid intake.
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
It is unlikely that primary prevention trials of dietary modification will be conducted owing to the large costs, the enormous numbers of participants required to conduct a trial of sufficient power and the difficulty of maintaining participants on an experimental diet for many years.
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

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Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

### Final comments

Please provide any final thoughts or comments below.

## Survey response 50

### General information

Family/last name
González Céspedes
Given/first name
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Sector
Government
Sector [Other]
Country
Paraguay

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
the reviews provide the necessary evidence to support the recommendations made in the guide

## Survey response 53

### General information

Family/last name
Eilat-Adar
Given/first name
Sigal
Organization/affiliation
The Academic College at Wingate
Sector
Academic/research
Sector [Other]
Country
Israel

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Page 16: 1. The leading cause for death in Israel since the 90s is cancer ( <a href="https://www.health.gov.il/PublicationsFiles/Leading_Causes_2015.pdf">https://www.health.gov.il/PublicationsFiles/Leading_Causes_2015.pdf</a> Accessed May 22nd 2018 (Hebrew)). 2. Since 2012 partially hydrogenated margarine for private consumption is rare in Israel. 3. Since 2014 it is obligatory to include trans fatty acids (TFA) on every packed product with 2% fat and more. A level
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
1. Me on behalf of the other Israeli advisory board want to draw into attention the fact that it is not mentioned how many trials and how many people were analyzed in the meta-analyses. 2. I couldnt find any comment referred to the PURE study and we suggest to mention some of its limitations (Prospective Urban Rural Epidemiology (PURE) study investigators. Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. Lancet. 2017 Nov 4;390(10107):2050-2062. doi: 10.1016/S0140-6736(17)32252-3. Epub 2017 Aug 29. ).
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)



Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
The Israeli committee thinks that it should be suggests to mention that trans fatty acids are replaced by interesterification. However, the influence of this process is still unknown (Sloop GD1, Weidman JJ2, St Cyr JA3.Perspective: interesterified triglycerides, the recent increase in deaths from heart disease, and elevated blood viscosity. Ther Adv Cardiovasc Dis. 2018 Jan;12(1):23-28. doi: 10.1177/1753944717745507.)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
This is a very detailed and important position paper and I (we) agree with its recommendations. The table of data contribute a lot to understanding the data. What I added are a few minor comments.

## Survey response 54

### General information

Family/last name
Das
Given/first name
VG Kumar
Organization/affiliation
Academician Emeritus
Sector
Non-governmental agency
Sector [Other]
Country
Malaysia

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
One should differentiate between sat fat from animal and plant sources. Animal food sources like butter, beef tallow and pork lard contain 40 - >50% C16:0 and C18:0, whereas sat fats in plant oils range from C6:0 to C18:0. Also with respect to CVD risk, one should also take into consideration the metabolic differences between medium-chain fats (C6 to C12) and long-chain saturated fats (C14:0 to C18:0). In addition, the positional distribution of fatty acids should be considered, as evidence shows that palm oil behaves comparatively like olive oil in regards to CVD risks, explained by the sn-2 hypothesis (Fattore et al., 2014; Sun et al 2015).
Recommendations (for saturated fatty acids only)
I wish to refer to the results of the meta analysis by Mensink (2016 (attached) Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis : Results of the multiple regression analysis indicated that effects on the serum lipoprotein profile of reducing SFA intake by replacing a mixture of SFA with cis-PUFA (predominantly linoleic acid and $\alpha$ -linolenic acid) or cis-MUFA (predominantly oleic acid) were more favourable than replacing SFA with a mixture of carbohydrates. Mensink's conclusion remains relevant nutrition-wise.

Remarks (for saturated fatty acids only)
In this context we must take cognizance that fatty acids are not randomly distributed among the three positions, sn-1, 2,3 of the triglyceride molecules (TG). This is an important consideration in that these TG molecules are subject to enzyme reactions in the stomach and intestine. This is especially the case with some plant oils where the sn-2 position of the triglyceride is dominantly occupied by oleic acid. Cocoa butter which is ca. 67% saturated is a prime example of this, as also palm oil. Digestion of such dietary triglycerides in the intestinal lumen selectively cleaves the fatty acids from the sn-1 and sn-3 positions to yield 2-monooleoyl glycerol (2-MAG), all of which enter the enterocytes where TAG is reformed from the 2-MAG precursor. This position effect of oleic acid, which predominates also in the fully unsaturated olive oil, has to be taken cognizance of the analysis of RCTs on "saturated fats"
Recently Dr Welma Stonehouse of CSIRO reported that cocoa butter (67% sat.) palm olein (ca. 40% sat) and virgin olive oil (ca. 15% sat) induced similar lipid profiles (abstract enclosed)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
RCTs on the long-term consumption of SFAs on circulating LDL-C morphology and distribution, to be conducted on populations from different geographical regions, and assessment of their effects on risk of cardiovascular diseases and stroke".
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

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## Final comments

Please provide any final thoughts or comments below.

## Survey response 56

### General information

Family/last name
Dombrow
Given/first name
Carol
Organization/affiliation
Heart & Stroke Foundation of Canada
Sector
Non-governmental agency
Sector [Other]
Country
Canada

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Heart & Stroke Foundation of Canada recently published a Saturated Fat Position Statement. I would like to highlight a couple of key messages that I did not see in the guidelines. In addition here is a link to the Position Statement for your reference- <a href="http://www.heartandstroke.ca/-/media/pdf-files/canada/2017-position-statements/saturatedfat-ps-eng">http://www.heartandstroke.ca/-/media/pdf-files/canada/2017-position-statements/saturatedfat-ps-eng</a>
Key Messages
To eat a healthy, balanced diet that consists of a variety of natural/whole and minimally processed foods.
Both the quality and quantity of fats you eat matters.
-Highly processed foods are a major source of saturated fat, and are also usually high in calories, sodium, sugar and sometimes trans fat.
-There is emerging evidence that it is not just the fat itself, but the type of food that fat is found in that can affect cardiovascular health.

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments
Thank you for the opportunity to share the Heart & Strokes Saturated Fat Position paper.

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 57

### General information

Family/last name
Kalergis
Given/first name
Maria
Organization/affiliation
Dairy Farmers of Canada
Sector
Private sector
Sector [Other]
Country
Canada

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
The Global Burden of Disease study 2016 (ie- reference # 1) is cited to make the case that cardiovascular diseases (CVDs) were leading causes of mortality in 2016 and several modifiable risk factors were identified including an unhealthy diet. However, this same study did not include saturated fat as a dietary risk factor amongst those listed. As well, the WHO commissioned systematic review by de Souza et al 2015 indicated that "This systematic review and meta-analysis of evidence from large generally well designed observational studies does not support a robust association of saturated fats with all-cause mortality, CHD, CHD mortality, ischemic stroke, or diabetes in healthy individuals."
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
No specific comment
Background (same for saturated fatty acids and trans-fatty acids)
<p>On page 17, the evidence for considering the food source of saturated fat, including that from dairy foods, is described as "emerging" and 5 references are cited in support of this comments (ie- references #38-42). However, this is not an accurate reflection of the vast and robust evidence that is currently available that indicates that dairy fat and higher fat dairy foods, including cheese, show either neutral or beneficial associations with cardiometabolic outcomes. This is well highlighted in the Systematic Review of Drouin-Chartier et al 2016 which consisted of 21 meta-analyses and found that dairy fat as well as various forms of dairy products (including higher fat dairy products) shows either favorable or neutral associations with cardiovascular-related clinical outcomes including: CVD, CHD, stroke, metabolic syndrome, hypertension and type 2 diabetes.</p> <p>Data with respect to cheese specifically indicates that cheese is not associated with an increased risk of CVD and is associated with a reduced risk of stroke and type 2 diabetes.</p> <p>On page 17, it is indicated that LDL cholesterol is a well-established surrogate endpoint for measuring the effects of interventions on CVD risk. However, according to the 2010 Institute of Medicine (IOM) report on biomarkers and surrogate endpoints in chronic disease, the effect of a food on LDL may not predict CVD risk.</p>
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Consideration should be given to other well conducted systematic reviews that were not commissioned by WHO in order to ensure other perspectives and reduce "perception of bias"

Summary of evidence (for saturated fatty acids only)
<p>While the recommendations are primarily based on the systematic review of RCTs by Hooper et al 2015 and the Meta-Regression analysis of Mensink 2016, data from systematic reviews and meta-analyses of prospective cohort studies, including the WHO commissioned systematic review (de Souza et al 2015), should also be considered as these have consistently found no association with higher intakes of SFA relative to lower intakes and increased risk of CVD-related outcomes amongst free-living individuals. While data derived from RCTs provides a high level of evidence, these also have important limitations especially several of the studies that are included in Hooper et al 2015 which have been conducted decades ago and have important design issues as outlined in the meta-analysis by Hamley 2017.</p> <p>There seems to be equal weight given to blood lipid markers (esp. LDL-C) and hard clinical outcomes (eg. CVD, CHD, stroke, etc.). This is not appropriate as blood lipid markers are only risk factors and only LDL-C is currently considered as a “surrogate marker” for CVD risk. However, even LDL-C is being questioned as a reliable surrogate endpoint for assessing the impact of food on CVD as outlined in the 2010 report by the Institute of Medicine (IOM 2010).</p> <p>The evidence with respect to the food source of saturated fat should also be considered as there is vast and robust evidence that dairy fat and higher fat dairy foods are not associated with increased CVD risk but may in fact be associated with benefits in this regard. This is well highlighted in the Systematic Review of Drouin-Chartier et al 2016 which consisted of 21 meta-analyses and found that dairy fat as well as various forms of dairy products (including higher fat dairy products) shows either favorable or neutral associations with cardiovascular-related clinical outcomes including: CVD, CHD, stroke, metabolic syndrome, hypertension and type 2 diabetes.</p> <p>The results from The PURE cohort study of 18 countries (Dehghan et al 2017) should also be considered. This study highlighted that a “high carbohydrate intake was associated with higher risk of total mortality, whereas total fat and individual types of fat were related to lower total mortality. Total fat and types of fat were not associated with cardiovascular disease, myocardial infarction, or cardiovascular disease mortality, whereas saturated fat had an inverse association with stroke. Global dietary guidelines should be reconsidered in light of these findings.”</p>
Recommendations (for saturated fatty acids only)
<p>The food source of saturated fat should be considered as there is vast and robust evidence that dairy fat and higher fat dairy foods are not associated with increased CVD risk and in fact are associated with benefits in this regard. This is well highlighted in the Systematic Review of Drouin-Chartier et al 2016 which consisted of 21 meta-analyses and found that dairy fat as well as various forms of dairy products (including higher fat dairy products) shows either favorable or neutral associations with cardiovascular-related clinical outcomes including: CVD, CHD, stroke, metabolic syndrome, hypertension and type 2 diabetes.</p> <p>With respect to the recommendation to replace SFA with PUFA, the findings from 2 recently published meta-analyses of RCTs should be considered (Hamley 2017; Hannon et al 2017). These meta-analyses question the benefits of replacing SFA with PUFA.</p>
Remarks (for saturated fatty acids only)
<p>Targeting SFA is not justified by the totality of available scientific evidence including the findings from the Global Burden of Disease Study 2016 which does not identify SFA as one of the “dietary risk factors” for disease burden.</p> <p>The food source of SFA is important to consider as SFA derived from dairy products has been shown to be neutral or beneficial with regards to CVD-related outcomes.</p> <p>The evidence for replacement of SFA by PUFA is not currently consistent or conclusive. Therefore, any recommendation to such an effect should not be made.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
<p>Recommendations should not be made where there is uncertainty that benefit will outweigh the risk and where there may still be “substantial debate” (ie- as per the conditional recommendations).</p> <p>As currently stated, there is a high risk that the “conditional recommendations” which include reducing SFA to &lt; 10% of energy and replacing SFA with PUFA, will be seen as being similar to “strong recommendations” and interpreted accordingly by policy makers. This distinction and what it means should be made more clear as it is currently only in the very fine print of the footnotes (ie- page 8 footnotes) as follows:</p> <p>“Conditional recommendations are those recommendations for which the WHO guideline development group is uncertain that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders.”</p>
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
<p>Several research needs that are identified involve RCTs with clinical outcomes. This type of design is not very feasible and highly unrealistic.</p>



## Annexes 1, 6, 7 (for saturated fatty acids only)

### Additional comments

The totality of the scientific evidence, including findings from the Global Burden of Disease study and the WHO commissioned systematic review of de Souza et al 2015, does not justify targeting SFA as a nutrient of concern. Furthermore, the food source of SFA is important to consider as there is a very large, robust body of scientific evidence that shows that dairy fat and higher fat dairy foods including cheese are neutral or beneficial for several cardiometabolic outcomes. Moreover, the evidence on replacement of SFA by PUFA is inconsistent and inconclusive. Finally, there is risk that the conditional recommendations will be misinterpreted and misapplied and recommendations to carry out RCTs with hard clinical end points are not feasible and very unrealistic and unlikely to occur.

### References:

de Souza RJ et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 2015;351:h3978.

Dehghan M et al. Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. *The Lancet*. [http://dx.doi.org/10.1016/S0140-6736\(17\)32241-9](http://dx.doi.org/10.1016/S0140-6736(17)32241-9)

Drouin-Chartier, JP et al. Systematic Review of the Association between Dairy Product Consumption and Risk of Cardiovascular-Related Clinical Outcomes. *Adv Nutr* 2016;7:1026–40.

GBD 2016 Causes of Death Collaborators. Global, regional, and national age-sex specific mortality for 264 causes of death, 1980–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet*. 2017;390(10100):1151–210.

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;16:30.

Hannon BA et al. Clinical outcomes of dietary replacement of saturated fatty acids with unsaturated fat sources in adults with overweight and obesity: a systematic review and meta-analysis of randomized control trials. *Ann Nutr Metab* 2017;71:107–117.

Hooper L et al. Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database Syst Rev*. 2015; 6:CD011737.

Institute of Medicine of the National Academies. Evaluation of Biomarkers and Surrogate Endpoints in Chronic Disease. May 2010.

## Trans-fatty acids

### Executive summary (same for saturated fatty acids and trans-fatty acids)

The findings from the WHO commissioned systematic review by de Souza et al 2015 are not cited. The findings of this review were as follows:

o Industrial, but not ruminant, trans fats were associated with CHD mortality and CHD.

o Ruminant trans-palmitoleic acid, considered as a biomarker of dairy intake, was inversely associated with type 2 diabetes. This finding is said to be “quite consistent and compatible with a 26-54% reduction in risk across an estimated threefold intake range.”

### Scope and purpose (same for saturated fatty acids and trans-fatty acids)

No specific comments.

### Background (same for saturated fatty acids and trans-fatty acids)

The rationale provided for including rTFA is not “evidence-based” (as indicated above) but based on speculation that levels may increase if iTFA is reduced. This is not a good reason and it is very unlikely that rTFA levels would increase to a level similar to iTFA. In fact, as pointed out in one of the included references in the draft guidelines (ref # 34), “if the only source of TFA in the diet is ruminant products, then TFA consumption would drop to less than 2g/day or less than 1% of energy.”

### Guideline development process (same for saturated fatty acids and trans-fatty acids)

No specific comments

Summary of evidence (for trans-fatty acids only)
<p>The WHO commissioned systematic review should be considered. The review found differential association of rTFA vs. iTFA as follows:</p> <ul style="list-style-type: none"> <li>o Industrial, but not ruminant, trans fats were associated with CHD mortality and CHD.</li> <li>o Ruminant trans-palmitoleic acid, considered as a biomarker of dairy intake, was inversely associated with type 2 diabetes. This finding is said to be "quite consistent and compatible with a 26-54% reduction in risk across an estimated threefold intake range."</li> </ul>
Recommendations (for trans-fatty acids only)
The totality of the evidence does not justify inclusion of rTFA with iTFA. While iTFA has consistently been shown to be harmful, this is not the case for rTFA. rTFA has not been shown to be harmful and, in fact, some specific rTFAs have been shown to have health benefits as outlined by a WHO commissioned systematic review (de Souza et al 2015).
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Recommendations should not be made where there is uncertainty that benefit will outweigh the risk and where there may still be "substantial debate" (ie- as per the conditional recommendations). As currently stated, there is high risk that these will be misinterpreted and misapplied.
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Several research needs that are identified involve RCTs with clinical outcomes. This type of design is not very feasible and highly unrealistic.
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
<p>The totality of the scientific evidence, including findings from the Global Burden of Disease study and the WHO commissioned systematic review of de Souza et al 2015, does not justify targeting SFA as a nutrient of concern. Furthermore, the food source of SFA is important to consider as there is a very large, robust body of scientific evidence that shows that dairy fat and higher fat dairy foods including cheese are neutral or beneficial for several cardiometabolic outcomes. Moreover, the evidence on replacement of SFA by PUFA is inconsistent and inconclusive.</p> <p>Also, the totality of the evidence does not justify inclusion of rTFA with iTFA. While iTFA has consistently been shown to be harmful, this is not the case for rTFA. rTFA has not been shown to be harmful and, in fact, some specific rTFAs have been shown to have health benefits as outlined by a WHO commissioned systematic review (de Souza et al 2015).</p> <p>Finally, there is risk that the conditional recommendations will be misinterpreted and misapplied and recommendations to carry out RCTs with hard clinical end points are not feasible and very unrealistic and unlikely to occur.</p>

## Survey response 58

### General information

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Sector [Other]
Country
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### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
We wish to express our concern regarding specific recommendations and statements in the draft WHO guidelines, in particular:  1.❑The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat. 2.❑“Saturated fat” is not the same as “animal fat.” 3.❑Coconut oil is a healthy medium-chain vegetable oil. There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity. The evidence against coconut oil is indirect and invalid. 4.❑The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
To express our concern regarding specific recommendations and statements in the draft WHO guidelines
Background (same for saturated fatty acids and trans-fatty acids)
There are two main groups of dietary fats and oils: vegetable oil and animal fats. Coconut oil is a vegetable oil that is predominantly saturated (92%) with a significant proportion of MCFA (65%), and is very different from animal fats. Animal fats are made up predominantly of long-chain fatty acids with considerable amounts of cholesterol: while coconut oil contains 0 to 3 ppm cholesterol, animal fats contain 1-2% cholesterol. Butter, which is often compared to coconut oil, contains 9% MCFA, 50% saturated LCFA, 3% trans fat and 2 % cholesterol. Therefore, any claim of similarity between coconut oil and butter, pork fat, and beef fat is erroneous. This invalidates comparisons made in most scientific articles regarding coconut oil and animal fat. An often-repeated recommendation is to replace saturated fat with unsaturated fat. This recommendation is based on an animal fat diet and has no relevance to coconut oil. There are several papers that contradict this recommendation. For example, a two-year randomized study comparing consumption of coconut oil with sunflower oil, which is rich in oleic acid and linoleic acid, showed no statistically significant difference in cardiovascular risk. A randomized-crossover dietary study showed that diets that are rich in saturated fatty acids (palm oil and coconut oil) and a diet rich in unsaturated fat (olive oil) gave no significant differences with respect to their effect on selected inflammatory markers. Several systematic reviews and meta-analyses do not support this recommendation.
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Review in the literature and discuss with colleagues

#### Summary of evidence (for saturated fatty acids only)

##### 1. The category of medium-chain fatty acids must be recognized separately from long-chain saturated fat.

Currently, saturated fatty acids (SFA) are considered as only one group. However, to properly account for the different physiological properties of the individual fatty acids, SFA should be further divided into two groups: medium-chain fatty acids (MCFA, C6 to C12) and saturated long-chain fatty acids (LCFA, C14:0 to C18:0). Although FAO recognized the nomenclature of MCFA and LCFA as different sub-classes of saturated fatty acids in its report entitled "Fats and fatty acids in nutrition", the FAO dietary recommendations still treated them as a single group, ignoring its own nomenclature.

In 1980, Bach and Babayan wrote a review where they concluded that MCFA "must be treated separately and differently in our understanding of fats and oils." There is much evidence for the molecular and cellular differences between MCFA and LCFA. For example, MCFAs do not require carnitine to cross the mitochondrial membrane unlike LCFAs which do. , This results in faster uptake of MCFAs into mitochondria. Once inside the mitochondria, MCFAs are more rapidly oxidized than LCFAs. , Unlike LCFAs, MCFAs are not incorporated into membrane phospholipids and are not stored in liver fat , and adipose tissue. These differentiating properties justify the recognition of a separate category of medium-chain fatty acids (C6 to C12) and long-chain saturated fatty acids (C14:0 to C18:0).

Some authors have contested the classification of lauric acid (C12) as a MCFA based on its incomplete absorption to the portal vein. This phenomenon may be explained by the position of C12 on the triglyceride – that is, whether it is in the sn-1/3 or sn-2 position – as this influences the rate at which C12 is released by lipases in the body. , Trilaurin is a triglyceride wherein all of the attached fatty acids are C12. Upon ingestion, C12 in the sn-1 and sn-3 positions are hydrolyzed most rapidly, leaving a C12 in the sn-2 position. This may explain why C12 is not completely channeled towards the portal vein since some C12 may remain bound to the glyceryl group and is brought into the lymphatic system. This distribution effect, however, does not change the cellular properties of C12 as a MCFA. Further, the distribution of MCFAs between the portal vein and lymph depends on the amount and type of fat that is being consumed by the subject in proportion to the total diet. , In coconut oil, over 10% of the triglycerides is trilaurin and in the oil, C12 is equally distributed between the sn-1/3 and sn-2 positions.

Unfortunately, most publications do not distinguish MCFA and LCFA and simply lump all saturated fat into one group. This is erroneous and must be corrected.

##### 2. "Saturated fat" is not the same as "animal fat."

The confusion regarding the use of the categories of "saturated fat" and "animal fat" may have originated from Ancel Keys when he used butter, margarine, and hydrogenated coconut oil to represent "saturated fat" in his early feeding studies. , Keys carried over this confusion in his 1986 Seven Countries Study (SCS) paper where he used the terms "saturated fat" and "animal fat" interchangeably. Although it was clear that it was animal fat that was being consumed, Keys mentioned "saturated fat" repeatedly and "animal fat" only once in the SCS paper. In fact, Keys is mistaken: almost half of the fatty acid composition of animal fat, in particular, beef and pork fat and butter, is unsaturated (see Table). Later, it was determined that the fats that were being consumed in the SCS actually included butter, lard and margarine which are all animal fats; margarine contains industrial trans fats, which are significant predictors of CHD mortality. Therefore, Keys's conclusion that "Death rates were related positively to average percentage of dietary energy from saturated fatty acids" is erroneous: in fact, the fats consumed in the SCS contained considerable amounts of unsaturated fat and trans fat. Thus, research papers which assume that animal fat and saturated fat are similar are also erroneous.

Unfortunately, most publications, including epidemiological surveys and feeding studies, use the terms "saturated fat" and "animal fat" interchangeably without presenting actual fatty acid profiles. Further, when the term "saturated fat" is used, these papers usually mean long-chain saturated fat from animal sources. Therefore, papers that refer to "saturated fat" and/or "animal fat" without an explicit definition of fatty acid profile should not be used in WHO guidelines.

##### 3. Coconut oil is a healthy medium-chain vegetable oil

Coconut oil is unique among the major vegetable oils in that it is made up of about 65% medium-chain fatty acids (MCFA) and 92% total saturated fatty acids, with negligible amounts of cholesterol (0 to 3 ppm). In comparison, animal fats (in particular beef and pork fat) are predominantly long-chain (C14 and longer), with significant amounts of unsaturated fatty acids and cholesterol (from 1-2%) (see Table). Thus, coconut oil and animal fats are very different and the basis for the claim that coconut oil is unhealthy is erroneous.

There is no direct evidence that coconut oil consumption is linked to heart disease, inflammatory diseases or obesity.

There is no direct evidence that coconut oil increases the risk of heart disease. In fact, all of the available evidence, from epidemiological, animal and human studies show that coconut- and coconut oil-based diets are healthy and superior to the western diet. Coconut milk, which contains 20% coconut oil, has been consumed by people in the tropics for thousands of years with no evidence of ill effects until the introduction of the western diet. Before the western diet was introduced, all of the inhabitants of remote Pacific islands consumed a lot of coconut and there was no evidence of heart disease and obesity. In the 1970s, Tokelauans and Pukapukans consumed coconut as the chief source of energy at 63% and 34% of their diet, respectively. Despite

this high coconut intake, vascular disease was uncommon in both populations. Similarly, the inhabitants of Kitava Island who maintained their traditional coconut diet uninfluenced by the western diet did not suffer from a high incidence of stroke and heart disease. Samoa is divided into two regions and provides a clear example of the contrasting effects of the western diet and traditional coconut-based diet. American Samoans who shifted to a western diet showed greater obesity and higher risk for heart disease as compared with Western Samoans who retained their traditional island diet. WHO reported that Pacific islanders were “2.2 times more likely to be obese and 2.4 times more likely to be diabetic if they ate imported fats than if they ate traditional fat sources.” The imported fats included vegetable oils and margarine while the traditional fats included coconut oil.

A number of human studies have shown that coconut and coconut oil-diet are not risk factors for heart disease, and in fact, are associated with healthy indicators. A study of regional diets in the Philippines in the 1980s showed that people in the Bicol region, which had the highest consumption of coconut, showed low incidence of atherosclerosis. The authors concluded that coconut oil does not increase the risk of CVD. In another Philippine study, coconut oil consumption and lipid profiles in a cohort of 1,839 Filipino women, aged 35–69 years, was positively associated with HDL levels. In a study conducted in Brazil, coconut oil showed favorable effects compared with soybean oil on the biochemical and anthropometric profiles of 40 obese women, aged 20 to 40 years. Observational studies in Indonesia likewise showed that consumption of coconut does not lead to adverse cardiovascular outcomes. In reviewing the effect of coconut consumption on cardiovascular risk factors and outcomes in humans, Eyres and co-workers concluded that: “Observational evidence suggests that consumption of coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes.”

A number of controlled studies on humans have shown that coconut oil does not raise risk factors for heart disease. For example, a randomized controlled study in Thailand showed that diets containing coconut milk of one to two meals per day with the fat content of no more than 30% of total energy did not increase either LDL level or cardiovascular risk factors. A 4-week randomised feeding study conducted at Cambridge University, UK, that compared coconut oil, olive oil and butter concluded that coconut oil did not significantly raise LDL concentrations compared with olive oil while butter significantly raised LDL concentrations compared with both coconut oil and olive oil. In addition, coconut oil significantly raised HDL concentrations compared with both butter and olive oil.

The evidence against coconut oil is indirect and invalid

While studies in coconut-consuming countries have consistently shown that coconut oil does not raise the risk of heart disease, the opposite conclusion is reported in studies done in the US and Europe where coconut oil consumption is low. Coconut oil was not part of the diet in Keys’ Seven Country Study. Recent evidence against coconut oil is based on correlation studies on lauric acid (C12) consumption in the US and not on coconut oil itself. In two influential studies, the amount of C12 consumed was less than 0.5% of total energy intake but was consumed from hydrogenated coconut oil and palm kernel oil; therefore, these conclusions are invalidated by the presence of trans fat. In fact, the vast majority of the studies that have been used to support the recommendations against coconut oil have been done in the US and Europe where consumption of (unhydrogenated) coconut oil is low.

Coconut has been consumed for millennia in the tropics and the Pacific with no indication of poor health outcomes. Coconut oil, which makes up about 20% of coconut meat, is part of this healthy tradition. Close to one billion people today consume coconut and coconut oil on a regular basis. If coconut oil caused heart disease, then there should be an epidemic of heart disease in all coconut consuming countries. Clearly, the evidence against coconut oil is indirect and invalid.

4. The recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% of energy will lead to an overconsumption of PUFA which will result in higher rates of obesity and inflammatory diseases.

From 1968 to 1973, Ancel Keys and Ivan Frantz, Jr., undertook a controlled human feeding study – called the Minnesota Coronary Survey (MCS) – which was meant to definitively prove the Keys paradigm. This study was the largest (n=9570), longest (5 years), and most rigorously implemented randomized controlled dietary study of cholesterol lowering by replacement of saturated fat with a vegetable oil rich in linoleic acid. It should be noted that the high saturated fat diet provided 18.5% of energy as saturated fat and 3.8% as unsaturated fat, while the high PUFA diet provided 9.2% of energy as saturated fat and 13.2% as omega-6 fat. This experiment showed that although the high omega-6 fat diet lowered serum cholesterol as predicted by Keys, the risk of death increased by 22% compared to the saturated fat diet. The results of this carefully designed and implemented experiment shows that the recommendation to replace saturated fat with unsaturated fat and to limit saturated fat to 10% will lead to higher risk of death from heart disease. Keys himself never published the results, although Frantz published partial results in 1989 which revealed the failure of the Keys hypothesis. These conclusions – that a high omega-6 fat diet reduces serum cholesterol but raises the incidence of heart disease – have been reproduced by a number of other independent studies, such as the Sydney Heart Study.

Although omega-6 and omega-3 polyunsaturated fatty acids are recognized as essential fatty acids, various international agencies have recommended intake levels ranging from 2-4% for omega-6 PUFA and 0.5-2.0% for omega-3, and a omega-6 to omega-3 ratio of about 4:1. However, the draft WHO guidelines do not recommend an upper limit and desirable ratio. As shown by the US experience, the recommendation to limit saturated fat to less than 10% of total energy and to replace saturated fat with PUFA with no guidance on PUFA intake may result in over-consumption of omega-6 fats which has been linked to the rise in obesity and inflammatory disease. This observation is supported mechanistically by a rat study that compared coconut oil with soybean oil, a

high omega-6 oil, which showed that soybean oil which upregulated genes for obesity, diabetes, inflammation, mitochondrial function and cancer while coconut oil gave favorable metabolic indicators.
After reviewing 25 years of the Dietary Guidelines for Americans, Cohen and co-workers (2015) observed that Americans have been religiously following the Dietary Guidelines and this has been coincident with a rise in obesity. This observation is supported by data from the US National Institutes of Health which show that the rise in obesity in the US starting in 1980 is coincident with the introduction of the American dietary guidelines.
Recommendations (for saturated fatty acids only)
To justify a revision of the draft WHO dietary recommendations, specifically: <ol style="list-style-type: none"> <li>1. To recognize the category of medium-chain fatty acids (MCFA, C6-C12);</li> <li>2. To correct the long-standing errors that saturated fat is inherently unhealthy and that animal fat is a saturated fat; and</li> <li>3. To delete the recommendations that saturated fat should be kept below 10% and should be replaced – without limit – with a high PUFA diet.</li> </ol>
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

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Annexes 1, 6, 7 (for trans-fatty acids only)

Additional comments

## Final comments

Please provide any final thoughts or comments below.
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several scientific arguments to justify a review of the dietary recommendations, as follows:

- 1.❑ Because of the distinct physiological properties of the various saturated fats, the category of SFA should be replaced with MCFA (C6 to C12) and LCFA (C14:0-C18:0).
- 2.❑ Coconut oil should not be classified as similar to butter and animal fats. Coconut oil is about 65% MCFA with negligible amounts of cholesterol, while animal fats are long-chain fats with considerable amounts of cholesterol. Data on the consumption of beef and pork fat and butter should not be used against coconut oil.
- 3.❑ The warning against coconut oil is based on flawed statistical correlations between C12 consumption and heart disease in the US.

## Survey response 59

### General information

Family/last name	
Logstrup	
Given/first name	
Susanne	
Organization/affiliation	
european heart network	
Sector	
Non-governmental agency	
Sector [Other]	
Country	
Belgium	

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)	
Scope and purpose (same for saturated fatty acids and trans-fatty acids)	
Background (same for saturated fatty acids and trans-fatty acids)	
Guideline development process (same for saturated fatty acids and trans-fatty acids)	
Summary of evidence (for saturated fatty acids only)	
Recommendations (for saturated fatty acids only)	
Remarks (for saturated fatty acids only)	
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)	
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)	
Annexes 1, 6, 7 (for saturated fatty acids only)	



Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>The European Heart Network welcomes the draft WHO guidelines on saturated fatty acid and trans fatty acid intake for adults and children. This detailed, up-to-date review of the latest evidence by WHO, along with the clear message conveyed by the draft recommendations, is a most welcome contribution to the ongoing commentary about this issue.</p> <p>The conclusions of EHN's most recent review of the evidence on foods, nutrients and cardiovascular disease are largely consistent with WHO's draft recommendations. Our 2017 policy paper Transforming European food and drink policies for cardiovascular health proposed population goals of less than 10% of energy for saturated fatty acids and less than one third of total fat. In the longer term, we advocate for a population goal of not more than 7% of energy from saturated fats. We also recommend that not more than 0.5% of energy should be derived from trans fatty acids, of which none should be from industrially produced trans fats.</p> <p>We appreciate the clarity of the recommendations and the transparency in relation to the evidence on which these are based. We look forward to national authorities and other stakeholders using these guidelines as a basis for the development and urgent implementation of policies and strategies to reduce intakes of trans fatty acids and limit intakes of saturated fats.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 60

### General information

Family/last name
Miranda
Given/first name
Laura
Organization/affiliation
ConMéxico
Sector
Private sector
Sector [Other]
Country
Mexico

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Background (same for saturated fatty acids and trans-fatty acids)
El sector alimentario de ConMéxico, agrupa 33 empresas líderes de alimentos y bebidas no alcohólicas, contribuye la solución integral de las ENT's del país, además de coadyuvar a la gestación y ejecución de políticas que fomenten hábitos de vida saludables.
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Summary of evidence (for saturated fatty acids only)
Sin comentarios

Recommendations (for saturated fatty acids only)
<p>Concordamos con las recomendaciones sobre la ingesta de ácidos grasos saturados y ácidos grasos trans en niños y adultos, estas basadas en la evidencia científica presentada en el documento.</p> <p>Recomendaciones Ácidos grasos Saturado</p> <ul style="list-style-type: none"> <li>• [?] En adultos y niños cuya ingesta de ácidos grasos saturados es superior al 10% del total de energía, la OMS recomienda la reducción de la ingesta de ácidos grasos saturado (recomendación fuerte).</li> <li>• [?] En adultos y niños, la OMS sugiere reducir la ingesta de ácidos grasos saturados a menos del 10% del consumo total de energía (recomendación condicional).</li> <li>• [?] La OMS sugiere usar ácidos grasos poliinsaturados en reemplazo como fuente de energía, si fuera necesario, cuando se reduzca la ingesta grasos saturados (recomendación condicional).</li> <li>• [?] En adultos y niños cuya ingesta de ácido graso saturados es inferior al 10% del consumo total de energía, la OMS no sugiere incrementar la ingesta de ácido grasos saturados (recomendación condicional).</li> </ul> <p>Recomendaciones sobre Ácidos Grasos Trans</p> <ul style="list-style-type: none"> <li>• [?] En adultos y niños cuya ingesta de ácido trans-graso es superior al 1% del consumo total de energía, la OMS recomienda reducir la ingesta de ácido trans-grasos (recomendación fuerte).</li> <li>• [?] En adultos y niños, la OMS sugiere reducir la ingesta de ácidos trans-grasos a menos del 1% del consumo total de energía (recomendación condicional).</li> <li>• [?] La OMS sugiere el uso de ácidos grasos poliinsaturados como sustitutos de ácidos grasos trans (recomendación condicional).</li> <li>• [?] En adultos y niños, cuya ingesta de ácido trans-graso es inferior al 1% del consumo total de energía, la OMS no sugiere aumento de la ingesta de ácido trans-graso (recomendación condicional)</li> </ul>
Remarks (for saturated fatty acids only)
<p>ConMéxico tiene observaciones particulares sobre la definición de "niños", la cual señala que estas recomendaciones se refieren a niños de 2 a 19 años, la propia OMS define a la adolescencia como el periodo de crecimiento y desarrollo humano que se produce después de la niñez, y antes de la edad adulta, entre los 10 y 19 años.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
<p>ConMéxico tienen preocupaciones puntuales en el apartado de "Traducción e Implementación" que señala, que en caso de ser necesario desarrollar medidas públicas para reducir la ingesta de ácidos grasos saturados y ácidos grasos trans, a través de intervenciones de política en salud, algunas de las medidas e intervenciones que los países pueden incluir: a) la restricción ventas y la promoción de alimentos y bebidas que son altas en ácidos grasos saturados y ácidos grasos trans en escuelas, incluyendo la prohibición en las ventas y la promoción de alimentos que contengan ácidos grasos trans de origen industrial y b) la utilización de medidas fiscales alimentos y bebidas que sean altos en ácidos grasos saturados y ácidos grasos trans.</p> <p>En cuanto a las grasas trans de origen industrial, de acuerdo con el Instituto Nacional de Salud Pública de México y los resultados de la Encuesta Nacional de Nutrición 2006 sobre la Ingestión de ácidos grasos en la dieta de la población mexicana, se concluyo que su nivel consumo nunca constituyó un riesgo para la población mexicana. Si a ello se agrega que todas las empresas asociadas han eliminado y reducido al máximo seguro el contenido de grasas trans en sus productos desde el 2008, satisfactoriamente podemos afirmar que la población mexicana está expuesta a un riesgo mínimo por su consumo.</p> <p>Concordamos con la OMS, en que se deben fomentar conductas basadas en una alimentación correcta y que consideren todas las fuentes de alimentos, ya que en conjunto forman una dieta diaria y con esto se logra el consumo adecuado de nutrimentos. Por ello, trasladar una recomendación de grasas saturadas de la dieta, a un alimento, platillo o producto en particular, podría no ser preciso e, incluso tener efectos distintos a los que se pretende en la conformación de una dieta correcta.</p> <p>En consonancia con lo anterior, sugerir la restricción de la venta y comercialización de alimentos con grasas saturadas, así como el uso de la política fiscal con el mismo objetivo, podría afectar la variedad, disponibilidad y asequibilidad de importantes fuentes de nutrientes para las poblaciones.</p>
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Annexes 1, 6, 7 (for saturated fatty acids only)
Sin comentarios
Additional comments
Sin comentarios

## Trans-fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Background (same for saturated fatty acids and trans-fatty acids)
El sector alimentario de ConMéxico, agrupa 33 empresas líderes de alimentos y bebidas no alcohólicas, contribuye la solución integral de las ENT's del país, además de coadyuvar a la gestación y ejecución de políticas que fomenten hábitos de vida saludables.
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Summary of evidence (for trans-fatty acids only)
Sin comentarios
Recommendations (for trans-fatty acids only)
<p>Concordamos con las recomendaciones sobre la ingesta de ácidos grasos saturados y ácidos grasos trans en niños y adultos, estas basadas en la evidencia científica presentada en el documento.</p> <p>Recomendaciones Ácidos grasos Saturado</p> <ul style="list-style-type: none"> <li>• <input type="checkbox"/> En adultos y niños cuya ingesta de ácidos grasos saturados es superior al 10% del total de energía, la OMS recomienda la reducción de la ingesta de ácidos grasos saturado (recomendación fuerte).</li> <li>• <input type="checkbox"/> En adultos y niños, la OMS sugiere reducir la ingesta de ácidos grasos saturados a menos del 10% del consumo total de energía (recomendación condicional).</li> <li>• <input type="checkbox"/> La OMS sugiere usar ácidos grasos poliinsaturados en reemplazo como fuente de energía, si fuera necesario, cuando se reduzca la ingesta grasos saturados (recomendación condicional).</li> <li>• <input type="checkbox"/> En adultos y niños cuya ingesta de ácido graso saturados es inferior al 10% del consumo total de energía, la OMS no sugiere incrementar la ingesta de ácido grasos saturados (recomendación condicional).</li> </ul> <p>Recomendaciones sobre Ácidos Grasos Trans</p> <ul style="list-style-type: none"> <li>• <input type="checkbox"/> En adultos y niños cuya ingesta de ácido trans-graso es superior al 1% del consumo total de energía, la OMS recomienda reducir la ingesta de ácido trans-grasos (recomendación fuerte).</li> <li>• <input type="checkbox"/> En adultos y niños, la OMS sugiere reducir la ingesta de ácidos trans-grasos a menos del 1% del consumo total de energía (recomendación condicional).</li> <li>• <input type="checkbox"/> La OMS sugiere el uso de ácidos grasos poliinsaturados como sustitutos de ácidos grasos trans (recomendación condicional).</li> <li>• <input type="checkbox"/> En adultos y niños, cuya ingesta de ácido trans-graso es inferior al 1% del consumo total de energía, la OMS no sugiere aumento de la ingesta de ácido trans-graso (recomendación condicional)</li> </ul>
Remarks (for trans-fatty acids only)
ConMéxico tiene observaciones particulares sobre la definición de "niños", la cual señala que estas recomendaciones se refieren a niños de 2 a 19 años, la propia OMS define a la adolescencia como el periodo de crecimiento y desarrollo humano que se produce después de la niñez, y antes de la edad adulta, entre los 10 y 19 años.

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
<p>ConMéxico tienen preocupaciones puntuales en el apartado de “Traducción e Implementación” que señala, que en caso de ser necesario desarrollar medidas públicas para reducir la ingesta de ácidos grasos saturados y ácidos grasos trans, a través de intervenciones de política en salud, algunas de las medidas e intervenciones que los países pueden incluir: a) la restricción de ventas y la promoción de alimentos y bebidas que son altas en ácidos grasos saturados y ácidos grasos trans en escuelas, incluyendo la prohibición en las ventas y la promoción de alimentos que contengan ácidos grasos trans de origen industrial y b) la utilización de medidas fiscales a alimentos y bebidas que sean altos en ácidos grasos saturados y ácidos grasos trans.</p> <p>En cuanto a las grasas trans de origen industrial, de acuerdo con el Instituto Nacional de Salud Pública de México y los resultados de la Encuesta Nacional de Nutrición 2006 sobre la Ingestión de ácidos grasos en la dieta de la población mexicana, se concluyó que su nivel de consumo nunca constituyó un riesgo para la población mexicana. Si a ello se agrega que todas las empresas asociadas han eliminado y reducido al máximo seguro el contenido de grasas trans en sus productos desde el 2008, satisfactoriamente podemos afirmar que la población mexicana está expuesta a un riesgo mínimo por su consumo.</p> <p>Concordamos con la OMS, en que se deben fomentar conductas basadas en una alimentación correcta y que consideren todas las fuentes de alimentos, ya que en conjunto forman una dieta diaria y con esto se logra el consumo adecuado de nutrimentos. Por ello, trasladar una recomendación de grasas saturadas de la dieta, a un alimento, platillo o producto en particular, podría no ser preciso e, incluso tener efectos distintos a los que se pretende en la conformación de una dieta correcta.</p> <p>En consonancia con lo anterior, sugerir la restricción de la venta y comercialización de alimentos con grasas saturadas, así como el uso de la política fiscal con el mismo objetivo, podría afectar la variedad, disponibilidad y asequibilidad de importantes fuentes de nutrientes para las poblaciones.</p>
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Sin comentarios
Annexes 1, 6, 7 (for trans-fatty acids only)
Sin comentarios
Additional comments
Sin comentarios

## Final comments

Please provide any final thoughts or comments below.

## Survey response 61

### General information

Family/last name
Rosales
Given/first name
Ashley
Organization/affiliation
Dairy Council of California
Sector
Other
Sector [Other]
quasi state of California government (not for profit)
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

#### Additional comments

WHO Guidelines: Saturated Fatty Acid and Trans-Fatty Intake for Adults and Children  
Comments by Ashley Rosales, RDN, on behalf of Dairy Council of California

The rising rate of obesity coupled with related chronic disease is a public health crisis impacting the lives of adults and, increasingly, children as well(1). Mitigating the devastating effects of chronic disease requires the collective action of multiple sectors working together to inspire changes in systems and community environments as well as individual behavior. It is commendable that respected organizations such as the World Health Organization are spearheading recommendations to improve healthy eating habits and ultimately reduce the risk of chronic disease.

Along with obesity and chronic disease is the interrelated issue of food insecurity, which reminds us that solving complex public health problems requires a broad range of solutions and a zealous application of credible nutrition science. It is with that notion that we have an opportunity to look at nutrition recommendations through the lens of whole foods and, ultimately, healthy eating patterns as the optimal way to obtain nutritional adequacy while also limiting consumption of nutrients that, in excess, may cause harm.

Emerging research shows that saturated fat consumption may not be directly linked to cardiovascular disease (CVD) risks(2). This suggests that saturated fat consumption on its own is an insufficient or overly simplistic metric for diet quality. Different food sources of fat can contribute additional nutrients and bioactive compounds to the diet that may impact disease risk, and studying individual nutrients may not account for the total food effects(3). Whole foods consist of numerous nutrients that are contained in a complex structure. These nutrients often interact in unique ways to impact various biological processes. The nature of the food structure and the nutrients therein—the food matrix—may exhibit different relationships with health indicators as compared to single nutrients studied in isolation.

More specifically, the effect of the food matrix was recently studied looking at the dairy matrix, and evidence was presented and discussed by an expert panel. Among the conclusions was that nutritional values of dairy products should be considered on the basis of the biofunctionality of the nutrients within dairy food structures rather than on nutrient content alone(4).

There is a growing body of evidence that links the consumption of milk and dairy foods to a wide range of health benefits, from well-studied associations like controlling blood pressure and improving bone health to newer associations like reducing the risk of diabetes and heart disease(5). Research looking specifically at fat in whole milk and reduced-fat dairy foods suggests that the fat may have unique properties that differentiate it from fat in other food sources(6). Some experts believe using the term saturated fat does not accurately describe the variety of fatty acids found in dairy fat. Whole milk contains about 3–4 percent dairy fat by weight, and dairy fat is made up of more than 400 different types of fatty acids, making it the most complex fat naturally occurring in a food(7).

Numerous observational studies over the past decade demonstrate that milk, cheese and yogurt consumption, regardless of fat content, is not associated with increased risk for CVD. An additional randomized controlled trial shows that regular (full-fat) dairy food consumption improves lipid biomarkers related to risk for CVD. These findings support the evidence that regular dairy food consumption may be linked to lower CVD risk(8). With strong consensus from a growing number of studies, the body of evidence supports the need to reaffirm the role of whole and reduced-fat dairy foods in healthy eating patterns. This is critical to ensure that future nutrition guidance continues to embrace dairy foods as an important part of healthy eating patterns that promote optimal health and reduce chronic disease(9).

In children's eating patterns, milk is an important source of essential nutrients that contribute to overall health, but by age 6 most children are not meeting the recommended daily servings from the dairy food group(10). Poor eating patterns, even in early childhood, can continue as habits in adulthood and increase the risk for overweight and the development of chronic conditions such as heart disease. Prospective studies show that greater consumption of milk or full-fat dairy foods in children is associated with lower measures of body fat as adolescents.(11,12) In the Avon Longitudinal Study of Parents and Children, those in the highest quartile of dairy fat intake at age 10 have lower risk of excess total body fat mass at age 13 and lower gains in body mass index compared with children in the lowest quartile(12).

There is compelling evidence linking food insecurity to poor health outcomes(13), heightening the urgency of the health sector to seek solutions to close this gap. With so many children and families worldwide living in poverty, access to nutritious and wholesome foods is essential to help children reach their full health potential as adults. The recommendations provided by reputable health organizations such as the World Health Organization serve as a catalyst for public policy that ultimately determines the food choices available to our most vulnerable populations through nutrition assistance programs. One example of this critical safety net is school meal programs. Research suggests that eating school breakfast every day is associated with healthier dietary intakes among U.S. schoolchildren, particularly increased intakes of fruits and vegetables, whole grains and dairy(14). Additionally, consumption specifically of fruits, vegetables or dairy products made readily available in school meal programs is associated with improved academic and health outcomes among children and adolescents(15).

The draft WHO Guidelines: Saturated fatty acid and trans-fatty intake for adults and children emphasizes the need to consider sustainability when implementing recommendations. Since sustainability and nutritious foods are inextricably linked, there is an opportunity for the agricultural, food systems and health sectors to continue efforts to use advances in science, innovation and

technology to become more efficient and sustainable while promoting optimal health for all. A recent report by the High Level Panel of Experts on Food Security and Nutrition (HLPE), which serves as the science-policy interface of the Committee on World Food Security, provides guidance on shaping food systems to ensure that food is produced and distributed in a sustainable manner and, ultimately, that all individuals have access to adequate nutritious foods(16). The conceptual framework proposed by HLPE highlights the central role of the food environment in facilitating healthy and sustainable consumer food choices. The report presents effective policies and programs that have the potential to shape food systems, contributing to improved food security and nutrition. Of the priorities for action, improving physical and economic access to healthy and sustainable diets and strengthening consumers' information and education to enable healthier food choices are critical. In addition to making changes to our food systems, the report stresses empowering individuals to make healthy food choices from a variety of nutrient-rich foods as opposed to nutrient-poor foods. Milk, cheese and yogurt provide many essential nutrients important for health. Consuming the recommended amount of dairy foods would go a long way in closing the gap on some nutrient intakes, including nutrients of concern calcium and vitamin D, as well as magnesium and vitamin A(17). The dairy agricultural community takes the roles of being sustainable and improving food and nutrition security seriously as demonstrated by its commitment to be part of the solution to continually improve sustainable production of nutrient-rich dairy foods worldwide.

In conclusion, recommendations for the public that generally restrict saturated fat without focusing on the whole food matrix or healthy eating patterns, could unintentionally limit access to and consumption of nutritious foods like milk and dairy foods. Ensuring that public health nutrition guidance is based in sound science makes it easier to create more opportunities for children and adults to access nutritious foods in the communities where they live, learn, work and play.

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## *Trans*-fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)

Summary of evidence (for trans-fatty acids only)

Recommendations (for trans-fatty acids only)

Remarks (for trans-fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for trans-fatty acids only)

Additional comments

## Final comments

Please provide any final thoughts or comments below.
Full comments attached as a PDF.

## WHO Guidelines: Saturated Fatty Acid and Trans-Fatty Intake for Adults and Children

*Comments by Ashley Rosales, RDN, on behalf of Dairy Council of California*

The rising rate of obesity coupled with related chronic disease is a public health crisis impacting the lives of adults and, increasingly, children as well.<sup>1</sup> Mitigating the devastating effects of chronic disease requires the collective action of multiple sectors working together to inspire changes in systems and community environments as well as individual behavior. It is commendable that respected organizations such as the World Health Organization are spearheading recommendations to improve healthy eating habits and ultimately reduce the risk of chronic disease.

Along with obesity and chronic disease is the interrelated issue of food insecurity, which reminds us that solving complex public health problems requires a broad range of solutions and a zealous application of credible nutrition science. **It is with that notion that we have an opportunity to look at nutrition recommendations through the lens of whole foods and, ultimately, healthy eating patterns as the optimal way to obtain nutritional adequacy while also limiting consumption of nutrients that, in excess, may cause harm.**

Emerging research shows that saturated fat consumption may not be directly linked to cardiovascular disease (CVD) risks.<sup>2</sup> This suggests that saturated fat consumption on its own is an insufficient or overly simplistic metric for diet quality. Different food sources of fat can contribute additional nutrients and bioactive compounds to the diet that may impact disease risk, and studying individual nutrients may not account for the total food effects.<sup>3</sup> Whole foods consist of numerous nutrients that are contained in a complex structure. These nutrients often interact in unique ways to impact various biological processes. The nature of the food structure and the nutrients therein—the food matrix—may exhibit different relationships with health indicators as compared to single nutrients studied in isolation.

More specifically, the effect of the food matrix was recently studied looking at the dairy matrix, and evidence was presented and discussed by an expert panel. Among the conclusions was that nutritional values of dairy products should be considered on the basis of the biofunctionality of the nutrients within dairy food structures rather than on nutrient content alone.<sup>4</sup>

**There is a growing body of evidence that links the consumption of milk and dairy foods to a wide range of health benefits, from well-studied associations like controlling blood pressure and improving bone health to newer associations like reducing the risk of diabetes and heart disease.<sup>5</sup>** Research looking specifically at fat in whole milk and reduced-fat dairy foods suggests that the fat may have unique properties that differentiate it from fat in other food sources.<sup>6</sup> Some experts believe using the term saturated fat does not accurately describe the variety of fatty acids found in dairy fat. Whole milk contains about 3–4 percent dairy fat by weight, and dairy fat is made up of more than 400 different types of fatty acids, making it the most complex fat naturally occurring in a food.<sup>7</sup>

Numerous observational studies over the past decade demonstrate that milk, cheese and yogurt consumption, regardless of fat content, is not associated with increased risk for CVD. An additional randomized controlled trial shows that regular (full-fat) dairy food consumption improves lipid biomarkers related to risk for CVD. These findings support the evidence that regular dairy food consumption may be linked to lower CVD risk.<sup>8</sup> **With strong consensus from a growing number of studies, the body of evidence supports the need to reaffirm the role of whole and reduced-fat dairy foods in healthy eating patterns.** This is critical to ensure that future nutrition guidance continues to embrace dairy foods as an important part of healthy eating patterns that promote optimal health and reduce chronic disease.<sup>9</sup>

In children's eating patterns, milk is an important source of essential nutrients that contribute to overall health, but by age 6 most children are not meeting the recommended daily servings from the Dairy food group.<sup>10</sup> Poor eating patterns, even in early childhood, can continue as habits in adulthood and increase the risk for overweight and the development of chronic conditions such as heart disease. Prospective studies show that greater consumption of milk or full-fat dairy foods in children is associated with lower measures of body fat as adolescents.<sup>11, 12</sup> In the Avon Longitudinal Study of Parents and Children, those in the highest quartile of dairy fat intake at age 10 have lower risk of excess total body fat mass at age 13 and lower gains in body mass index compared with children in the lowest quartile.<sup>12</sup>

There is compelling evidence linking food insecurity to poor health outcomes,<sup>13</sup> heightening the urgency of the health sector to seek solutions to close this gap. With so many children and families worldwide living in poverty, access to nutritious and wholesome foods is essential to help children reach their full health potential as adults. The recommendations provided by reputable health

organizations such as the World Health Organization serve as a catalyst for public policy that ultimately determines the food choices available to our most vulnerable populations through nutrition assistance programs. One example of this critical safety net is school meal programs. Research suggests that eating school breakfast every day is associated with healthier dietary intakes among U.S. schoolchildren, particularly increased intakes of fruits and vegetables, whole grains and dairy.<sup>14</sup> Additionally, consumption specifically of fruits, vegetables or dairy products made readily available in school meal programs is associated with improved academic and health outcomes among children and adolescents.<sup>15</sup>

The draft **WHO Guidelines: Saturated fatty acid and trans-fatty intake for adults and children** emphasizes the need to consider sustainability when implementing recommendations. Since sustainability and nutritious foods are inextricably linked, there is an opportunity for the agricultural, food systems and health sectors to continue efforts to use advances in science, innovation and technology to become more efficient and sustainable while promoting optimal health for all. A recent report by the High Level Panel of Experts on Food Security and Nutrition (HLPE), which serves as the science-policy interface of the Committee on World Food Security, provides guidance on shaping food systems to ensure that food is produced and distributed in a sustainable manner and, ultimately, that all individuals have access to adequate nutritious foods.<sup>16</sup> The conceptual framework proposed by HLPE highlights the central role of the food environment in facilitating healthy and sustainable consumer food choices. The report presents effective policies and programs that have the potential to shape food systems, contributing to improved food security and nutrition. Of the priorities for action, **improving physical and economic access to healthy and sustainable diets and strengthening consumers' information and education to enable healthier food choices are critical.** In addition to making changes to our food systems, the report stresses empowering individuals to make healthy food choices from a variety of nutrient-rich foods as opposed to nutrient-poor foods. Milk, cheese and yogurt provide many essential nutrients important for health. Consuming the recommended amount of dairy foods would go a long way in closing the gap on some nutrient intakes, including nutrients of concern calcium and vitamin D, as well as magnesium and vitamin A.<sup>17</sup> The dairy agricultural community takes the roles of being sustainable and improving food and nutrition security seriously as demonstrated by its commitment to be part of the solution to continually improve sustainable production of nutrient-rich dairy foods worldwide.

In conclusion, **recommendations for the public that generally restrict saturated fat without focusing on the whole food matrix or healthy eating patterns,**

**could unintentionally limit access to and consumption of nutritious foods like milk and dairy foods.** Ensuring that public health nutrition guidance is based in sound science makes it easier to create more opportunities for children and adults to access nutritious foods in the communities where they live, learn, work and play.

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## Survey response 62

### General information

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### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

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Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments



Please provide any final thoughts or comments below.

#### MYTHS ABOUT EDIBLE FATS

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What are saturated and unsaturated fats?

According to the lipid hypothesis postulating a link between hypercholesterolemia and atherosclerosis, edible fat are classified into two groups:

- saturated fats of animal origin which are putatively associated with atherosclerosis,
- unsaturated fats of plant origin which deliver putative health benefits.

In reality, there is no such thing as saturated or unsaturated fats. All edible fats contain saturated, monounsaturated and polyunsaturated fatty acids (FAs) in various proportions. Animal fats contain more saturated FAs, whereas vegetable oils contain more unsaturated FAs. Coconut oil is most abundant in saturated FAs (around 90%); therefore, it should be classified as an animal fat in view of the above criteria (Gunstone 2011).

Do saturated fats cause atherosclerosis?

Hypercholesterolemia is one of the many and one of the least important risk factors of atherosclerosis. Saturated FAs and artificial trans fatty acid (TFA) isomers induce a similar increase in total cholesterol and LDL cholesterol levels (Mensink et al. 2003). However, unlike TFA isomers, saturated FAs also contribute to an increase in HDL cholesterol levels, and they do not compromise the LDL/HDL ratio. Artificial trans isomers decrease HDL cholesterol levels and significantly compromise the LDL/HDL ratio. According to a 1994 report of the Danish Nutrition Council, the intake of artificial TFAs as compared with saturated FAs is associated with an approximately 10-fold higher risk of cardiovascular diseases (Das 2006).

Unlike vegetable oils, animal fats are characterized by oxidative stability (are not susceptible to oxidation) due to a lower content of polyunsaturated FAs, a high content of oleic acid (up to 35% in milk fat, up to 50% in lard) and the presence of antioxidants (conjugated linoleic acid, CLA; coenzyme Q10, phospholipids,  $\beta$ -carotene, vitamins E, A, D3) (Cichosz et al. 2017). In addition to lipophilic antioxidants, animal fats also contain substances that enhance intestinal barrier integrity. Both phospholipids and short-chain and medium-chain saturated FAs have anti-inflammatory and immunomodulatory properties, and they minimize allergic reactions to foods. These compounds exert metabolic programming effects, and they are particularly desirable in the diets of young children.

Animal fats are abundant in compounds that are vital for neurogenesis. Polyunsaturated n-3 FAs, phospholipids and cholesterol play important roles in the development of the brain, retina and intellectual potential of children. Brain and neural development begins in the fetal period and continues until 18 months of age, and these processes are suppressed in children whose diets are deficient in the above compounds. Cholesterol deficiency inhibits the development of neural synapses, impairs cognitive ability and memory. Children deprived of cholesterol in early life are at greater risk of developing brain dysfunctions, poor eyesight and even aggressive behavior. Phospholipids play the key role in transmitting neural impulses, synthesizing neurotransmitters and preventing dendrite degradation. Cholesterol and phospholipid deficiencies disrupt the formation of the myelin sheath, which increases the risk of brain dysfunctions, autism and multiple sclerosis. It is not a coincidence that human milk is abundant in cholesterol and phospholipids (Delplanque et al. 2015).

High intake of saturated FAs of animal (butter, lard, beef tallow) or plant origin (palm oil, coconut oil) inhibits carcinogenesis in animals with chemically induced tumors. Animal fats contain bioactive compounds with antioxidant, immunostimulatory, anticarcinogenic and neuroprotective effects which enhance endogenous immune responses in humans.

The growing popularity of vegetable oils has led to a several-fold decrease in the dietary intake of lipophilic antioxidants such as CLA, coenzyme Q10, phospholipids and vitamins E, A and D3. Unsaturated FAs are incorporated into cellular structures, and they undergo oxidation when deprived of antioxidant protection. Lipid peroxidation, a chain reaction that damages cell structures and genes, poses a particular threat and contributes to various diseases, including malignant transformations.

Vegetable oils increase the risk of atherosclerosis and cancer

Vegetable oils are particularly susceptible to oxidation because they are abundant in polyunsaturated FAs and completely devoid of antioxidants which are removed in the refining process. In the human body, the demand for antioxidants increases exponentially with a rise in the concentration of polyunsaturated FAs and the number of double and triple bonds. Diets rich in vegetable oils do not provide antioxidant protection, especially because most fruit and vegetable antioxidants are not active in a lipophilic environment. The oxidation of vegetable oils cannot be reliably evaluated in a sensory analysis because lipid degradation products are characterized by very high sensory perception thresholds. For this reason, the safety of edible oils cannot be guaranteed based on their use-by date.

Excessive consumption of vegetable oils disrupts the pro-oxidant-antioxidant balance. Oxidative stress increases the risk of atherosclerosis, cancer and neurological disorders. LDL cholesterol, the main component of atherosclerotic plaque, is oxidized only in the absence of antioxidants. The above suggests that plaque formation is the result, and not the cause of atherosclerosis. The oxidation of polyunsaturated FAs is inevitable, in particular at high temperature (Romero et al. 2000; Sebastian et al. 2014).. Primary oxidation products (hydroperoxides and peroxides) are converted to harmless hydroxy acids, whereas secondary oxidation products (aldehydes, ketones, acids, hydrocarbons) damage cell membranes and intracellular structures, suppress enzyme activity, and exert cytotoxic, atherogenic and mutagenic effects. Peroxidation products modify DNA and cause genetic instability, which increases the number of somatic mutations in the genetic material of cells and initiates malignant transformations.

Artificial trans isomers, cyclic monomers and polymers are produced by subjecting the highly reactive double bonds of unsaturated FAs to high temperature (Moreno et al. 1999). Oxidation is also inevitable in phytosterols. Oxidized phytosterols disrupt cell membrane function, inhibit DNA synthesis, increase the rate of cholesterol oxidation, and exert cytotoxic, immunosuppressive, angiotoxic, mutagenic and carcinogenic effects (Tabee 2008).

The oxidation products of polyunsaturated FAs and phytosterols are active at every stage of carcinogenesis in humans. The

correlation between the intake of vegetable oils and carcinogenesis has been postulated by numerous animal experiments and epidemiological studies. Mutagenic transformation is not induced by diets that are high in fat, but by diets rich in n-6 linoleic acid, which is found mainly in vegetable oil, and elaidic acid, the main artificial trans fatty acid in margarine and convenience foods.

Are vegetable oils a source of essential unsaturated FAs?

n-6 linoleic acid and n-3  $\alpha$ -linolenic acid are not biologically active despite the fact that they are regarded as essential unsaturated FAs. They can play various biological roles only after conversion to long-chain polyunsaturated derivatives: n-6 arachidonic acid (AA), n-3 eicosapentaenoic acid (EPA) and n-3 docosahexaenoic acid (DHA). The human body synthesizes enzymes (elongase and desaturase), but they are largely suppressed when zinc, magnesium and vitamin B6 are deficient and when insulin secretion is compromised. These enzymes are not highly active in premature infants, elderly persons and people who take anticoagulant and hypotensive drugs.

In the human body, the conversion of n-3  $\alpha$ -linolenic acid is also inhibited by excess n-6 polyunsaturated FAs from vegetable oils. n-6 linoleic acid and n-3  $\alpha$ -linolenic acid are converted by the same enzymes. However, n-6 FAs are converted first, which is why the n-6/n-3 PUFA ratio is very important (the optimal ratio is 4:1). Regardless of the above, desaturase activity is suppressed by artificial trans isomers from margarines and convenience foods, which implies that the conversion of both n-6 linoleic acid and n-3  $\alpha$ -linolenic acid is inhibited. The conversion of n-6 linoleic acid and n-3  $\alpha$ -linolenic acid to biologically active long-chain derivatives is possible, but it is highly unlikely in people consuming typical Western diets which abound in polyunsaturated n-6 FAs and artificial trans isomers (Simopoulos 2002; Lavie et al. 2009).

In reality, most edible vegetable oils (rice, sunflower, sesame, corn, primrose, pumpkin seed and grape seed oil) contain n-6 linoleic acid whose excess has been correlated with a higher risk of atherosclerosis, cancer and neurological disorders (Łoźna, 2012; Rose, 1997). Fish and marine oils are a source of biologically active long-chain polyunsaturated FAs that are readily assimilated by the human body.

Margarine – the unsafe “healthy” alternative to butter

The results of numerous experiments, clinical studies and epidemiological observations indicate that margarines, confectionery fats, deep frying fats and convenience foods are sources of artificial TFAs which cause all diet-dependent metabolic disorders (obesity, type 2 diabetes, atherosclerosis, cancer, neurological disorders) (Bakker et al. 1997; Slattery et al. 2001; Stender et al. 2006). Industrially produced trans fatty acids are more rapidly incorporated into cell membrane phospholipids than natural cis isomers. Their melting temperature is around 20 °C higher in comparison with cis isomers, which is why artificial trans isomers have solid consistency at body temperature. The above facilitates the formation of atherosclerotic plaque and disrupts cell function by decreasing the elasticity and fluidity of cell membranes, and suppressing the activity of receptors and enzymes.

Artificial trans fatty acids inhibit transacetylase, an enzyme responsible for cholesterol esterification, which disrupts lipid metabolism and promotes atherosclerotic changes. In people consuming diets where artificial FTAs accounted for approximately 10% of daily energy intake, an increase in LDL cholesterol and a significant decrease in HDL cholesterol were observed already after 3 weeks. Regardless of the above, artificial trans isomers increase the risk of heart arrhythmia, raise triglyceride levels and compromise lipoprotein metabolism. Atherosclerotic plaques contain elaidic acid, a compound found in hydrogenated vegetable oils, which provides further evidence for the atherosclerosis-promoting effects of margarine (Stachowska et al., 2004).

The Nurses' Health Study demonstrated that elaidic acid contributes to insulin resistance and type 2 diabetes. Artificial trans fatty acids stimulate the accumulation of visceral fat which alters metabolic processes inside adipocytes. Adipose tissue produces leptin which intensifies atherosclerotic processes. The changes in the structure and function of cell membranes make cells less sensitive to insulin, which increases the risk of type 2 diabetes (Salmeron et al. 2001, Lopez-Garcia et al., 2005).

The Euramic study demonstrated a correlation between the intake of artificial FTAs and the incidence of breast, prostate and colorectal cancer. Industrially produced trans fatty acids induce changes in cell membrane function and activate cytochromes P450, which disrupts immune system function, intensifies the proliferation of cancer cells and promotes tumor growth (Aro et al. 1995; Simonsen et al., 1998 Lopez-Garcia et al. 2005; Liu et al. 2007; Mozaffarian & Clarke, 2009). The Issac program revealed a correlation between the consumption of artificial FTAs and the incidence of allergies and asthma in 13- and 14-year-olds treated in 155 medical centers around the world (Welland, 1999).

Artificial trans isomers have a particularly negative influence on infants and young children. They block enzymes which convert n-6 linoleic acid (from vegetable oils) and n-3  $\alpha$ -linolenic acid to biologically active long-chain polyunsaturated derivatives (AA, EPA, DHA) which are essential for brain and retina development. Artificial trans isomers are metabolized in a completely different manner than natural trans isomers from milk fat. Industrially produced FTAs cross the placenta and reach the fetus, and they are also present in the mother's milk, which disrupts brain development and weakens the child's immune system (Cichon & Stolyhwo, 1999; Jamiol-Milc et al. 2009). According to Professor Walter Willett, a health and nutrition expert of the Harvard Medical School, artificial trans isomers are “the biggest food processing disaster ever” (Willett, 2006).

Can artificial trans isomers be equated with saturated FAs?

No other dietary component is as damaging to health as industrially produced trans FAs. For this reason, nutrition and health labels which equate artificial FTAs with saturated FAs are misleading for consumers. Saturated FAs and artificial trans FAs exert completely different effects on the blood lipid profile. Saturated FAs increase total cholesterol and LDL and HDL cholesterol levels, whereas artificial trans isomers increase total cholesterol and LDL cholesterol levels, but decrease HDL cholesterol, increase triglyceride and atherogenic lipoprotein levels, and promote the synthesis of blood coagulation factors. Artificial FTAs also increase the risk of heart arrhythmia (Kromhout et al. 1995).

Artificial trans isomers disrupt cell metabolism. When incorporated into cell membrane phospholipids, they decrease membrane elasticity and fluidity, disrupt ion channel function and block receptors and enzymes. Membrane function and cell metabolism deteriorate proportionally to the concentration of artificial FTAs. Cell membrane rigidity decreases sensitivity to insulin and increases the risk of type 2 diabetes. Artificial trans isomers activate cytochromes P450, which promotes the synthesis of free radicals and reactive oxygen species. The changes in membrane channels and enzymes weaken cellular defense systems, which intensifies free radical processes, mutagenic transformations and promotes carcinogenesis (Lopez-Garcia et al. 2005; Liu et al.

2007; Mozaffarian & Clarke, 2009).

Unlike artificial trans isomers, saturated FAs have antibacterial, antiviral and immunostimulatory properties. In addition to saturated FAs, animal fats also contain compounds with documented anticarcinogenic and neuroprotective effects. The products of animal fat oxidation possess unique sensory qualities which intensify olfactory and gustatory perception.

Can artificial trans isomers be equated with natural isomers?

No other component of the human diet delivers equally potent anticarcinogenic effects as natural trans isomers from the fat and meat of ruminants. Meanwhile, food and health labels equate natural trans fats with artificial trans isomers that are extremely harmful for the human metabolism.

Naturally-occurring trans fats, including CLA and vaccenic acid, are produced by rumen microflora and deliver a host of health benefits. Conjugated linoleic acid enhances cell membrane function and modulates cellular defense mechanisms. It increases cell sensitivity to insulin, normalizes glucose tolerance, reduces hyperinsulinemia and free fatty acid levels in the blood (Risérus et al. 2001; Laso et al. 2007). Conjugated linoleic acid is a potent antioxidant which protects lipids and phospholipids against free radicals, and delivers anti-inflammatory, immunostimulatory and bacteriostatic effects. It suppresses atherosclerotic changes and the development of cancer cells (Lee et al. 2005). Conjugated linoleic acid exerts anticarcinogenic effects at every stage of carcinogenesis. It inhibits angiogenesis, promotes the synthesis of DNA and eicosanoids which stimulate cell growth and division (Angel, 2004; Elias & Innis, 2001).

The intake of natural trans FAs should not be limited – in fact, it should be actively promoted. Food producers should ensure that the milk and meat of ruminants are abundant in CLA and other bioactive components (Lock & Barman, 2004). The fact that artificial and natural trans isomers are equated testifies to the hypocrisy of food industry lobbyists and legislators.

The latest lie – “trans-free” margarine

The use of more oxidatively stable oils, such as palm oil instead of sunflower oil, in the production of margarines has decreased the content of artificial trans isomers in these products. However, food producers’ and industry lobbyists’ claims regarding the alleged health benefits of “trans-free” margarines are completely ungrounded in fact.

Very few countries have introduced regulations limiting the content of artificial trans fats in foods despite the fact that the harmful effects of margarines have been scientifically proven more than 30 years ago. The EU has failed to introduce a legal limit on the content of artificial trans fats in foods, which confirms that “trans-free” margarines are not, in fact, free of industrial TFAs. The esterification technology is significantly more expensive, which is why the market will continue to be flooded with the cheapest and most harmful margarines unless food companies are legally obliged to state the content of artificial trans isomers on product labels.

The absence of laws regulating the content of trans isomers in foods seems to be the best proof that business triumphs over common sense. It demonstrates that the food industry disregards public health concerns or even consciously contributes to the development of diet-dependent metabolic disorders.

Do food laws protect consumers?

Revising the consensus on dietary fats

There has never been reliable scientific evidence that animal fats are unhealthy or that vegetable oils, in particularly margarines, deliver health benefits. In many countries, decision-makers have reached a consensus on dietary fats with food producers by assuming a priori that all vegetable oils are healthy. The fact that animal fats are a valuable source of healthy lipophilic antioxidants has been conveniently disregarded.

The following facts have also been ignored in the fat consensus:

- the real demand for polyunsaturated FAs in human nutrition,
- high demand for antioxidants in diets rich in vegetable oils,
- very high susceptibility of polyunsaturated FAs and phytosterols to oxidation,
- health risks posed by the carcinogenic properties of secondary oxidation products,
- healthy dietary n-6/n-3 PUFA ratio.

The fat consensus is completely unreliable for the above reasons. The results of many epidemiological studies indicate that the fat consensus not only fails to promote healthy nutrition, but it poses an evident risk for public health. This dietary experiment which advocated the replacement of animal fats with much cheaper vegetable oils has led to a 5-fold increase in the incidence of cancer and neurological disorders since the 1970s.

Despite the above, dietary guidelines continue to state that fats should account for 30% of daily energy intake, with equal proportions (10% each) of saturated, monounsaturated and polyunsaturated FAs. Thus, the recommended intake of polyunsaturated FAs exceeds nutritional requirements 4-fold.

The illusive safety of edible oils

The FAO/WHO Codex Committee on Fats and Oils relies only on one parameter – peroxide value – in the process of elaborating standards for fats. This parameter varies considerably because peroxides are converted to secondary oxidation products. For this reason, peroxide value is not a reliable measure of lipid oxidation. In addition, the cytotoxic and genotoxic effects of secondary lipid oxidation products have been recognized a long time ago.

The labeling requirements for edible fats should be expanded to include the following parameters which pose a threat to consumer health:

- concentration of lipid hydroperoxides,
- number of conjugated double and triple bonds,
- content of aldehydes from the degradation of unsaturated FAs.

The content of harmful secondary lipid oxidation products is not taken into account even in evaluations of the oxidative stability of milk replacement formulas for infants which are classified as foods for special dietary uses. The fact that fat analyses are limited to a single, non-stable parameter benefits food producers and provides further evidence that the declared safety of edible vegetable oils is illusive.

## Nutrition and health claims mislead consumers

Research has long demonstrated that dietary guidelines which postulate a link between hypercholesterolemia and atherosclerosis have contributed to an increase in the incidence of diet-dependent diseases (obesity, type 2 diabetes, atherosclerosis, cancer, neurological disorders). The above can be attributed mainly to the replacement of oxidatively stable animal fats, a source of valuable bioactive components, with vegetable oils and margarine.

Despite the above, Regulation (EC) No. 1924/2006 of the European Parliament and of the Council of 20 December 2006 classifies both natural trans isomers (which deliver health benefits) and artificial trans isomers (the most harmful component of the human diet) as saturated fatty acids whose dietary intake should be limited.

Food labels containing information about the total content of saturated FAs and trans isomers testify to the hypocrisy of the food industry. Food companies discredit natural and biologically active foods which reduce the risk of diet-dependent diseases.

Saturated FAs cannot be equated with trans FA isomers. Natural trans isomers deliver numerous health benefits, and they should never be linked with artificial trans isomers which have disastrous health consequences.

Could it be possible that under the guise of concern for public health, food companies consciously mislead consumers and prevent them from making smart food choices?

Who really benefits from food laws?

The lipid hypothesis postulating a link between hypercholesterolemia and atherosclerosis has been fabricated by the producers of vegetable oils and margarines, and it has never been backed by clinical research or epidemiological studies. The existing body of knowledge clearly indicates that the risk of diet-dependent diseases is not magnified by the intake of dietary fat, but by its composition.

The growing incidence of diet-dependent diseases is proportional to the intake of edible vegetable oils for the following reasons:

- the intake of n-6 polyunsaturated FAs exceeds healthy levels several-fold,
- nutritional guidelines advocate diets with an unhealthy ratio of n-6/n-3 polyunsaturated FAs,
- high intake of unsaturated FAs disrupts the pro-oxidant-antioxidant balance,
- unsaturated FAs are oxidized during thermal processing,
- oxidized polyunsaturated FAs and phytosterols have carcinogenic properties,
- convenience foods abound in artificial trans isomers.

The lipid hypothesis contributes to the incidence and prevalence of obesity, type 2 diabetes, cancer and neurological disorders, without minimizing the risk of atherosclerosis. It does not confirm the alleged health benefits of vegetable oils or the adequacy of dietary guidelines. Sound nutritional recommendations should be based on the real demand for n-6 and n-3 polyunsaturated FAs and their optimal dietary ratio (4:1). They should account for the importance of lipophilic antioxidants which are readily available in animal fat and fish oil.

Food laws should guarantee food and nutrition safety, and should resolve public health issues instead of generating new problems. However, the existing laws equate natural and artificial trans isomers and saturated FAs despite the fact that they play completely different biological roles in the human body. The facts speak for themselves: food laws and dietary guidelines are not concerned with health protection. The failed attempts to change the existing laws and limit the intake of artificial trans isomers, and the growing incidence of diet-dependent diseases clearly indicate that effective lobbying leads to disregard for public health. Absurd food laws and dietary guidelines (developed based on the fabricated lipid theory of atherosclerosis) would have been long verified if public health were to take precedence over the profits of food and pharmaceutical companies. Despite the low effectiveness of health care systems (not only in Poland), the real causes of diet-dependent diseases continue to be ignored, and successive governments show very little concern for public health.

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## Survey response 63

### General information

Family/last name
Flynn
Given/first name
Mary
Organization/affiliation
Food safety Authority of Ireland
Sector
Government
Sector [Other]
Country
Ireland

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Page 11 last two lines under Remarks: Ireland would question making no distinction between ruminant and industrially produced trans fatty acids in the recommendations given the low intake of ruminant trans fatty acids. Ireland acknowledges the intent is to prevent replacement of industrial trans with ruminant trans but feels the recommendations should clarify this. Ireland is concerned that without this distinction foods that are important in the Irish diet for calcium and iron (i.e. dairy and meat) will be negatively affected.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
No comment
Background (same for saturated fatty acids and trans-fatty acids)
See page 16 - last sentence in relation to the comment above in Executive Summary
Guideline development process (same for saturated fatty acids and trans-fatty acids)
no comment
Summary of evidence (for saturated fatty acids only)
no comment
Recommendations (for saturated fatty acids only)
1. Ireland agrees with the recommendations but would ask for acknowledgement that a target of 10% saturated fat intake for children from age 2 to 5 years may not be achievable given their significant involvement of their food intake at this age range which transition from infancy to school-aged children. Ireland has concerns that a target of 10% saturated fat intake at this age may compromise iron and calcium intakes. 2. Ireland acknowledges that the approach taken by WHO re effectiveness on CVD risk and blood lipid levels resulted in PUFA as the most effective replacement for saturated fats. Nonetheless, Ireland has some concerns about recommending PUFA as the replacement source of energy without any upper limits. This is due to uncertainty about the long-term, inter-generational safety of high intakes of PUFA. In addition there is no guidance on ratio of n6 and n3 PUFA. Ireland would favour an approach where MUFA and unrefined carbohydrate is included.
Remarks (for saturated fatty acids only)
Page 38 top of page: see above

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
<p>Page 44 Ireland supports all recommendations for saturated fat but has concerns about those for trans fatty acids. It is difficult to assess intakes of trans fats accurately due to variation in fats used by the food industry and the fact that food composition tables cannot reflect this variability. In the EU trans fatty acids are not on food labelling</p> <p>Ireland supports a ban on industrially produced trans fatty acids in food in similar to the one in place in Denmark.</p> <p>Ireland would suggest that the restricting the sales and promotion of foods and beverages high in trans fatty acids should be restricted for all age groups and not just in schools</p>
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
<p>Page 46 first bullet point:</p> <p>Ireland strongly supports the recommendation to build evidence on what is best for replacing energy from reduction in saturated fat</p>
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
<p>Page 32: Ireland notes that the evidence from prospective studies was not strong for the association of ruminant trans fatty acids with cardiovascular disease and mortality. Furthermore in RCTs the beneficial effects on blood lipids was only evident when ruminant trans fatty acids were replaced with PUFA. This suggests that this may relate to the strong lipid lowering effects of PUFA rather than the deleterious effects of ruminant trans fatty acids. Ireland would like this to be discussed</p>
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
<p>Page 40:</p> <p>Ireland would question making no distinction between ruminant and industrially produced trans fatty acids in the recommendations given the low intake of ruminant trans fatty acids. Ireland acknowledges the intent is to prevent replacement of industrial trans with ruminant trans but feels the recommendations should clarify this. Ireland is concerned that without this distinction foods that are important in the Irish diet for calcium and iron (i.e. dairy and meat) will be negatively affected.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

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## Final comments

Please provide any final thoughts or comments below.
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<p>Thank you for all the hard work that went into this report. Public Health and Healthcare Professionals in Ireland have had to undertake significant work in recent years to disentangle the mixed findings reported in scientific literature about saturated fat. Our research is supported by this report. A cornerstone of the evidence-base for Healthy Eating in Ireland is the reduction in trans fatty acids from industrial sources and a reduction in saturated fat to 10% of energy intake (this saturated fat target ensures ruminant trans fatty acids remain low)</p>
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## Survey response 64

### General information

Family/last name	Hanson
Given/first name	Chacanne
Organization/affiliation	Nestlé S.A.
Sector	Private sector
Sector [Other]	
Country	Switzerland

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>We would like to thank the World Health Organization (WHO) for the opportunity to comment on the “Draft Guidelines on Saturated Fatty Acid and Trans-Fatty Acid Intake for Adults and Children” and provide feedback.</p> <p>We support the recommendation that 10 per cent or less of total energy intake comes from saturated fats and 1 per cent or less from trans-fats. In line with our global commitment on product formulation and innovation (<a href="https://www.nestle.com/csv/impact/commitments">https://www.nestle.com/csv/impact/commitments</a>), we fully support the efforts of the WHO, governments and the public health community to reduce the overall consumption of saturated fat and to eliminate iTFA from partially hydrogenated oils (PHOs).</p> <p>This is line with our commitment as part of the International Food and Beverage Alliance, which pledged to have no more than one gram trans-fatty acid per 100 grams of product by the end of 2018 at the latest. In support of both WHO Guidelines recommending a maximum 1% total energy intake from all trans-fat and an intake of saturated fat not exceeding 10% of total energy intake, IFBA member companies will seek wherever possible to replace PHOs with unsaturated fats. PHOs can also be found in thermal processing, processing aids (release agents), food additives (emulsifiers), and carriers; however, these are used in very small amounts during production and resulting PHO traces in a final product are incidental and nutritionally negligible. Nonetheless, we will work with our supply chain partners with the aim of removing PHOs also from these sources wherever feasible. See the complete IFBA-STATEMENT here: <a href="http://thenest-eur-hq.nestle.com/EM/EM_EMPL/Life-at-my-Workplace/Events-and-Local-News/Events%20%20Local%20News%20documents/IFBA%20-%20statement.pdf">http://thenest-eur-hq.nestle.com/EM/EM_EMPL/Life-at-my-Workplace/Events-and-Local-News/Events%20%20Local%20News%20documents/IFBA%20-%20statement.pdf</a></p> <p>IFBA member companies, including Nestlé, are further committed to working in collaboration with governments, health authorities, civil society and food and beverage industry associations to share best practices and help guide other companies, particularly Small and Medium Enterprises, through the process of substituting PHOs. We call on food producers in all sectors to take prompt action and we stand ready to support effective measures to work toward the elimination of industrially produced trans fat and to ensure a level playing field in this area.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
-

Background (same for saturated fatty acids and trans-fatty acids)
<p>p3; We suggest to reference the range of intake for ruminant trans-FA to give a better appreciation on the extent to which intake levels are very low (ref 9). Data are available in ref. 57</p> <p>p17: It would be important to emphasize the difference between effects of whole grain vs. refined</p> <p>p17: Recommendations to further limit SFA&lt; 10% E and TFA&lt; 1% E may have important unintended consequences on the consumption of whole/partially skimmed milk and dairy products, and may lead to their further decrease of intake in the general population. Dairy products are a valuable source of calcium and high quality proteins. We recommend including a comment on milk /dairy consumption, for example in the Remarks, both for SFA and TFA.</p>
Guideline development process (same for saturated fatty acids and trans-fatty acids)
-
Summary of evidence (for saturated fatty acids only)
<p>p6; The evidence for SFA and risk of CVDs and mortality in adults is taken from one meta-analysis (MA) of RCTs that identified 15 trials (ref 24); this review is driving the recommendations made for SFAs. Other MA with slightly different inclusion criteria are existing (e.g. ref 4), leading to different trials being included, and thus different conclusions. We noted that these MA were conducted with high quality methodology but provided only evidence of "moderate" quality and thus they do not allow to conclude that decreasing SFA intake will decrease CVD risk as a general statement. Indeed the available nutritional studies were multifactorial by design: control and intervention diets are not isoenergetic, the control SFA group is not always at a physiological level (e.g. until 35%), there is no information on which SFA has been decreased (SFA are not all atherogenic and some may have different beneficial effects).</p> <p>In the same way that fat encompasses a broad family of compounds, carbohydrates include a wide variety of representatives. It would be misleading to group them under the denomination of "carbohydrates". We plead for a higher level of details, i.e. making wherever possible the distinction between available and non-available carbohydrates. We recommend to use the sentence "Although replacement with carbohydrates ... not possible" (page 10) earlier in the text, eg page 6.</p> <p>p22 Our remark refers to the section re "Meta-analysis of RCTs that included a control group with SFA intake greater than 10%": in the studies identified in Ref 24, the data on the SFA intake in the control group were available for only 10 of the studies. We recommend to mention this lack of information on SFA intake (the meta-analysis is made on more than 10 studies, therefore there is uncertainty on the level of SFA intake in some of the studies).</p> <p>p28: We recommend NUGAG reviews the results of individual SFAs. Indeed, lauric and myristic acid intake are low, but intake of palmitic and stearic are relatively high, therefore recommendations related to these individual SFAs would be meaningful from a public health perspective. Individual saturated fatty acids have different health impact and should be considered separately. In particular stearic acid does not have a significant impact on blood lipids as was also concluded in ref. 59 (effect evaluated on 52 trials).</p>
Recommendations (for saturated fatty acids only)
<p>Same comments as that for the executive summary</p> <p>p8 . While we support the recommendation 1 (decrease SFA intake when intake greater than 10%), we think the difference between recommendation 1 and 2 (suggesting to further decrease SFA&lt; 10% E) is not clear. In addition, the grading of the recommendation as "conditional" is not easy to translate into practical guidance for consumers nor actionable industry guidelines.</p>
Remarks (for saturated fatty acids only)
<p>p8: We suggest to include definitions and examples of "surrogate endpoints" and "intermediate outcome markers"</p> <p>We question the extrapolation of adult data to children as a basis for the recommendations (especially the data on CVD) and recommend further research in this area in particular building health data for children, as it has the potential to drive innovation in children products.</p> <p>p9: The 3rd paragraph mentions that in RCTs assessing CV outcomes, when SFA were replaced with PUFA they were replaced primarily with plant-based oils rich in linoleic acid. However we wonder if this statement can be made if: (1) many studies do not inform on the PUFA that were consumed; (2) in the studies that mention what is the replacement PUFA, 3 studies replace with mostly n-6 (e.g. corn oil) while 2 studies replace with a mix of n-6 and n-3 (soy oil and soy oil + fish) and one study mentions EPA (i.e. n-3)</p> <p>p10. The sentence "None of the RCTs included .... compared direct replacement nutrients to one another" would require further explanations</p> <p>p10 Second bullet point: we suggest to indicate if the studies with CV events were conducted with either LA (n-6) or ALA or EPA + DHA (n-3) as PUFA</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
-
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
-

Annexes 1, 6, 7 (for saturated fatty acids only)
p 51, 59; In these profiles related to children population, it is not clear why outcome assessed in adults are reported. In addition, they are not commented in the corresponding text.
p 51, 55, 59: Data for total cholesterol, although available and statistically significant (see e.g. page 28) are lacking in the tables.
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>We would like to thank the World Health Organization (WHO) for the opportunity to comment on the “Draft Guidelines on Saturated Fatty Acid and Trans-Fatty Acid Intake for Adults and Children” and provide feedback.</p> <p>We support the recommendation that 10 per cent or less of total energy intake comes from saturated fats and 1 per cent or less from trans-fats. In line with our global commitment on product formulation and innovation (<a href="https://www.nestle.com/csv/impact/commitments">https://www.nestle.com/csv/impact/commitments</a>), we fully support the efforts of the WHO, governments and the public health community to reduce the overall consumption of saturated fat and to eliminate iTFA from partially hydrogenated oils (PHOs).</p> <p>This is line with our commitment as part of the International Food and Beverage Alliance, which pledged to have no more than one gram trans-fatty acid per 100 grams of product by the end of 2018 at the latest. In support of both WHO Guidelines recommending a maximum 1% total energy intake from all trans-fat and an intake of saturated fat not exceeding 10% of total energy intake, IFBA member companies will seek wherever possible to replace PHOs with unsaturated fats. PHOs can also be found in thermal processing, processing aids (release agents), food additives (emulsifiers), and carriers; however, these are used in very small amounts during production and resulting PHO traces in a final product are incidental and nutritionally negligible. Nonetheless, we will work with our supply chain partners with the aim of removing PHOs also from these sources wherever feasible. See the complete IFBA-STATEMENT here: <a href="http://thenest-eur-hq.nestle.com/EM/EM_EMPL/Life-at-my-Workplace/Events-and-Local-News/Events%20%20Local%20News%20documents/IFBA%20-%20statement.pdf">http://thenest-eur-hq.nestle.com/EM/EM_EMPL/Life-at-my-Workplace/Events-and-Local-News/Events%20%20Local%20News%20documents/IFBA%20-%20statement.pdf</a></p> <p>IFBA member companies, including Nestlé, are further committed to working in collaboration with governments, health authorities, civil society and food and beverage industry associations to share best practices and help guide other companies, particularly Small and Medium Enterprises, through the process of substituting PHOs. We call on food producers in all sectors to take prompt action and we stand ready to support effective measures to work toward the elimination of industrially produced trans fat and to ensure a level playing field in this area.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
-
Background (same for saturated fatty acids and trans-fatty acids)
<p>p3; We suggest to reference the range of intake for ruminant trans-FA to give a better appreciation on the extent to which intake levels are very low (ref 9). Data are available in ref. 57</p> <p>p17: It would be important to emphasize the difference between effects of whole grain vs. refined</p> <p>p17: Recommendations to further limit SFA&lt; 10% E and TFA&lt; 1% E may have important unintended consequences on the consumption of whole/partially skimmed milk and dairy products, and may lead to their further decrease of intake in the general population. Dairy products are a valuable source of calcium and high quality proteins. We recommend including a comment on milk /dairy consumption, for example in the Remarks, both for SFA and TFA.</p>
Guideline development process (same for saturated fatty acids and trans-fatty acids)
-
Summary of evidence (for trans-fatty acids only)
<p>p32: Footnote 4: it seems that the references should be 75 (Motard-Belanger) and 76 (Gebauer) (and not 74 and 75); also, at the end of footnote 4, ... "reported larger reductions in LDL cholesterol" - the comparison should be explained clearly. does it mean larger reduction with ruminant TFA as compared to industrially-produced TFA?</p> <p>p35: The second paragraph of this section is a repetition of the second paragraph of this page and relates to the previous section on “TFA intakes of less than 1% of total energy intake”.</p> <p>p36: Results of regression analyses of increasing intakes should be opposite but of the same magnitude as results of decreasing intakes. It seems that results related to industrial TFA were used in some instances (ex. in the text and in GRADE evidence profile 15 and 16, from ref 73) and results of total TFA were used in other instances (profile 17).</p>

Recommendations (for trans-fatty acids only)
<p>Same comments as that for the executive summary</p> <p>p11: Recommendations should also mention to what extent MUFA could be considered as a replacement solution. Indeed, the available evidence clearly demonstrates that replacement with MUFAs lowers LDL-C to roughly the same extent PUFAs do; also, the 3rd recommendation (to replace with PUFA) could have the undesirable consequence to increase the intake of linoleic acid, a tendency that is currently debated as potentially decreasing EPA and DHA in body tissues (Blasbalg TL, Hibbeln JR, Ramsden CE, Majchrzak SF, Rawlings RR. Changes in consumption of omega-3 and omega-6 fatty acids in the United States during the 20th century. Am J Clin Nutr. 2011 May;93(5):950-62. doi: 10.3945/ajcn.110.006643); finally, proposing more than one type of replacement would be a plus in term of feasibility (more than one choice, use of MUFA rather than PUFA in products more sensitive to oxidation).</p> <p>p 40: In note 1, the study of ref. 28 is presented as a meta-analysis, but it is not. Ref. 28 is a single study that administered high doses of CLA, not representative of normal intake. The right reference should be added or CLA should be excluded from TFA definition.</p> <p>An additional recommendation to reduce iTFA by removing partially hydrogenated oils (PHOs) in foods products should be added. 1. Elimination of Partially Hydrogenated Oils (PHOs) in the Food Supply to help consumers reaching the WHO recommendation to limit the daily consumption of trans fat to at most 1% of total energy intake within the daily diet. As Nestlé, we already committed to do so.</p>
Remarks (for trans-fatty acids only)
<p>p12: In the remark related to the second recommendation for TFA, the notion of “confidence” would need to be more clearly explained. For example, for recommendation 2, it is not clear why the confidence is “diminished because of the few events occurring in studies with up to 21 years follow-up “ while the number of events was not challenged for the TFA recommendation 1 while being comparable. Indeed, from the GRADE evidence profiles 15 and 16, the study event rates do not appear very different.</p> <p>p13: In the remark related to the 4th recommendation for TFA, same comment as for 2nd recommendation above: when comparing GRADE evidence profiles 15 and 21, the study event rates does not appear very different.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
<p>p 51, 59; In these profiles related to children population, it is not clear why outcome assessed in adults are reported. In addition, they are not commented in the corresponding text.</p> <p>p 51, 55, 59: Data for total cholesterol, although available and statistically significant (see e.g. page 28) are lacking in the tables.</p>
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 65

### General information

Family/last name
Garg
Given/first name
Manohar
Organization/affiliation
University of Newcastle
Sector
Academic/research
Sector [Other]
Country
Australia

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
My comments will be submitted as part of a group response led by Professor Arne Astrup.

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## Survey response 66

### General information

Family/last name
Lawrence
Given/first name
Anita
Organization/affiliation
Dairy Australia
Sector
Non-governmental agency
Sector [Other]
Country
Australia

### Saturated fatty acids

## Executive summary (same for saturated fatty acids and trans-fatty acids)

Paragraph 1, page 1: in 2016, Mozaffarian (1) stated 'A cardioprotective diet pattern must be characterized by the healthful foods that are included, not simply specific items to be avoided.'

While noncommunicable diseases (NCDs) are important, health is more than the absence of NCDs. With any dietary guidance, it is important to 'first do no harm' and to ensure that nutritional adequacy is not compromised. This does not appear to have been fully considered in the development of these guidelines. For example, there appears to have been a lack of consideration of the impact of reduced intake of dairy foods (as a result of these foods being labelled unhealthy due to their saturated fat content) on intakes of key nutrients such as calcium (which is important for bone accrual in children(2)) and iodine (which is important for preventing cognitive impairment in children(3,4)).

The statement in paragraph 1, page 1: 'Dietary saturated fatty acids and trans-fatty acids are of particular concern because high levels of intake are correlated with increased risk of CVDs (2)' should be amended, as causation cannot be implied from correlations.

Paragraph 2, page 5 (The evidence): The report should clearly report that three primary outcomes and a number of secondary outcomes were set in the 2015 review by Hooper and colleagues, and that there was no clear effect of reducing saturated fat intake for two out of three of the primary outcomes (total mortality and cardiovascular mortality). At present, it is not clear from the text which results are the primary and secondary outcomes. It would also be helpful to reference the systematic reviews being referred to in this paragraph and the next.

Paragraphs 1 and 2, page 7: it would be helpful to include the references for the meta-analysis of prospective cohort studies and the meta-regression analysis of RCTs.

Paragraph 3, page 7: it is quite remarkable that trans-fatty acid recommendations are being proposed for children and yet no studies were identified that met the inclusion criteria established for the systematic review of studies conducted in children.

Page 8, saturated fat recommendation 1: 'In adults and children whose saturated fatty acid intake is greater than 10% of total energy intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake (strong recommendation)'. This suggests any person over the age of two years with a saturated fat intake higher than 10.0% of energy should reduce their saturated fat intake and that it does not matter which foods are targeted. We are concerned about possible unintended consequences associated with this advice, such as intakes of under-consumed nutrients being further compromised.

We would also question the appropriateness of this recommendation for frail elderly people. The Australian Dietary Guidelines (5) make it clear that their guidelines are 'not appropriate for frail elderly people or those with complex health conditions and an appropriate health professional should be consulted' (page 27).

We suggest that if this recommendation goes ahead, the wording of this recommendation is amended to the following:

'In adults and children over 2 years whose saturated fatty acid intake is greater than 10% of total energy intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake in a way that ensures that the individual's other nutritional and dietary requirements are met (strong recommendation<sup>2</sup>). This recommendation does not apply to frail elderly people or those with complex health conditions.'

However, we would also question the scientific basis for this recommendation as the majority of studies that form the evidence for this were undertaken in adults who already had cardiovascular disease or diabetes. Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, 70% of the data behind the strong recommendation for all adults and children 'to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake' comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children.

Page 8, saturated fat recommendations 2-4: According to footnote 3, 'Conditional recommendations are those recommendations for which the WHO guideline development group is uncertain that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders (22)'.

If it is the case that there is uncertainty about whether desirable consequences outweigh undesirable consequences, we think it would be better to make no recommendations. WHO's reputation and credibility are likely to be damaged if it recommends a dietary change that is later shown to have undesirable consequences. The sugar recommendations released by the WHO in 2015 have been widely referenced by policy makers around the world, but little distinction has been made between the strong and the conditional recommendations.

Page 8, Remark 2: It is good that this remark makes clear that the recommendations for children are based on blood lipid and blood pressure measures and that the rest of the evidence is an extrapolation of adult data to children. However, we do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).



A 2015 study by Janiaud and colleagues (6) found different treatment benefits estimated by clinical trials in adults compared with those performed in children for 11% of drugs (14 out of 124). A commentary (7) on the paper pointed out that 'kids are no little adults and not all kids are the same.' They added 'although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.' We agree – the world's children deserve better evidence for the dietary recommendations affecting them.

Paragraph 2, page 9: It is stated that 'The evidence indicates that children's growth is not compromised by reduction of saturated fatty acid intake.' Growth is more than the variables measured (height, body weight, BMI and other measures of adiposity). There was not consideration of other aspects of growth such as bone accrual – indeed the authors of the Special Turku Coronary Risk Factor Intervention Project (STRIP) commented that 'further studies are needed to draw further conclusions about the influence of dietary counselling on bone health'. Therefore, we feel that the statement should be amended to:

'The evidence indicates that children's height and body weight are not compromised by reduction of saturated fatty acid intake. Effects on other aspects of growth such as bone accrual are unknown.'

Paragraph 3, page 9: Here the text states that: 'in the RCTs that assessed cardiovascular outcomes and mortality outcomes (Hooper et al., 2015) saturated fatty acids were largely replaced by polyunsaturated fatty acids, the polyunsaturated fatty acids were primarily from plant-based oils, rich in linoleic acid'.

This statement is not consistent with Ramsden and colleagues (9) Webtable 6, which indicates that three studies selectively increased n-6 linoleic acid (LA) without a concurrent increase in n-3 PUFAs and five studies were mixed n-3/n-6 PUFA interventions that increased n-3 PUFAs and n-6 LA. Ramsden and colleagues conclude that: 1) the specific PUFA composition of dietary interventions is a critical determinant of clinical CVD outcomes, and 2) selective substitution of n-6 LA for SFA is unlikely to be beneficial, particularly in patients with established CHD.' Commenting on this paper, Professor Philip Calder wrote 'The more cautious UK dietary recommendations on fat and fatty acids, which include the statement, "There is reason to be cautious about high intakes of omega 6 PUFAs" seem fully justified in light of the current study's findings.' In contrast, the Hooper meta-analysis did not appear to consider differences between the effects of n-6 LA and mixed n-3/n-6 PUFA interventions. We believe that there should be greater consideration of the results in the context of the concerns expressed about n-6 LA and the statement in question should be amended to:

'In the RCTs that assessed cardiovascular outcomes and mortality outcomes (Hooper et al., 2015) saturated fatty acids were largely replaced by polyunsaturated fatty acids, the polyunsaturated fatty acids were from plant-based oils, rich in linoleic acid and mixtures of n-3 and n-6'.

Paragraph 5, page 9: Here is a classic example of 'cherry picking' findings to suit a particular argument, rather than presenting a balanced view. Three pre-determined primary outcomes were selected and the evidence for all three outcomes was judged to be of moderate quality using GRADE (page 3 of Hooper et al., 2015).

These three results (all-cause mortality, CVD mortality and combined CV events) should always be given equal weight in the WHO recommendations. At present, there is disproportionate emphasis on the finding that reducing saturated fat leads to reduced combined CV events and not on the fact that it makes no significant difference to the risk of all-cause mortality or CVD mortality. In light of this, it is difficult to see how it is justifiable to make a strong recommendation to reduce saturated fatty acids when it is likely to make no difference to two out of the three pre-specified main outcomes. Paragraph 5 on page 9 even refers to the 'totality of evidence reviewed' but then fails to report the results for two out of three of the main outcomes. We therefore recommend that this paragraph be re-written in a way that provides a more balanced representation of the results.

The WHO-commissioned systematic review and meta-analysis of observational studies examining intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes (10) concluded that 'the certainty of associations between saturated fat and all outcomes was "very low"' yet the draft WHO recommendations are suggesting that there is enough evidence for a strong recommendation to reduce saturated fat intake. Again, this appears to be cherry picking the results to support preferred outcomes.

Paragraph 6, page 9: This paragraph justifies reducing saturated fatty acid intake on the basis of LDL cholesterol levels in adults and children. However, consideration of LDL cholesterol levels in isolation of other factors is meaningless. For example, 16:0 raises LDL cholesterol, but also raises HDL-cholesterol, reduces triglyceride-rich lipoproteins and remnants and has little effect on Apo-B. Mozaffarian (2016) commented: 'Continued prioritization of saturated fat reduction appears to rely on selected evidence: e.g. effects on LDL-cholesterol alone (discounting the other, complex lipid and lipoprotein effects); historical ecological trends in certain countries (e.g. Finland) but not in others; and expedient comparisons with polyunsaturated fat, the most healthful macronutrient.'

Paragraph 2, page 10: The 2013 Australian Dietary Guidelines (11) state, 'Foods exert certain health effects because of the nutrients they contain. As such, dietary recommendations are often couched in terms of individual nutrients (such as requirements of vitamins and minerals). However, people eat foods rather than single nutrients, so such advice can be difficult to put into practice. For this reason, these Guidelines make recommendations based only on whole foods, such as vegetables and meats,

rather than recommendations related to specific components and individual nutrients'. We recommend that the WHO also focuses on making recommendations based on whole foods rather than nutrients. We believe that the recommendation to replace saturated fatty acids with polyunsaturated fatty acids is difficult to put into practice. For example, people would not have a vegetable oil sandwich rather than a cheese sandwich, they would not drink vegetable oil rather than milk and they would not eat a vegetable oil dessert rather than a yogurt. The focus on individual nutrient plays into the promotion of ultra-processed foods, which in turn are linked with chronic diseases (12).

We also recommend that greater consideration be given to the differing effects of n-3 and n-6 PUFA reported by Ramsden and colleagues (13) before a recommendation is given to simply replace saturated fatty acids with (unspecified) PUFA.

Paragraph 3, page 10: The omission of the PREDIMED study from the review by Hooper and colleagues (2015) highlights the narrowness of the work used to underpin these guidelines. While we appreciate the rationale for the work, we think it would have been better to have focused on dietary patterns and to make dietary recommendations in relation to these, not single nutrients. We recommend that greater consideration is given to dietary patterns and that any recommendations related to saturated fat intake are set within the context of these findings.

Paragraph 4, page 10: Although it is stated that 'the recommendation to not increase saturated fatty acid intake if intake is already below 10% of total energy intake (fourth recommendation) is based on the totality of evidence reviewed, a major omission is consideration of the impact of such a recommendation on the frail elderly. It is well acknowledged that the frail elderly do not require a low fat diet. Focus on the saturated fat intake of such people, as a result of this proposed WHO recommendation, could well reduce their access to nutrient-dense, protein-providing foods such as cheese and whole milk. Also, children between the ages of 2 and 5 years often consume higher amounts of dairy foods than older children and recommendation four has the potential to adversely impact on the provision of these foods in pre-school settings.

Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out (14), they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed WHO saturated fat guidelines will adversely impact on intake of milk and dairy foods around the world.

Paragraph 2, page 11: As pointed out by Mozaffarian (15), 'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.' We recommend that the WHO makes future recommendations in relation to dietary patterns rather than single nutrients.

We also believe that the statement: 'public health interventions should aim to reduce saturated fat intake, while reducing total fat intake where necessary, and without increasing free sugar intake' could detrimentally impact on the nutritional quality of the diet and should be amended to:

'public health interventions aimed at reducing saturated fat intake should ensure that nutritional adequacy is not compromised and that free sugar intake is not increased'

Recommendation 1, page 11: 'In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing trans-fatty acid intake (strong recommendation<sup>2</sup>)'. In light of Figure 4 from the WHO-commissioned systematic review by de Souza and colleagues which clearly demonstrates that consumption of ruminant trans fats over 5-20 years is not associated with any adverse health outcomes and that its consumption is associated with reduced risk of type 2 diabetes, we suggest that this recommendation be amended to:

'In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing industrial trans-fatty acid intake (strong recommendation)'.<sup>1</sup>

The conclusions of the second WHO-commissioned systematic review (16) of RCTs relies on theoretical calculations ('coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA') and regression analysis that was not weighted for study size. Why was such a different analysis technique used for the systematic review of RCTs for saturated fatty acids and the systematic review of RCTs for trans fatty acids?

We would also highlight the fact that the RCTs lasted for between 14 and 56 days yet it expected that people will follow the WHO recommendations for years.

Our suggested revised wording would be less confusing to the general public, as both the WHO's dietary recommendations and the WHO's REPLACE initiative announced in May 2018, would focus on artificial trans fatty acids.

Recommendations 2-4, page 11: Similarly, we recommend that industrial trans-fatty acids rather than all trans-fatty acids are specified in the three conditional recommendations.

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1. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225
  2. Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. *Osteoporosis Int* 27,1281-1386
  3. Velasco I et al., (2018) Iodine as essential nutrient during the first 1000 days of life. *Nutrients* 10, 290
  4. Bath SC et al., (2017) Iodine concentration of milk-alternative drinks available in the UK. *Br J Nutr* 118, 525-32
  5. National Health and Medical Research Council (2013) Australian Dietary Guidelines. Canberra: National Health and Medical Research Council.
  6. Janiaud P et al., (2015) Different treatment benefits were estimated by clinical trials performed in adults compared with those performed in children. *Journal of Clinical Epidemiology* 68, 1221-31.
  7. Oostenbrink R & de Wildt SN, (2016) Drug trials: Kids are no little adults and not all kids are the same. *Journal of Clinical Epidemiology* 71, 111-12.
  8. Paakkunainen U et al., (2002) The Impact of Low-Saturated Fat, Low Cholesterol Diet on Bone Properties Measured Using Calcaneal Ultrasound in Prepubertal Children. *Calcified Tissue International*, 71, 219-226.
  9. Ramsden C et al., (2013) Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. *BMJ* 2013, 346 e8707.
  10. De Souza RJ et al., (2015) Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 351:h3978.
  11. National Health and Medical Research Council (2013) Australian Dietary Guidelines. Canberra: National Health and Medical Research Council.
  12. Monterio C et al., (2017) The UN decade of nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutrition* 21, 5-17.
  13. Ramsden C et al., (2013) Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. *BMJ* 2013, 346 e8707.
  14. Food and Agriculture Organization of the United Nations (2013) Milk and dairy products in human nutrition. Rome: FAO.
  15. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225
  16. Brouwer IA (2016) Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis. Geneva: World Health Organization.

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Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Paragraph 2, page 15: We believe that the scope and purpose of the guidelines require an update. As pointed out by Mozaffarian (17), 'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.'

We recommend that the WHO makes future recommendations in relation to dietary patterns rather than single nutrients. Focusing on a single nutrient to prevent CVD in adults and children is taking a rather 20th rather than 21st century approach. Mozaffarian added 'Thus, the present period is one of exciting, rapid transition away from single-nutrient theories and simple surrogate outcomes and toward foods, dietary patterns and evaluation of clinical end points. This transition forms the basis for our modern understanding of diet and cardio-metabolic health.'

Paragraph 3, page 15: The recommendations state 'It is hoped that the guidelines will also help to accelerate the implementation of actions to promote healthy diets, improve health and nutritional status of all people, and ultimately reduce the burden of NCDs to help accelerate achievement of the Sustainable Development Goals.' We share this hope, but fear that the proposed guidelines will reduce the health and nutritional status of all people by labelling nutritious and healthy foods such as milk, yogurt and cheese as foods to limit, in order to reduce saturated fat intake. Unless changes are made to the wording of the recommendations, they have the potential to promote unhealthy diets and reduce the health and nutritional status of all people by for example, reducing intake of nutritious foods that contain saturated fat such as whole milk, yogurt and cheese.

17. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

#### Background (same for saturated fatty acids and trans-fatty acids)

Paragraph 4, page 16: While saturated fatty acids are found in the foods listed (butter, cow's milk, meat, salmon and egg yolks), just 3.2% of total saturated fat comes from butter in Australia, cows', sheep's and goats' milk provide 8.7%, meat, poultry and game products and dishes supply 19.6%, fish and seafood products and dishes supply 1.8% and egg products and dishes supply 1.8%. Thus, in total, these foods account for just 35% of the saturated fat in the Australian diet. In contrast, discretionary choices (foods and drinks not necessary to provide the nutrients the body needs, many of which are high in saturated fats, sugars, salt and/or alcohol) provided 49% of the saturated fat within the Australian diet of children aged 2-18 years (18). We suggest that the text focuses more on these sources of saturated fat and less on those that are naturally occurring, particularly butter, milk, fish and eggs.

Paragraph 4, page 16: it would be helpful to point out that not all saturated fatty acids are the same. As stated by Professor Mozaffarian (19):

"Saturated fat represents a highly heterogeneous category of fatty acids, with chain lengths ranging from 6 to 24 carbons, deriving from diverse foods, and possessing dissimilar biology. For instance, palmitic acid (16:0) exhibits in vitro adverse effects; whereas medium-chain (6:0–12:0), odd-chain (15:0, 17:0), and very long-chain (20:0–24:0) saturated fats may have metabolic benefits. This biological and metabolic diversity does not support the grouping together of all saturated fatty acids based on only one chemistry characteristic: the absence of double bonds."

"Judging a food or a person's diet as harmful because it contains more saturated fat, or as beneficial because it contains less, is unsound. This is consistent with the many longitudinal cohort studies demonstrating largely neutral effects of overall saturated fat intake. Consistent with this, meats higher in processing and sodium, rather than saturated fat, are most strongly linked to CHD. Cheese, a leading source of saturated fat, is also linked to neutral or even beneficial effects on CHD and diabetes mellitus. In sum, these lines of evidence – complex lipid effects including little influence on ApoB, no relation of overall intake with CHD, and no observed cardiovascular harm for most major food sources – provide powerful and consistent evidence for absence of appreciable harms of total saturated fat."

Paragraph 1, page 17: We are pleased to see acknowledgement that 'different saturated fatty acid-containing foods, such as dairy foods, may have differing effects on risk of cardiovascular diseases and type 2 diabetes, either as a result of differing composition of saturated fatty acids across foods, other constituents of the foods, or a combination of the two' however, we are disappointed that no consideration of this is apparent in the resulting recommendations.

Paragraph 3, page 17: The bottom paragraph on page 17 cites five papers. Four were from the previous century and the other is more than 10 years old. We recommend that more recent scientific papers are used to support the statements made, in order to take into account contemporary scientific thinking on the relationship between childhood diet and risk of future CVD, particularly in light of rising rates of obesity and type 2 diabetes.

18. Johnson BJ et al., (2017) Contribution of discretionary foods and drinks to Australian children's intake of energy, saturated fat, added sugars and salt. *Children (Basel)* 4, 104 doi: 10.3390/children4120104

19. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

#### Guideline development process (same for saturated fatty acids and trans-fatty acids)

Paragraph 2, page 19: With the focus on reduction of CVD, it is surprising that no cardiologists were invited to participate in discussions. In light of the aging population of many countries, it would have also been helpful to include a specialist in elderly nutrition in the subject matter experts.

Paragraph 4, page 19: It is mentioned that the role of the NUGAG Subgroup on Diet and Health included advice on the 'balance of benefits and harms.' We believe that this is an area that requires greater consideration. The official endorsement of the low-fat diet in the 1980s and 1990s led to rising rates of overweight and type 2 diabetes, as consumers incorrectly interpreted the advice as being a licence to eat what you want, when you want as long as it is low in fat.

Consideration is required about how consumers and policy makers will interpret the proposed guidelines related to saturated and trans fatty acid intakes. It could well lead to the promotion of diets that are lacking in essential nutrients such as calcium and iodine, as milk and cheese consumption are discouraged due to their saturated and trans fat contents (20).

20. <https://www.foodnavigator.com/Article/2018/05/07/WHO-guidelines-on-saturated-fats-and-trans-fats> visited 31/5/18

#### Summary of evidence (for saturated fatty acids only)

Paragraph 2, page 19: It is difficult to understand why type 2 diabetes and adiposity were priority health outcomes for children but not adults. We recommend that further work be undertaken considering these outcomes for adults. We also recommend that further consideration be given to the impact of reducing saturated and trans fat intake on frail elderly people (e.g. in terms of sarcopenia, falls, fractures and bone health), and on bone accrual in children. In addition, greater consideration is required of the impact on the nutritional adequacy of the diet when a general recommendation to reduce saturated and trans fat intake is given. Does it lead to lower meat and dairy intake? What are the impacts on nutrient intakes?

Footnote 1, page 21: The rationale for not considering the results of the WHO commissioned systematic review of cohort studies (21) in the formulation of recommendations on saturated fat appears to be weak, particularly as it was considered in the formulation of recommendations for trans fats. The systematic review of RCTs (22) and the systematic review of prospective cohort studies both concluded that saturated fat intake was not related to risk of all-cause mortality or CVD mortality. This appears to be another example of 'cherry picking' the results to support a preferred position.

Footnote 2, page 21: The rationale for including LDL as a 'critical outcome' is somewhat questionable. As Professor Ron Krauss explained to the Committee on Quantification of Biomarkers and Surrogate Endpoints in Chronic Disease (23): 'CVD is a very complex disease that is increasingly recognized as a spectrum of pathologic and pathophysiologic effects, only one of which is primarily related to progression of the cholesterol content of plaques as a function of LDL-C in the blood'. Atherosclerosis, he added, is "often indolent, progressive over time, and then is complicated by a number of additional factors that can convert a cholesterol-rich plaque to a more malignant form that destabilizes and is involved with both inflammation and thrombosis; immune changes can occur that could be critical." Thus, he concluded, "it is rather simplistic to consider either LDL or HDL, or even the two of them together, as sufficient to explain these complex mechanisms."

Professor Kraus also noted that the predictive value of LDL-C for CVD events varies considerably as a function of health status, and risk for cardiovascular events associated with high LDL-C in patients without diabetes and CVD was found to be significantly lower than LDL-C-associated event risk in patients with both conditions (see figure from Robinson and Stone, 2006 (24)).

Page 22, paragraph 2: This paragraph notes that of the 17 comparisons made in the systematic review of RCTs, six included only people at high risk of CVDs, four included people at moderate risk and five included people at low risk. However, when it came to the evidence base for the primary outcome that is the basis for the strong recommendation (CV events), it should be noted that the vast majority of studies that form the evidence for this were undertaken in adults who already had cardiovascular disease or diabetes. Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, 70% of the data behind the strong recommendation for all adults and children 'to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake' comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children. Recommendations to the general (healthy population) should be based on the results of studies in the general (healthy population) – not on the results of secondary prevention trials.

Page 22, paragraph 2: Along with information about the saturated fat content of the diets consumed by both the intervention and control groups, we suggest that it should be noted that trans fat intake was either unclear or not reported in all of the comparisons apart from two (the WHI with CVD and the WHI without CVD). As it is well established that industrially produced trans fatty acids increase risk of CVD and that in many countries, industrially produced trans fat was present in margarines in the 1960s and 1970s – at the same time when some of these trials were undertaken, there should be greater consideration of trans fatty acids as a confounding factor. Discussing this issue in relation to the Sydney Diet Heart Study, Ramsden and colleagues commented that 'the restriction of common margarines and shortenings (major sources of trans fatty acids) in the intervention group would be expected to substantially reduce consumption of trans fatty acids compared with the control group.' However, as the composition of the margarine used in the intervention was unknown, it is possible that 'nutrients other than n-6 LA and SFAs could have contributed to, or reduced, the observed unfavourable effects of the LA intervention.' In light of the possibility of confounding by industrially produced trans fatty acid intake in all but two comparisons, greater emphasis should be placed on the results of these two comparisons (from the Women's Health Initiative) where the trans fat intake of the subjects is known and can be adjusted for.

Page 25, paragraph 1: It is stated that 'Significant reductions in risk of CVD mortality (RR 0.69; 95% CI: 0.51, 0.94) and cardiovascular events (RR 0.79; 95% CI: 0.62, 0.99) were observed in meta-analysis of two trials with 979 participants in which saturated fatty acid intake was reduced to less than 9% of total energy intake.' Again, this is another example of cherry picking the data. Out of the 46 comparisons made in table 23 of Hooper et al., 2015, just these two results did not have confidence intervals spanning 1.0. The text fails to mention that no clear effect on any cardiovascular or mortality outcome was apparent when saturated fatty acid intake was less than 8 or 7% E. Similarly, there is no mention that a saturated fatty acid intake of less than 9% E had no effect on all cause mortality, myocardial infarction, non fatal myocardial infarction, stroke, CHD mortality and CHD events. It seems odd that no adjustments appear to have been made for multiple comparisons.

Page 28, paragraph 2. We also recommend that further consideration be given to the impact of reducing saturated and trans fat intake on bone accrual in children. In addition, greater consideration is required of the impact on the nutritional adequacy of the diet when a general recommendation to reduce saturated and trans fat intake is given. Does it lead to lower meat and dairy intake? What are the impacts on nutrient intakes?

Page 28, paragraph 2. We note that the trials considered in the systematic review of RCTs included some children and adolescents with normal cholesterol levels and some with raised levels. Looking in more detail at Table 1 from Te Morenga & Montez (25), it would appear that 54% of participants were hyperlipidaemic and 46% were the general population that had not been tested, or were known to be normolipidaemic. As with the adults, we believe that the results from primary and secondary prevention trials should be considered separately, not together. Extrapolating the findings from hyperlipidaemic children to the general population may not be appropriate. Recommendations to the general population should be based on evidence from the general population – not those with specific medical conditions.

Page 29, paragraph 3. We find it quite remarkable that just one trial has appropriately tested the effects of a reduction in saturated fatty acid intake to 9% of total energy and yet the WHO is proposing to recommend that 7.5 billion children reduce their intake of saturated fat if it is above 10% E. It is unclear if the children who participated in the trial in question by Denke et al., (2000) were hyperlipidemic or not. The range of ages for the children tested was 8-16 years, so the results could well not be applicable to younger children. For all eight measures of bias, the study scored 'unclear risk of bias' (see Figure 6 from Morenga & Montez (26)). In addition, the trial lasted five weeks, yet the WHO recommendations are likely to be in place for years.

Page 30, paragraph 2. It is stated that 'evidence for cardiovascular, mortality and blood lipid outcomes from adults was also considered when formulating the recommendations for children, without downgrading for indirectness.' According to the GRADE handbook 'Direct evidence consists of research that directly compares the interventions which we are interested in, delivered to the populations in which we are interested, and measures the outcomes important to patients.' We do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues (27) found different treatment benefits estimated by clinical trials in adult compared with those performed in children for 11% of drugs (14 out of 124). A commentary (28) on the paper pointed out that 'kids are no little adults and not all kids are the same.' They added 'although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.' We agree – the world's children deserve better evidence for the dietary recommendations affecting them.

21. De Souza RJ et al., (2015) Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 351:h3978.

22. Hooper L et al., (2015) Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database Syst Rev* 6, CD011737

23. <https://www.ncbi.nlm.nih.gov/books/NBK209569/#ddd00018> visited 31/5/18

24. Robinson JG & Stone NJ (2006) Identifying patients for aggressive cholesterol lowering: the risk curve concept. *Am J Cardiol* 98, 1405-8.

25. Te Morenga L & Montez JM (2017) Health effects of saturated and trans-fatty acid intake in children and adolescents: systematic review and meta-analysis. *PLOS one* Nov 17, 2017.

26. Te Morenga L & Montez JM (2017) Health effects of saturated and trans-fatty acid intake in children and adolescents: systematic review and meta-analysis. *PLOS one* Nov 17, 2017.

27. Janiaud P et al., (2015) Different treatment benefits were estimated by clinical trials performed in adults compared with those performed in children. *Journal of Clinical Epidemiology* 68, 1221-31.

28. Oostenbrink R & de Wildt SN, (2016) Drug trials: Kids are no little adults and not all kids are the same. *Journal of Clinical Epidemiology* 71, 111-12.

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#### Recommendations (for saturated fatty acids only)

Page 37, Recommendation 1: 'In adults and children whose saturated fatty acid intake is greater than 10% of total energy intake, WHO recommends reducing saturated fatty acid intake (strong recommendation)'. This suggests any person over the age of two years with a saturated fat intake higher than 10.0% of energy should reduce their saturated fat intake and that it does not matter which foods are targeted. We are concerned about possible unintended consequences associated with this advice, such as intakes of under-consumed nutrients being further compromised.

We would also question the appropriateness of this recommendation for frail elderly people. The Australian Dietary Guidelines make it clear that their guidelines are 'not appropriate for frail elderly people or those with complex health conditions and an appropriate health professional should be consulted' (page 27).

We suggest that if this recommendation goes ahead, the wording of this recommendation is amended to the following:

'In adults and children over 2 years whose saturated fatty acid intake is greater than 10% of total energy intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake in a way that ensures that the individual's other nutritional and dietary requirements are met (strong recommendation<sup>2</sup>). This recommendation does not apply to frail elderly people or those with complex health conditions.'

However, we would also question the scientific basis for this recommendation as the majority of studies that form the evidence for this were undertaken in adults who already had cardiovascular disease or diabetes. Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, 70% of the data behind the strong recommendation for all adults and children 'to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake' comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children.

Page 37, Recommendations 2-4. According to footnote 3, 'Conditional recommendations are those recommendations for which the WHO guideline development group is uncertain that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders'.

If it is the case that there is uncertainty about whether desirable consequences outweigh undesirable consequences, we think it would be better to make no recommendations. WHO's reputation and credibility are likely to be damaged if it recommends a dietary change that is later shown to have undesirable consequences. The sugar recommendations released by the WHO in 2015 have been widely referenced by policy makers around the world, but little distinction has been made between the strong and the conditional recommendations.



Remarks (for saturated fatty acids only)
<p>Page 37, Remark 2: It is good that this remark makes clear that the recommendations for children are based on blood lipid and blood pressure measures and that the rest of the evidence is an extrapolation of adult data to children. However, we do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).</p> <p>A 2015 study by Janiaud and colleagues (31) found different treatment benefits estimated by clinical trials in adult compared with those performed in children for 11% of drugs (14 out of 124). A commentary (32) on the paper pointed out that 'kids are no little adults and not all kids are the same.' They added 'although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.' We agree – the world's children deserve better evidence for the dietary recommendations affecting them.</p> <p>Page 38, Paragraph 1: It is stated that 'The evidence indicates that children's growth is not compromised by reduction of saturated fatty acid intake.' Growth is more than the variables measured (height, body weight, BMI and other measures of adiposity). There was not consideration of other aspects of growth such as bone accrual – indeed the authors of the Special Turku Coronary Risk Factor Intervention Project (STRIP) commented (33) that 'further studies are needed to draw further conclusions about the influence of dietary counselling on bone health'. Therefore, we feel that the statement should be amended to:</p> <p>'The evidence indicates that children's height and body weight are not compromised by reduction of saturated fatty acid intake. Effects on other aspects of growth such as bone accrual are unknown.'</p> <p>We recommend that greater consideration be given to bone accrual prior to any recommendations being released.</p> <p>Paragraph 4, page 38: Here is a classic example of 'cherry picking' findings to suit a particular argument, rather than presenting a balanced view. Three pre-determined primary outcomes were selected and the evidence for all three outcomes was judged to be of moderate quality using GRADE (page 3 of Hooper et al., 2015).</p> <p>These three results (all-cause mortality, CVD mortality and combined CV events) should always be given equal weight in the WHO recommendations. At present, there is disproportionate emphasis on the finding that reducing saturated fat leads to reduced combined CV events and not on the fact that it makes no significant difference to the risk of all-cause mortality or CVD mortality. In light of this, it is difficult to see how it is justifiable to make a strong recommendation to reduce saturated fatty acids when it is likely to make no difference to two out of three main outcomes. Paragraph 4 on page 38 even refers to the 'totality of evidence reviewed' but then fails to report the results for two out of three of the main results. We therefore recommend that this paragraph be re-written in a way that provides a more balanced representation of the results.</p> <p>The WHO-commissioned systematic review and meta-analysis of observational studies examining intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes (34) concluded that 'saturated fats are not associated with all-cause mortality, CVD, CHD, ischaemic stroke, or type 2 diabetes' yet the draft WHO recommendations are suggesting that there is enough evidence for a strong recommendation to reduce saturated fat intake. Again, this appears to be cherry picking the results to support preferred outcomes.</p> <p>Paragraph 5, page 38: This paragraph justifies reducing saturated fatty acid intake on the basis of LDL cholesterol levels in adults and children. However, consideration of LDL cholesterol levels in isolation of other factors is meaningless. For example, 16:0 raises LDL cholesterol, but also raises HDL-cholesterol, reduces triglyceride-rich lipoproteins and remnants and has little effect on Apo-B. Mozaffarian (2016) commented: 'Continued prioritization of saturated fat reduction appears to rely on selected evidence: e.g. effects on LDL-cholesterol alone (discounting the other, complex lipid and lipoprotein effects); historical ecological trends in certain countries (e.g. Finland) but not in others; and expedient comparisons with polyunsaturated fat, the most healthful macronutrient.'</p> <p>Paragraph 3, page 39: Although it is stated that 'the recommendation to not increase saturated fatty acid intake if intake is already below 10% of total energy intake (fourth recommendation) is based on the totality of evidence reviewed, a major omission is consideration of the impact of such a recommendation on the frail elderly. It is well acknowledged that the frail elderly do not require a low fat diet. Focus on the saturated fat intake of such people, as a result of this proposed WHO recommendation, could well reduce their access to nutrient-dense, protein providing foods such as cheese and whole milk. Also, children between the ages of 2 and 5 years often consume higher amounts of dairy foods than older children and recommendation four has the potential to adversely impact on the provision of these foods in pre-school settings.</p> <p>Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out (35), they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed WHO saturated fat guidelines will adversely impact on intake of milk and dairy foods around the world.</p> <p>Paragraph 1, page 40: As pointed out by Mozaffarian (36), 'Methodological advances in nutrition science now demonstrate that</p>

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nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.' We recommend that the WHO makes future recommendations in relation to dietary patterns rather than single nutrients.

We also believe that the statement: 'public health interventions should aim to reduce saturated fat intake, while reducing total fat intake where necessary, and without increasing free sugar intake' could detrimentally impact on the nutritional quality of the diet and should be amended to:

'public health interventions aimed at reducing saturated fat intake should ensure that nutritional adequacy is not compromised and that free sugar intake is not increased'

31. Janiaud P et al., (2015) Different treatment benefits were estimated by clinical trials performed in adults compared with those performed in children. *Journal of Clinical Epidemiology* 68, 1221-31.

32. Oostenbrink R & de Wildt SN, (2016) Drug trials: Kids are no little adults and not all kids are the same. *Journal of Clinical Epidemiology* 71, 111-12.

33. Paakkunainen U et al., (2002) The Impact of Low-Saturated Fat, Low Cholesterol Diet on Bone Properties Measured Using Calcaneal Ultrasound in Prepubertal Children. *Calcified Tissue International*, 71, 219-226.

34. De Souza RJ et al., (2015) Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 351:h3978.

35. Food and Agriculture Organization of the United Nations (2013) Milk and dairy products in human nutrition. Rome: FAO.

36. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Page 44, paragraph 2 (Translation and implementation) We note that the text states that these guidelines (on saturated and trans fatty acids) should be used in conjunction with other guidance on dietary goals and nutrition guidelines. We would encourage the WHO to make future recommendations in relation to dietary patterns rather than single nutrients. This is because, as explained by Mozaffarian (47), 'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.'

While use of guidelines by policy-makers and programme managers to assess current intake of saturated fatty acids and trans fatty acids relative to a benchmark is important, it is critical that accompanying guidance from WHO stresses to these groups that these are not the only criteria on which to assess the healthiness of the diet 'a cardioprotective diet pattern must be characterized by the healthful foods that are included, not simply specific items to be avoided (48).'

The problem with using single naturally present nutrients as criteria for front-of-pack labelling, restricting sales of foods in schools, fiscal policies targeting foods and reformulation is that it encourages consumption of ultra-processed foods, which in turn are linked with chronic diseases, including breast cancer (49,50). There is a need to distinguish between foods that naturally contain saturated fatty acids and trans fatty acids, and those with added saturated fatty acids and industrially produced trans fats. It is the nutrient-poor, energy-rich (discretionary) foods that contain added saturated fatty acids and industrially produced trans fats that should be labelled, restricted in schools, taxed and reformulated. These policies should be developed in a way that does not restrict consumption of healthy, nutritious foods that naturally contain saturated and ruminant trans fats.

It is always important to 'first do no harm' and simply focusing on total saturated fat and total trans fat content of a food is likely to lead to a reduction in intake of milk, yogurt and cheese, which is likely to negatively impact on the nutritional adequacy and healthfulness of the overall diet. For example, reduced intake of dairy foods (as a result of these foods being labelled unhealthy due to their saturated fat content) would be likely to negatively impact on intakes of key nutrients such as calcium (which is important for bone accrual in children (51) and iodine (which is important for preventing cognitive impairment in children (52,53). Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out (54), they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed translation of the WHO saturated fat and trans fat guidelines will adversely impact on intake of milk and dairy foods around the world. Any translation of the finalised guidelines needs to ensure that this does not occur.

Page 45, paragraph 4: The text suggest that in relation to overall dietary guidance, it is 'feasible to achieve the recommendations in these guidelines because a wide variety of fresh foods are naturally low in saturated fatty acids and trans-fatty acids'. We would point out that while eating a wide variety of fresh foods that are naturally low in saturated fatty acids and trans fatty acids is beneficial for many aspects of health, consumption of dairy foods (which are NOT naturally low in saturated fatty acids) is associated with a 6% lower risk of hip fracture (55) per daily serving in men and women (80,600 women and 43,306 men, up to 32 years follow up, RR=0.94, CI 0.90 to 0.98). This is important as the United Nations recently highlighted: 'the number of older persons — those aged 60 years or over — is expected to more than double by 2050 and to more than triple by 2100, rising from 962 million globally in 2017 to 2.1 billion in 2050 and 3.1 billion in 2100. Globally, population aged 60 or over is growing faster than all younger age groups.'

Similarly, there is level A evidence for calcium intake having a positive effect on bone accrual in children and adolescents. Weaver and colleagues (57) state 'The evidence since 2000 builds on earlier evidence, with additional RCTs showing a benefit to bone owing to the inclusion of dairy products in the diet. Dairy products contain colloidal calcium phosphate protein complexes in the form of casein micelles that have the minerals and nutrients needed for bone growth.'

47. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

48. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

49. Monterio C et al., (2017) The UN decade of nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutrition* 21, 5-17.

50. Fiolet T et al., (2018) Consumption of ultra-processed foods and cancer risk: results from NutriNet-Sante prospective cohort. *BMJ* 360: k322

51. Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. *Osteoporosis Int* 27,1281-1386

52. Velasco I et al., (2018) Iodine as essential nutrient during the first 1000 days of life. *Nutrients* 10, 290

53. Bath SC et al., (2017) Iodine concentration of milk-alternative drinks available in the UK. *Br J Nutr* 118, 525-32
54. Food and Agriculture Organization of the United Nations (2013) Milk and dairy products in human nutrition. Rome: FAO.
55. Feskanich D et al., (2018) Milk and other dairy foods and risk of hip fracture in men and women. *Osteopros Int* 92, 385-96
56. <http://www.un.org/en/sections/issues-depth/ageing/> visited 31/5/18
57. Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. *Osteoporosis Int* 27,1281-1386

#### Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Page 46, research needed on saturated fatty acids and trans fatty acids: All of the identified research relates to a reductionist, single nutrient paradigm. As pointed out by Mozaffarian (58), 'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.' We recommend that the WHO makes these and future recommendations in relation to dietary patterns rather than single nutrients. Within a dietary pattern framework, we suggest that RCTs are conducted in the population that future guidelines are made for (i.e. no extrapolation of results from secondary prevention trials to the general population, and no extrapolation of results from adults to children). In light of the rapidly ageing population, we recommend that greater focus is placed on determining a healthy diet for the elderly, not just avoidance of NCDs.

It is difficult to understand why type 2 diabetes and adiposity were priority health outcomes for children but not adults. We recommend that further work be undertaken considering these outcomes for adults. We also recommend that further consideration be given to the impact of reducing saturated and trans fat intake on frail elderly people (e.g. in terms of sarcopenia, falls, fractures and bone health), and on bone accrual in children. In addition, greater consideration is required of the impact on the nutritional adequacy of the diet when a general recommendation to reduce saturated and trans fat intake is given. Does it lead to lower meat and dairy intake? What are the impacts on nutrient intakes?

It is mentioned that the role of the NUGAG Subgroup on Diet and Health included advice on the 'balance of benefits and harms.' We believe that this is an area that requires greater consideration. The official endorsement of the low-fat diet in the 1980s and 1990s led to rising rates of overweight and type 2 diabetes, as consumers incorrectly interpreted the advice as being a licence to eat what you want, when you want as long as it is low in fat. Research is required that investigates how consumers and policy makers are likely to interpret the proposed guidelines related to saturated and trans fatty acid intakes.

58. Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

#### Annexes 1, 6, 7 (for saturated fatty acids only)

#### Additional comments

## *Trans-fatty acids*

#### Executive summary (same for saturated fatty acids and trans-fatty acids)

See comments made previously in the saturated fatty acid submission

#### Scope and purpose (same for saturated fatty acids and trans-fatty acids)

See comments made previously in the saturated fatty acid submission

#### Background (same for saturated fatty acids and trans-fatty acids)

See comments made previously in the saturated fatty acid submission

#### Guideline development process (same for saturated fatty acids and trans-fatty acids)

See comments made previously in the saturated fatty acid submission

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#### Summary of evidence (for trans-fatty acids only)

Paragraph 1, page 31: The WHO-commissioned systematic review of prospective cohort studies (37) is a key piece of evidence for the trans fatty acid recommendations, but (as stated in footnote 1 on page 21), not for the saturated fatty acid recommendations. This appears to be inconsistent and gives the impression that commissioned work is downgraded if it does not provide the expected results.

Paragraph 2, page 31: The conclusions of the second WHO-commissioned systematic review (38) of RCTs rely on theoretical calculations ('coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA') and regression analysis unweighted for study size. Why was such a different analysis technique used for the systematic review of RCTs for saturated fatty acids and the systematic review of RCTs for trans fatty acids?

We would also highlight the fact that the RCTs lasted for between 14 and 56 days yet it is expected that people will follow the WHO recommendations for years. In the systematic review of prospective cohort studies by de Souza and colleagues, Figure 4 clearly demonstrates that consumption of ruminant trans fats are not associated with any adverse health outcomes (all-cause mortality, CHD mortality, total CHD or ischemic stroke) and that its consumption is associated with reduced risk of type 2 diabetes (5/5 studies comprising 12,942 participants, RR 0.58 95% CI 0.46-0.74 P

#### Recommendations (for trans-fatty acids only)

Page 40, Recommendation 1: 'In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing trans-fatty acid intake (strong recommendation<sup>2</sup>)'. In light of Figure 4 from the WHO-commissioned systematic review by de Souza and colleagues which clearly demonstrates that consumption of ruminant trans fats over 5-20 years is not associated with any adverse health outcomes and that its consumption is associated with reduced risk of type 2 diabetes, we suggest that this recommendation be amended to:

'In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing industrial trans-fatty acid intake (strong recommendation)'.

The updated wording would be less confusing to the general public as both the WHO dietary recommendations and the WHO's REPLACE initiative announced in May 2018, would focus on industrial trans fatty acids.

Page 40, Recommendations 2-4: Similarly, we recommend that industrial trans fatty acids rather than all trans fatty acids are specified in the three conditional recommendations.

Remarks (for trans-fatty acids only)
<p>Page 41, Paragraph 1: The text indicates that the recommendations for trans fatty acid intake in children are based on extrapolation of adult data on CVD risk and blood lipids. As stated previously, we do not believe that it is appropriate to extrapolate results from adults to recommendations for children lasting years. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).</p> <p>A 2015 study by Janiaud and colleagues (43) found different treatment benefits estimated by clinical trials in adults compared with those performed in children for 11% of drugs (14 out of 124). A commentary (44) on the paper pointed out that 'kids are no little adults and not all kids are the same.' They added 'although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.' We agree – the world's children deserve better evidence for the dietary recommendations affecting them.</p> <p>Page 41, Paragraph 2: We believe that this paragraph should point out that theoretical models and regression analyses not weighted for study size, not actual comparisons, were the basis for the observations made in this paragraph (45) ('coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA'). Why was such a different analysis technique used for the systematic review of RCTs for saturated fatty acids and the systematic review of RCTs for trans fatty acids?</p> <p>We would also highlight the fact that the RCTs lasted for between 14 and 56 days yet it is expected that people will follow the WHO recommendations for years.</p> <p>Page 41, paragraphs 3-4: It does not appear that the totality of the evidence reviewed is the basis for the recommendation to reduce total trans fatty acid intake, as there is no mention that the WHO-commissioned systematic review by de Souza and colleagues clearly demonstrates that consumption of ruminant trans fats over 5-20 years is not associated with any adverse health outcomes and that its consumption is associated with reduced risk of type 2 diabetes.</p> <p>Page 42, paragraph 1: We believe that this paragraph should point out that theoretical models and regression analyses, not actual comparisons, were the basis for the observations made in this paragraph (46) ('coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA').</p> <p>43. Janiaud P et al., (2015) Different treatment benefits were estimated by clinical trials performed in adults compared with those performed in children. <i>Journal of Clinical Epidemiology</i> 68, 1221-31.</p> <p>44. Oostenbrink R &amp; de Wildt SN, (2016) Drug trials: Kids are no little adults and not all kids are the same. <i>Journal of Clinical Epidemiology</i> 71, 111-12.</p> <p>45. Brouwer IA (2016) Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis. Geneva: World Health Organization.</p> <p>46. Brouwer IA (2016) Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis. Geneva: World Health Organization.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
See comments made previously in the saturated fatty acid submission
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
See comments made previously in the saturated fatty acid submission
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

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Please provide any final thoughts or comments below.

## Comments on the Executive Summary

**Paragraph 1, page 1:** in 2016, Mozaffarian<sup>1</sup> stated 'A cardioprotective diet pattern must be characterized by the healthful foods that are included, not simply specific items to be avoided.'

While noncommunicable diseases (NCDs) are important, health is *more* than the absence of NCDs. With any dietary guidance, it is important to 'first do no harm' and to ensure that nutritional adequacy is not compromised. This does not appear to have been fully considered in the development of these guidelines. For example, there appears to have been a lack of consideration of the impact of reduced intake of dairy foods (as a result of these foods being labelled unhealthy due to their saturated fat content) on intakes of key nutrients such as calcium (which is important for bone accrual in children<sup>2</sup>) and iodine (which is important for preventing cognitive impairment in children<sup>3,4</sup>).

The statement in paragraph 1, page 1: 'Dietary saturated fatty acids and *trans*-fatty acids are of particular concern because high levels of intake are correlated with increased risk of CVDs (2)' should be amended, as *causation* cannot be implied from correlations.

**Paragraph 2, page 5** (The evidence): The report should clearly report that three primary outcomes and a number of secondary outcomes were set in the 2015 review by Hooper and colleagues, and that there was no clear effect of reducing saturated fat intake for two out of three of the primary outcomes (total mortality and cardiovascular mortality). At present, it is not clear from the text which results are the primary and secondary outcomes. It would also be helpful to reference the systematic reviews being referred to in this paragraph and the next.

**Paragraphs 1 and 2, page 7:** it would be helpful to include the references for the meta-analysis of prospective cohort studies and the meta-regression analysis of RCTs.

**Paragraph 3, page 7:** it is quite remarkable that *trans*-fatty acid recommendations are being proposed for children and yet no studies were identified that met the inclusion criteria established for the systematic review of studies conducted in children.

**Page 8, saturated fat recommendation 1:** '*In adults and children whose saturated fatty acid intake is greater than 10% of total energy intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake (strong recommendation<sup>2</sup>)*'. This suggests any person over the age of two years with a saturated fat intake higher than 10.0% of energy should reduce their saturated fat intake and **that it does not matter which foods are targeted**. We are concerned about possible unintended consequences associated with this advice, such as intakes of under-consumed nutrients being further compromised.

We would also question the appropriateness of this recommendation for **frail elderly people**. The Australian Dietary Guidelines<sup>5</sup> make it clear that their guidelines are 'not appropriate for frail elderly people or those with complex health conditions and an appropriate health professional should be consulted' (page 27).



We suggest that if this recommendation goes ahead, the wording of this recommendation is amended to the following:

*‘In adults and children over 2 years whose saturated fatty acid intake is greater than 10% of total energy intake<sup>i</sup>, WHO recommends reducing saturated fatty acid intake in a way that ensures that the individual’s other nutritional and dietary requirements are met (strong recommendation<sup>2</sup>). This recommendation does not apply to frail elderly people or those with complex health conditions.’*

However, we would also question the **scientific basis for this recommendation** as the majority of studies that form the evidence for this were undertaken in adults who *already* had cardiovascular disease or diabetes.<sup>i</sup> Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, **70% of the data** behind the strong recommendation for all adults and children ‘to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake’ comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children.

**Page 8, saturated fat recommendations 2-4:** According to footnote 3, ‘**Conditional** recommendations are those recommendations for which the WHO guideline development group is **uncertain** that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders (22)’.

If it is the case that there is uncertainty about whether desirable consequences outweigh undesirable consequences, we think it would be better to make **no recommendations**. WHO’s reputation and credibility are likely to be damaged if it recommends a dietary change that is later shown to have undesirable consequences. The sugar recommendations released by the WHO in 2015 have been widely referenced by policy makers around the world, but little distinction has been made between the strong and the conditional recommendations.

**Page 8, Remark 2:** It is good that this remark makes clear that the recommendations for children are based on blood lipid and blood pressure measures and that the rest of the evidence is an **extrapolation** of adult data to children. However, we do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues<sup>6</sup> found *different* treatment benefits estimated by clinical trials in adults compared with those performed in children for 11% of drugs (14 out of 124). A commentary<sup>7</sup> on the paper pointed out that ‘kids are no little adults and not all kids are the same.’ They added ‘although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.’ We agree – the world’s children deserve better evidence for the dietary recommendations affecting them.

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<sup>i</sup> DART participants were men recovering from a MI, participants in the study by Houtsmuller were adults with newly diagnosed diabetes, participants in the study by Ley were people with impaired glucose tolerance or high normal blood glucose, participants in Moy 2001 had at least one CVD risk factor, participants in the MRC trial were men who had survived a first MI, those in the Oslo Diet Heart trial were men who had a previous MI, the Rose corn oil and olive trials involved men with angina or following a MI, the participants in the STARS trial were men with angina and those taking part in the WHI with CVD all had CVD at baseline.

**Paragraph 2, page 9:** It is stated that ‘The evidence indicates that children’s growth is not compromised by reduction of saturated fatty acid intake.’ Growth is **more** than the variables measured (height, body weight, BMI and other measures of adiposity). There was not consideration of other aspects of growth such as bone accrual – indeed the authors of the Special Turku Coronary Risk Factor Intervention Project (STRIP) commented<sup>8</sup> that ‘further studies are needed to draw further conclusions about the influence of dietary counselling on bone health’. Therefore, we feel that the statement should be amended to:

*‘The evidence indicates that children’s height and body weight are not compromised by reduction of saturated fatty acid intake. Effects on other aspects of growth such as bone accrual are unknown.’*

**Paragraph 3, page 9:** Here the text states that: ‘in the RCTs that assessed cardiovascular outcomes and mortality outcomes (Hooper et al., 2015) saturated fatty acids were largely replaced by polyunsaturated fatty acids, the polyunsaturated fatty acids were primarily from plant-based oils, **rich in linoleic acid**’.

This statement is not consistent with Ramsden and colleagues<sup>9</sup> Webtable 6, which indicates that three studies selectively increased n-6 linoleic acid (LA) without a concurrent increase in n-3 PUFAs and five studies were mixed n-3/n-6 PUFA interventions that increased n-3 PUFAs and n-6 LA. Ramsden and colleagues conclude that: 1) the specific PUFA composition of dietary interventions is a critical determinant of clinical CVD outcomes, and 2) selective substitution of n-6 LA for SFA is unlikely to be beneficial, particularly in patients with established CHD.’ Commenting on this paper, Professor Philip Calder wrote ‘The more cautious UK dietary recommendations on fat and fatty acids, which include the statement, “*There is reason to be cautious about high intakes of omega 6 PUFAs*” seem fully justified in light of the current study’s findings.’ In contrast, the Hooper meta-analysis did not appear to consider differences between the effects of n-6 LA and mixed n-3/n-6 PUFA interventions. We believe that there should be greater consideration of the results in the context of the concerns expressed about n-6 LA and the statement in question should be amended to:

‘In the RCTs that assessed cardiovascular outcomes and mortality outcomes (Hooper et al., 2015) saturated fatty acids were largely replaced by polyunsaturated fatty acids, the polyunsaturated fatty acids were from plant-based oils, rich in linoleic acid and mixtures of n-3 and n-6’.

**Paragraph 5, page 9:** Here is a classic example of ‘cherry picking’ findings to suit a particular argument, rather than presenting a balanced view. Three pre-determined **primary** outcomes were selected and the evidence for all three outcomes was judged to be of moderate quality using GRADE (page 3 of Hooper et al., 2015).

These three results (all-cause mortality, CVD mortality and combined CV events) should always be given equal weight in the WHO recommendations. At present, there is disproportionate emphasis on the finding that reducing saturated fat leads to reduced combined CV events and not on the fact that it makes **no significant difference** to the risk of all-cause mortality or CVD mortality. In light of this, it is difficult to see how it is justifiable to make a strong recommendation to reduce saturated fatty acids when it is likely to make no difference to two out of the three pre-specified main outcomes. Paragraph 5 on page 9 even refers to the ‘totality of evidence reviewed’ but then fails to report the results for two out of three of the main outcomes. We therefore recommend that this paragraph be re-written in a way that provides a more balanced representation of the results.

The *WHO-commissioned* systematic review and meta-analysis of observational studies examining intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes<sup>10</sup> concluded that *'the certainty of associations between saturated fat and all outcomes was "very low"'* yet the draft WHO recommendations are suggesting that there is enough evidence for a *strong* recommendation to reduce saturated fat intake. Again, this appears to be cherry picking the results to support preferred outcomes.

**Paragraph 6, page 9:** This paragraph justifies reducing saturated fatty acid intake on the basis of LDL cholesterol levels in adults and children. However, consideration of LDL cholesterol levels in isolation of other factors is meaningless. For example, 16:0 raises LDL cholesterol, but also raises HDL-cholesterol, reduces triglyceride-rich lipoproteins and remnants and has little effect on Apo-B. Mozaffarian (2016) commented: 'Continued prioritization of saturated fat reduction appears to rely on selected evidence: e.g. effects on LDL-cholesterol alone (discounting the other, complex lipid and lipoprotein effects); historical ecological trends in certain countries (e.g. Finland) but not in others; and expedient comparisons with polyunsaturated fat, the most healthful macronutrient.'

**Paragraph 2, page 10:** The 2013 Australian Dietary Guidelines<sup>11</sup> state, *'Foods exert certain health effects because of the nutrients they contain. As such, dietary recommendations are often couched in terms of individual nutrients (such as requirements of vitamins and minerals). However, people eat foods rather than single nutrients, so such advice can be difficult to put into practice. For this reason, these Guidelines make recommendations based only on whole foods, such as vegetables and meats, rather than recommendations related to specific components and individual nutrients'*. We recommend that the WHO also focuses on making recommendations based on whole foods rather than nutrients. We believe that the recommendation to replace saturated fatty acids with polyunsaturated fatty acids is difficult to put into practice. For example, people would not have a vegetable oil sandwich rather than a cheese sandwich, they would not drink vegetable oil rather than milk and they would not eat a vegetable oil dessert rather than a yogurt. The focus on individual nutrient plays into the promotion of ultra-processed foods, which in turn are linked with chronic diseases.<sup>12</sup>

We also recommend that greater consideration be given to the differing effects of n-3 and n-6 PUFA reported by Ramsden and colleagues<sup>13</sup> before a recommendation is given to simply replace saturated fatty acids with (unspecified) PUFA.

**Paragraph 3, page 10:** The omission of the PREDIMED study from the review by Hooper and colleagues (2015) highlights the narrowness of the work used to underpin these guidelines. While we appreciate the rationale for the work, we think it would have been better to have focused on dietary patterns and to make dietary recommendations in relation to these, not single nutrients. We recommend that greater consideration is given to dietary patterns and that any recommendations related to saturated fat intake are set within the context of these findings.

**Paragraph 4, page 10:** Although it is stated that 'the recommendation to not increase saturated fatty acid intake if intake is already below 10% of total energy intake (fourth recommendation) is based on the totality of evidence reviewed, a major omission is consideration of the impact of such a recommendation on the frail elderly. It is well acknowledged that the frail elderly do not require a low fat diet. Focus on the saturated fat intake of such people, as a result of this proposed WHO recommendation, could well reduce their access to nutrient-dense, protein-providing foods such as cheese and whole milk.

Also, children between the ages of 2 and 5 years often consume higher amounts of dairy foods than older children and recommendation four has the potential to adversely impact on the provision of these foods in pre-school settings.

Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out<sup>14</sup>, they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed WHO saturated fat guidelines will adversely impact on intake of milk and dairy foods around the world.

**Paragraph 2, page 11:** As pointed out by Mozaffarian<sup>15</sup> (2016), *'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.'* We recommend that the WHO makes future recommendations in relation to dietary patterns rather than single nutrients.

We also believe that the statement: *'public health interventions should aim to reduce saturated fat intake, while reducing total fat intake where necessary, and without increasing free sugar intake'* could detrimentally impact on the nutritional quality of the diet and should be amended to:

*'public health interventions aimed at reducing saturated fat intake should ensure that nutritional adequacy is not compromised and that free sugar intake is not increased'*

**Recommendation 1, page 11:** *'In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing trans-fatty acid intake (strong recommendation<sup>2</sup>)'*. In light of Figure 4 from the WHO-commissioned systematic review by de Souza and colleagues which clearly demonstrates that consumption of ruminant trans fats over 5-20 years is **not** associated with any adverse health outcomes and that its consumption is associated with reduced risk of type 2 diabetes, we suggest that this recommendation be amended to: '

*In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing industrial trans-fatty acid intake (strong recommendation<sup>2</sup>)'*.

The conclusions of the second WHO-commissioned systematic review<sup>16</sup> of RCTs relies on theoretical calculations (*'coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA'*) and regression analysis that was not weighted for study size. Why was such a different analysis technique used for the systematic review of RCTs for saturated fatty acids and the systematic review of RCTs for *trans* fatty acids?

We would also highlight the fact that the RCTs lasted for between **14 and 56 days** yet it expected that people will follow the WHO recommendations for years.

Our suggested revised wording would be less confusing to the general public, as both the WHO's dietary recommendations and the WHO's REPLACE initiative announced in May 2018, would focus on artificial *trans* fatty acids.

**Recommendations 2-4, page 11:** Similarly, we recommend that industrial *trans*-fatty acids rather than all *trans*-fatty acids are specified in the three conditional recommendations.

## Comments on the Scope and Purpose

**Paragraph 2, page 15:** We believe that the scope and purpose of the guidelines require an update. As pointed out by Mozaffarian<sup>17</sup> (2016), *'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.'* We recommend that the WHO makes future recommendations in relation to dietary patterns rather than single nutrients. Focusing on a single nutrient to prevent CVD in adults and children is taking a rather 20<sup>th</sup> rather than 21<sup>st</sup> century approach. Mozaffarian added *'Thus, the present period is one of exciting, rapid transition away from single-nutrient theories and simple surrogate outcomes and toward foods, dietary patterns and evaluation of clinical end points. This transition forms the basis for our modern understanding of diet and cardio-metabolic health.'*

**Paragraph 3, page 15:** The recommendations state 'It is hoped that the guidelines will also help to accelerate the implementation of actions to promote healthy diets, improve health and nutritional status of all people, and ultimately reduce the burden of NCDs to help accelerate achievement of the Sustainable Development Goals.' We share this hope, but fear that the proposed guidelines will *reduce* the health and nutritional status of all people by labelling nutritious and healthy foods such as milk, yogurt and cheese as foods to limit, in order to reduce saturated fat intake. Unless changes are made to the wording of the recommendations, they have the potential to promote unhealthy diets and reduce the health and nutritional status of all people by for example, reducing intake of nutritious foods that contain saturated fat such as whole milk, yogurt and cheese.

## Comments on the Background

**Paragraph 4, page 16:** While saturated fatty acids are found in the foods listed (butter, cow's milk, meat, salmon and egg yolks), just 3.2% of total saturated fat comes from butter in Australia, cows', sheep's and goats' milk provide 8.7%, meat, poultry and game products and dishes supply 19.6%, fish and seafood products and dishes supply 1.8% and egg products and dishes supply 1.8%. Thus, in total, these foods account for just 35% of the saturated fat in the Australian diet. In contrast, discretionary choices (foods and drinks not necessary to provide the nutrients the body needs, many of which are high in saturated fats, sugars, salt and/or alcohol) provided 49% of the saturated fat within the Australian diet of children aged 2-18 years<sup>18</sup>. We suggest that the text focuses more on these sources of saturated fat and less on those that are naturally occurring, particularly butter, milk, fish and eggs.

**Paragraph 4, page 16:** it would be helpful to point out that not all saturated fatty acids are the same. As stated by Professor Mozaffarian<sup>19</sup>:

"Saturated fat represents a highly heterogeneous category of fatty acids, with chain lengths ranging from 6 to 24 carbons, deriving from diverse foods, and possessing dissimilar biology. For instance, palmitic acid (16:0) exhibits in vitro adverse effects; whereas medium-chain (6:0–12:0), odd-chain (15:0, 17:0), and very long-chain (20:0–24:0) saturated fats may have metabolic benefits. This biological and metabolic diversity **does not support** the grouping together of all saturated fatty acids based on **only one chemistry characteristic**:

the absence of double bonds.”

“Judging a food or a person’s diet as harmful because it contains more saturated fat, or as beneficial because it contains less, is **unsound**. This is consistent with the many longitudinal cohort studies demonstrating largely neutral effects of overall saturated fat intake. Consistent with this, meats higher in processing and sodium, rather than saturated fat, are most strongly linked to CHD. Cheese, a leading source of saturated fat, is also linked to neutral or even beneficial effects on CHD and diabetes mellitus. In sum, these lines of evidence – complex lipid effects including little influence on ApoB, no relation of overall intake with CHD, and no observed cardiovascular harm for most major food sources – provide powerful and consistent evidence for absence of appreciable harms of total saturated fat.”

**Paragraph 1, page 17:** We are pleased to see acknowledgement that ‘different saturated fatty acid-containing foods, such as dairy foods, may have differing effects on risk of cardiovascular diseases and type 2 diabetes, either as a result of differing composition of saturated fatty acids across foods, other constituents of the foods, or a combination of the two’ however, we are disappointed that no consideration of this is apparent in the resulting recommendations.

**Paragraph 3, page 17:** The bottom paragraph on page 17 cites five papers. Four were from the previous century and the other is more than 10 years old. We recommend that more recent scientific papers are used to support the statements made, in order to take into account contemporary scientific thinking on the relationship between childhood diet and risk of future CVD, particularly in light of rising rates of obesity and type 2 diabetes.

## Comments on the Guideline Development Process

**Paragraph 2, page 19:** With the focus on reduction of CVD, it is surprising that no cardiologists were invited to participate in discussions. In light of the aging population of many countries, it would have also been helpful to include a specialist in elderly nutrition in the subject matter experts.

Paragraph 4, page 19: It is mentioned that the role of the NUGAG Subgroup on Diet and Health included advice on the ‘balance of benefits and harms.’ We believe that this is an area that requires greater consideration. The official endorsement of the low-fat diet in the 1980s and 1990s led to rising rates of overweight and type 2 diabetes, as consumers incorrectly interpreted the advice as being a licence to eat what you want, when you want as long as it is low in fat.

Consideration is required about how consumers and policy makers will interpret the proposed guidelines related to saturated and *trans* fatty acid intakes. It could well lead to the promotion of diets that are lacking in essential nutrients such as calcium and iodine, as milk and cheese consumption are discouraged due to their saturated and *trans* fat contents<sup>20</sup>.

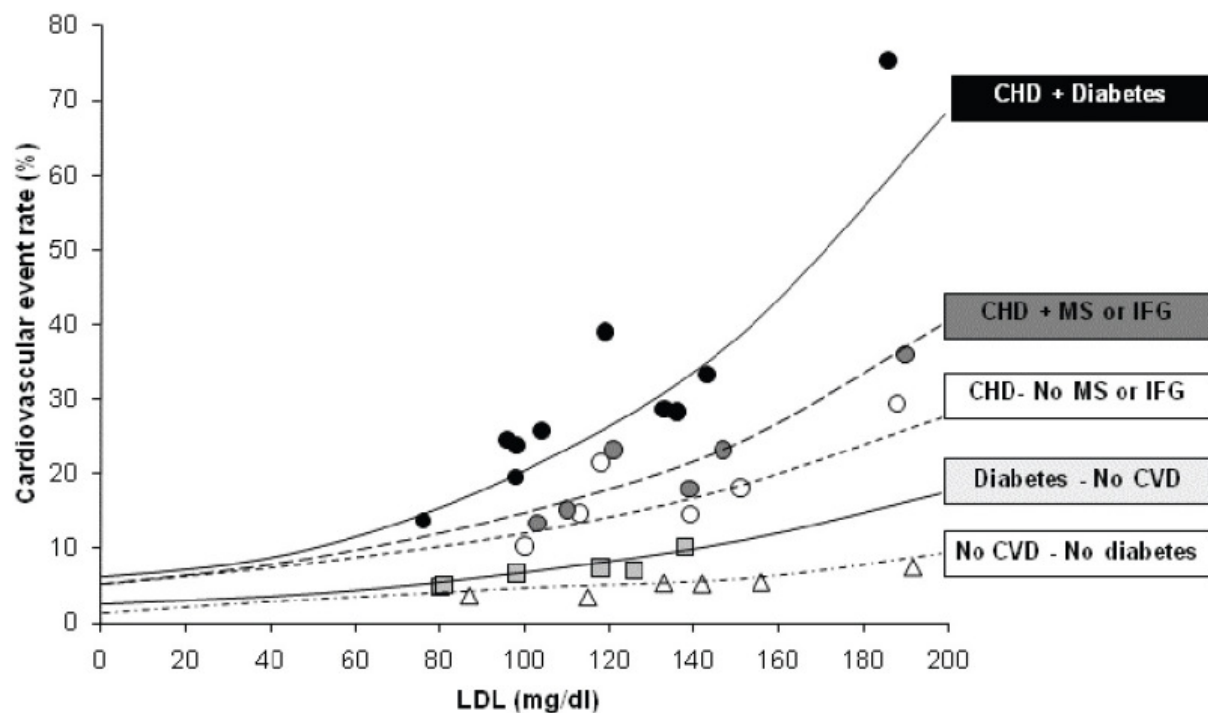
## Comments on the Summary of Evidence for Saturated Fatty Acids

**Paragraph 2, page 19:** It is difficult to understand why type 2 diabetes and adiposity were priority health outcomes for children but not adults. We recommend that further work be undertaken considering these outcomes for adults. We also recommend that further consideration be given to the impact of reducing saturated and *trans* fat intake on frail elderly people (e.g. in terms of sarcopenia, falls, fractures and bone health), and on bone accrual in children. In addition, greater consideration is required of the impact on the nutritional adequacy of the diet when a general recommendation to reduce saturated and *trans* fat intake is given. Does it lead to lower meat and dairy intake? What are the impacts on nutrient intakes?

**Footnote 1, page 21:** The **rationale** for not considering the results of the WHO commissioned systematic review of cohort studies<sup>21</sup> in the formulation of recommendations on saturated fat appears to be weak, particularly as it was considered in the formulation of recommendations for *trans* fats. The systematic review of RCTs<sup>22</sup> and the systematic review of prospective cohort studies both concluded that saturated fat intake was not related to risk of all-cause mortality or CVD mortality. This appears to be another example of ‘cherry picking’ the results to support a preferred position.

**Footnote 2, page 21:** The rationale for including LDL as a ‘critical outcome’ is somewhat questionable. As Professor Ron Krauss explained to the Committee on Quantification of Biomarkers and Surrogate Endpoints in Chronic Disease<sup>23</sup>, ‘CVD is a very complex disease that is increasingly recognized as a spectrum of pathologic and pathophysiologic effects, **only one** of which is primarily related to progression of the cholesterol content of plaques as a function of LDL-C in the blood’. Atherosclerosis, he added, is “often indolent, progressive over time, and then is complicated by a number of additional factors that can convert a cholesterol-rich plaque to a more malignant form that destabilizes and is involved with both inflammation and thrombosis; immune changes can occur that could be critical.” Thus, he concluded, “it is rather **simplistic** to consider either LDL or HDL, or even the two of them together, as sufficient to explain these complex mechanisms.”

Professor Kraus also noted that the predictive value of LDL-C for CVD events **varies** considerably as a function of health status, and risk for cardiovascular events associated with high LDL-C in patients without diabetes and CVD was found to be significantly lower than LDL-C-associated event risk in patients with both conditions (see figure below from Robinson and Stone, 2006<sup>24</sup>).



From Robinson and Stone, 2006<sup>25</sup>

**Page 22, paragraph 2:** This paragraph notes that of the 17 comparisons made in the systematic review of RCTs, six included only people at high risk of CVDs, four included people at moderate risk and five included people at low risk. However, when it came to the evidence base for the primary outcome that is the basis for the strong recommendation (CV events), it should be noted that the vast majority of studies that form the evidence for this were undertaken in adults who *already* had cardiovascular disease or diabetes.<sup>ii</sup> Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, **70% of the data** behind the strong recommendation for all adults and children ‘to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake’ comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children. Recommendations to the general (healthy population) should be based on the results of studies in the general (healthy population) – **not on the results of secondary prevention trials.**

**Page 22, paragraph 2:** Along with information about the saturated fat content of the diets consumed by both the intervention and control groups, we suggest that it should be noted that *trans* fat intake was either unclear or not reported in all of the comparisons apart from two (the WHI with CVD and the WHI without CVD). As it is well established that industrially produced *trans* fatty acids increase risk of CVD and that in many countries, industrially produced *trans* fat was present in margarines in the 1960s and 1970s – at the same time when some of these trials were undertaken,

<sup>ii</sup> DART participants were men recovering from a MI, participants in the study by Houtsmuller were adults with newly diagnosed diabetes, participants in the study by Ley were people with impaired glucose tolerance or high normal blood glucose, participants in Moy 2001 had at least one CVD risk factor, participants in the MRC trial were men who had survived a first MI, those in the Oslo Diet Heart trial were men who had a previous MI, the Rose corn oil and olive trials involved men with angina or following a MI, the participants in the STARS trial were men with angina and those taking part in the WHI with CVD all had CVD at baseline.



there should be greater consideration of *trans* fatty acids as a confounding factor. Discussing this issue in relation to the Sydney Diet Heart Study, Ramsden and colleagues commented that ‘the restriction of common margarines and shortenings (major sources of *trans* fatty acids) in the intervention group would be expected to substantially reduce consumption of *trans* fatty acids compared with the control group.’ However, as the composition of the margarine used in the intervention was unknown, it is possible that ‘nutrients other than n-6 LA and SFAs could have contributed to, or reduced, the observed unfavourable effects of the LA intervention.’ In light of the possibility of **confounding** by industrially produced *trans* fatty acid intake in **all but two** comparisons, greater emphasis should be placed on the results of these two comparisons (from the Women’s Health Initiative) where the *trans* fat intake of the subjects is known and can be adjusted for.

**Page 25, paragraph 1:** It is stated that ‘Significant reductions in risk of CVD mortality (RR 0.69; 95% CI: 0.51,0.94) and cardiovascular events (RR 0.79; 95% CI: 0.62, 0.99) were observed in meta-analysis of two trials with 979 participants in which saturated fatty acid intake was reduced to less than 9% of total energy intake.’ Again, this is another example of cherry picking the data. **Out of the 46** comparisons made in table 23 of Hooper et al., 2015, just these **two** results did not have confidence intervals spanning 1.0. The text fails to mention that **no** clear effect on any cardiovascular or mortality outcome was apparent when saturated fatty acid intake was less than 8 or 7% E. Similarly, there is **no** mention that a saturated fatty acid intake of less than 9% E had **no** effect on all cause mortality, myocardial infarction, non fatal myocardial infarction, stroke, CHD mortality and CHD events. It seems odd that no adjustments appear to have been made for multiple comparisons.

**Page 28, paragraph 2.** We also recommend that further consideration be given to the impact of reducing saturated and *trans* fat intake on bone accrual in children. In addition, greater consideration is required of the impact on the nutritional adequacy of the diet when a general recommendation to reduce saturated and *trans* fat intake is given. Does it lead to lower meat and dairy intake? What are the impacts on nutrient intakes?

**Page 28, paragraph 2.** We note that the trials considered in the systematic review of RCTs included some children and adolescents with normal cholesterol levels and some with raised levels. Looking in more detail at Table 1 from Te Morenga & Montez 2017<sup>26</sup>, it would appear that 54% of participants were hyperlipidaemic and 46% were the general population that had not been tested, or were known to be normolipidaemic. As with the adults, we believe that the results from primary and secondary prevention trials should be considered separately, not together. Extrapolating the findings from hyperlipidaemic children to the general population may not be appropriate. Recommendations to the general population should be based on evidence from the general population – not those with specific medical conditions.

**Page 29, paragraph 3.** We find it quite remarkable that just one trial has appropriately tested the effects of a reduction in saturated fatty acid intake to 9% of total energy and yet the WHO is proposing to recommend that 7.5 billion children reduce their intake of saturated fat if it is above 10% E. It is unclear if the children who participated in the trial in question by Denke et al., (2000) were hyperlipidemic or not. The range of ages for the children tested was 8-16 years, so the results could well not be applicable to younger children. For all eight measures of bias, the study scored ‘unclear risk of bias’ (see Figure 6 from Morenga & Montez 2017<sup>27</sup>). In addition, the trial lasted **five weeks**, yet the WHO recommendations are likely to be in place for **years**.

**Page 30, paragraph 2.** It is stated that ‘evidence for cardiovascular, mortality and blood lipid outcomes from adults was also considered when formulating the recommendations for children, without downgrading for indirectness.’ According to the GRADE handbook ‘Direct evidence consists of research that directly compares the interventions which we are interested in, delivered to the populations in which we are interested, and measures the outcomes important to patients.’ We do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues<sup>28</sup> found *different* treatment benefits estimated by clinical trials in adult compared with those performed in children for 11% of drugs (14 out of 124). A commentary<sup>29</sup> on the paper pointed out that ‘kids are no little adults and not all kids are the same.’ They added ‘although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.’ We agree – the world’s children deserve better evidence for the dietary recommendations affecting them.

## Recommendations and remarks – Saturated fatty acids

**Page 37, Recommendation 1:** ‘*In adults and children whose saturated fatty acid intake is greater than 10% of total energy intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake (strong recommendation<sup>2</sup>)*’. This suggests any person over the age of two years with a saturated fat intake higher than 10.0% of energy should reduce their saturated fat intake and **that it does not matter which foods are targeted**. We are concerned about possible unintended consequences associated with this advice, such as intakes of under-consumed nutrients being further compromised.

We would also question the appropriateness of this recommendation for **frail elderly people**. The Australian Dietary Guidelines<sup>30</sup> make it clear that their guidelines are ‘not appropriate for frail elderly people or those with complex health conditions and an appropriate health professional should be consulted’ (page 27).

We suggest that if this recommendation goes ahead, the wording of this recommendation is amended to the following:

*‘In adults and children over 2 years whose saturated fatty acid intake is greater than 10% of total energy intake<sup>1</sup>, WHO recommends reducing saturated fatty acid intake in a way that ensures that the individual’s other nutritional and dietary requirements are met (strong recommendation<sup>2</sup>). This recommendation does not apply to frail elderly people or those with complex health conditions.’*

However, we would also question the **scientific basis for this recommendation** as the majority of studies that form the evidence for this were undertaken in adults who *already* had cardiovascular disease or diabetes.<sup>iii</sup> Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed,

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<sup>iii</sup> DART participants were men recovering from a MI, participants in the study by Houtsmuller were adults with newly diagnosed diabetes, participants in the study by Ley were people with impaired glucose tolerance or high normal blood glucose, participants in Moy 2001 had at least one CVD risk factor, participants in the MRC trial were men who had survived a first MI, those in the Oslo Diet Heart trial were men who had a previous MI, the Rose corn oil and olive trials involved men with angina or following a MI, the participants in the STARS trial were men with angina and those taking part in the WHI with CVD all had CVD at baseline.

**70% of the data** behind the strong recommendation for all adults and children ‘to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake’ comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children.

**Page 37, Recommendations 2-4.** According to footnote 3, ‘**Conditional** recommendations are those recommendations for which the WHO guideline development group is **uncertain** that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders (22)’.

If it is the case that there is uncertainty about whether desirable consequences outweigh undesirable consequences, we think it would be better to make **no recommendations**. WHO’s reputation and credibility are likely to be damaged if it recommends a dietary change that is later shown to have undesirable consequences. The sugar recommendations released by the WHO in 2015 have been widely referenced by policy makers around the world, but little distinction has been made between the strong and the conditional recommendations.

**Page 37, Remark 2:** It is good that this remark makes clear that the recommendations for children are based on blood lipid and blood pressure measures and that the rest of the evidence is an **extrapolation** of adult data to children. However, we do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues<sup>31</sup> found *different* treatment benefits estimated by clinical trials in adult compared with those performed in children for 11% of drugs (14 out of 124). A commentary<sup>32</sup> on the paper pointed out that ‘kids are no little adults and not all kids are the same.’ They added ‘although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.’ We agree – the world’s children deserve better evidence for the dietary recommendations affecting them.

**Page 38, Paragraph 1:** It is stated that ‘The evidence indicates that children’s growth is not compromised by reduction of saturated fatty acid intake.’ Growth is **more** than the variables measured (height, body weight, BMI and other measures of adiposity). There was not consideration of other aspects of growth such as bone accrual – indeed the authors of the Special Turku Coronary Risk Factor Intervention Project (STRIP) commented<sup>33</sup> that ‘further studies are needed to draw further conclusions about the influence of dietary counselling on bone health’. Therefore, we feel that the statement should be amended to:

*‘The evidence indicates that children’s height and body weight are not compromised by reduction of saturated fatty acid intake. Effects on other aspects of growth such as bone accrual are unknown.’*

We recommend that greater consideration be given to bone accrual prior to any recommendations being released.

**Paragraph 4, page 38:** Here is a classic example of ‘cherry picking’ findings to suit a particular argument, rather than presenting a balanced view. Three pre-determined **primary** outcomes were selected and the evidence for all three outcomes was judged to be of moderate quality using GRADE (page 3 of Hooper *et al.*, 2015).

These three results (all-cause mortality, CVD mortality and combined CV events) should always be given equal weight in the WHO recommendations. At present, there is disproportionate emphasis on the finding that reducing saturated fat leads to reduced combined CV events and not on the fact that it makes **no significant difference** to the risk of all-cause mortality or CVD mortality. In light of this, it is difficult to see how it is justifiable to make a strong recommendation to reduce saturated fatty acids when it is likely to make no difference to two out of three main outcomes. Paragraph 4 on page 38 even refers to the 'totality of evidence reviewed' but then fails to report the results for two out of three of the main results. We therefore recommend that this paragraph be re-written in a way that provides a more balanced representation of the results.

The WHO-commissioned systematic review and meta-analysis of observational studies examining intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes<sup>34</sup> concluded that '*saturated fats are not associated with all-cause mortality, CVD, CHD, ischaemic stroke, or type 2 diabetes*' yet the draft WHO recommendations are suggesting that there is enough evidence for a *strong* recommendation to reduce saturated fat intake. Again, this appears to be cherry picking the results to support preferred outcomes.

**Paragraph 5, page 38:** This paragraph justifies reducing saturated fatty acid intake on the basis of LDL cholesterol levels in adults and children. However, consideration of LDL cholesterol levels in isolation of other factors is meaningless. For example, 16:0 raises LDL cholesterol, but also raises HDL-cholesterol, reduces triglyceride-rich lipoproteins and remnants and has little effect on Apo-B. Mozaffarian (2016) commented: 'Continued prioritization of saturated fat reduction appears to rely on selected evidence: e.g. effects on LDL-cholesterol alone (discounting the other, complex lipid and lipoprotein effects); historical ecological trends in certain countries (e.g. Finland) but not in others; and expedient comparisons with polyunsaturated fat, the most healthful macronutrient.'

**Paragraph 3, page 39:** Although it is stated that 'the recommendation to not increase saturated fatty acid intake if intake is already below 10% of total energy intake (fourth recommendation) is based on the totality of evidence reviewed, a major omission is consideration of the impact of such a recommendation on the frail elderly. It is well acknowledged that the frail elderly do not require a low fat diet. Focus on the saturated fat intake of such people, as a result of this proposed WHO recommendation, could well reduce their access to nutrient-dense, protein providing foods such as cheese and whole milk.

Also, children between the ages of 2 and 5 years often consume higher amounts of dairy foods than older children and recommendation four has the potential to adversely impact on the provision of these foods in pre-school settings.

Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out<sup>35</sup>, they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed WHO saturated fat guidelines will adversely impact on intake of milk and dairy foods around the world.

**Paragraph 1, page 40:** As pointed out by Mozaffarian<sup>36</sup> (2016), '*Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.*' We recommend that the WHO makes future recommendations in relation to dietary patterns rather than single nutrients.

We also believe that the statement: *'public health interventions should aim to reduce saturated fat intake, while reducing total fat intake where necessary, and without increasing free sugar intake'* could detrimentally impact on the nutritional quality of the diet and should be amended to:

*'public health interventions aimed at reducing saturated fat intake should ensure that nutritional adequacy is not compromised and that free sugar intake is not increased'*

## Comments on the Summary of Evidence for *Trans* Fatty Acids

**Paragraph 1, page 31:** The WHO-commissioned systematic review of prospective cohort studies<sup>37</sup> is a key piece of evidence for the *trans* fatty acid recommendations, but (as stated in footnote 1 on page 21), **not** for the saturated fatty acid recommendations. This appears to be inconsistent and gives the impression that commissioned work is downgraded if it does not provide the expected results.

**Paragraph 2, page 31:** The conclusions of the second WHO-commissioned systematic review<sup>38</sup> of RCTs rely on theoretical calculations (*'coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminant TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA'*) and regression analysis unweighted for study size. Why was such a different analysis technique used for the systematic review of RCTs for saturated fatty acids and the systematic review of RCTs for *trans* fatty acids?

We would also highlight the fact that the RCTs lasted for between **14 and 56 days** yet it is expected that people will follow the WHO recommendations for years. In the systematic review of prospective cohort studies by de Souza and colleagues, Figure 4 clearly demonstrates that consumption of ruminant *trans* fats are **not** associated with any adverse health outcomes (all-cause mortality, CHD mortality, total CHD or ischemic stroke) and that its consumption is associated with reduced risk of type 2 diabetes (5/5 studies comprising 12,942 participants, RR 0.58 95% CI 0.46-0.74 P<0.001). The observation in relation to type 2 diabetes was based on a median of **14 years** of follow up (5-20 years).

**Paragraph 2, page 32:** The text states *'it was therefore determined that the available evidence did not support making a distinction between industrial and ruminant trans-fatty acids, and data solely from analyses of total trans-fatty acids were considered when formulating the recommendations on trans fatty acid intake.'* As the objective of these guidelines is to reduce the risk of NCDs (as stated on page 4), it is quite remarkable that the observation from five prospective cohort studies in which almost 13,000 adults have been followed for a median of 14 years<sup>39</sup> is completely disregarded in favour of theoretical mathematical calculations and unweighted regressions derived from studying 680 adults for 2 to 8 weeks<sup>40</sup>.

Figure 3 from the 2016 systematic review by Brouwer highlights the limited evidence behind the decision to make not distinction between industrial and ruminant *trans* fatty acids – there are just 5 data points for ruminant *trans* fatty acids and there are 17 data points for industrial *trans* fatty acids from short-term trials lasting 2 to 8 weeks. We believe that the available evidence does not support restricting intake of naturally occurring *trans* fats and that by doing so, may increase risk of type 2 diabetes – first do no harm.

**Paragraphs 3 and 4, page 36:** The text indicates that there were no studies conducted in children in relation to *trans* fatty acids and that evidence from adults was considered when formulating the recommendations for children ‘without downgrading for indirectness.’ As stated previously, we do not believe that it is appropriate to extrapolate results from RCTs in adults lasting 2 to 8 weeks to recommendations for children lasting years. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues<sup>41</sup> found *different* treatment benefits estimated by clinical trials in adults compared with those performed in children for 11% of drugs (14 out of 124). A commentary<sup>42</sup> on the paper pointed out that ‘kids are no little adults and not all kids are the same.’ They added ‘although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.’ We agree – the world’s children deserve better evidence for the dietary recommendations affecting them.

All of the eight references cited to support the view that abnormal changes in blood lipids are associated with early stages of CVD and are linked to future CV events, are over ten years old. Children with abnormal changes in blood lipids should be treated using modern, evidence-based techniques – their medical needs do not provide a rationale to treat the entire population in the same way. It is a bit like recommending statins to the entire population just because they benefit the health of those with elevated LDL cholesterol.

## Recommendations and remarks – *Trans* fatty acids

**Page 40, Recommendation 1:** ‘*In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing trans-fatty acid intake (strong recommendation<sup>2</sup>)*’. In light of Figure 4 from the WHO-commissioned systematic review by de Souza and colleagues which clearly demonstrates that consumption of ruminant *trans* fats over 5-20 years is **not** associated with any adverse health outcomes and that its consumption is associated with reduced risk of type 2 diabetes, we suggest that this recommendation be amended to: ‘

*In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing industrial trans-fatty acid intake (strong recommendation<sup>2</sup>)*’.

The updated wording would be less confusing to the general public as both the WHO dietary recommendations and the WHO’s REPLACE initiative announced in May 2018, would focus on industrial *trans* fatty acids.

**Page 40, Recommendations 2-4:** Similarly, we recommend that industrial *trans* fatty acids rather than all *trans* fatty acids are specified in the three conditional recommendations.

**Page 41, Paragraph 1:** The text indicates that the recommendations for *trans* fatty acid intake in children are based on extrapolation of adult data on CVD risk and blood lipids. As stated previously, we do not believe that it is appropriate to extrapolate results from adults to recommendations for children lasting years. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues<sup>43</sup> found *different* treatment benefits estimated by clinical trials in adults compared with those performed in children for 11% of drugs (14 out of 124). A

commentary<sup>44</sup> on the paper pointed out that ‘kids are no little adults and not all kids are the same.’ They added ‘although in older children treatment effects may be similar to adults, this may not be the case in younger children. Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.’ We agree – the world’s children deserve better evidence for the dietary recommendations affecting them.

**Page 41, Paragraph 2:** We believe that this paragraph should point out that theoretical models and regression analyses not weighted for study size, not actual comparisons, were the basis for the observations made in this paragraph<sup>45</sup> (*‘coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA’*). Why was such a different analysis technique used for the systematic review of RCTs for saturated fatty acids and the systematic review of RCTs for *trans* fatty acids?

We would also highlight the fact that the RCTs lasted for between **14 and 56 days** yet it is expected that people will follow the WHO recommendations for years.

**Page 41, paragraphs 3-4:** It does not appear that the totality of the evidence reviewed is the basis for the recommendation to reduce total *trans* fatty acid intake, as there is no mention that the WHO-commissioned systematic review by de Souza and colleagues clearly demonstrates that consumption of ruminant *trans* fats over 5-20 years is **not** associated with any adverse health outcomes and that its consumption is associated with *reduced* risk of type 2 diabetes.

**Page 42, paragraph 1:** We believe that this paragraph should point out that theoretical models and regression analyses, not actual comparisons, were the basis for the observations made in this paragraph<sup>46</sup> (*‘coefficients of Mensink were subsequently used to recalculate the effects of replacing industrial or ruminal TFA with either carbohydrates, a mix of SFA or a mix of cis-PUFA’*).

## Dissemination, translation and implementation, and monitoring and evaluation

**Page 44, paragraph 2 (Translation and implementation)** We note that the text states that these guidelines (on saturated and *trans* fatty acids) should be used in conjunction with other guidance on dietary goals and nutrition guidelines. We would encourage the WHO to make future recommendations in relation to **dietary patterns** rather than single nutrients. This is because, as explained by Mozaffarian<sup>47</sup> (2016), *‘Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.’*

While use of guidelines by policy-makers and programme managers to assess current intake of saturated fatty acids and *trans* fatty acids relative to a benchmark is important, it is critical that accompanying guidance from WHO stresses to these groups that these are not the only criteria on which to assess the healthiness of the diet ‘a cardioprotective diet pattern must be characterized by the healthful foods that are **included**, not simply specific items to be **avoided**<sup>48</sup>.’

The problem with using single **naturally present** nutrients as criteria for front-of-pack labelling, restricting sales of foods in schools, fiscal policies targeting foods and reformulation is that it encourages consumption of ultra-processed foods, which in turn are linked with chronic diseases,

including breast cancer.<sup>49, 50</sup> There is a need to distinguish between foods that naturally contain saturated fatty acids and *trans* fatty acids, and those with added saturated fatty acids and industrially produced *trans* fats. It is the nutrient-poor, energy-rich (discretionary) foods that contain added saturated fatty acids and industrially produced *trans* fats that should be labelled, restricted in schools, taxed and reformulated. These policies should be developed in a way that does not restrict consumption of healthy, nutritious foods that naturally contain saturated and ruminant *trans* fats.

It is always important to ‘first do no harm’ and simply focusing on total saturated fat and total *trans* fat content of a food is likely to lead to a reduction in intake of milk, yogurt and cheese, which is likely to negatively impact on the nutritional adequacy and healthfulness of the overall diet. For example, reduced intake of dairy foods (as a result of these foods being labelled unhealthy due to their saturated fat content) would be likely to negatively impact on intakes of key nutrients such as calcium (which is important for bone accrual in children<sup>51</sup>) and iodine (which is important for preventing cognitive impairment in children<sup>52,53</sup>). Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out<sup>54</sup>, they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed translation of the WHO saturated fat and *trans* fat guidelines will adversely impact on intake of milk and dairy foods around the world. Any translation of the finalised guidelines needs to ensure that this does not occur.

**Page 45, paragraph 4:** The text suggest that in relation to overall dietary guidance, it is ‘feasible to achieve the recommendations in these guidelines because a wide variety of fresh foods are naturally low in saturated fatty acids and *trans*-fatty acids’. We would point out that while eating a wide variety of fresh foods that are naturally low in saturated fatty acids and *trans* fatty acids is beneficial for many aspects of health, consumption of dairy foods (which are NOT naturally low in saturated fatty acids) is associated with a 6% lower risk of hip fracture<sup>55</sup> per daily serving in men and women (80,600 women and 43,306 men, up to 32 years follow up, RR=0.94, CI 0.90 to 0.98). This is important as the United Nations recently highlighted: ‘the number of older persons — those aged 60 years or over — is expected to more than double by 2050 and to more than triple by 2100, rising from 962 million globally in 2017 to 2.1 billion in 2050 and 3.1 billion in 2100. Globally, population aged 60 or over is growing faster than all younger age groups<sup>56</sup>.’

Similarly, there is level A evidence for calcium intake having a positive effect on bone accrual in children and adolescents<sup>57</sup>. Weaver and colleagues state ‘*The evidence since 2000 builds on earlier evidence, with additional RCTs showing a benefit to bone owing to the inclusion of dairy products in the diet. Dairy products contain colloidal calcium phosphate protein complexes in the form of casein micelles that have the minerals and nutrients needed for bone growth.*’

## Research gaps and future initiatives

**Page 46, research needed on saturated fatty acids and *trans* fatty acids:** All of the identified research relates to a reductionist, single nutrient paradigm. As pointed out by Mozaffarian<sup>58</sup> (2016), ‘*Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.*’ We



recommend that the WHO makes these and future recommendations in relation to dietary patterns rather than single nutrients. Within a dietary pattern framework, we suggest that RCTs are conducted in the population that future guidelines are made for (i.e. no extrapolation of results from secondary prevention trials to the general population, and no extrapolation of results from adults to children). In light of the rapidly ageing population, we recommend that greater focus is placed on determining a healthy diet for the elderly, not just avoidance of NCDs.

It is difficult to understand why type 2 diabetes and adiposity were priority health outcomes for children but not adults. We recommend that further work be undertaken considering these outcomes for adults. We also recommend that further consideration be given to the impact of reducing saturated and *trans* fat intake on frail elderly people (e.g. in terms of sarcopenia, falls, fractures and bone health), and on bone accrual in children. In addition, greater consideration is required of the impact on the nutritional adequacy of the diet when a general recommendation to reduce saturated and *trans* fat intake is given. Does it lead to lower meat and dairy intake? What are the impacts on nutrient intakes?

It is mentioned that the role of the NUGAG Subgroup on Diet and Health included advice on the 'balance of benefits and harms.' We believe that this is an area that requires greater consideration. The official endorsement of the low-fat diet in the 1980s and 1990s led to rising rates of overweight and type 2 diabetes, as consumers incorrectly interpreted the advice as being a licence to eat what you want, when you want as long as it is low in fat. Research is required that investigates how consumers and policy makers are likely to interpret the proposed guidelines related to saturated and *trans* fatty acid intakes.

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<sup>1</sup> Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

<sup>2</sup> Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. *Osteoporosis Int* 27,1281-1386

<sup>3</sup> Velasco I et al., (2018) Iodine as essential nutrient during the first 1000 days of life. *Nutrients* 10, 290

<sup>4</sup> Bath SC et al., (2017) Iodine concentration of milk-alternative drinks available in the UK. *Br J Nutr* 118, 525-32

<sup>5</sup> National Health and Medical Research Council (2013) Australian Dietary Guidelines. Canberra: National Health and Medical Research Council.

<sup>6</sup> Janiaud P et al., (2015) Different treatment benefits were estimated by clinical trials performed in adults compared with those performed in children. *Journal of Clinical Epidemiology* 68, 1221-31.

<sup>7</sup> Oostenbrink R & de Wildt SN, (2016) Drug trials: Kids are no little adults and not all kids are the same. *Journal of Clinical Epidemiology* 71, 111-12.

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- <sup>8</sup> Paakkunainen U et al., (2002) The Impact of Low-Saturated Fat, Low Cholesterol Diet on Bone Properties Measured Using Calcaneal Ultrasound in Prepubertal Children. *Calcified Tissue International*, 71, 219-226.
- <sup>9</sup> Ramsden C et al., (2013) Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. *BMJ* 2013, 346 e8707.
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- <sup>12</sup> Monterio C et al., (2017) The UN decade of nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutrition* 21, 5-17.
- <sup>13</sup> Ramsden C et al., (2013) Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. *BMJ* 2013, 346 e8707.
- <sup>14</sup> Food and Agriculture Organization of the United Nations (2013) Milk and dairy products in human nutrition. Rome: FAO.
- <sup>15</sup> Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225
- <sup>16</sup> Brouwer IA (2016) Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis. Geneva: World Health Organization.
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- <sup>18</sup> Johnson BJ et al., (2017) Contribution of discretionary foods and drinks to Australian children's intake of energy, saturated fat, added sugars and salt. *Children (Basel)* 4, 104 doi: [10.3390/children4120104](https://doi.org/10.3390/children4120104)
- <sup>19</sup> Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225
- <sup>20</sup> <https://www.foodnavigator.com/Article/2018/05/07/WHO-guidelines-on-saturated-fats-and-trans-fats> visited 31/5/18
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- <sup>48</sup> Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225
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## Survey response 67

### General information

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### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

Hearing response to WHO draft guidelines on saturated fatty acid and trans-fatty acid intake for adults and children

#### Summary

The recommendation to reduce the intake of saturated fatty acids (SAT), and replace them with polyunsaturated fat (PUFA) and monounsaturated fat (MUFA) in order to reduce the risk and incidence of cardiovascular disease (CVD) needs to take recent evidence showing that the health effects of SAT is entirely depending on the specific food source of SAT into consideration. The WHO draft report does acknowledge that "...emerging evidence has led to the suggestion that different saturated fatty acid-containing foods, such as dairy foods, may have different effects on risk of cardiovascular diseases and type 2 diabetes, either as a result of differing composition of saturated fatty acids across foods, other constituents of the foods, or a combination of the two (38-42). However, many questions remain to be answered before a clear understanding can be reached and firm conclusions drawn." and that "... there is a need for more studies comparing the effects of saturated fatty acids from different food sources (e.g. plant, animal and dairy) on CVD and mortality".

However, we would like to draw attention to the existing comprehensive research on this topic, and advice against ignoring evidence that shows beneficial effects of various foods containing SAT on CVD risk. The global recommendation to reduce the intake of SAT without pointing at specific food sources may cause the consumer to reduce intake of nutrient-dense foods that reduce the risk of CVD, type 2 diabetes, and other serious non-communicable diseases. Such a recommendation might work against the intentions of the WHO, and weaken the impact of the recommendations on the incidence and mortality of important diseases.

Today there is robust evidence to show that the food matrix in which SAT exists is a stronger determinant of health effects than SAT content alone, and a number of important food sources of SAT, such as eggs, cheese, yogurt and dark chocolate, are either neutral or protective against cardiovascular disease and type 2 diabetes. The forms of evidence provided are food-based meta-analyses of prospective observational studies, randomized controlled trials with well-established surrogate end-points, and experimental human mechanistic studies. This totality of evidence has demonstrated that the food matrix is more important for health effects than single ingredients. The general advice to reduce the intake of SAT, without pointing at specific foods sources, is very likely to be counterproductive as consumers who aim at reducing SAT in order to reduce CVD risk may well replace some of these nutrient-dense foods and choose less healthy alternatives. We strongly recommend a more food based translation of the recommendations on how to achieve the reduction in SAT intake. This will avoid the unnecessary reduction or exclusion of foods such as eggs, dark chocolate, cheese, and other fermented dairy, which are key sources of important nutrients such as high quality protein, calcium etc., and also have known health benefits. This has been recently extensively reviewed with regard to dairy foods (1).

We acknowledge that there is a lack of RTC's to show the effect these foods have on hard CVD end-points and mortality. However, in the absence of such trials WHO needs to consider the existing comprehensive evidence. To recommend that the public reduce intake of specific foods without evidence to substantiate that this will improve health is inconsistent with the principle of "do no harm". Governments are responsible for delivering population-directed dietary advice, which must generate benefit for most, and minimal collateral hazard for even very small subgroups, unless they can be identified and guided separately.

How robust is the evidence linking saturated fat to cardiovascular disease?

#### 1. Evidence from randomized controlled trials with hard end-points

The evidence to show that SAT is more harmful than PUFA and MUFA has weakened over recent years, and has been challenged by several meta-analyses of observational studies and RCT's. Even the most positive Cochrane analysis of RCT's from 2015 (the key WHO reference, Hooper et al. (2)) could not find any significant effect of high vs low SAT on 1) total mortality, 2) Cardiovascular mortality 3) Myocardial infarction 4) Non-fatal myocardial Infarctions 5) Stroke 6) Coronary Heart Disease events, 7) Coronary Heart Disease mortality. Only 8) The composite end-point "Combined cardiovascular events", provided a significant result [RR 0.83 (0.72 to 0.96)].

A 2017 meta-analysis of RCT's by Hamley assessed the effect of replacement of SAT with mostly n-6 PUFA with an aim to reducing the risk of coronary heart disease (3). They found, on the basis of results from the adequately controlled trials, that there was no effect for major CHD events (RR = 1.06, CI = 0.86-1.31), total CHD events (RR = 1.02, CI = 0.84-1.23), CHD mortality (RR = 1.13, CI = 0.91-1.40) and total mortality (RR = 1.07, CI = 0.90-1.26). The scientists concluded: "Available evidence from adequately controlled randomised controlled trials suggests replacing SAT with mostly n-6 PUFA is unlikely to reduce CHD events, CHD mortality or total mortality. The suggestion of benefits reported in earlier meta-analyses is due to the inclusion of inadequately controlled trials. These findings have implications for current dietary recommendations."

This analysis is consistent with the meta-analyses by Schwingshackl et al. (2013) and Ramsden et al. (2016), both of which found that replacement of saturated fat with linoleic acid did not lower risk of death from coronary heart disease or all causes (4,5).

#### 2. Evidence from randomized controlled trials with surrogate end-points

The WHO report excludes observational studies, and puts weight on a meta-analysis of RCT's that have assessed surrogate end-points such as serum lipids and lipoproteins, including total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglyceride, LDL cholesterol to HDL cholesterol ratio, total cholesterol to HDL cholesterol ratio, triglyceride to HDL cholesterol ratio, apolipoprotein A-I (ApoA-I) and apolipoprotein B (ApoB) total-cholesterol (6). The main findings are that replacement of SAT with PUFA results in small but significant reductions in total cholesterol, LDL cholesterol, in HDL cholesterol, triglycerides, and in total cholesterol to HDL cholesterol ratio, and LDL cholesterol to HDL cholesterol ratio of 0.034.

However, it is unclear if these changes in serum lipoproteins translate into a reduction in cardiovascular end-points and mortality. There is increasing recognition of the limitation of using LDL-cholesterol concentration as a marker of diet effects on CVD risk, as

atherogenicity of the LDL-particle is also determined by resistance against oxidation, size, composition and cytotoxicity. There is evidence that SAT increases the LDL-particle size, and thus potentially reduces CVD risk (7).

The weakness of serum LDL-cholesterol concentration and ratio to reflect changes in CVD risk following dietary interventions have been highlighted by RCT's showing that replacement of SAT with PUFA in the diet effectively lowers serum cholesterol but does not translate into lower risk of death from coronary heart disease or all causes(5). Therefore, caution should be exercised in interpreting the findings of the Mensink meta-analysis as supporting evidence for a beneficial effect of replacement of SAT with PUFA (6). In 2010 a consensus panel concluded that "Single risk factors have limitations when considered on their own because the effects of diet on CVD risk are mediated by many pathways, with blood lipids being only one. Although elevated LDL cholesterol is one of the major risk factors known, there is still a need for clinical endpoints for assessing the effects of diet on CVD risk." (8).

### 3. Evidence from observational studies

WHO does not assess the evidence derived from meta-analyses of prospective cohort studies, arguing that the quality of evidence for relevant outcomes is lower than in the analyses of RCTs, and that it was not possible to assess potential differential effects of replacing SAT with different nutrients in the meta-analysis of the cohort studies. However, in the assessment of totality of evidence all types of evidence need to be considered, and to exclude high-quality observational studies is a breach of the traditions of evidence-based medicine.

Indeed, we consider the observational studies very valuable for assessing the effect of SAT on CVD end-points, and particularly for the food-based analyses (see below). To summarize the outcome of the meta-analysis mentioned in the WHO report (9), they found that SAT are not associated with all cause mortality, CVD, CHD, ischemic stroke, or type 2 diabetes, and concluded that "dietary guidelines must carefully consider the health effects of recommendations for alternative macronutrients to replace trans fats and saturated fats". This is in line with the analysis by Siri-Tarino et al. (2010) and Harcombe Z et al. (2017) that showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD (10,11).

### 4. Evidence from food-based analyses of cardiovascular disease risks

In 2010 a consensus panel of experts concluded that: "There is increasing evidence to support that the total matrix of a food is more important than just its fatty acid content when predicting the effect of a food on CHD risk, e.g., the effect of SFAs from cheese on blood lipids and CHD may be counterbalanced by the content of protein, calcium, or other components in cheese. In addition, the special fatty acid profile (rumenic acid, trans vaccenic acid, and short-chain fatty acids) may modify the effect on CHD risk. Another example is dark chocolate, which has a high content of stearic acid, oleic acid, and polyphenols, and observational studies, mechanistic studies, and RCTs show that dark chocolate reduces risk factors of CVD." (8). Since then several food-based studies have been conducted, and today a number of meta-analyses have assessed the health effects of various foods containing significant amounts of SAT and concluded that future recommendations should be food based (1).

#### 4.1 Butter

Butter is the most SAT dense food and a meta-analysis by Pimpin et al. (2016) assessed observational studies linking butter intake to major disease outcomes and mortality (12). Based on 9 publications, together reporting on 636,151 unique participants with 6.5 million person-years of follow-up, and including 28,271 total deaths, 9,783 cases of incident cardiovascular disease, and 23,954 cases of incident diabetes, they found that butter consumption was weakly associated with all-cause mortality (N = 9 country-specific cohorts; per 14g (1 tablespoon)/day: RR = 1.01, 95%CI = 1.00, 1.03, P = 0.045), but was not associated with any cardiovascular disease (N = 4; RR = 1.00, 95%CI = 0.98, 1.02; P = 0.704), coronary heart disease (N = 3; RR = 0.99, 95%CI = 0.96, 1.03; P = 0.537), or stroke (N = 3; RR = 1.01, 95%CI = 0.98, 1.03; P = 0.737), and was inversely associated with incidence of diabetes (N = 11; RR = 0.96, 95%CI = 0.93, 0.99; P = 0.021).

#### 4.2 Eggs

Egg contains ~2.6 grams SAT/100 grams, and can easily be a significant contributor to daily SAT intake. However, eggs are also nutrient-dense foods that provide a wide range of important nutrients that are limitedly available in many other foods. High quality prospective, population based studies and a number of meta-analyses of prospective studies have found that egg consumption is not associated with risk of coronary heart disease, but with a lower risk of stroke, though subgroup analyses found an increase of CHD risk in diabetic populations (13-15). More recent studies have shown that the apparent increased risk of CVD among patients with type 2 diabetes is most likely due to lack of adjustment for confounding (16). Evidence from RCT's has shown neutral or beneficial effects on markers of diabetes and cardiovascular disease. For example, Fuller et al. compared the effects of an high-egg diet (12 eggs/wk) with a low-egg diet (



## Survey response 68

### General information

Family/last name
Selvaduray
Given/first name
Kanga Rani
Organization/affiliation
Malaysian Palm Oil Board
Sector
Government
Sector [Other]
Country
Malaysia

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Comments on Summary of Evidence 1. Only moderate quality evidence found in reduced SFA intake on overall CVD risk. Recommendation should be made with strong quality evidence from RCTs. 2. Source of SFAs, whether from animals or vegetables were not mentioned. 3. Type of CARBs also not included and mentioned. 4. Recommendations were only based blood lipid outcomes, stroke and CHD (how about other NCDs) 5. Reduction of SFA also reduced the good cholesterol, HDL. 6. The amount (%) and type (omega 3 OR omega 6) of PUFA to be considered when replacing SFA in adults and children 7. Evidence also found that cognitive development and iron status were limited with reduced intake of SFAs (item 3-SFA recommendation for children) and evidence should be studied further. 8. Recommendation for TFA intake was based on adult data on CVD risk. Further evidence in children is needed.
Recommendations (for saturated fatty acids only)
Several current studies have highlighted that it's not fats but carbohydrates is the major culprit for CVD and many more health ailments [1, 2]. Besides, there are more emerging evidence grounded on epidemiological and systematic review that there is no association between saturated fatty acids and all-cause mortality [3, 4]. Based on recent epidemiological cohort study of 18 countries, it was reported that high fat intake (35% E) had lower risk of early death (23%) and stroke (18%) as compared to low fat intake. Besides saturated fat (SFA) intake (

Remarks (for saturated fatty acids only)
There is increasing evidence over the recent years to show that there is no association of saturated fats to all-cause mortality including cardiovascular disease, coronary heart disease and ischemic stroke. A recent prospective cohort study from 18 countries in five continents (Dehghan et al, 2017), published in The Lancet dated 4th November 2017, reported that saturated fat consumption shows no association with cardiovascular disease, myocardial infarction, or cardiovascular disease mortality, instead saturated fat had an inverse association with stroke. However, there is an association between high carbohydrate intake and mortality especially in countries where the intakes of carbohydrates are particularly high. It was reported that high fat intake (35% E) had lower risk of early death (23%) and stroke (18%) as compared to low fat intake. Besides saturated fat (SFA) intake (
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

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## Final comments

Please provide any final thoughts or comments below.
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Evidence based on changes in surrogate markers may not necessarily relate to clinical disease end-point and mortality. There is increasing evidence over the recent years to show that there is no association of saturated fats to all-cause mortality including cardiovascular disease, coronary heart disease and ischemic stroke. The upper limit and types of PUFAs should be taken into account when recommending replacing SFA with PUFA. The effects of carbohydrate should be factored in when making recommendations of limiting SFA intake.
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## Survey response 69

### General information

Family/last name
Fode
Given/first name
Peder
Organization/affiliation
Danish Drink and Food Federation
Sector
Private sector
Sector [Other]
Country
Denmark

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
We found that the inclusion of CLA (conjugated linoleic acid) to the definition of trans fats (see draft p.11 and p.40) is planned here. This is not acceptable from our perspective, as scientifically not justified and legally contradictory to EU law (Definition for trans fats as laid down in Reg (EU) No 1169/2011, Annex I. We think the CLA topic would also be of interest for all companies who deal with milk based specialties or infant products on milk basis. If this TFA definition will stay as is in the draft, it will be a major burden for the presentation and marketing of milk based products also.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>We found that the inclusion of CLA (conjugated linoleic acid) to the definition of trans fats (see draft p.11 and p.40) is planned here. This is not acceptable from our perspective, as scientifically not justified and legally contradictory to EU law (Definition for trans fats as laid down in Reg (EU) No 1169/2011, Annex I.</p> <p>We think the CLA topic would also be of interest for all companies who deal with milk based specialties or infant products on milk basis. If this TFA definition will stay as is in the draft, it will be a major burden for the presentation and marketing of milk based products also.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 70

### General information

Family/last name
Wassell
Given/first name
Paul
Organization/affiliation
Golden Agri-Resources (PT SMART Tbk)
Sector
Private sector
Sector [Other]
Country
Indonesia

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Think the background does not highlight that Palm oil is a rich source of MUFA. Palm oil has many derivatives and it can be more informative to mention the main source of fatty acid coming from which palm oil derivative.
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
page 28 For Individual fatty acids, it will be more appropriate to review evidence on impact of isomer position of the fatty acid (which position with TAG)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
<p>I believe, this is a fair draft.</p> <p>Palm oil derivatives, have played major role to switch product formulas away from trans fatty acids or animal fat products. It would be beneficial for everyone to know, precisely which types of fatty acid are coming from which palm oil derivatives. The olein is rich in MUFA. Super oleins are over 50% MUFA.</p> <p>As regards intake of palmitic acid. This is interesting - but more information is needed to know about the position of palmitic acid within human diet.</p>

## Survey response 72

### General information

Family/last name
Mensik
Given/first name
Petr
Organization/affiliation
EU Specialty Food Ingredients
Sector
Private sector
Sector [Other]
Country
Belgium

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
<p>EU Specialty Food Ingredients welcomes the WHO initiative to develop guidelines for saturated fatty acids and trans fatty acids intake for adults and children. EU Specialty Food Ingredients supports the WHO recommendation to reduce the intake of saturated fatty acids and trans-fatty acids in adults and children, which we believe is consistent with the current science.</p> <p>?</p> <p>We noted that for the recommendations on saturated fatty acids intake, evidence from prospective cohort studies (PSCs) was not taken into account and focus in the assessment has been put only on randomized controlled trials (RCTs). Nevertheless, newer PCSs not included in this assessment confirm findings from RCTs, specifically when addressing saturated fatty acids replacement regimes (PUFA, MUFA or carbohydrates).</p>
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)



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Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

### *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)

#### Summary of evidence (for trans-fatty acids only)

EU Specialty Food Ingredients welcomes the WHO initiative to develop guidelines for saturated fatty acids and trans fatty acids intake for adults and children. EU Specialty Food Ingredients supports the WHO recommendation to reduce the intake of saturated fatty acids and trans-fatty acids in adults and children, which we believe is consistent with the current science.

As regards trans fatty acids intake, results from both RCTs and PCSs were considered in the evidence evaluation, and the included meta-analyses reflect well the scientific status quo. However, we oppose to the inclusion of conjugated linoleic acid (CLA) to the definition of trans fatty acids for the following reasons:

Current global rules and legislation as regards trans fatty acids definition should be taken into account.

The following CODEX ALIMENTARIUS definition of trans fatty acids clearly excludes conjugated forms: "For the purpose of the Codex Guidelines on Nutrition Labelling and other related Codex Standards and Guidelines, trans fatty acids are defined as all the geometrical isomers of monounsaturated and polyunsaturated fatty acids having non-conjugated interrupted by at least one methylene group (-CH<sub>2</sub>-CH<sub>2</sub>-) carbon-carbon double bonds in the trans configuration." (Codex 2017)

The definition of trans fatty acids in Regulation (EU) No. 1169/2011 on Food Information to Consumers, Annex I, Specific Definitions expressly includes only those forms of polyunsaturated fatty acids where double bonds are interrupted by at least one methylene group: "4. 'trans fat' means fatty acids with at least one non-conjugated (namely interrupted by at least one methylene group) carbon-carbon double bond in the trans configuration". (EU 2011)

While several countries do not explicitly rule conjugated or non-conjugated forms in their legislation (i.e. Australia, Argentina, India, Latvia), most countries with provisions to trans fatty acids labelling and reducing strategies to industrially produced trans fatty acids in the EU and Europe, but also in Asia, Africa and South-America follow exactly this definition – clearly excluding conjugated forms, i.e. Austria, Countries of the Cooperation Council for the Arab States of the Gulf, Colombia, Denmark, Hungary, Iceland, Norway, the Philippines, Singapore, Slovenia, South Africa, South-Korea, Sweden, Switzerland.

The United States and Canada repeatedly differentiated conjugated forms from the definition of trans fatty acids, especially when setting legal implementations to the reduction of industrially produced trans fatty acids originating from fat hydrogenation: US FDA expressly excludes conjugated forms of fatty acids with a trans double bond from their definition of trans fatty acids for food labelling (US FDA 2003). In addition, while the latest ban of partially hydrogenated oils, the main source for industrially produced trans fatty acids, there was again no inclusion of CLA into the definition, simply as the evidence for adverse health effects goes back to the trans fatty acids coming from partially hydrogenated oils. "This order does not apply to the use of conjugated linoleic acid (CLA) as a food ingredient." (USA FDA 2015)

In Canada, Food and Drug Regulation, B.01.001(1) by Minister of Justice gives the definition to trans fats: "trans fatty acids [...] means unsaturated fatty acids that contain one or more isolated or non-conjugated double bonds in a trans-configuration." (Canada 2018). In addition, Health Canada expressly excluded CLA as food ingredient from the scope of prohibition of partially hydrogenated oils in 2017 (Canada 2017).

Current scientific evidence to CLA and adverse health effects related to industrially produced trans fatty acids shall be taken into account.

In general, adverse health effects that have been ascribed to trans fatty acids are not scientifically proven to be a general feature of CLA. There is a chemical difference between those fatty acids with isolated and those with conjugated double bonds. Differences in chemical structure usually lead to different reactivities. To our knowledge, there is a clear clinical and epidemiological evidence to the adverse health effects of industrially produced trans fatty acids from partial fat hydrogenation, which is yet not the case for the chemically differing molecule of CLA. Taking today's scientific data into account, we realize, that there is yet no convincing evidence from clinical studies that CLA would behave like trans fatty acids in the human body.

Already in 2004, the European Food Safety Authority (EFSA) stated that "In conclusion, while there is some evidence of adverse effects of supplemental CLA in humans for the trans-10,cis-12 isomer, no such effects were observed for CLA supplements containing mixtures of the trans-10,cis-12 and cis-9,trans-11 isomers. Furthermore, the adverse effects of the trans-10,cis-12 isomer were observed only at intake levels one or two orders of magnitude higher than those corresponding to intake from foods. Few studies have investigated the health effects in humans of naturally occurring CLA from foods and evidence is weak and conflicting with respect any health effects at current levels of intake."

Specifically, for high-quality specified CLA products on the market, a relative 1:1 mixture of cis9, trans11 and trans10, cis12 conjugated linoleic acid isomers obtained from vegetable oils by a distinct manufacturing process is used mainly in the form of food supplements and similar applications for use as an adjunct in body fat reduction regimes. As stated by EFSA in 2010 to these products, there is no evidence of adverse effects on blood lipids, i.e. biomarkers of CVD risk by the consumption of this specified form of CLA: "The Panel considers that consumption of the 1:1 CLA mixture under the proposed conditions of use has no significant effect on LDLcholesterol concentrations, and that the magnitude of the changes observed in HDL- and triglyceride concentrations is unlikely to have an impact on coronary heart disease risk." (EFSA 2010 a and b)

In 2010, EFSA issued its Scientific Opinion on Dietary Reference Values for fats, including saturated fatty acids, polyunsaturated

fatty acids, monosaturated fatty acids, trans fatty acids, and cholesterol. In this scientific opinion a clear differentiation of CLA from the adverse effects caused by industrially produced trans-fatty acids is stated: "There is no convincing evidence that any of the conjugated linoleic acids isomers in the diet play a role in prevention or promotion of diet-related diseases. The Panel therefore proposes not to set any Dietary Reference Value for conjugated linoleic acids." (EFSA 2010 c)

In 2012, after reviewing the data as available from the studies Wanders et al. 2010 (Effect of a high intake of conjugated linoleic acid on lipoprotein levels in healthy human subjects) and Brouwer et al. 2010 (Effect of animal and industrial trans fatty acids on HDL and LDL cholesterol levels in humans – a quantitative review), which are referred to in the draft WHO guideline to be the justification for inclusion of CLA to the trans fatty acids definition, EFSA confirmed its prior position that "the additional information provided does not contain evidence that would modify the previous conclusions reached by the Panel regarding the effects of CLA on blood lipids and lipoproteins". (EFSA 2012)

In addition, one of the most recent reviews referred to in the draft WHO guideline, the reference (73) by Brouwer published 2016 under WHO, Geneva (Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis) to our understanding does not provide new evidence to CLA: Studies referred to in this review in context with CLA have either already been part of EFSA's 2012 opinion (Brouwer et al 2010. PLoS One 2010;5(3):e9434, Desroches et al 2005. Am J Clin Nutr 2005;82(2):309-3019, and Brown et al 2011. Nutr Res 2011;31(1):33-41) or do not deal with CLA particularly (Zock Katan 1992. J Lipid Res 1992;33(3):399-410).

We would also like to point out that neither in the draft WHO guideline nor in the referred to review by Brouwer (WHO) 2016 the recent review 'Current issues surrounding the definition of trans-fatty acids: implications for health, industry and food labels' by Wang and Proctor from 2013 was mentioned. Even though the underlying studies used in this might be considered in the draft WHO guidelines and the Brouwer (WHO) 2016 review, it is questionable whether the findings of this review in differentiation of CLA and TFA's were taken into account.

Finally, this review by Brouwer/WHO 2016 has already been part of Health Canada's evaluation to prohibition of partially hydrogenated oils in 2017 with the result of excluding CLA from the trans fatty acids prohibition (see above).

#### Summary

Trans fatty acids are those fatty acids with trans double bonds that originate from industrial partial oil hydrogenation processes. The current global regulatory framework overall clearly excludes conjugated fatty acids from the definition of trans fatty acids.

We believe that the argumentation for inclusion of CLA to the trans fatty acids definition in the draft WHO guidelines does not provide convincing scientific evidence that would allow for such inclusion.

While the evidence for negative health effects with regard to CVD risk factors is strong for industrially produced trans fatty acids originating from partial oil hydrogenation, it is unlikely that CLA has such negative effect.

Overall, we conclude that CLA has to be excluded from definition of trans fatty acids in this guidelines. Neither is CLA a trans fatty acid according to relevant international definitions, nor does the available data justify conclusion on negative effects on CVD system.

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Nutrient Content Claims, and Health Claims

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Br J Nutr. 2013 Oct;110(8):1369-83

Recommendations (for trans-fatty acids only)

Remarks (for trans-fatty acids only)

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Already in 2004, the European Food Safety Authority (EFSA) stated that "In conclusion, while there is some evidence of adverse effects of supplemental CLA in humans for the trans-10,cis-12 isomer, no such effects were observed for CLA supplements containing mixtures of the trans-10,cis-12 and cis-9,trans-11 isomers. Furthermore, the adverse effects of the trans-10,cis-12 isomer were observed only at intake levels one or two orders of magnitude higher than those corresponding to intake from foods. Few studies have investigated the health effects in humans of naturally occurring CLA from foods and evidence is weak and conflicting with respect any health effects at current levels of intake."

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In 2010, EFSA issued its Scientific Opinion on Dietary Reference Values for fats, including saturated fatty acids, polyunsaturated fatty acids, monosaturated fatty acids, trans fatty acids, and cholesterol. In this scientific opinion a clear differentiation of CLA from the adverse effects caused by industrially produced trans-fatty acids is stated: "There is no convincing evidence that any of the conjugated linoleic acids isomers in the diet play a role in prevention or promotion of diet-related diseases. The Panel therefore proposes not to set any Dietary Reference Value for conjugated linoleic acids." (EFSA 2010 c)

In 2012, after reviewing the data as available from the studies Wanders et al. 2010 (Effect of a high intake of conjugated linoleic acid on lipoprotein levels in healthy human subjects) and Brouwer et al. 2010 (Effect of animal and industrial trans fatty acids on HDL and LDL cholesterol levels in humans – a quantitative review), which are referred to in the draft WHO guideline to be the justification for inclusion of CLA to the trans fatty acids definition, EFSA confirmed its prior position that “the additional information provided does not contain evidence that would modify the previous conclusions reached by the Panel regarding the effects of CLA on blood lipids and lipoproteins”. (EFSA 2012)

In addition, one of the most recent reviews referred to in the draft WHO guideline, the reference (73) by Brouwer published 2016 under WHO, Geneva (Effect of trans-fatty acid intake on blood lipids and lipoproteins: a systematic review and meta-regression analysis) to our understanding does not provide new evidence to CLA: Studies referred to in this review in context with CLA have either already been part of EFSA’s 2012 opinion (Brouwer et al 2010. PLoS One 2010;5(3):e9434, Desroches et al 2005. Am J Clin Nutr 2005;82(2):309-3019, and Brown et al 2011. Nutr Res 2011;31(1):33-41) or do not deal with CLA particularly (Zock Katan 1992. J Lipid Res 1992;33(3):399-410).

We would also like to point out that neither in the draft WHO guideline nor in the referred to review by Brouwer (WHO) 2016 the recent review ‘Current issues surrounding the definition of trans-fatty acids: implications for health, industry and food labels’ by Wang and Proctor from 2013 was mentioned. Even though the underlying studies used in this might be considered in the draft WHO guidelines and the Brouwer (WHO) 2016 review, it is questionable whether the findings of this review in differentiation of CLA and TFA’s were taken into account.

Finally, this review by Brouwer/WHO 2016 has already been part of Health Canada’s evaluation to prohibition of partially hydrogenated oils in 2017 with the result of excluding CLA from the trans fatty acids prohibition (see above).

#### Summary

Trans fatty acids are those fatty acids with trans double bonds that originate from industrial partial oil hydrogenation processes. The current global regulatory framework overall clearly excludes conjugated fatty acids from the definition of trans fatty acids.

We believe that the argumentation for inclusion of CLA to the trans fatty acids definition in the draft WHO guidelines does not provide convincing scientific evidence that would allow for such inclusion.

While the evidence for negative health effects with regard to CVD risk factors is strong for industrially produced trans fatty acids originating from partial oil hydrogenation, it is unlikely that CLA has such negative effect.

Overall, we conclude that CLA has to be excluded from definition of trans fatty acids in this guidelines. Neither is CLA a trans fatty acid according to relevant international definitions, nor does the available data justify conclusion on negative effects on CVD system.

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#### References

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- EFSA 2010 c. Scientific Opinion on Dietary Reference Values for fats, including saturated fatty acids, polyunsaturated fatty acids, monosaturated fatty acids, trans fatty acids, and cholesterol. The EFSA Journal 2010; 8 (5): 1601
- EFSA 2012. Statement on the safety of the "conjugated linoleic acid (CLA)-rich oils" Clarinol® and Tonalin® TG 80 as Novel Food ingredients. The EFSA Journal 2012 10 (5): 2700
- EU 2011. Regulation (EU) No. 1169/2011 on Food Information to Consumers, Annex I, Specific Definitions
- US FDA 2003. Federal Register, Vol. 68, Issue 133, July 11, 2003, Food Labeling: Trans Fatty Acids in Nutrition Labeling, Nutrient Content Claims, and Health Claims
- US FDA 2015. Federal Register Vol. 80 Issue 116, June 17, 2015, Final Determination Regarding Partially Hydrogenated Oils
- Wang Y, Proctor SD. Current issues surrounding the definition of trans-fatty acids: implications for health, industry and food labels. Br J Nutr. 2013 Oct;110(8):1369-83

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Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

### Final comments

Please provide any final thoughts or comments below.

## Survey response 73

### General information

Family/last name	Bahmanpour
Given/first name	Salmeh
Organization/affiliation	Shiraz University of Medical Sciences
Sector	Academic/research
Sector [Other]	
Country	Iran (Islamic Republic of)

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Evidence summaries and GRADE assessments were discussed and reviewed by the WHO Nutrition Guidance Expert Advisory Group (NUGAG) Subgroup on Diet and Health as part of WHO's guideline development process(1) The PICO questions and priority health outcomes guiding this review were discussed and developed by the NUGAG Subgroup on Diet and Health. A PRISMA checklist was been completed(1) .  1. Te Morenga L, Montez JM. Health effects of saturated and trans-fatty acid intake in children and adolescents: Systematic review and meta-analysis. PloS one. 2017;12(11):e0186672.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
- To assess the effect of reducing saturated fat intake and replacing it with carbohydrate (CHO), polyunsaturated (PUFA) or monounsaturated fat (MUFA) and/or protein on NCD mortality and cardiovascular morbidity,(2)  2-Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. The Cochrane Library. 2015.
Background (same for saturated fatty acids and trans-fatty acids)
Public health dietary advice on prevention of cardiovascular disease (CVD) suggests reducing the amount of saturated fat we eat, by cutting down on animal fats, is good for our health and leads to a reduced risk of dying or getting cardiovascular disease (heart disease or stroke)(3).  3. Mensink RP, Organization WH. Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis. 2016.



Guideline development process (same for saturated fatty acids and trans-fatty acids)
<ul style="list-style-type: none"> <li>•Protein and alcohol intake , (hydrogenated) very long chain (n-3) PUFA (fish oils) and medium-chain fatty acids (MCFA) or behenic acid were also excluded(2)</li> <li>•total PUFA in these studies can be considered to equal PUFA with 18 carbon atoms (linoleic acid plus -linolenic acid)(2).</li> <li>•TFA intake of 2% of total energy intake or less were included(2).</li> <li>•All review was conducted in accordance with the WHO's guideline development process,based on the Cochrane Collaboration approach<sup>24</sup> and reported according to the MOOSE and PRISMA checklist(3)</li> </ul> <p>2.Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. The Cochrane Library. 2015.</p> <p>3.Mensink RP, Organization WH. Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis. 2016.</p>
Summary of evidence (for saturated fatty acids only)
<ul style="list-style-type: none"> <li>-the effect of SFA reduction on serum lipids and lipoproteins is highly dependent on the nature of replacement.</li> <li>-different replacement scenarios have different effects on serum lipids and lipoproteins</li> <li>-Replacing the energy from saturated fat with polyunsaturated fat appears to be a useful strategy, and replacement with carbohydrate appears less useful, but effects of replacement with monounsaturated fat were unclear(3)</li> <li>-The most recent American Heart Association guidelines suggest that Americans should "Aim for a dietary pattern that achieves 5% to 6% of calories from saturated fat" and "Reduce percent of calories from saturated fat"(3).</li> <li>-Dietary guidelines for children and adolescents should continue to recommend diets low in saturated fat(1).</li> </ul> <p>1.Te Morenga L, Montez JM. Health effects of saturated and trans-fatty acid intake in children and adolescents: Systematic review and meta-analysis. PloS one. 2017;12(11):e0186672.</p> <p>3.Mensink RP, Organization WH. Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis. 2016.</p>
Recommendations (for saturated fatty acids only)
<ul style="list-style-type: none"> <li>-Dietary periods of intervention no shorter than 13 days</li> <li>-Results of the multiple regression analysis indicated that effects on the serum lipoprotein profile of reducing SFA intake by replacing a mixture of SFA with cis-PUFA or cis-MUFA more favourable than replacing SFA with a mixture of carbohydrates(2).</li> <li>-</li> <li>-Dietary guidelines for children and adolescents should continue to recommend diets low in saturated fat(1).</li> <li>-Interventions targeting reduction in saturated fat intakes amongst children and adolescents should focus on the core components of children's diets(1).</li> </ul> <p>1.Te Morenga L, Montez JM. Health effects of saturated and trans-fatty acid intake in children and adolescents: Systematic review and meta-analysis. PloS one. 2017;12(11):e0186672.</p> <p>2.Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. The Cochrane Library. 2015</p>
Remarks (for saturated fatty acids only)
<ul style="list-style-type: none"> <li>-Dietary and policy recommendations frequently focus on reducing saturated fatty acid consumption for improving cardiometabolic health, based largely on ecologic and animal studies. The main three SFA in Human Diet are myristic acid, palmitic acid and Stearic Acid. it is well-established that Dietary saturated fatty acids (C12-C16) are hypercholesterolemic and increase CHD risk. Evidence also suggests that SFA intake may correlate with increased colorectal cancer, breast cancer , diabetes and insulin resistance, and stroke. The US Dietary Guidelines recommend consuming less than 10%E (percentage of total energy intake) from SFA , and the American Heart Association less than 7%E . Health effects of reducing SFA consumption vary depending on whether the replacement nutrient is carbohydrate (CHO), monounsaturated fat (MUFA), or polyunsaturated fat (PUFA)?</li> </ul>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
<ul style="list-style-type: none"> <li>-effects of SFA with less than 12 carbon atoms or more than 18 carbon atoms could not be estimated due to lack of information(2).</li> </ul> <p>2- Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. The Cochrane Library. 2015</p>

Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)
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- trans fatty acids are associated with increased all cause mortality.(1)

1. De Souza RJ, Mente A, Maroleanu A, Cozma AI, Ha V, Kishibe T, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *Bmj*. 2015;351:h3978.

Guideline development process (same for saturated fatty acids and trans-fatty acids)
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- Findings in prospective cohorts were generally consistent with those from case-control studies, which found that higher exposure to trans fats (whether measured by food frequency questionnaire or biomarker) was associated with a 51% increased odds of CHD(1)

- The World Cancer Fund panel, however, found insufficient evidence to implicate trans fats specifically for any type of cancer.(1)

1. De Souza RJ, Mente A, Maroleanu A, Cozma AI, Ha V, Kishibe T, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *Bmj*. 2015;351:h3978.

Summary of evidence (for trans-fatty acids only)

Recommendations (for trans-fatty acids only)

Remarks (for trans-fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

Annexes 1, 6, 7 (for trans-fatty acids only)

Additional comments

## Final comments

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Please provide any final thoughts or comments below.

-[?] Dietary and policy recommendations frequently focus on reducing saturated fatty acid consumption for improving cardiometabolic health, based largely on ecologic and animal studies. The main three SFA in Human Diet are myristic acid, palmitic acid and Stearic Acid. it is well-established that Dietary saturated fatty acids (C12-C16) are hypercholesterolemic and increase CHD risk. Evidence also suggests that SFA intake may correlate with increased colorectal cancer, breast cancer , diabetes and insulin resistance, and stroke. The US Dietary Guidelines recommend consuming less than 10%E (percentage of total energy intake) from SFA , and the American Heart Association less than 7%E . Health effects of reducing SFA consumption vary depending on whether the replacement nutrient is carbohydrate (CHO), monounsaturated fat (MUFA), or polyunsaturated fat (PUFA)?

## Survey response 74

### General information

Family/last name
Strilich
Given/first name
Tracy
Organization/affiliation
Stepan Company
Sector
Private sector
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

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Additional comments

*Trans*-fatty acids

#### Executive summary (same for saturated fatty acids and trans-fatty acids)

We support the recommendations by WHO to reduce the trans fatty acid intake in adults and children. However, we disagree with the inclusion of conjugated linoleic acid (CLA) in the definition of a trans fatty acid (TFA), as found on page 11 of the Executive Summary, for the following reasons:

- When evaluating all data leading to the conclusion that plasma cholesterol would be the cause of cardiovascular disease, it is quite evident that cardiovascular disease is not so much caused by cholesterol itself, but instead by a low grade inflammatory reaction in the body for which plasma cholesterol is a biomarker. This argumentation was published in *Lipids in Health and Disease* 2012; 11:149. (<https://lipidworld.biomedcentral.com/articles/10.1186/1476-511X-11-149>)

This paper explains why plasma cholesterol is a good predictor for cardiovascular disease, but not a cause. Cardiovascular disease is more likely caused by an activation of HMGCoA reductase which promotes inflammatory reactions. For that reason, statins (HMGCoA inhibitors) are cardioprotective. However, some drugs lower plasma cholesterol by a mechanism which coincides with an increasing HMGCoA reductase activity. Those drugs promote cardiovascular disease as published in *N Engl J Med* 2007; 356:1620-1630. (<https://www.nejm.org/doi/full/10.1056/NEJMoa071359>).

In conclusion, plasma cholesterol is not the cause of cardiovascular disease. Therefore, the WHO should be cautious in translating effects on plasma cholesterol into cardiovascular risk.

- The negative conclusions on saturated fats that were presented in the WHO draft guidelines are mainly based on evidence that saturated fats increase plasma cholesterol. Actual evidence that it causes cardiovascular disease is close to absent, as supported by *Journal of the American College of Nutrition* 2014; 33:79 – 88 and *The American Journal of Clinical Nutrition* 1 March 2010; Volume 91, Issue 3, Pages 535 – 546.

(<https://www.tandfonline.com/doi/abs/10.1080/07315724.2014.878633?journalCode=uacn20and>  
<https://academic.oup.com/ajcn/article/91/3/535/4597110>)

The studies as referred to above do not support the notion that there is a clear relationship between diet-induced increases in plasma cholesterol and cardiovascular disease.

- In view of common structure-response relationships, it is very unlikely that all trans fats cause cardiovascular disease, regardless of the number of double bonds in the fatty acid or if the trans bond is conjugated. From a structure point of view this group of molecules is simply too diverse to activate a common receptor involved in causing cardiovascular disease. In general, from a physiological point of view and shown in various studies, isolated trans fats or saturated fats do not behave as a group at all. Therefore, it is very unlikely that they share the potential of causing cardiovascular disease

- It is much more likely that specific trans fatty acids, like elaidic acid, cause health issues. For industrial trans fats the evidence is relatively clear that they are involved in causing cardiovascular disease. However, the effect of these fats on plasma cholesterol is by far not enough to explain the size of the effect. It is unclear why trans fats cause cardiovascular disease, but plasma cholesterol does not seem to play a crucial role. The assumed structure-effect relationship between all saturated and all trans fats with cardiovascular disease is therefore likely to be incorrect. Since industrial trans fats do cause cardiovascular disease, but this effect cannot be explained effects on plasma cholesterol, it is illogical to use the plasma cholesterol biomarker to ban all fatty acids with a trans bond in its structure.

- As very related saturated fatty acids have very different biological effects in many biological models and behave very differently regarding uptake and metabolism a common guideline to treat them all as unsafe fats based on a common effect on plasma cholesterol only is scientifically incorrect.

- In addition to having negligible effects on plasma cholesterol, exposure to CLA was associated with a lower risk of myocardial infarction in basic and multivariate models as published in *The American Journal of Clinical Nutrition* 1 July 2010; Volume 92, Issue 1, Pages 34 – 40. (<https://academic.oup.com/ajcn/article/92/1/34/4597390>). In view of this, of the above and the fact that the trans bond of CLA is present in a conjugated form (which is structurally very different from an unconjugated trans-bond), we consider it logical to exclude CLA from the current trans-fat discussion.

Stepan Lipid Nutrition is in favor of protecting the world population against unhealthy food ingredients. However, in our view this guideline is too much guided by biomarkers for diseases, whereas risks should be based on effects of foods on the disease itself. Therefore, we conclude that CLA should be excluded from the definition of TFA in this guideline. Also we believe that MCTs are a completely separate group of saturated fats which do not pose any risk at all on cardiovascular disease.

#### Scope and purpose (same for saturated fatty acids and trans-fatty acids)

#### Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

## Survey response 75

### General information

Family/last name	Hajjar
Given/first name	Kalila
Organization/affiliation	FEDIOL
Sector	Private sector
Sector [Other]	
Country	Belgium

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>FEDIOL is the European federation representing the interests of the European vegetable oil and protein meal industry. Directly and indirectly, FEDIOL covers about 150 processing sites that crush oilseeds and/or refine crude vegetable oils. These plants belong to around 35 companies. It is estimated that over 80% of the EU crushing and refining activity is covered by the FEDIOL membership structure.</p> <p>FEDIOL welcomes the possibility to provide feedback to the WHO Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children.</p> <p>FEDIOL has carefully been through the document. Overall, FEDIOL appreciates the document, which is in line with current scientific knowledge and provides a comprehensive overview and new insights from published science. It represents a huge work in assessing available science and deriving guidelines.</p> <p>FEDIOL members will continue working on reformulation practices in partnership with industry upper in the chain to further improve the nutrition composition of food products.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
<p>Overall, FEDIOL appreciates the document, which is in line with current scientific knowledge and provides a comprehensive overview and new insights from published science. It represents a huge work in assessing available science and deriving guidelines.</p>



Recommendations (for saturated fatty acids only)
Overall, FEDIOL appreciates the document, which is in line with current scientific knowledge and provides a comprehensive overview and new insights from published science. It represents a huge work in assessing available science and deriving guidelines.
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
We have however noted an error in the retranscription of the recommendations provided in page 91 , where we understand, in light of all evidence highlighted in the WHO draft guidelines that the mentioned 1% should be read as “10%” and should hence be corrected accordingly.
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
<p>FEDIOL welcomes the possibility to provide feedback to the WHO Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children.</p> <p>FEDIOL has carefully been through the document. Overall, FEDIOL appreciates the document, which is in line with current scientific knowledge and provides a comprehensive overview and new insights from published science. It represents a huge work in assessing available science and deriving guidelines.</p> <p>FEDIOL members will continue working on reformulation practices in partnership with industry upper in the chain to further improve the nutrition composition of food products.</p>
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
<p>FEDIOL has carefully been through the document. Overall, FEDIOL appreciates the document, which is in line with current scientific knowledge and provides a comprehensive overview and new insights from published science. It represents a huge work in assessing available science and deriving guidelines.</p> <p>FEDIOL further welcomes the definition of TFAs as including all fatty acids with a double bond in the trans configuration regardless of whether they come from ruminant sources or are produced industrially .</p>
Recommendations (for trans-fatty acids only)
FEDIOL has carefully been through the document. Overall, FEDIOL appreciates the document, which is in line with current scientific knowledge and provides a comprehensive overview and new insights from published science. It represents a huge work in assessing available science and deriving guidelines.
Remarks (for trans-fatty acids only)

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Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

### Final comments

Please provide any final thoughts or comments below.

## Survey response 76

### General information

Family/last name
Baka
Given/first name
Athanasia
Organization/affiliation
IMACE
Sector
Private sector
Sector [Other]
Country
Belgium

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
IMACE welcomes the WHO SFA-TFA Open Consultation and is in line with the draft guidelines proposed. IMACE is agreement and strongly supports the non-discrimination between industrially produced and ruminant sources of TFA.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
No Comments
Background (same for saturated fatty acids and trans-fatty acids)
IMACE highlights the efforts made by our members, over the last 20 years and has been supporting the industry's initiatives to reducing TFA in margarines and spreads, via reformulation, optimisation of refining processes in accordance with the IMACE Code of Practice on TFA. Following these numerous industry actions, significant overall reductions in the industrially produced TFA content of food products sold to the final consumers have been achieved and are already far below 2% on fat basis or 1% on energy basis.
Guideline development process (same for saturated fatty acids and trans-fatty acids)
No Comments
Summary of evidence (for saturated fatty acids only)
No Comments
Recommendations (for saturated fatty acids only)
<p>The WHO Guidelines document suggests using polyunsaturated fatty acids as a source of replacement energy, "if needed", when reducing saturated fatty acid intake (conditional recommendation).</p> <p>IMACE finds that the addition of 'if needed' is confusing, either it should be removed, or the statement should be aligned with the text on page 10 which stated "when a replacement is needed".</p> <p>IMACE recommends that the statement could be formulated as following:</p> <ul style="list-style-type: none"><li>- WHO suggests using polyunsaturated fatty acids as a source of replacement energy when reducing saturated fatty acid intake</li><li>- WHO suggests replacing saturated fatty acids by polyunsaturated fatty acids when a replacement is needed</li></ul>
Remarks (for saturated fatty acids only)
No Comments

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
No Comments
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
No Comments
Annexes 1, 6, 7 (for saturated fatty acids only)
No Comments
Additional comments
No Additional Comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
As indicated in the previous section, IMACE welcomes the WHO SFA-TFA Open Consultation and is in line with the draft guidelines proposed. IMACE is in agreement and strongly supports the non-discrimination between industrially produced and ruminant sources of TFA.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
IMACE acknowledges and strongly agrees with the fact that ruminant sources of TFA are included in the scope of the guideline document.
Background (same for saturated fatty acids and trans-fatty acids)
<p>IMACE highlights the efforts made by our members, over the last 20 years and has been supporting the industry's initiatives to reduce TFA in margarines and spreads, via reformulation, optimisation of refining processes in accordance with the IMACE Code of Practice on TFA. Following these numerous industry actions, significant overall reductions in the industrially produced TFA content of food products sold to the final consumers have been achieved and are already far below 2% on fat basis or 1% on energy basis.</p> <p>Consequently, the intake of industrially produced TFA in the EU has decreased considerably over recent years to a level, where there is no longer a general public health concern. This decrease was also highlighted by EFSA in its opinions of 2004(2) and 2009(3), based on data analysis at national level.</p>
Guideline development process (same for saturated fatty acids and trans-fatty acids)
No Comments
Summary of evidence (for trans-fatty acids only)
No Comments
Recommendations (for trans-fatty acids only)
IMACE is in agreement and supports the recommendation as laid down in the Guideline document
Remarks (for trans-fatty acids only)
No Comments
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
IMACE is in favour of regulatory measures (specifically in the form of a legal maximum limit), restraining the intake of industrially produced TFA that should aim to significantly contribute to the TFA public health objective. In addition, a regulatory measure should be easy to implement in practice.
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
No Comments
Annexes 1, 6, 7 (for trans-fatty acids only)
No Comments
Additional comments
No Additional Comments

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## Final comments

Please provide any final thoughts or comments below.
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Over the past 20 years, IMACE members have been actively and successfully reformulating the vegetable fat-based spreads to reduce industrially produced TFA. Therefore, the intake of non-ruminant TFA in the majority of EU Member States is below the limits of public health concern.
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IMACE is totally aligned with the proposed WHO guidelines document. In particular, we welcome the inclusion of both sources of TFA (i.e. industrially produced and ruminant) as they both have the same health effects and should therefore be treated in a non-discriminatory way. This would ensure that the public health issue concerning total TFA intake is addressed properly.
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## Survey response 77

### General information

Family/last name
Pottie
Given/first name
Siska
Organization/affiliation
IMACE
Sector
Private sector
Sector [Other]
Country
Belgium

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
IMACE welcomes the WHO SFA-TFA Open Consultation and is in line with the draft guidelines proposed. IMACE is agreement and strongly supports the non-discrimination between industrially produced and ruminant sources of TFA.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
No Comments
Background (same for saturated fatty acids and trans-fatty acids)
<p>IMACE highlights the efforts made by our members, over the last 20 years and has been supporting the industry's initiatives to reduce TFA in margarines and spreads, via reformulation, optimisation of refining processes in accordance with the IMACE Code of Practice on TFA. Following these numerous industry actions, significant overall reductions in the industrially produced TFA content of food products sold to the final consumers have been achieved and are already far below 2% on fat basis or 1% on energy basis.</p> <p>Consequently, the intake of industrially produced TFA in the EU has decreased considerably over recent years to a level, where there is no longer a general public health concern. This decrease was also highlighted by EFSA in its opinions of 2004(2) and 2009(3), based on data analysis at national level.</p>
Guideline development process (same for saturated fatty acids and trans-fatty acids)
No Comments
Summary of evidence (for saturated fatty acids only)
No Comments
Recommendations (for saturated fatty acids only)
<p>WHO guidelines document suggests using polyunsaturated fatty acids as a source of replacement energy "if needed", when reducing saturated fatty acid intake (conditional recommendation).</p> <p>IMACE finds that the addition of 'if needed' is confusing. We suggest that it should either be removed, or the statement should be aligned with the text on page 10 which states "when a replacement is needed".</p> <p>IMACE recommends that the statement could be formulated as following:</p> <ul style="list-style-type: none"><li>- WHO suggests using polyunsaturated fatty acids as a source of replacement energy when reducing saturated fatty acid intake</li><li>- WHO suggests replacing saturated fatty acids by polyunsaturated fatty acids when a replacement is needed".</li></ul>

Remarks (for saturated fatty acids only)
No Comments
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
No Comments
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
No Comments
Annexes 1, 6, 7 (for saturated fatty acids only)
No Comments
Additional comments
No Additional Comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
As indicated in the previous section, IMACE welcomes the WHO SFA-TFA Open Consultation and is in line with the draft guidelines proposed. IMACE is in agreement and strongly supports the non-discrimination between industrially produced and ruminant sources of TFA.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
IMACE acknowledges and strongly agrees with the fact that ruminant sources of TFA are included in the scope of the guideline document.
Background (same for saturated fatty acids and trans-fatty acids)
<p>IMACE highlights the efforts made by our members, over the last 20 years and has been supporting the industry's initiatives to reduce TFA in margarines and spreads, via reformulation, optimisation of refining processes in accordance with the IMACE Code of Practice on TFA. Following these numerous industry actions, significant overall reductions in the industrially produced TFA content of food products sold to the final consumers have been achieved and are already far below 2% on fat basis or 1% on energy basis.</p> <p>Consequently, the intake of industrially produced TFA in the EU has decreased considerably over recent years to a level, where there is no longer a general public health concern. This decrease was also highlighted by EFSA in its opinions of 2004(2) and 2009(3), based on data analysis at national level.</p>
Guideline development process (same for saturated fatty acids and trans-fatty acids)
No Comments
Summary of evidence (for trans-fatty acids only)
No Comments
Recommendations (for trans-fatty acids only)
IMACE is in agreement and supports the recommendation as laid down in the Guideline document.
Remarks (for trans-fatty acids only)
No Comments
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
IMACE is in favour of regulatory measures (specifically in the form of a legal maximum limit), restraining the intake of industrially produced TFA that should aim to significantly contribute to the TFA public health objective. In addition, a regulatory measure should be easy to implement in practice.
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
No Comments
Annexes 1, 6, 7 (for trans-fatty acids only)
No Comments

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Additional comments
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No Additional Comments
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## Final comments

Please provide any final thoughts or comments below.
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<p>Over the past 20 years, IMACE members have been actively and successfully reformulating the vegetable fat-based spreads to reduce industrially produced TFA. Therefore, the intake of non-ruminant TFA in the majority of EU Member States is below the limits of public health concern.</p>
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<p>IMACE is totally aligned with the proposed WHO guidelines document. In particular, we welcome the inclusion of both sources of TFA (i.e. industrially produced and ruminant) as they both have the same health effects and should therefore be treated in a non-discriminatory way. This would ensure that the public health issue concerning total TFA intake is addressed properly.</p>
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## Survey response 78

### General information

Family/last name
Renaldi
Given/first name
Rocco
Organization/affiliation
International Food and Beverage Alliance
Sector
Private sector
Sector [Other]
Country
Switzerland

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)

Remarks (for saturated fatty acids only)
<p>IFBA would like to thank the World Health Organization (WHO) for the opportunity to comment on the “Draft Guidelines on Saturated Fatty Acid and Trans-Fatty Acid Intake for Adults and Children” and provide feedback.</p> <p>We support the WHO strong recommendations to reduce saturated fatty acid intake in adults and children to no more than 10% of total energy intake, and trans-fatty acid intake to no more than 1% of total energy intake.</p> <p>IFBA also fully supports the efforts of the WHO, governments, and the public health community to reduce the global intake of industrially produced trans-fatty acids (iTFA). IFBA members have committed to reducing iTFA from partially hydrogenated oils (PHOs) in their products worldwide to no more than one gram trans-fatty acid per 100 grams of product by the end of 2018 at the latest.</p> <p>In support of both WHO Guidelines recommending no more than 1% total energy intake from all trans-fat and an intake of saturated fat not exceeding 10% of total energy intake, IFBA member companies will seek wherever possible to replace PHOs with unsaturated fats.</p> <p>PHOs can also be formed during thermal processing or be found in processing aids (release agents), food additives (emulsifiers), and carriers; however, these incidental uses result in trace amounts of PHO and are nutritionally negligible. Nonetheless, we will work with our supply chain partners with the aim of removing PHOs also from these sources wherever feasible.</p> <p>IFBA member companies are further committed to working in collaboration with governments, health authorities, civil society and food and beverage industry associations to share best practices and help guide other companies, particularly Small and Medium Enterprises, through the process of substituting PHOs. We call on food producers in all sectors to take prompt action and we stand ready to support effective measures to work toward the elimination of industrially produced trans-fats and to ensure a level playing field in this area. Please see here for more details.</p> <p>Finally, we would suggest rephrasing the following recommendation, for greater clarity:</p> <p>“WHO suggests using polyunsaturated fatty acids as a source of replacement energy, if needed, when reducing saturated fatty acid intake (conditional recommendation).”</p> <p>As follows:</p> <p>“WHO suggests replacing saturated fatty acids with [poly]unsaturated fatty acids when a replacement is needed”</p> <p>This would be aligned with the language used in this regard on p. 10 of the draft Guidelines: a replacement may be needed but not necessarily, or even likely, as a source of replacement energy. Furthermore, replacement with polyunsaturated fats is a very specific recommendation; replacement with unsaturated fats in general may be a stronger recommendation.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
<p>IFBA would like to thank the World Health Organization (WHO) for the opportunity to comment on the “Draft Guidelines on Saturated Fatty Acid and Trans-Fatty Acid Intake for Adults and Children” and provide feedback.</p> <p>We support the WHO strong recommendations to reduce saturated fatty acid intake in adults and children to no more than 10% of total energy intake, and trans-fatty acid intake to no more than 1% of total energy intake.</p> <p>IFBA also fully supports the efforts of the WHO, governments, and the public health community to reduce the global intake of industrially produced trans-fatty acids (iTFA). IFBA members have committed to reducing iTFA from partially hydrogenated oils (PHOs) in their products worldwide to no more than one gram trans-fatty acid per 100 grams of product by the end of 2018 at the latest.</p> <p>In support of both WHO Guidelines recommending no more than 1% total energy intake from all trans-fat and an intake of saturated fat not exceeding 10% of total energy intake, IFBA member companies will seek wherever possible to replace PHOs with unsaturated fats.</p> <p>PHOs can also be formed during thermal processing or be found in processing aids (release agents), food additives (emulsifiers), and carriers; however, these incidental uses result in trace amounts of PHO and are nutritionally negligible. Nonetheless, we will work with our supply chain partners with the aim of removing PHOs also from these sources wherever feasible.</p> <p>IFBA member companies are further committed to working in collaboration with governments, health authorities, civil society and food and beverage industry associations to share best practices and help guide other companies, particularly Small and Medium Enterprises, through the process of substituting PHOs. We call on food producers in all sectors to take prompt action and we stand ready to support effective measures to work toward the elimination of industrially produced trans-fats and to ensure a level playing field in this area. Please see here for more details.</p> <p>Finally, we would suggest rephrasing the following recommendation, for greater clarity:</p> <p>“WHO suggests using polyunsaturated fatty acids as a source of replacement energy, if needed, when reducing saturated fatty acid intake (conditional recommendation).”</p> <p>As follows:</p> <p>“WHO suggests replacing saturated fatty acids with [poly]unsaturated fatty acids when a replacement is needed”</p> <p>This would be aligned with the language used in this regard on p. 10 of the draft Guidelines: a replacement may be needed but not necessarily, or even likely, as a source of replacement energy. Furthermore, replacement with polyunsaturated fats is a very specific recommendation; replacement with unsaturated fats in general may be a stronger recommendation.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

Final comments

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Please provide any final thoughts or comments below.

## Survey response 79

### General information

Family/last name
Griffiths
Given/first name
James
Organization/affiliation
Council for Responsible Nutrition (CRN)
Sector
Other
Sector [Other]
Non-profit trade assn
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
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The Council for Responsible Nutrition (CRN) is the leading trade association for the dietary supplement and nutritional products industry, representing manufacturers of dietary ingredients and of national brand name and private label dietary supplements, many of which are multinational and already actively selling ingredients, finished products and services globally.

CRN in consultation with its member companies reviewed the published draft World Health Organization (WHO) guidelines on “Saturated fatty acid and trans-fatty intake for adults and children”, and per the request for public input via draft consultation (for a period ending June 1, 2018) offer the following.

CRN and CRN members support the discussion and activity of WHO on the subject of trans-fatty intake and trans-fatty acids.

CRN and CRN members support the recommendations of WHO on the need to reduce and/or prevent the increase of Trans-Fatty Acid (TFA) intake in adults and children. However, there needs to be a clear distinction regarding the manufactured TFA produced industrially through the partial hydrogenation of oils (TFA by PHOs).

CRN and CRN members want to draw WHO’s attention to the inappropriate grouping of naturally-occurring TFA and artificially-generated TFA (through the partial hydrogenation of oils (PHOs)). The US Food and Drug Administration (FDA) recognizes the fact that trans-fats occur naturally in meat and dairy products from ruminant animals and that naturally-occurring trans-fat is unavoidable in ordinary non-vegan diets. FDA has excluded naturally-occurring TFA and specifically, Conjugated Linoleic Acid (CLA) from the recent FDA notice on PHOs (Final Determination Regarding Partially Hydrogenated Oils; 80 FR (116) 34650), meaning CLA and other naturally-occurring TFA continue to be permitted as food ingredients in the US and many other countries.

Scope and purpose (same for saturated fatty acids and trans-fatty acids)
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Background (same for saturated fatty acids and trans-fatty acids)
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Current global rules and legislations to the TFA definition must be taken into account.

Following the Codex Alimentarius (Codex) definition “For the purpose of the Codex Guidelines on Nutrition Labelling and other related Codex Standards and Guidelines, trans-fatty acids are defined as all the geometrical isomers of monounsaturated and polyunsaturated fatty acids having non-conjugated, interrupted by at least one methylene group (-CH<sub>2</sub>-CH<sub>2</sub>-), carbon-carbon double bonds in the trans configuration” (Codex, 2017). This definition excludes specifically the trans-fats (vaccenic acid and conjugated linoleic acid) that are present especially in human milk, dairy products, and beef.

The TFA definition from European Union (EU) Regulation No. 1169/2011 on Food Information to Consumers, Annex I, Specific Definitions expressly only includes those forms of polyunsaturated fatty acids where double bonds are interrupted by at least one methylene group: i.e., “‘trans fat’ means fatty acids with at least one non-conjugated (namely interrupted by at least one methylene group) carbon-carbon double bond in the trans configuration” (EU 2011).

In accordance with the definition as described above, individual countries in the EU and elsewhere in Europe (i.e., Austria, Denmark, Hungary, Iceland, Norway, Slovenia, Sweden, Switzerland) have implemented or plan to implement rules for reducing and/or labeling of TFA, where the majority of them follows exactly this definition.

In Canada, Food and Drug Regulations by the Minister of Justice includes the definition from B.01.001(1) about trans fats, i.e., “trans fatty acids [...] means unsaturated fatty acids that contain one or more isolated or non-conjugated double bonds in a trans-configuration.” (Canada 2018). In addition, Health Canada expressly excluded CLA used as a food ingredient from the scope of the Canadian PHO Prohibition in 2015 (Canada 2015).

The US FDA expressly excludes conjugated forms of fatty acids with a trans double bond from their definition of trans-fatty acids for food labelling (US FDA 2003). In addition, with the latest ban of partially hydrogenated oils (PHO), the main source for industrially-produced TFA, there was again no inclusion of CLA into the definition, simply as the evidence for adverse health effects goes back to only the TFA coming from PHO. “This order does not apply to the use of conjugated linoleic acid (CLA) as a food ingredient” (USA FDA 2015).

Guideline development process (same for saturated fatty acids and trans-fatty acids)
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Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
<p>Conclusion</p> <p>CRN and CRN members consistently focus on the need to let “good science” drive dietary recommendations and regulatory decisions. It is our suggestion that the WHO guidance document make a distinction between industrially-generated TFA and “naturally-occurring” TFA. Conjugated linoleic acid and vaccenic should be exempted from the definition of TFA as CLA and vaccenic acids are present in human milk, dairy products and meat.</p> <p>Should WHO have further questions that CRN and CRN members could address, please do not hesitate to contact me at your earliest convenience.</p>
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments
<p>References</p> <p>Canada 2015, Health Canada, September 15, 2017, Notice of Modification - Prohibiting the Use of Partially Hydrogenated Oils (PHOs) in Foods, Reference Number: [NOM/ADM-C-2017-3]</p> <p>Canada 2018, Minister of Justice, Food and Drug regulation, C.R.C., c. 870, Part B Foods, Division 1 General, B.01.001.(1), current to April 24, 2018</p> <p>Codex 2017. GUIDELINES ON NUTRITION LABELLING - CAC/GL 2-1985, Adopted in 1985. Revised in 1993 and 2011. Amended in 2003, 2006, 2009, 2010, 2012, 2013, 2015, 2016 and 2017. ANNEX adopted in 2011. Revised in 2013, 2015, 2016 and 2017.</p> <p>EFSA 2004. Opinion of the scientific panel on dietetic products, nutrition and allergies on a request from the Commission related to the presence of trans fatty acids in foods and the effect on human health of the consumption of trans fatty acids. The EFSA Journal 81: 1-49.</p> <p>EFSA 2010 a. Scientific Opinion on the safety of "conjugated linoleic acid (CLA)-rich oil" (Tonalin® TG 80) as a Novel Food ingredient. The EFSA Journal 2010 8 (5): 1600</p> <p>EFSA 2010 b. Scientific Opinion on the safety of "conjugated linoleic acid (CLA)-rich oil" (Clarinol®) as a Novel Food ingredient. The EFSA Journal 2010 8 (5): 1601</p> <p>EFSA 2012. Statement on the safety of the "conjugated linoleic acid (CLA)-rich oils" Clarinol® and Tonalin® TG 80 as Novel Food ingredients. The EFSA Journal 2012 10 (5): 2700</p> <p>EU 2011. Regulation (EU) No. 1169/2011 on Food Information to Consumers, Annex I, Specific Definitions</p> <p>US FDA 2003. Federal Register, Vol. 68, Issue 133, July 11, 2003, Food Labeling: Trans Fatty Acids in Nutrition Labeling, Nutrient Content Claims, and Health Claims</p> <p>US FDA 2015. Federal Register Vol. 80 Issue 116, June 17, 2015, Final Determination Regarding Partially Hydrogenated Oils</p>

## Final comments

Please provide any final thoughts or comments below.

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## Survey response 80

### General information

Family/last name
Forouhi
Given/first name
Nita
Organization/affiliation
MRC Epidemiology Unit, University of Cambridge School of Clinical Medicine
Sector
Academic/research
Sector [Other]
Country
United Kingdom

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
With the considerable progress that has been made in advancing the research and scientific understanding, it is important to not limit the focus of this report to nutrient intake (saturated fatty acids), but also to include an appraisal of and provide evidence based recommendations on foods that are rich in saturated fats but differ in their health effects.
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)



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#### Summary of evidence (for saturated fatty acids only)

1. In the evidence review for RCTs of SFAs and CVD endpoints among adults, it seems that only one systematic review (Hooper et al, 2015) is included, but the reason for this is not provided. Other reviews of RCT evidence for CHD (or CVD) events exist, such as by (but not limited to) Mozaffarian D (PLOS Med, 2010; PMID: 20351774), Chowdhury R (Annals Int Med 2014; PMID: 24723079), Hamley S (J Nutrients, 2017; PMID: 28526025), Schwingshackl (BMJ Open, 2014; PMID: 24747790). There is also an extensive literature of prospective cohort studies on this topic, but one systematic review (De Souza et al, BMJ 2015 as ref 61) was mentioned, but not included in evidence appraisal formally. As also highlighted later (in our comments on recommendations for future research), valuable information can be obtained from prospective studies.
2. The description of the evidence on SFA intake and CVD (e.g. on page 5, page ) is misleading when it states (as in the original publication - ref 24 in the report – Hooper et al, 2015) that “non-significant reductions in risk of CHD events and MI were also observed with reduced SFA intake”. It is better (and more correct) to make a clear statement that the only significant finding was for the endpoint of combined (composite of) CVD events, and that the results were null (non-significant) for all other cardiovascular (fatal or non-fatal) endpoints including MI, CHD, or stroke.
3. On page 25-26, an explanation is provided about sub-group analyses that were applied to assess effects of replacing SFA with other macronutrients. A critique of the pros and cons of the decision to group trials based on statistical significance of the difference in SFA intake and replacement nutrient between the intervention and control groups regardless of whether or not that macronutrient was the main replacement for SFAs is warranted. This decision seems post-hoc.
4. On page 17, the report acknowledges the importance of food sources of nutrients when it states that “emerging evidence has led to the suggestion that different saturated fatty acid-containing foods, such as dairy foods, may have different effects on risk of cardiovascular diseases and type 2 diabetes, either as a result of differing composition of saturated fatty acids across foods, other constituents of the foods, or a combination of the two. However, many questions remain to be answered before a clear understanding can be reached and firm conclusions drawn”. But such evidence is not appraised, and the recommendations do not link back SFA intake (ie nutrient intake) to types of food sources of the SFAs. Indeed there is a strong body of evidence from prospective cohort studies that different foods that are rich in SFAs vary in their effects on NCD endpoints such as CVD or type 2 diabetes – such as, positive (adverse) associations with processed red meat intake but inverse associations with fermented dairy products (e.g. reviewed in Mozaffarian D, Circulation 2016; PMID: 26746178, and elsewhere). Such messaging is very important because it is the overall foods and dietary patterns, not the nutrient solely, that exert health effects. There is also increasing evidence from fatty acid biomarkers studies that different individual saturated fatty acids relate differently with health endpoints (see below). In light of considerable progress in research and scientific understanding on the importance of food sources of saturated fats, it will be a missed opportunity to not explicitly make statements about the food sources when making recommendations on SFAs. For policy makers and stakeholders clear advice is needed on what foods people might safely consume, and which ones are beneficial, harmful or neutral for health.
5. It is stated on page 21 that one of the questions appraised was on “lower intake of individual saturated fatty acids relative to higher intake” – (a) at this point, the footnote labelled as #1 (page 21) does not refer to individual SFAs, and (b) the report specifically reviewed this topic (page 27-28) with a single systematic review of RCTs as included in reference #59. There is additional evidence from prospective studies, that includes some of the fatty acids reported in those RCTs but also others such as odd-chain saturated fatty acids (15:0 and 17:0, rich in dairy fat) and others, in relation to clinical end points such as type 2 diabetes and CVD (some – not all – examples include Forouhi NG et al, Lancet DE 2014, PMID:25107467; Chowdhury R et al, Annals Int Med 2014, PMID:24723079; Khaw KT et al, PLOS Med 2012, PMID:22802735; Liang J et al, Crit Rev Food Sci Nutr, PMID:28001085). It is not clear why such evidence was not reviewed, as this supplements the RCT evidence in a meaningful way.

Recommendations (for saturated fatty acids only)
<p>1. There is no recommendation on food sources rich in different types of SFAs. A statement (or explanation) on different sources of foods that are rich in SFA but have differential health effects should be incorporated, as not all SFA-rich foods are equal or homogeneous in overall effects because of different mix of types of fatty acids within foods in different proportions (mix of SFA, PUFA, MUFA, trans-FA from industrial or ruminant sources), different individual SFA (e.g. odd chain or even chain) within foods, and other non-SFA or non-fat components in whole foods (e.g. vitamins, minerals, probiotic compounds, antioxidants, phytochemicals fibre, etc).</p> <p>2. The first and second recommendations, as presented, are confusing and misleading (both for SFA and trans-FA).</p> <p>(i) It is unclear how health professionals, policy makers, other stakeholders (or the general public should they access these guidelines) will be able to implement the first and second recommendations (for both SFA and trans-FA – at pages 8 and 11 and also at pages 37 and 40). Though the evidence review makes a distinction, in practice the two seem highly linked. The first (strong) recommendation (e.g. for SFA) advises reducing SFA intake among those with SFA intake greater than 10% of total energy intake, while the second recommendation (which is conditional) advises reducing SFA intake to less than 10% of TEI. The second statement would imply a reduction if the intake is indeed greater than the stated threshold. The two statements, as presented, are confusing.</p> <p>(ii) It is unclear if the first recommendation has the implication of individual level assessment of SFA intake as it states this applies to adults or children whose SFA intake that is greater than 10% of TEI (and similarly with a 1% of TEI threshold for trans-FA). If this is the case, information should be provided on how this might be assessed in practice. The second recommendation seems to be directed at a population level. Clarity is needed.</p> <p>(iii) The advice to reduce SFA intake, without considering a replacement nutrient is at odds with prior evidence reviews and guidelines, that also included the Hooper et al review (that was used in this report). This will add to prevailing confusion on this topic.</p> <p>(iv) Given the progress in scientific understanding, a recommendation, or at the very least an explanation, on differences between specific individual SFAs should be incorporated (and linked to food sources – see earlier comment).</p> <p>3. The third recommendation (for SFA intake) is confusing: “using PUFA as a source of replacement energy, if needed, when reducing SFA intake”. This stipulation (“if needed”) is confusing as the reason is unclear, particularly in the context of isocaloric substitutions within a diet. What criteria should be applied to judge when replacement is needed? This is also at variance with other existing guidelines and evidence reviews that promote the importance of replacing a reduction in SFA intake with PUFA intake (and not with carbohydrate – particularly refined carbohydrate – intake).</p>
Remarks (for saturated fatty acids only)
<p>Some key strengths of this report include the evidence review and recommendations for adults as well as children, both for saturated and for trans-fatty acids intake, and the clear specification of the quality of the appraised evidence. The comments below are intended to help improve the final report, highlighting areas of concern. Two main concerns are (i) the lack of linking the nutrient (saturated fat, SFA) to food sources of the nutrient, which is critical for meaningful translation of the evidence review; (ii) the report’s recommendations, as currently presented, may lead to more confusion on this topic than already exists.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

## Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)

### A. Saturated fatty acids

1. For SFAs, the sole focus for future research is highlighted as the need for future RCTs as follows: (i) large RCTs of replacement of SFAs with other nutrient types on CVD incidence and mortality in different geographical regions, (ii) RCTs comparing the effects of saturated fatty acids from different food sources (e.g. plant, animal and dairy), and (iii) RCTs in settings with low saturated fatty acid intake (i.e. less than 10% of total energy intake) to assess lower thresholds, above which saturated fatty acid intake increases the risk of CVD and mortality.

The recommendation to conduct large RCTs does not weigh up the issues of logistics including massive cost (prohibitive in many geographical contexts) and the length of time that would be needed to accrue sufficient events for 'hard' clinical endpoints (or mortality) among healthy populations or even at-risk populations, as well as the common challenges of RCTs of nutritional factors – adherence, blinding, contamination, suitable control group, intensity of 'treatment' in intervention and control arms, sustainability of specific diets over a long time till events develop, the difference between dietary advice and actual intake, the issue of energy intake (restricted or ad libitum), and so on. The current recommendations therefore seem aspirational, despite the mention on page 5 about considerations on feasibility, cost, and other logistical issues.

It is important to acknowledge that in nutritional research (as in much of public health research that is not of pharmaceutical products or devices), the classical 'hierarchy of evidence' with double-blind placebo controlled randomised trials as the gold standard, needs to be considered alongside logistical issues and the strengths of other study designs including (a) well conducted large prospective studies that account for confounding factors, and also (b) RCTs of intermediate risk factors for the hard clinical end-points – these can include but are not limited to blood pressure, weight, a variety of lipid markers, inflammation, glycaemia, and other risk factors. It is important to highlight that in many countries prospective studies of NCDs including CVD endpoints are currently either lacking altogether, or when they do exist, diet/nutrition are not assessed due to the (perceived or real) complexity of dietary assessment and interpretation. This is an important research gap.

2. In research on lipid endpoints, there has been a focus on examining LDL-c, HDL-c, and sometimes their ratio and triglycerides, but researchers should be encouraged to include assessment of non-HDL cholesterol and also include apolipoproteins. This is important to enable the study of and comparisons across a wide range of lipids/lipoproteins, which is commensurate with the increasing understanding that LDL-c is only one marker of cardiovascular risk (e.g. recent article from the PURE study, Mente A et al, Lancet Diabetes Endocrinol 2017; PMID:28864143; Emerging Risk Factor Collaboration Di Angelantonio, JAMA 2012; PMID:22797450; Emerging Risk Factor Collaboration Di Angelantonio, JAMA 2012; PMID:19903920). More research into the role of LDL particle size and of particles like lipoprotein (a) is also warranted in a global context.

### B. Trans fatty acids

1. We noted the evidence review reported on pages 30-36, and the statement on page 32-33 that "it was determined that the available evidence did not support making a distinction between industrial and ruminant trans-fatty acids, and data solely from analyses of total trans-fatty acids were considered when formulating the recommendations on trans-fatty acid intake". The research needs section would benefit from acknowledging this limitation, particularly in light of epidemiological evidence of inverse associations between types of dairy products and cardiometabolic disease incidence and from using blood biomarker fatty acid levels. Specifically there is evidence from prospective cohort studies that blood trans palmitoleic acid is related inversely with some NCD outcomes [e.g. type 2 diabetes – Mozaffarian et al, PMID:23407305 (AJCN, 2013) and also PMID:21173413 (Annals Int Med, 2010)] and with null association with stroke [e.g. PMID:25411278 (AJCN, 2014)].

2. It is important to explore ways to differentiate between dietary intakes of industrially produced trans fatty acids and those from different 'natural' trans fatty acids from ruminant animal related meat or dairy products. The role of combining information from dietary self-report and fatty acid biomarkers should be assessed.

### C. Both SFA and trans-FA

1. The recommendation to conduct RCTs among those at high CVD risk who are on lipid-lowering medication is welcome, but (as above) the important and sometimes critical contribution of well conducted prospective studies should not be overlooked.

2. The recommendation to use improved methods of analysis, including development of robust biomarkers is welcome, but some further remarks to support/expand this would be helpful.

3. Additional research needs, not currently stated.

3a. Greater clarity and consistency (and standardisation) is needed on clinical endpoints that are studied and reported, as currently some research focuses on total CVD (with varying definitions and conditions included), others only on CHD or coronary artery disease or stroke individually, and some more specifically report myocardial infarction or heart failure. This makes comparability of the results between studies very challenging.

3b. Further research is needed on effects of substitutions of SFA with different types of PUFA (total n-3, plant-based n-3 (ALA-rich) or seafood-based n-3 (EPA, DHA rich) and n-6, and specifically LA), MUFA, carbohydrates (total, rich in whole-grain, or refined, high- or low-GI, fibre-rich or poor) and protein (animal-source or plant-source). This is needed both for disease events and for effects on lipids.

## Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments
<p>Other points (minor comments)</p> <p>1. In some critical places where the main evidence is summarised for both SFA and trans-FA (such as on page 21, top para; page 21 – the statement “The key research questions guiding the systematic reviews undertaken...”; or page 30, first para on trans-FA), the wording may give the impression that systematic reviews were specifically conducted for this evidence review by the WHO. For the WHO guidelines on free sugars (2015) it was clearly stated that two systematic reviews were commissioned, to assess the effects of increasing or decreasing intake of free sugars on excess weight gain and dental caries. For the current report, either it should be stated that evidence review highlighted published (existing) systematic reviews that assessed the effects of SFA intake and health outcomes, or clarify explicitly that they were specifically undertaken under the WHO umbrella.</p> <p>2. In several places (e.g. page 4 (objective section); page 15, 2nd para; page 16, background section; page 21, in the framing of the review key question), mention is made of providing recommendations on the intake of SFA and trans-FA to reduce the risk of NCDs in adults and children, particularly CVDs which are a leading cause of NCD mortality. Since no other NCD health conditions other than CVD are appraised for the link with SFA intake, and there is no evidence review of the role of the considered macronutrients for weight or obesity, type 2 diabetes (except in children), cancer or others, it is more appropriate to state up front (or be clearer) that the current report addresses the link between SFA and CVD incidence or mortality. (it is noted that for trans-FA where prospective evidence was appraised, type 2 diabetes was included as a stated endpoint, and that among children intermediate endpoints were considered).</p> <p>3. It is stated (e.g. on page 21) that the primary health outcomes (in adults) included “CHD (incidence, mortality and morbidity), CVD (incidence, mortality and morbidity), stroke (incidence, mortality and morbidity) and blood lipids”. It is unclear how “morbidity” was assessed in the evidence review. There is a footnote on page 23 that expands on the definition of the “CVD events”, namely non-fatal MI, angina, stroke, heart failure, peripheral vascular events or atrial fibrillation and unplanned interventions such as CABG or angioplasty. But, this does not explain why for instance for stroke, “morbidity” is included additional to incidence and mortality. Also, were sub-types of stroke, for instance, included in the review (i.e. atherosclerotic or haemorrhagic)? This should be clarified.</p> <p>4. On page 22 there is a helpful summary of key characteristics of the trials included in the systematic review by Hooper et al (ref #24). It should also include information on (a) participation in the trials by sex (men only, women only or both men and women), (b) how long ago the trials were conducted – the majority were old trials published in the 1960s to late 1990s, with only a couple published in 2004 or 2006, and (c) clarity to highlight that those considered at high risk were secondary prevention trials among people with existing CVD (usually MI or angina), while those with moderate or low risk were primary prevention trials.</p>

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)

Remarks (for trans-fatty acids only)
<p>1. We noted the evidence review reported on pages 30-36, and the statement on page 32-33 that “it was determined that the available evidence did not support making a distinction between industrial and ruminant trans-fatty acids, and data solely from analyses of total trans-fatty acids were considered when formulating the recommendations on trans-fatty acid intake”. The research needs section would benefit from acknowledging this limitation, particularly in light of epidemiological evidence of inverse associations between types of dairy products and cardiometabolic disease incidence and from using blood biomarker fatty acid levels. Specifically there is evidence from prospective cohort studies that blood trans palmitoleic acid is related inversely with some NCD outcomes [e.g. type 2 diabetes – Mozaffarian et al, PMID:23407305 (AJCN, 2013) and also PMID:21173413 (Annals Int Med, 2010)] and with null association with stroke [e.g. PMID:25411278 (AJCN, 2014)].</p> <p>2. It is important to explore ways to differentiate between dietary intakes of industrially produced trans fatty acids and those from different ‘natural’ trans fatty acids from ruminant animal related meat or dairy products. The role of combining information from dietary self-report and fatty acid biomarkers should be assessed.</p>
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
<p>The WHO have a tremendous opportunity to help clear the currently very confused debate and understanding about the harms or benefits of saturated fatty acids intake, particularly by providing clarity on the role of different foods that are rich in types of saturated fatty acids but differ in their health effects. There is considerable evidence that some SFA-rich foods have adverse health effects, some are neutral and some have benefits, and therefore the emphasis should be placed on foods and all SFA should not be considered as homogeneous.</p>

## Survey response 81

### General information

Family/last name
Wong
Given/first name
Samantha
Organization/affiliation
Food Industry Asia
Sector
Other
Sector [Other]
Industry association
Country
Singapore

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments
Food Industry Asia welcomes the recommendation that 10 per cent or less of total energy intake comes from saturated fats and 1 per cent or less from trans-fats (pages 8 and 11). However, the potential impact of the recommendations need to be considered; the consumption of saturated fatty acids and trans-fatty acids should be regarded as a part of a food matrix, not in isolation, to account for foods that contain essential nutrients or reduce the risk of chronic diseases. Foods such as milk, cheese and beef are rich in nutrients, but could be discriminated due to their naturally-occurring saturated fat and trans-fat contents.

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
In reference to the trans-fatty acids recommendations "In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake, WHO recommends reducing trans-fatty acid intake" and "In adults and children, WHO suggests reducing the intake of trans-fatty acids to less than 1% of total energy intake" on page 40 of the guidelines, FIA would support these recommendations and noted that the term "trans-fatty acids" includes all fatty acids with a double bond in the trans-configuration regardless of whether they come from ruminant sources or are industrially-produced (page 11). As mentioned in the guidelines, there is no evidence to show that ruminant trans-fats are less harmful to health than industrially-produced trans-fatty acids, although ruminant trans-fats are typically consumed at low levels (page 32 to 33).
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments
From the perspective of the food industry, we wish to highlight that many of our member companies have been working to reduce or eliminate trans-fat in their products globally. Initial findings from a reformulation study carried out by the Institute of Grocery Distribution (IGD) in Singapore show that removing trans-fat was one of the key priorities for food companies previously. At present, many companies have achieved their objective of reducing or eliminating trans-fats from their products and continue to work in this area.

## Final comments

Please provide any final thoughts or comments below.

Food Industry Asia (FIA) wishes to thank the World Health Organization (WHO) for the opportunity to comment on the “Draft Guidelines on Saturated Fatty Acid and Trans-Fatty Acid Intake for Adults and Children” and provide feedback on behalf of the food industry in Asia through the e-consultation process.

FIA is a non-profit industry association established in Asia to represent the view of the food industry as a trusted partner for multi-stakeholder dialogue.

The goal of FIA is to harness the expertise of major food and beverage companies and respond to the regions complex challenges in food safety, regulatory harmonisation and health & nutrition. Our members share common values on the responsible promotion of balanced diets and lifestyles.

Together, we work with a broad range of stakeholders in Asia to promote the role of public-private partnership (PPP) as a cost-effective mechanism for delivering positive socio-economic outcomes. At the heart of our philosophy lies a belief that the private sector can play a more positive role in civil society on many of the complex challenges associated with health & nutrition. To this end, FIA is committed to working collaboratively with governments, policy makers, civil societies and academia throughout Asia, either directly or through existing local industry groups.

Industry Commitments and Actions to Tackle Non-Communicable Diseases (NCDs): To facilitate better collaborative action to tackle NCDs, we highlight three examples where FIA works with the public sector and civil societies that adhere to the UN SDGs’ call to foster and develop inclusive partnerships:

#### 1. Responsible Marketing to Children

In line with 2010 WHO Recommendations on the Marketing of Foods and Non-alcoholic Beverages to Children, FIA recognises the role of responsible marketing and we support these recommendations with continued commitment to strengthen and adapt self-regulatory measures.

FIA is playing an important role in the implementation and monitoring of this commitment in Asia by driving adoption in the region and working with governments to codify the policy into national guidelines. This approach was designed within a framework in which robust industry-led standards can be easily incorporated in regional and national regulatory policies to create a system responsive to the unique needs of different countries. In Asia, FIA and our members are driving these self-regulatory commitments that are effective and measurable at the national level in Singapore, India, Malaysia, Thailand, and the Philippines, through multi-stakeholder partnerships.

#### 2. Front of Pack Guideline Daily Amounts (GDA) Nutrition Labelling

Front-of-pack GDA labelling is a valuable tool that was developed based on sound science, seeking to provide simplified nutrition information to facilitate consumer understanding and guide their purchase decisions. FIA has worked with its members to promote this voluntary scheme in Asia and “Fast Facts on Packs”, FIA’s GDA Nutrition Labelling Report published in 2016; revealed that significant progress has been made in driving the awareness and adoption of the GDA nutrition labelling scheme as an industry initiative to tackle obesity and NCDs. FIA has worked closely with governments in the region including India, Thailand and Taiwan to promote this science-based nutrition labelling scheme.

#### 3. ARoFIIN

The Asia Roundtable on Food Innovation for Improved Nutrition (ARoFIIN) was convened by the Health Promotion Board, an agency of the Ministry of Health in Singapore, A\*STAR (Singapore’s lead government research agency), Singapore Institute for Clinical Sciences (SICS) and FIA. ARoFIIN was set up to leverage multi-stakeholder partnerships by bringing together experts from across the government, academia, industry, and civil society to initiate and sustain a regional, multi-stakeholder dialogue on tackling the double burden of malnutrition through the role of food innovation and nutrition education.

In June 2017, ARoFIIN launched findings from a study carried out by the Economist Intelligence Unit – Tackling Obesity in ASEAN – Prevalence, Impact and Guidance on Interventions. Commissioned by ARoFIIN, the study responds to the challenges and threats posed by obesity in the region. The aim of the report is to guide policymakers, health organisations and industry to collectively tackle the rising threat of obesity in the region.

Following the study, ARoFIIN convened a series of follow-up workshops with academia, civil society, government bodies and industry, to recommend and develop scalable country-specific solutions and interventions which are now in the final proposal stage.



## Survey response 82

### General information

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de Groot	
Given/first name	
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Sector	
Academic/research	
Sector [Other]	
Country	
Netherlands	

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)	
Scope and purpose (same for saturated fatty acids and trans-fatty acids)	
Background (same for saturated fatty acids and trans-fatty acids)	
Guideline development process (same for saturated fatty acids and trans-fatty acids)	
Summary of evidence (for saturated fatty acids only)	
Recommendations (for saturated fatty acids only)	
Remarks (for saturated fatty acids only)	
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)	
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)	
Annexes 1, 6, 7 (for saturated fatty acids only)	

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Additional comments

## *Trans*-fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

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## Survey response 83

### General information

Family/last name
Ng
Given/first name
Tony Kock Wai
Organization/affiliation
University Tunku Abdul Rahman
Sector
Academic/research
Sector [Other]
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Malaysia

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
A very comprehensive review by WHO of the evidence for the effects of SFA and TFA intake on blood lipids and NCDs in adults and children.
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)

Summary of evidence (for saturated fatty acids only)
<p>While the evidence for lowering LDLC and TC by replacing SFA/TFA with PUFA is moderate to strong, any beneficial effect on all-cause mortality, CVD and stroke is at best, WEAK. The Report was conservative/cautious by stating many times that although LDLC is a “well-established surrogate endpoint of CVD, it is not a physical manifestation or confirmation of disease. Therefore, although the evidence for LDL cholesterol reduction was of high quality, a conservative approach was taken in the WHO Report.</p> <p>In connection to the above, the Report also stated that “No clear effect on any cardiovascular or mortality outcome was observed when reducing saturated fatty acid intake to less than 10% of total energy intake” which means that this recommendation, although stated by in the WHO Report as “conditional”, appears premature and can be misleading and certainly not fully justified.</p> <p>Throughout the WHO Report PUFA was recommended to replace either SFA or TFA but nowhere in the Report is an UPPER LIMIT recommended for PUFA intake. We note from the Sydney Diet Heart Study, (Ramsden et al., FASEB J, Vol. 27, No. 1 Suppl. April 2013), very high dietary levels of PUFA (15% en., esp. C18:2) in antioxidant-compromised persons (eg. IHD patients) can be very harmful or even kill!</p> <p>It may be pertinent to note that high blood cholesterol levels are not related to CVD mortality (Xu et al., 2018)- the Diet-Heart Disease myth which was debunked a long time ago!</p> <p>REFERENCES</p> <p>Ramsden et al. The Sydney Diet Heart Study: a randomised controlled trial of linoleic acid for secondary prevention of coronary heart disease and death. FASEB J, Vol. 27, No. 1 Suppl. April 2013),</p> <p>Xu et al. Egg consumption and the risk of cardiovascular disease and all-cause mortality: Guangzhou Biobank Cohort Study and meta-analyses. Eur J Nutr. 2018 Apr 21. doi: 10.1007/s00394-018-1692-3.</p>
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)

Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

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## Survey response 84

### General information

Family/last name
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Organization/affiliation
International Dairy Federation
Sector
Private sector
Sector [Other]
Country
Belgium

### Saturated fatty acids

## Executive summary (same for saturated fatty acids and trans-fatty acids)

The objective of this work is to ensure that WHO guidelines on these critical nutrients and associated dietary practices are comprehensive and therefore should be informed by the most recent scientific data, a 'whole food' and 'dietary approach', rather than an 'isolated nutrients' approach should be taken.

Because:

1. Foods and diets are clearly far more than the sum of their single nutrients. Single nutrients are not consumed in isolation – many factors within a food influence the effects of a single nutrient (for saturated fat this could be the presence of other nutrients such as calcium and protein and carbohydrates, the structures/matrix of the food, the position of individual fatty acids on the glycerol molecule, the presence of other factors influencing the absorption of the fat, the precise type and chain length of fatty acids, the ratio of SFA and other types of fatty acids, etc.) and it is inaccurate to generalise about the effects of a single nutrient without considering the food it is present in.

2. Foods are not a matrix that we can adjust at will. Their composition is sometimes mainly defined by the raw material meaning by nature itself. Milk is just one example of that. Thus, in recommending decreasing or increasing the consumption of a single nutrient it will result in a modification of the diet itself. However, this major issue is not addressed if a nutrient approach is taken. The WHO evidence review should focus on foods and dietary patterns (rather than isolated nutrients). Overall, no single food or nutrient creates a healthy dietary pattern, but instead, it is the combination of nutrient-dense foods that is emphasized: "...dietary components of an eating pattern can have interactive, synergistic, and potentially cumulative relationships, such that the eating pattern may be more predictive of overall health status and disease risk than individual foods or nutrients..." (2015-2020 Dietary Guidelines for Americans).

Secondly the review should have focused on actual disease risk, rather than considering various markers of risk in isolation. Although the report states that some food sources of SFA may be different such as dairy foods (page 17), we disagree with the fact that this is "emerging evidence". We do note that WHO only reviewed evidence coming from RCTs, but systematic reviews and meta-analyses from observational studies are strong evidence as well that should have been taken into consideration to provide more complete recommendations. There is a very large and growing body of evidence, including several systematic reviews and meta-analyses, related to the role of dairy fat and higher fat dairy foods on cardiovascular-related outcomes which should be considered. (Drouin-Chartier et al 2016). Not acknowledging this substantial research could prevent the scientific process and moving to its rightful position as consensus science, and therefore we would request consideration of this apparent in the resulting recommendations

The phrase "saturated fat" is a useful description of the chemical structure of a fatty acid, but it does not help illustrate the variety of fatty acids. As stated by Professor Mozaffarian :

"Saturated fat represents a highly heterogeneous category of fatty acids, with chain lengths ranging from 6 to 24 carbons, deriving from diverse foods, and possessing dissimilar biology. For instance, palmitic acid (16:0) exhibits in vitro adverse effects; whereas medium-chain (6:0–12:0), odd-chain (15:0, 17:0), and very long-chain (20:0–24:0) saturated fats may have metabolic benefits. This biological and metabolic diversity does not support the grouping together of all saturated fatty acids based on only one chemistry characteristic: the absence of double bonds."

"Judging a food or a person's diet as harmful because it contains more saturated fat, or as beneficial because it contains less, is unsound. This is consistent with the many longitudinal cohort studies demonstrating largely neutral effects of overall saturated fat intake (Figure 7). 39,157,344 Consistent with this, meats higher in processing and sodium, rather than saturated fat, are most strongly linked to CHD (see Meats, above). Cheese, a leading source of saturated fat, is also linked to neutral or even beneficial effects on CHD and diabetes mellitus (see Milk, Cheese, Yogurt, above). In sum, these lines of evidence – complex lipid effects including little influence on ApoB, no relation of overall intake with CHD, and no observed cardiovascular harm for most major food sources – provide powerful and consistent evidence for absence of appreciable harms of total saturated fat."

With regards to dairy, whole milk contains about 3-4% dairy fat by weight, and dairy fat is made up of more than 400 distinct types of fatty acids, making it the most complex fat naturally occurring in a food (Mansson HL, 2008 ). About two-thirds of the fatty acids in milk are saturated, and the remaining one third that are unsaturated include monounsaturated and polyunsaturated fatty acids (USDA, 2015), though the types and proportions of fatty acids in milk can vary (Mansson HL, 2008; Benbrook CM, 2013)

There is much less evidence relating to children specifically and care should be taken in the extrapolation of evidence for adults to children. We do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues found different treatment benefits estimated by clinical trials in adult compared with those performed in children for 11% of drugs (14 out of 124) (Janiaud, 2015). A commentary on the paper pointed out that 'kids are no little adults and not all kids are the same.' They added 'although in older children treatment effects may be similar to adults, this may not be the case in younger children (Oostenbrink, 2016). Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.'

Our concern that with extrapolation of adult data to children particularly from the age of 2 years it may result in a reduced intake of nutritionally sound sources of energy such as dairy products and meats in children due to requirements for energy and other nutrients packaged in these whole foods.

Recommendation 1 suggest that any person over the age of two years with a saturated fat intake higher than 10.0% of energy should reduce their saturated fat intake and that it does not matter which foods are targeted. We are concerned about possible unintended consequences associated with this advice, such as intakes of under-consumed nutrients being further compromised. We would also question the appropriateness of this recommendation for frail elderly people.

We would also question the scientific basis for this recommendation as the majority of studies that form the evidence for this were

undertaken in adults who already had cardiovascular disease or diabetes. Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, 70% of the data behind the strong recommendation for all adults and children 'to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake' comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children.

We also note that the format of the report is hard to follow, with the summary of the evidence, recommendations, research gaps, grade evidence profiles etc for each of the research questions being quite spread out.

Finally we recommend that it should be made clear to member states, policy makers, media etc how conditional recommendations should be interpreted ie that they are based on non-conclusive evidence and therefore it is uncertain if the recommendation would provide a benefit; and a substantial debate is required before considering as part of policy making.

## References

Benbrook CM et al., (2013) Organic production enhances milk nutritional quality by shifting fatty acid composition: A United States-wide, 18-month study. PLOS One 8:1-13.

Drouin-Chartier et al., (2016) Comprehensive Review of the Impact of Dairy Foods and Dairy Fat on Cardiometabolic Risk. Adv Nutr 7:1041–51

Janiaud P et al., (2015) Different treatment benefits were estimated by clinical trials performed in adults compared with those performed in children. Journal of Clinical Epidemiology 68, 1221-31.

Mansson HL., (2008) Fatty acids in bovine milk fat. Food Nutr Res 2008. Epub 2008 Jun 11.

Mozaffarian D., (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. Circulation 133, 187-225

Oostenbrink R & de Wildt SN, (2016) Drug trials: Kids are no little adults and not all kids are the same. Journal of Clinical Epidemiology 71, 111-12.

U.S. Department of Health and Human Services and U.S. Department of Agriculture. (2015) 2015-2020 Dietary Guidelines for Americans. 8th Edition, December 2015. Available at <http://health.gov/dietaryguidelines/2015/guidelines/>.

US Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. (2015) USDA National Nutrient Database for Standard Reference, Release 28. Version Current: September 2015. Internet: <http://ndb.nal.usda.gov>.

Velasco I et al., (2018) Iodine as essential nutrient during the first 1000 days of life. Nutrients 10, 290

Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. Osteoporosis Int 27,1281-1386

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)



#### Summary of evidence (for saturated fatty acids only)

We note that WHO own commissioned Systematic Reviews (ref#61\_ de Souza et al 2015) has not been included in the analysis .

o[?] de Souza RJ, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ* 2015;351:h3978.

The authors concluded that "This systematic review and meta-analysis of evidence from large generally well designed observational studies does not support a robust association of saturated fats with all-cause mortality, CHD, CHD mortality, ischemic stroke, or diabetes in healthy individuals."

Moreover, a specific fatty acid found in dairy products (ie-trans-palmitoleic acid) was associated with reduced risk of type 2 diabetes. This finding is said to be "quite consistent and compatible with a 26-54% reduction in risk across an estimated threefold intake range.

The food source, as indicated previously, should have been addressed as there is a large body of evidence that shows that dairy fat and higher fat dairy foods such as cheese do not increase CVD-related outcomes and in fact show protective associations for some outcomes. This is well highlighted in the Systematic Review of Drouin-Chartier et al 2016 as described below:

o[?]Drouin-Chartier, JP et al. Systematic Review of the Association between Dairy Product Consumption and Risk of Cardiovascular-Related Clinical Outcomes. *Adv Nutr* 2016;7:1026-40.

A comprehensive, systematic "umbrella review" of meta-analyses of prospective cohort studies of the association of dairy consumption with cardiovascular-related clinical outcomes: cardiovascular disease (CVD), coronary artery disease (CAD), stroke, hypertension, metabolic syndrome (MetS), and type 2 diabetes (T2D). This review included a rating of quality as well as a grading of the evidence using GRADE.

Data from this comprehensive study which includes 21 individual meta-analyses indicates that the consumption of various forms of dairy products (including higher fat dairy products) shows either favorable or neutral associations with cardiovascular-related clinical outcomes.

Data with respect to cheese specifically indicated that cheese is not associated with an increased risk of CVD and is associated with a reduced risk of stroke and type 2 diabetes.

2 recently published meta-analyses of RCTs that cast doubt on the benefits of replacing Sat Fat with PUFA were not considered. These are described below:

o[?]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;16:30.

The aim of this study was to systematically review and conduct a meta-analysis of all the randomized controlled trials that looked at replacing saturated fat (SFA) by mostly n-6 polyunsaturated fat (PUFA) and accounting for the major confounding variables (which earlier meta-analyses did not sufficiently do) and emphasize the results from those trials that most accurately test the effect of replacing SFA with mostly n-6 PUFA.

The author concludes: "Available evidence from adequately controlled randomised controlled trials suggest replacing SFA with mostly n-6 PUFA is unlikely to reduce CHD events, CHD mortality or total mortality. The suggestion of benefits reported in earlier meta-analyses is due to the inclusion of inadequately controlled trials. These findings have implications for current dietary recommendations."

o[?]Hannon BA et al. Clinical outcomes of dietary replacement of saturated fatty acids with unsaturated fat sources in adults with overweight and obesity: a systematic review and meta-analysis of randomized control trials. *Ann Nutr Metab* 2017;71:107-117.

The purpose of this systematic review and meta-analysis of randomized controlled trials was to assess the evidence of the effect of saturated fatty acids (SFA) replacement with unsaturated fatty acids (UFA) in metabolically healthy adults with overweight and obesity on markers of dyslipidemia and body composition.

There is no strong evidence that replacement of SFA with UFA may benefit lipid profiles in this population.

Finally another study that should be included is:

o[?] Chowdhury et al. 2014 Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis.

The purpose of this study was to summarize evidence about associations between fatty acids and coronary disease.

The author concludes: "Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats."

Equal weight for hard clinical outcomes (eg. CVD, CHD, stroke, etc.) was given to blood lipid markers (esp. LDL-C) and this is not appropriate. Blood lipid markers are (inconsistent or unsubstantiated) risk factors and only LDL-C is currently considered as a “surrogate marker” for CVD risk and isolation of other factors meaningless. And even LDL-C is being questioned as a reliable biomarker for assessing the impact of food on CVD as outlined in the 2010 report by the Institute of Medicine. For example, 16:0 raises LDL cholesterol, but also raises HDL-cholesterol, reduces triglyceride-rich lipoproteins and remnants and has little effect on Apo-B. Mozaffarian (2016) commented ‘Continued prioritization of saturated fat reduction appears to rely on selected evidence: e.g. effects on LDL-cholesterol alone (discounting the other, complex lipid and lipoprotein effects); historical ecological trends in certain countries (e.g. Finland) but not in others; and expedient comparisons with polyunsaturated fat, the most healthful macronutrient.’

We do note a discrepancy between the statement on page 5 with respect to the evidence for replacement of Sat Fat by PUFA and what is being recorded on page 10. On page 5, the evidence is described for replacement of Sat Fat by PUFA as “low quality evidence” while on page 10 it is described as “moderate to high quality evidence.”

Although it is stated that ‘the recommendation to not increase saturated fatty acid intake if intake is already below 10% of total energy intake (fourth recommendation) is based on the totality of evidence reviewed, a major omission is consideration of the impact of such a recommendation on the frail elderly. It is well acknowledged that the frail elderly do not require a low fat diet. Focus on the saturated fat intake of such people, as a result of this proposed WHO recommendation, could well reduce their access to nutrient-dense, protein providing foods such as cheese, whole milk and yoghurt.

Also, children between the ages of 2 and 5 years often consume higher amounts of dairy foods than older children and recommendation four has the potential to adversely impact on the provision of these foods in pre-school settings.

Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out, they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed WHO saturated fat guidelines will adversely impact on intake of milk and dairy foods around the world.

## References

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Mozaffarian D., (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 133, 187-225

## Recommendations (for saturated fatty acids only)

We question the scientific rationale(s) justifying that SFA intake should be lower than the maximum target of 10% of the energy within the context of a nutritionally adequate diet based on food based dietary guidelines. This question also applies to gender, age and health status. With regard to current world wide SFA intake levels it would be relevant to have at least intake levels ranging from 4 to 20 Energy%. If a relation between intake and a specific health outcome is present in this intake range, then the type of relation should be considered as well (e.g. linear, non-linear, etc.). Baseline nutritional status should be viewed in relation to malnutrition and imprinting in early life; factors to consider are adequate energy intake with a good balance between fat and (complex) carbohydrates, high quality protein, vitamin and mineral adequacy, nutrient dense food patterns. With regard to SFA replacement, careful consideration should be given to the ratio between protein, carbohydrates and fat, and the type of protein, carbohydrate and fat.

The introduction of the draft guideline states that “ the debate has continued as to whether the available evidence for adverse health effects related to consumption of saturated fatty acids warrants appreciable reduction in intake. “

Remarks (for saturated fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
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We note that the text states that these guidelines (on saturated and trans fatty acids) should be used in conjunction with other guidance on dietary goals and nutrition guidelines. We would encourage the WHO to make future recommendations in relation to dietary patterns rather than single nutrients. This is because, as explained by Mozaffarian (2016), 'Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.'

Therefore focusing on total saturated fat and total trans fat content of a food is likely to lead to a reduction in intake of milk, yogurt and cheese, which is likely to negatively impact on the nutritional adequacy and healthfulness of the overall diet. For example, reduced intake of dairy foods (as a result of these foods being labelled unhealthy due to their saturated fat content) would be likely to negatively impact on intakes of key nutrients such as calcium (which is important for bone accrual in children (Weaver CM, 2016)) and iodine (which is important for preventing cognitive impairment in children (Velasco, 2018; Bath, 2017)). Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out (FAO, 2013), they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed translation of the WHO saturated fat and trans fat guidelines will adversely impact on intake of milk and dairy foods around the world. Any translation and policy measure development of the finalised guidelines needs to ensure that this does not occur.

There is a high risk that the "conditional recommendations" which include reducing Sat Fat to < 10% of energy and replacing Sat Fat with PUFA, will be seen as similar as the "strong recommendations" and interpreted accordingly by policy makers, which could impact legislation without having the opportunity for critical discussions from expert stakeholders. This distinction and what it means should be made more clear as it is currently only in the very fine print of the footnotes (see page 8 footnotes) as follows: "Conditional recommendations are those recommendations for which the WHO guideline development group is uncertain that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders."

#### References

Bath SC et al., (2017) Iodine concentration of milk-alternative drinks available in the UK. Br J Nutr 118, 525-32

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Velasco I et al., (2018) Iodine as essential nutrient during the first 1000 days of life. Nutrients 10, 290

Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. Osteoporosis Int 27,1281-1386

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
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Several research needs that are identified involve RCTs with clinical outcomes. This type of design is not very feasible and highly unrealistic.

Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments
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IDF is questioning the recommendations made in this report. Nutrition research has shifted focus to examine the relationship of whole foods with health. This is based on the premise that we do not eat nutrients in isolation but as foods, and meals, and part of dietary patterns. From this research, a different picture has emerged in some cases than might be predicted from the nutrient content of the foods investigated.

Furthermore, the potential impact of these recommendations needs to be considered beyond just consumption of SFAs and TFAs, which are consumed as part of a food matrix, not in isolation. For example continuing to promote the consumption of dairy products such as milk, cheese and yoghurt which provide an important package of essential nutrients and components which provide a range of health benefits, including supporting cardiovascular health and helping to reduce the risk of associated risk factors such as type 2 diabetes despite their inherent SFA and TFA content (Thorning et al 2017) .

Given the amount of evidence that does exist exploring the association of dairy consumption and risk of cardiovascular disease and associated risk factors, this warrants further exploration and discussion in the guidelines.

#### Reference

Thorning TK et al. (2017) Whole dairy matrix or single nutrients in assessment of health effects: current evidence and knowledge gaps. Am J Clin Nutr.105(5):1033-1045

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## *Trans*-fatty acids

## Executive summary (same for saturated fatty acids and trans-fatty acids)

The objective of this work is to ensure that WHO guidelines on these critical nutrients and associated dietary practices are comprehensive and therefore should be informed by the most recent scientific data, a 'whole food' and 'dietary approach', rather than an 'isolated nutrients' approach should be taken.

Because:

1. Foods and diets are clearly far more than the sum of their single nutrients. Single nutrients are not consumed in isolation – many factors within a food influence the effects of a single nutrient (for saturated fat this could be the presence of other nutrients such as calcium and protein and carbohydrates, the structures/matrix of the food, the position of individual fatty acids on the glycerol molecule, the presence of other factors influencing the absorption of the fat, the precise type and chain length of fatty acids, the ratio of SFA and other types of fatty acids, etc.) and it is inaccurate to generalise about the effects of a single nutrient without considering the food it is present in.

2. Foods are not a matrix that we can adjust at will. Their composition is sometimes mainly defined by the raw material meaning by nature itself. Milk is just one example of that. Thus, in recommending decreasing or increasing the consumption of a single nutrient it will result in a modification of the diet itself. However, this major issue is not addressed if a nutrient approach is taken. The WHO evidence review should focus on foods and dietary patterns (rather than isolated nutrients). Overall, no single food or nutrient creates a healthy dietary pattern, but instead, it is the combination of nutrient-dense foods that is emphasized: "...dietary components of an eating pattern can have interactive, synergistic, and potentially cumulative relationships, such that the eating pattern may be more predictive of overall health status and disease risk than individual foods or nutrients..." (2015-2020 Dietary Guidelines for Americans).

Secondly the review should have focused on actual disease risk, rather than considering various markers of risk in isolation. Although the report states that some food sources of SFA may be different such as dairy foods (page 17), we disagree with the fact that this is "emerging evidence". We do note that WHO only reviewed evidence coming from RCTs, but systematic reviews and meta-analyses from observational studies are strong evidence as well that should have been taken into consideration to provide more complete recommendations. There is a very large and growing body of evidence, including several systematic reviews and meta-analyses, related to the role of dairy fat and higher fat dairy foods on cardiovascular-related outcomes which should be considered. (Drouin-Chartier et al 2016). Not acknowledging this substantial research could prevent the scientific process and moving to its rightful position as consensus science, and therefore we would request consideration of this apparent in the resulting recommendations

The phrase "saturated fat" is a useful description of the chemical structure of a fatty acid, but it does not help illustrate the variety of fatty acids. As stated by Professor Mozaffarian :

"Saturated fat represents a highly heterogeneous category of fatty acids, with chain lengths ranging from 6 to 24 carbons, deriving from diverse foods, and possessing dissimilar biology. For instance, palmitic acid (16:0) exhibits in vitro adverse effects; whereas medium-chain (6:0–12:0), odd-chain (15:0, 17:0), and very long-chain (20:0–24:0) saturated fats may have metabolic benefits. This biological and metabolic diversity does not support the grouping together of all saturated fatty acids based on only one chemistry characteristic: the absence of double bonds."

"Judging a food or a person's diet as harmful because it contains more saturated fat, or as beneficial because it contains less, is unsound. This is consistent with the many longitudinal cohort studies demonstrating largely neutral effects of overall saturated fat intake (Figure 7). 39,157,344 Consistent with this, meats higher in processing and sodium, rather than saturated fat, are most strongly linked to CHD (see Meats, above). Cheese, a leading source of saturated fat, is also linked to neutral or even beneficial effects on CHD and diabetes mellitus (see Milk, Cheese, Yogurt, above). In sum, these lines of evidence – complex lipid effects including little influence on ApoB, no relation of overall intake with CHD, and no observed cardiovascular harm for most major food sources – provide powerful and consistent evidence for absence of appreciable harms of total saturated fat."

With regards to dairy, whole milk contains about 3-4% dairy fat by weight, and dairy fat is made up of more than 400 distinct types of fatty acids, making it the most complex fat naturally occurring in a food (Mansson HL, 2008 ). About two-thirds of the fatty acids in milk are saturated, and the remaining one third that are unsaturated include monounsaturated and polyunsaturated fatty acids (USDA, 2015), though the types and proportions of fatty acids in milk can vary (Mansson HL, 2008; Benbrook CM, 2013)

There is much less evidence relating to children specifically and care should be taken in the extrapolation of evidence for adults to children. We do not believe that extrapolating evidence from adults to children is appropriate in this instance. Indeed, regulations in both the US and the EU require new pharmaceutical drugs to be studied in children (not just adults).

A 2015 study by Janiaud and colleagues found different treatment benefits estimated by clinical trials in adult compared with those performed in children for 11% of drugs (14 out of 124) (Janiaud, 2015). A commentary on the paper pointed out that 'kids are no little adults and not all kids are the same.' They added 'although in older children treatment effects may be similar to adults, this may not be the case in younger children (Oostenbrink, 2016). Hence, to advocate extrapolation of adult data to children based on the results of this study is not warranted in the youngest age groups.'

Our concern that with extrapolation of adult data to children particularly from the age of 2 years it may result in a reduced intake of nutritionally sound sources of energy such as dairy products and meats in children due to requirements for energy and other nutrients packaged in these whole foods.

Recommendation 1 suggest that any person over the age of two years with a saturated fat intake higher than 10.0% of energy should reduce their saturated fat intake and that it does not matter which foods are targeted. We are concerned about possible unintended consequences associated with this advice, such as intakes of under-consumed nutrients being further compromised. We would also question the appropriateness of this recommendation for frail elderly people.

We would also question the scientific basis for this recommendation as the majority of studies that form the evidence for this were

undertaken in adults who already had cardiovascular disease or diabetes. Out of the 13 comparisons of SFA reduction and CV events, just three (Black 1994, Veterans Admin and WHI without CVD) were from studies involving healthy adults. Indeed, 70% of the data behind the strong recommendation for all adults and children 'to reduce their saturated fatty acid intake if it is greater than 10% of total energy intake' comes from individuals who already had CVD or diabetes. Just 30% of the data relates to healthy adults and none of the data relates to children.

We also note that the format of the report is hard to follow, with the summary of the evidence, recommendations, research gaps, grade evidence profiles etc for each of the research questions being quite spread out.

Finally we recommend that it should be made clear to member states, policy makers, media etc how conditional recommendations should be interpreted ie that they are based on non-conclusive evidence and therefore it is uncertain if the recommendation would provide a benefit; and a substantial debate is required before considering as part of policy making.

## References

Benbrook CM et al., (2013) Organic production enhances milk nutritional quality by shifting fatty acid composition: A United States-wide, 18-month study. PLOS One 8:1-13.

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Weaver CM et al., (2016) The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. Osteoporosis Int 27,1281-1386

Scope and purpose (same for saturated fatty acids and trans-fatty acids)

Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)

Summary of evidence (for trans-fatty acids only)
<p>Eliminating all TFA from diet without considering the source of TFA potentially leads to poorer diet, as dairy is a rich source of essential nutrients (several minerals, vitamins and protein). The WHO Scientific update on Trans fatty acids concluded that "Although ruminant TFAs cannot be removed entirely from the diet, their intake is low in most populations and to date there is no conclusive evidence supporting an association with CHD risks in the amounts usually consumed." (R Uauy, 2009).</p> <p>We do have strong concerns regarding the interpretation that ruminant TFA (rTFA) are defined in a similar category as industrial TFA (iTFA). The report cites evidence of no effect of ruminant trans fatty acid intake on a host of cardiometabolic outcomes. It excludes the findings from a 2015 Systematic Reviews (de Souza et al 2015), which indicated the following:</p> <ul style="list-style-type: none"> <li>o Industrial, but not ruminant, trans fats were associated with CHD mortality and CHD.</li> <li>o Ruminant trans-palmitoleic acid, considered as a biomarker of dairy intake, was inversely associated with type 2 diabetes. This finding is said to be "quite consistent and compatible with a 26-54% reduction in risk across an estimated threefold intake range."</li> </ul> <p>The rationale provided for including rTFA is based on speculation that levels may increase if iTFA is reduced. It is very unlikely that rTFA levels would increase to a level similar to iTFA. In fact, as pointed out in one of the used references (ref # 34), "if the only source of TFA in the diet is ruminant products, then TFA consumption would drop to less than 2g/day or less than 1% of energy." But the implementation measures including regulation/restriction of marketing, sales and promotion of, and fiscal policies targeting food and beverages 'high' in trans-fatty acids may well unfairly and detrimentally target milk and dairy products, unless clear distinctions are made in recommendations about industrial produced trans fatty acids, as done for the WHO global plan to eliminate all industrially produced TFA.</p> <p>Therefore we would suggest that the recommendation be amended to:</p> <p>In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing industrial trans-fatty acid intake (strong recommendation<sup>2</sup>).</p> <p>Similarly, we recommend that industrial trans fatty acids rather than all trans fatty acids are specified in the three conditional recommendations.</p> <p>Reference</p> <p>De Souza RJ et al., (2015) Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. <i>BMJ</i> 351:h3978.</p> <p>Uauy R et al., (2009). WHO Scientific update on trans fatty acids: summary and conclusions. <i>EJCN</i> 63, S68-75</p>
Recommendations (for trans-fatty acids only)
<p>we would suggest that the recommendation be amended to:</p> <p>In adults and children whose trans-fatty acid intake is greater than 1% of total energy intake<sup>1</sup>, WHO recommends reducing industrial trans-fatty acid intake (strong recommendation<sup>2</sup>).</p> <p>Similarly, we recommend that industrial trans fatty acids rather than all trans fatty acids are specified in the three conditional recommendations.</p>
Remarks (for trans-fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
<p>We note that the text states that these guidelines (on saturated and trans fatty acids) should be used in conjunction with other guidance on dietary goals and nutrition guidelines. We would encourage the WHO to make future recommendations in relation to dietary patterns rather than single nutrients. This is because, as explained by Mozaffarian (2016), ‘Methodological advances in nutrition science now demonstrate that nutrient-focused metrics are inadequate to explain most effects of diet on chronic diseases. Rather cardio-metabolic diseases are largely influenced not by single nutrients, but by specific foods and overall diet patterns.’</p> <p>Therefore focusing on total saturated fat and total trans fat content of a food is likely to lead to a reduction in intake of milk, yogurt and cheese, which is likely to negatively impact on the nutritional adequacy and healthfulness of the overall diet. For example, reduced intake of dairy foods (as a result of these foods being labelled unhealthy due to their saturated fat content) would be likely to negatively impact on intakes of key nutrients such as calcium (which is important for bone accrual in children (Weaver CM, 2016)) and iodine (which is important for preventing cognitive impairment in children (Velasco, 2018; Bath, 2017)). Milk and dairy products are a concentrated source of macro- and micronutrients. As FAO points out (FAO, 2013), they can play a particularly important role in human nutrition in developing countries where the diets of poor people frequently lack diversity and consumption of animal-source foods may be limited. We are concerned that the proposed translation of the WHO saturated fat and trans fat guidelines will adversely impact on intake of milk and dairy foods around the world. Any translation and policy measure development of the finalized guidelines needs to ensure that this does not occur.</p> <p>There is a high risk that the “conditional recommendations” which include reducing Sat Fat to &lt; 10% of energy and replacing Sat Fat with PUFA, will be seen as similar as the “strong recommendations” and interpreted accordingly by policy makers, which could impact legislation without having the opportunity for critical discussions from expert stakeholders. This distinction and what it means should be made more clear as it is currently only in the very fine print of the footnotes (see page 8 footnotes) as follows: “Conditional recommendations are those recommendations for which the WHO guideline development group is uncertain that the desirable consequences of implementing the recommendation outweigh the undesirable consequences. Policy-making related to conditional recommendations therefore may require substantial debate and involvement of various stakeholders.”</p> <p>References</p> <p>Bath SC et al., (2017) Iodine concentration of milk-alternative drinks available in the UK. <i>Br J Nutr</i> 118, 525-32</p> <p>Food and Agriculture Organization of the United Nations (2013) Milk and dairy products in human nutrition. Rome: FAO.</p> <p>Mozaffarian D., (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. <i>Circulation</i> 133, 187-225</p> <p>Velasco I et al., (2018) Iodine as essential nutrient during the first 1000 days of life. <i>Nutrients</i> 10, 290</p> <p>Weaver CM et al., (2016) The National Osteoporosis Foundation’s position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. <i>Osteoporosis Int</i> 27,1281-1386</p>
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Several research needs that are identified involve RCTs with clinical outcomes. This type of design is not very feasible and highly unrealistic.
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments
<p>IDF is questioning the recommendations made in this report. Nutrition research has shifted focus to examine the relationship of whole foods with health. This is based on the premise that we do not eat nutrients in isolation but as foods, and meals, and part of dietary patterns. From this research, a different picture has emerged in some cases than might be predicted from the nutrient content of the foods investigated.</p> <p>Furthermore, the potential impact of these recommendations needs to be considered beyond just consumption of SFAs and TFAs, which are consumed as part of a food matrix, not in isolation. For example continuing to promote the consumption of dairy products such as milk, cheese and yoghurt which provide an important package of essential nutrients and components which provide a range of health benefits, including supporting cardiovascular health and helping to reduce the risk of associated risk factors such as type 2 diabetes despite their inherent SFA and TFA content (Thorning et al 2017) .</p> <p>Given the amount of evidence that does exist exploring the association of dairy consumption and risk of cardiovascular disease and associated risk factors, this warrants further exploration and discussion in the guidelines.</p> <p>Reference</p> <p>Thorning TK et al. (2017) Whole dairy matrix or single nutrients in assessment of health effects: current evidence and knowledge gaps. <i>Am J Clin Nutr.</i>105(5):1033-1045</p>



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## Final comments

Please provide any final thoughts or comments below.

International Dairy Federation is questioning the recommendations made in this report. Nutrition research has shifted focus to examine the relationship of whole foods with health. This is based on the premise that we do not eat nutrients in isolation but as foods, and meals, and part of dietary patterns. From this research, a different picture has emerged in some cases than might be predicted from the nutrient content of the foods investigated.

Furthermore, the potential impact of these recommendations needs to be considered beyond just consumption of SFAs and TFAs, which are consumed as part of a food matrix, not in isolation. For example continuing to promote the consumption of dairy products such as milk, cheese and yoghurt which provide an important package of essential nutrients and components which provide a range of health benefits, including supporting cardiovascular health and helping to reduce the risk of associated risk factors such as type 2 diabetes despite their inherent SFA and TFA content (Thorning et al 2017) .

Given the amount of evidence that does exist exploring the association of dairy consumption and risk of cardiovascular disease and associated risk factors, this warrants further exploration and discussion in the guidelines.

Thorning TK et al. (2017) Whole dairy matrix or single nutrients in assessment of health effects: current evidence and knowledge gaps. Am J Clin Nutr.105(5):1033-1045

## Survey response 85

### General information

Family/last name
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Sector [Other]
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Austria

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
The executive summary gives a very clear overview. Nothing to add
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Very good description of scope and purpose.  It is not clear, if "policy Actions" means legislation which is an important topic for trans-fatty acids.
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
The development process is transparent and comprehensible.
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.

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## Survey response 86

### General information

Family/last name
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Private sector
Sector [Other]
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### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)

## Summary of evidence (for saturated fatty acids only)

June 1, 2018

Comment to the WHO Department of Nutrition for Health and Development (NHD), Nutrition Guidance Expert Advisory Group (NUGAG) Subgroup on Diet and Health

RE: [Public consultation on the WHO Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children](#)

To Whom It May Concern:

National Dairy Council® (NDC) appreciates the opportunity to submit comments regarding WHO's Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children. NDC, the non-profit organization founded by the U.S. dairy farmers, is committed to nutrition research and education about dairy's role in the diet and health and wellness. NDC provides science-based dairy nutrition information to, and in collaboration with, a variety of stakeholders committed to fostering a healthier nation, including health professionals, educators, school nutrition directors, academia and industry. Established in 1915, NDC comprises a staff of registered dietitians and nutrition research and communications experts across the country.

Dietary patterns may be more predictive of health than individual nutrients

The randomized controlled trial evidence WHO reviewed when drafting guidance on saturated fat provides important insights about the health effects of saturated fat consumption (1, 2). Nutrition science, however, is evolving to include the whole eating pattern when considering the impacts of diet on chronic disease outcomes. Eating patterns capture the synergistic and cumulative effects that combinations of foods and beverages – and the nutrients they contain – can have on health (3). Because eating patterns contain multiple foods and beverages that work together in relation to health, they may be more predictive of health than any one food or nutrient (4).

The Canadian Heart and Stroke Foundation, in their statement on saturated fat, heart disease and stroke, concur: "The science of nutrition is ever-evolving with new evidence emerging all the time. It is becoming increasingly clear that what has the most impact on health is the overall quality of one's diet, combined with the types and quantity of food consumed." Their saturated fat recommendations "do not include a threshold or limit for saturated fat and instead focus on a healthy balanced dietary pattern, which can help Canadians reduce consumption of saturated fats (5)."

New observational evidence doesn't find link between saturated fat consumption and CVD risk

Recommendations to limit saturated fat consumption are based on evidence that a high consumption of saturated fat increases plasma LDL-cholesterol (LDL-C) levels, which has been linked to increased risk for heart disease (6). Recent prospective cohort studies, however, have found that populations consuming higher amounts of saturated fat do not have higher risk for cardiovascular disease (CVD) than those who consume lower amounts (7-9). One meta-analysis and systematic review by de Souza et al, commissioned by the WHO and published in 2015, found "no clear association between higher intake of saturated fats and all-cause mortality, coronary heart disease (CHD), CHD mortality, ischemic stroke, or type 2 diabetes among apparently healthy adults" (10).

Current authoritative recommendations to reduce saturated fat can be accompanied by guidance to replace saturated fat with unsaturated fat (4, 11, 12). While several large prospective studies and a meta-analysis have found that modeling replacement of saturated fat with polyunsaturated fat is linked to reduced risk for CVD, replacing saturated fat with carbohydrates has shown little or no benefit (6, 13-15). Recommendations to reduce saturated fat and replace with other macronutrients, however, may not be straight-forward. A cross sectional, multi-modeling analysis of the association of nutrients with risk factors for CVD from 18 countries in North America, South America, Europe, Africa, and Asia found that replacing saturated fatty acids with unsaturated fatty acids improved some risk factors, such as LDL-C, but worsened others, such as HDL-cholesterol (HDL-C) and triglycerides. Substituting saturated fatty acids with carbohydrate had an adverse effect on blood lipids (16). This study concluded that current recommendations to reduce total and saturated fats are not supported and cautioned that focusing on a single lipid marker for CVD, such as LDL-C, may not capture the impact of various dietary nutrients on CVD (16).

Dairy foods consumption is associated with neutral or beneficial outcomes

Another growing body of evidence indicates that consumption of dairy foods has neutral or inverse associations with risk for CVD. Between 2009 and 2017, at least eight systematic reviews and/or meta-analyses and 15 cohort studies have been published about the links between dairy foods and CVD (17-39). The evidence indicates that consumption of milk, cheese and yogurt, regardless of fat content, is not linked to higher risk for CVD, CHD or stroke, and in some cases, consumption is linked to lower risk. To illustrate, a systematic review based on 12 meta-analyses published beginning in 2004, plus 11 additional prospective cohort studies published between 2004 and 2016, concluded (39):

o Total dairy food consumption as well as yogurt consumption is not associated with increased risk for CVD (moderate-quality evidence).

o Cheese consumption is not associated with increased risk for CVD (high-quality evidence).

o Total dairy food consumption as well as cheese consumption is associated with reduced risk for stroke (moderate-quality evidence).

o Milk consumption is not associated with risk for stroke (moderate quality-evidence).

Industrially produced trans fat has different biological effects than ruminant trans fat

There are two main dietary sources of trans fatty acids (TFA): industrially produced TFA (iTFA) formed during the partial hydrogenation of vegetable oils, and naturally occurring TFA produced by ruminants such as cows - also referred to as ruminant TFA (rTFA) - that are naturally present in meat and milk (40). Ruminant TFA cannot be removed entirely from most healthy diets and its intake is low in most populations (41). Additionally, emerging evidence indicates that the biological activities of iTFA and rTFA differ. To date there is no conclusive evidence supporting an association of rTFA with CHD risks in the amounts usually consumed, while iTFA consumption has been linked to specific negative health consequences:

o The FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition concluded in 2010: "There is convincing evidence that TFA from commercial partially hydrogenated vegetable oils (PHVO) increase CHD risk factors and CHD events - more so than had been thought in the past. There also is probable evidence of an increased risk of fatal CHD and sudden cardiac death in addition to an increased risk of metabolic syndrome components and diabetes" (42).

o The 2015 systematic review and meta-analysis by de Souza et al., mentioned above, concluded that industrially produced trans fats are associated with risk of CHD and CHD mortality, but not ruminant derived (10).

o The WHO consultation defined rTFA in the same category as iTFA and suggested that rTFA have the same health effects as iTFA. It was speculated that by reducing iTFA consumption, rTFA intake might increase. However, that scenario is very unlikely to occur as indicated in the 2009 WHO Scientific update on trans fatty acids, which stated that, "Although ruminant TFAs cannot be removed entirely from the diet, their intake is low in most populations and to date there is no conclusive evidence supporting an association with CHD risks in the amounts usually consumed" (41).

o A publication reporting worldwide consumption of TFA stated, "If the only source of TFA in the diet is ruminant products, then TFA consumption would drop to less than 2 g/day or less than 1% of energy (43)." This level is unlikely to have an adverse effect on CVD risk markers in healthy people (40).

In addition, certain rTFA may be associated with beneficial health effects. Findings from the 2015 systematic review and meta-analysis by de Souza et al., and prospective studies, found that ruminant trans-palmitoleic acid was inversely associated with insulin resistance, type 2 diabetes and atherogenic dyslipidemia (10, 44, 45).

Given the above, it is important to differentiate rTFA from iTFA in dietary recommendations. Nutrient-rich milk and other dairy foods play a key role in healthy eating patterns throughout life, and growth and development, especially in childhood (46-48). Eliminating rTFA from healthy eating patterns will be virtually impossible and may yield questionable benefits, while a reduction in iTFA consumption has been demonstrated to have cardiovascular benefits. To recommend reducing all TFAs without differentiation between sources may lead to decreased nutrient intake if consumers are discouraged from eating dairy products.

## Conclusion

The full breadth of evidence available to inform recommendations may not have been considered by the WHO in their draft guidelines on saturated and trans fat. Nutrient-rich dairy foods, including milk, cheese and yogurt, are part of eating patterns worldwide (4, 46, 49-52). Making recommendations to restrict dietary saturated and trans fat without considering the source and the potential health benefits associated with healthy foods and eating patterns that naturally contain various forms of saturated and trans fat may have unintended consequences for diet quality and long-term health.

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Recommendations (for saturated fatty acids only)

Remarks (for saturated fatty acids only)

Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)



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Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

### *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)

## Summary of evidence (for trans-fatty acids only)

June 1, 2018

Comment to the WHO Department of Nutrition for Health and Development (NHD), Nutrition Guidance Expert Advisory Group (NUGAG) Subgroup on Diet and Health

RE: [Public consultation on the WHO Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children](#)

To Whom It May Concern:

National Dairy Council® (NDC) appreciates the opportunity to submit comments regarding WHO's Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children. NDC, the non-profit organization founded by the U.S. dairy farmers, is committed to nutrition research and education about dairy's role in the diet and health and wellness. NDC provides science-based dairy nutrition information to, and in collaboration with, a variety of stakeholders committed to fostering a healthier nation, including health professionals, educators, school nutrition directors, academia and industry. Established in 1915, NDC comprises a staff of registered dietitians and nutrition research and communications experts across the country.

Dietary patterns may be more predictive of health than individual nutrients

The randomized controlled trial evidence WHO reviewed when drafting guidance on saturated fat provides important insights about the health effects of saturated fat consumption (1, 2). Nutrition science, however, is evolving to include the whole eating pattern when considering the impacts of diet on chronic disease outcomes. Eating patterns capture the synergistic and cumulative effects that combinations of foods and beverages – and the nutrients they contain – can have on health (3). Because eating patterns contain multiple foods and beverages that work together in relation to health, they may be more predictive of health than any one food or nutrient (4).

The Canadian Heart and Stroke Foundation, in their statement on saturated fat, heart disease and stroke, concur: "The science of nutrition is ever-evolving with new evidence emerging all the time. It is becoming increasingly clear that what has the most impact on health is the overall quality of one's diet, combined with the types and quantity of food consumed." Their saturated fat recommendations "do not include a threshold or limit for saturated fat and instead focus on a healthy balanced dietary pattern, which can help Canadians reduce consumption of saturated fats (5)."

New observational evidence doesn't find link between saturated fat consumption and CVD risk

Recommendations to limit saturated fat consumption are based on evidence that a high consumption of saturated fat increases plasma LDL-cholesterol (LDL-C) levels, which has been linked to increased risk for heart disease (6). Recent prospective cohort studies, however, have found that populations consuming higher amounts of saturated fat do not have higher risk for cardiovascular disease (CVD) than those who consume lower amounts (7-9). One meta-analysis and systematic review by de Souza et al, commissioned by the WHO and published in 2015, found "no clear association between higher intake of saturated fats and all-cause mortality, coronary heart disease (CHD), CHD mortality, ischemic stroke, or type 2 diabetes among apparently healthy adults" (10).

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o Total dairy food consumption as well as yogurt consumption is not associated with increased risk for CVD (moderate-quality evidence).

o Cheese consumption is not associated with increased risk for CVD (high-quality evidence).

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## Conclusion

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Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)

Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments

## Final comments

Please provide any final thoughts or comments below.
<p>National Dairy Council® (NDC) appreciates the opportunity to submit comments regarding WHO's Draft guidelines on saturated fatty acid and trans-fatty acid intakes for adults and children. NDC, the non-profit organization founded by the U.S. dairy farmers, is committed to nutrition research and education about dairy's role in the diet and health and wellness. NDC provides science-based dairy nutrition information to, and in collaboration with, a variety of stakeholders committed to fostering a healthier nation, including health professionals, educators, school nutrition directors, academia and industry. Established in 1915, NDC comprises a staff of registered dietitians and nutrition research and communications experts across the country.</p> <p>The full breadth of evidence available to inform recommendations may not have been considered by the WHO in their draft guidelines on saturated and trans fat. Nutrient-rich dairy foods, including milk, cheese and yogurt, are part of eating patterns worldwide (4, 46, 49-52). Making recommendations to restrict dietary saturated and trans fat without considering the source and the potential health benefits associated with healthy foods and eating patterns that naturally contain various forms of saturated and trans fat may have unintended consequences for diet quality and long-term health.</p>

## Survey response 87

### General information

Family/last name
McCullough
Given/first name
KatieRose
Organization/affiliation
North American Meat Institute
Sector
Other
Sector [Other]
Country
United States of America

### Saturated fatty acids

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)

Additional comments
All comments can be found in the attachment.

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
Recommendations (for trans-fatty acids only)
Remarks (for trans-fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for trans-fatty acids only)
Additional comments
All comments can be found in the attachment.

## Final comments

Please provide any final thoughts or comments below.
All comments can be found in the attachment. Thank you.



June 1, 2018

Department of Nutrition for Health and Development  
World Health Organization  
Avenue Appia 20  
1202 Geneva

**Re: Request for Public Consultation on Guidelines: Saturated fatty acid and *trans*-fatty acid intake for adults and children.**

Dear Sir/Madam:

The North American Meat Institute (NAMI or Meat Institute) is the leading voice for the meat and poultry industry. The Meat Institute has a rich, century-long history and provides essential member services including legislative, regulatory, scientific, international, and public affairs representation. Together, the Meat Institute's members produce the vast majority of U.S. beef, pork, lamb, and poultry in facilities ranging in size from very small to large, in addition to the equipment, ingredients, and services needed to produce the safest, highest quality and most nutritious products.

Eating a balanced, healthful diet from all food groups and engaging in moderate exercise are the keys to a healthy lifestyle and providing wholesome, high quality, and nutrient dense products that can be part of a healthy dietary pattern is a priority for the meat industry. Meat Institute members are committed to supporting healthy dietary patterns and lifestyles for consumers by providing them with a variety of products to meet specific individual dietary needs. NAMI appreciates the opportunity to comment on the World Health Organization's (WHO) Guidelines for Saturated fatty acid and *trans*-fatty acid intake for adults and children (guidelines).

### **Policies should be based on the Totality of the Scientific Evidence.**

Policy and other guidance recommendations should be based on the strongest scientific evidence available. The totality of the scientific evidence must be considered when developing nutrition policy because emerging scientific evidence may provide additional insights into more appropriate dietary recommendations. Science by its very nature is constantly evolving. As new evidence becomes available, it should be incorporated into the body of scientific evidence because the totality of that evidence may change existing conclusions.

The current WHO guideline vilifies saturated fatty-acids. Regarding saturated fatty acids, however, a 2010 meta-analysis of prospective epidemiologic studies showed there is no significant evidence to conclude dietary saturated fat is associated with an increased risk of coronary heart disease (CHD) or cardiovascular disease (CVD).<sup>1</sup> Although these findings were controversial, there is a growing body of evidence that saturated fats may not be as harmful to humans as previously believed. Accordingly, it is important that all scientific evidence is reviewed to provide meaningful guidance by the WHO.

### **Vilifying Individual Nutrients Has the Potential for Adverse Unintended Consequences.**

Over the last four decades, several nutrients have been highlighted for concern, *e.g.*, fat and cholesterol. Yet, today, more robust scientific evidence shows those concerns are not justified or are at best questionable. During those decades, however, consumers reacted by avoiding those nutrients -- but at what expense? Does not drinking chocolate milk because of the added sugars outweigh the benefits of not drinking milk? Recommendations to eat a low-fat diet often led to overconsuming other foods, which meant one over-consumed dietary component merely replaced another.

A thoughtful, measured approach must be considered when drawing attention to certain nutrients. WHO should foster a total diet approach, where all foods can fit in moderation when combined with physical activity, for optimal health outcomes instead of vilifying individual nutrients.

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<sup>1</sup> Siri-Tarino, Patty W, Sun, Q. Hu, F., Krauss, R.M. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *The American Journal of Clinical Nutrition*, Volume 91, Issue 3, 1 March 2010, Pages 535–546, <https://doi.org/10.3945/ajcn.2009.27725>. <https://academic.oup.com/ajcn/article/91/3/535/4597110>. Accessed April 22, 2018.

## **Science Supports the Safety of Naturally Occurring *Trans*-Fatty Acids and Saturated Fatty Acids in the Diet.**

*Trans* fats naturally occur in ruminants and products derived from ruminants, *e.g.* meat products. Observational studies indicate that, in the amounts that people typically consume naturally occurring, or ruminant, *trans* fats, the negative effects on lipids are not seen. The public health concern regarding *trans* fats primarily involves those that are industrially produced, which are distinguishable from those involving *trans* fats in meat.<sup>2</sup> Indeed, the United States Food and Drug Administration (FDA) revoked the generally recognized as safe (GRAS) status of partially hydrogenated oils (PHOs), the main source of industrially produced *trans* fat and FDA will consider whether some PHOs are safe and at what levels.

Several systematic reviews and meta-analyses of epidemiological and clinical studies have been conducted and the results consistently reveal that usual consuming food sources of naturally occurring *trans* fat does not adversely affect cardiovascular disease risk.<sup>3</sup> An expert review article concluded studies distinguishing the effects of naturally occurring from industrially produced *trans* fat, or total *trans* fat in general, are needed, and that unnecessarily restricting naturally occurring *trans* fat intake could cause people to miss their possible health benefits.<sup>4</sup> WHO should recognize the health benefits of meat products with naturally occurring *trans* fats and not recommend that people consume less of these products because it could cause adverse health outcomes. Instead, WHO should consider naturally occurring and industrial *trans*-fatty acids separately in the guidelines.

## **It is Not Always Possible to Reduce Both Saturated Fatty Acids and *Trans*-Fatty Acids in Products and Retain the Same Product Functionality.**

In many products, fats are added not just for sensory reasons, but for product functionality. One cannot reduce both saturated fatty acids and *trans*-fatty acids in food products that require a high melting for functionality. For example, *trans*-fatty acids have been replaced by mixtures of butter and canola oil in margarine (now called "spreads") to maintain melting points. WHO needs to recognize the practical

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<sup>2</sup> Uauy, R., Aro, A., Clarke, R., Ghafoorunissa, R., L'Abbé, M., Mozaffarian, D., Skeaff, M., Stender, S., & Tavella, M. (2009). WHO Scientific Update on *trans* fatty acids: summary and conclusions. *European Journal of Clinical Nutrition*, 63, pp. S68-S75.

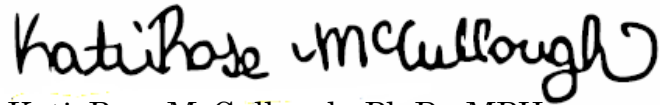
<sup>3</sup> Wang, Y., & Proctor, S. D. (2013). Current issues surrounding the definition of *trans*-fatty acids: implication for health, industry and food labels. *British Journal of Nutrition*, 110, pp. 1369-1383.

<sup>4</sup> Dawczynski, C., & Lorkowski, S. (2016). *Trans*-fatty acids and cardiovascular risk: does origin matter? *Expert Review of Cardiovascular Therapy*, 14, pp. 1001-1005.

problems associated with reducing both saturated fatty acids and *trans*-fatty acids in food products.

The Meat Institute appreciates the opportunity to submit these comments. If you have questions about these comments or would like to discuss them, please contact me at [kmccullough@meatinstitute.org](mailto:kmccullough@meatinstitute.org). Thank you.

Respectfully submitted,

A handwritten signature in black ink that reads "Katie Rose McCullough". The signature is written in a cursive, flowing style.

KatieRose McCullough, Ph.D., MPH  
Director, Regulatory and Scientific Affairs

cc: Barry Carpenter  
Mark Dopp  
Susan Backus

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## Survey response 88

### General information

Family/last name
Willett
Given/first name
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Organization/affiliation
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Sector
Academic/research
Sector [Other]
Country
United States of America

### Saturated fatty acids

## Executive summary (same for saturated fatty acids and trans-fatty acids)

All my comments are pasted here:

This review represents a major effort and the broad conclusions that saturated fat intake should be low and replaced with polyunsaturated fat or whole grains, and that trans fat intake should be low, are well supported by abundant evidence. The executive summary text is thoughtful and appropriate. That said, the rest of the review in this document reads as very mechanical and superficial; important details and nuances are not addressed. The report would be strengthened if each section included a deep discussion about the strengths, limitations, and interpretation of the evidence, and particularly the importance of considering the alternative substitutions for saturated and trans fat. The original meta-analyses published in BMJ in 2015 does contain a good discussion of the evidence from the meta-analysis, and importing some of that into this document could be useful.

It is appreciated that the review included the weight of evidence from RCT's, prospective cohort studies, and controlled feeding studies with CVD risk factors as outcomes. However, for saturated fat and CHD, the substantial body of evidence from prospective cohort studies is omitted except in the executive summary. As noted in the executive summary, these have been summarized in an analysis of primary data, with substitutions with either PUFA or carbohydrate, by Jakobsen et al. (reference #3).

One central issue not addressed is the basis for using 10% of energy as a cut point for saturated fat or 1% of energy for trans fat. This is of course challenging because the dose-response relationships appear to be fairly linear, and not a clear basis for using a specific cut point; at a minimum this should be acknowledged. For trans fat this is particularly important because trans fat from partial hydrogenation has no nutritional benefit and only harms; in this case it would usually be appropriate and prudent to have some margin of safety below the level where harm has been documented. Also, this is important because 18:2 trans has been more strongly associated with CHD than 18:1 trans, and interventions to reduce, rather than eliminate, industrial trans fat can actually increase 18:2 trans. Thus, the most conservative and prudent objective should be to eliminate partial hydrogenation (as has been done in several countries), and is clearly feasible.

The finding for trans fat and total mortality (Low quality) was based on 1 study with about 1500 deaths and 18,000 persons. A far larger study (over 125,000 men and women with 33,000 deaths), based on many repeated measures of trans fat with dynamically updated food composition data) showed a very strong and highly significant positive association between trans fat and total mortality (Wang DD. JAMA Internal Medicine, 2016). The repeated measures of trans fat intake are particularly important because food composition has changed greatly over the last several decades.

Page 34 The conclusion about trans fat and diabetes risk (very low quality of evidence) is likely to be wrong. The largest study using repeated measurements of trans fat intake (Hu FB et al. NEJM 2001, including 83,000 women and 3,300 cases) showed a strong and highly significant positive association (a report from the same cohort with fewer cases was included). Also, in controlled feeding studies, compared to other types of fat, trans fat uniquely reduces HDL cholesterol, increases triglycerides, and increases inflammatory factors, which are all components of the metabolic syndrome, adding important biological support for effects on diabetes risk. The relation between trans fat and diabetes risk is important because intakes and diabetes risk are high in many low income countries.

Page 11—The comment Re “Public health interventions should aim to reduce saturated fatty acid intake, while reducing total fat intake where necessary, and without increasing free sugars intake. “ is strange and not supported by any data because there is no good evidence that total fat needs to be limited more than total carbohydrate. The same comment pertains to trans fat on page 14.

Page 46 The statements about research needed (below) seem naïve. Given the available evidence, would an IRB approve randomizing people to replacing saturated fat with refined starch vs whole grains to look at total mortality? Would people be willing to be randomized if they were fully informed of the available evidence? Can we ethically continue intervention to examine mortality if there is an effect on incidence of diabetes or heart disease? Also, the experience with adherence to assigned diets in studies of this type, such as the MRFIT and WHI, has been dismal. The reality is that such studies are highly unlikely to be feasible.

“Research needed on saturated fatty acids:

- large RCTs that include populations from different geographical regions and assess the effects on risk of cardiovascular diseases and mortality of replacing saturated fatty acids with MUFA and different types of carbohydrates (e.g. refined and unrefined) and PUFA (e.g. n-3 PUFA, n-6 PUFA) “

Again, the overall conclusions of this report are on target, and it is hoped that the above comments may strengthen them.

## Scope and purpose (same for saturated fatty acids and trans-fatty acids)

## Background (same for saturated fatty acids and trans-fatty acids)

Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for saturated fatty acids only)
Recommendations (for saturated fatty acids only)
Remarks (for saturated fatty acids only)
Dissemination, translation and implementation, and monitoring and evaluation (same for saturated fatty acids and trans-fatty acids)
Research gaps and future initiatives (same for saturated fatty acids and trans-fatty acids)
Annexes 1, 6, 7 (for saturated fatty acids only)
Additional comments

## *Trans-fatty acids*

Executive summary (same for saturated fatty acids and trans-fatty acids)
Scope and purpose (same for saturated fatty acids and trans-fatty acids)
Background (same for saturated fatty acids and trans-fatty acids)
Guideline development process (same for saturated fatty acids and trans-fatty acids)
Summary of evidence (for trans-fatty acids only)
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Additional comments

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## Final comments

Please provide any final thoughts or comments below.