Saturated fatty acid and *trans*-fatty acid intake for adults and children

WHO guideline summary
Saturated fatty acid and trans-fatty acid intake for adults and children: WHO guideline summary

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Note

This document is a summary of a WHO guideline, the full version of which is available only in English and can be accessed at https://www.who.int/publications/i/item/9789240073630. This summary document does not contain all of the information found in the full guideline, but what it does contain has been extracted directly from the full guideline and is therefore identical to the information found in the full guideline. Anywhere in this summary document where “this guideline” or “the guideline” is used in the text, it refers to the full guideline document.
Background

Noncommunicable diseases (NCDs) are the world’s leading cause of death, responsible for an estimated 41 million of the 55 million deaths in 2019 (1). Nearly half of these deaths were premature (i.e. in people aged less than 70 years) and occurred in low- and middle-income countries. Of the major NCDs, cardiovascular diseases (CVDs) were the leading cause of mortality in 2019, responsible for more than 18 million deaths (2). Modifiable risk factors such as unhealthy diets, physical inactivity, tobacco use and harmful use of alcohol are major risk factors for CVDs. Dietary saturated fatty acids (SFA) and trans-fatty acids (TFA) are of particular concern because high levels of intake have been correlated with increased risk of CVDs (3).

SFA are fatty acids containing only single carbon–carbon bonds (i.e. no double bonds). They are found primarily in foods from animal sources (e.g. dairy foods, meat, egg yolks, hard fats), as well as in some plant-derived fats and oils.

TFA are unsaturated fatty acids with at least one double carbon–carbon bond in the trans configuration. TFA can be produced industrially by the partial hydrogenation of vegetable and fish oils, but also occur naturally in meat and dairy products from ruminant animals (e.g. cattle, sheep, goats, camels) as a result of the conversion of cis double bonds in unsaturated fatty acids to the trans position by bacterial enzymes in the stomach (rumen) of the animals. Although the sources are different, the individual isomers in industrially produced and ruminant TFA are largely the same, but present in differing proportions (4–6). Industrially produced TFA are the predominant source of dietary TFA in many populations. They can be found in partially hydrogenated cooking oils and fats which are often used at home, in restaurants, or in the informal sector (e.g. street vendors), and in ready-made baked and fried foods (e.g. doughnuts, cookies, crackers and pies) and other pre-packaged snacks and foods. Although current intakes of ruminant TFA are generally low, ruminant TFA may become the predominant dietary source of TFA in populations where industrially produced TFA are being phased out of the food supply (7–9).

Reduced intake of SFA has been associated with a significant reduction in the risk of coronary heart disease when SFA are replaced with polyunsaturated fatty acids or carbohydrates from whole grains (10–13). However, an apparent lack of effect is often observed in studies in which the macronutrients replacing SFA are unknown, are not accounted for or consist largely of refined carbohydrates (10, 13–15). Studies have also demonstrated that high intakes of industrially produced TFA are strongly associated with increased risk of coronary heart disease and related mortality (16, 17). Few studies have identified an association between intake of ruminant TFA and CVDs; however, to date, ruminant TFA intake in most study populations has been very low (18). Efforts to understand the effects of SFA intake in greater detail have shown that individual SFA may have differing effects on blood lipids (19). In addition, growing evidence has led to the suggestion that different SFA-containing foods, such as dairy foods, may have differential effects on risk of CVDs and type 2 diabetes, as a result of either differing compositions of SFAs across foods, other constituents of the foods (i.e. the “food matrix”) or a combination of the two (20–26).

The reduction in CVD risk observed with decreased intake of SFA and TFA is believed to occur primarily through an effect on blood lipids, because intakes of both are associated with increases in levels of total cholesterol and low-density lipoprotein (LDL) cholesterol (19, 27), and decreases in high-density lipoprotein (HDL) cholesterol in the case of TFA (27). Other physiological mechanisms, such as inflammation, may also play a role (28, 29). Increased total cholesterol is associated with increased risk of coronary heart disease (30). LDL cholesterol is a well-established surrogate end-point (i.e. biomarker) for measuring the effects of
interventions on CVD risk (31, 32), and is considered by many to be a causal factor for atherosclerosis and coronary heart disease (33). Other lipid measures – such as non-HDL cholesterol, triglycerides, cholesterol ratios and cholesterol particle number – have also been suggested as possible predictors of CVD risk.

Although CVDs typically present later in life, preclinical signs of atherosclerosis in the form of atherosclerotic lesions in the aorta and coronary arteries can begin to appear in childhood (34, 35), and are positively associated with abnormal blood lipid levels and other CVD risk factors (36, 37). Elevated total and LDL cholesterol in childhood are associated with an increase in CVD risk factors in adulthood (38), including thickening of the carotid artery intima-media (39–41), which is a marker of subclinical atherosclerosis and a predictor of future cardiovascular events (42). Dietary intervention studies conducted in children have demonstrated significant reductions in total or LDL cholesterol when SFA were replaced with polyunsaturated fatty acids (43–48). Despite the positive effect of such replacement on blood lipids, concern has been raised about the possible negative impact of a reduced-fat diet or a diet intended to reduce blood lipids on normal growth and development in children (49, 50), although the primary concern has generally been the potential for inadequate caloric or micronutrient intake rather than any effects related to SFA itself.

Studies of TFA intake in children are limited; nevertheless, there is no evidence to suggest that the effects on blood lipids would be different from those observed in adults, and intake may therefore lead to preclinical signs of atherosclerosis (34–37), as described in the preceding paragraph.

Despite longstanding dietary advice to limit SFA intake and a limited number of focused efforts to reduce intake at the population level, SFA intake remains high in many parts of the world (51). And while more consistent efforts to reduce the level of industrially produced TFA in the food supply at the local to national levels have led to decreased intake in some countries (52), the global average intake of TFA in 2010 (51) was estimated to exceed the population nutrient intake goal of 1% of total energy intake established by the 1989 World Health Organization (WHO) Study Group on Diet, Nutrition and the Prevention of Chronic Diseases (53) and updated by the 2002 Joint WHO/Food and Agriculture Organization of the United Nations (FAO) Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases (3). Efforts to reduce the level of industrially produced TFA in the food supply received a boost in 2018, when their elimination was identified as one of the priority targets in the WHO 13th General Programme of Work. The WHO REPLACE action package was launched in 2018 to help countries eliminate industrially produced TFA from their food supplies.¹

**Objective, scope and methods**

The objective of this guideline is to provide updated guidance on the intake of SFA and TFA, to be used by policy-makers, programme managers, health professionals and other stakeholders in efforts to promote healthy diets. The guideline was developed by the WHO Nutrition Guidance Expert Advisory Group (NUGAG) Subgroup on Diet and Health following the WHO guideline development process, as outlined in the *WHO handbook for guideline development* (54). This process includes a review of systematically gathered evidence by an international, multidisciplinary group of experts; assessment of the quality of that evidence via the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework²; and consideration of additional, potentially mitigating factors³ when translating the evidence into recommendations. The guideline was reviewed by a group of external experts and feedback was solicited from interested stakeholders during public consultations. The guidance in this guideline replaces previous WHO guidance on SFA and TFA intake, including that from the 1989 WHO Study Group on Diet, Nutrition and the Prevention of Chronic Diseases (53) and the 2002 Joint WHO/FAO Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases (3).

¹ https://www.who.int/teams/nutrition-and-food-safety/replace-trans-fat
² http://www.gradeworkinggroup.org/
³ These 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.
The evidence

SFA

Evidence from recent systematic reviews of randomized controlled trials (RCTs) and prospective observational studies conducted in adults (55–57) suggests the following.

▶ Lowering SFA intake reduces low-density lipoprotein (LDL) cholesterol (high certainty evidence) and CVD risk (moderate certainty evidence), and may be associated with reduced risk of all-cause mortality (i.e. death from any cause) and coronary heart disease (both very low certainty evidence).

▶ Consuming 10% or less of daily calories (i.e. total energy intake) as SFA reduces LDL cholesterol (high certainty evidence), is associated with reduced risk of all-cause mortality (low certainty evidence), and may be associated with reduced risk of coronary heart disease (very low certainty evidence).

▶ Replacing SFA with unsaturated fatty acids and carbohydrates lowers LDL cholesterol (high certainty evidence) and is associated with reduced risk of all-cause mortality (low to moderate certainty evidence).

▶ Replacing SFA with polyunsaturated fatty acids, monounsaturated fatty acids from plant-based foods, and carbohydrates from foods containing naturally occurring dietary fibre (e.g. whole grains, vegetables, fruits, pulses) is associated with additional health benefits including reduced risk of coronary heart disease (very low to low certainty evidence).

▶ Replacing SFA with mixed protein or animal protein (but not plant protein) is associated with an increase in risk of coronary heart disease (very low to low certainty evidence).

Although beneficial effects of lowering SFA intake were not observed for all outcomes assessed, there was no indication that lower SFA intake increased risk for any critical outcome (except when SFA were replaced by mixed or animal protein), nor were there any other significant undesirable effects identified in the systematic reviews.

Evidence from a systematic review of RCTs conducted in children (58) found that reducing SFA intake reduced total cholesterol, LDL cholesterol and diastolic blood pressure (high certainty evidence). A small number of trials suggest that the effect was strongest when SFA were replaced primarily with polyunsaturated fatty acids or a mixture of polyunsaturated fatty acids and monounsaturated fatty acids, and when SFA intake was reduced to a level less than 10% of total energy intake (high certainty evidence). Significant effects were not observed for other outcomes, and there were no indications of any adverse effects from reduced SFA intake.

TFA

Evidence from recent systematic reviews of RCTs and prospective observational studies conducted in adults (56, 59) suggests the following.

▶ Lowering TFA intake reduces LDL cholesterol (high certainty evidence), and is associated with reduced risk of all-cause mortality, CVDs and coronary heart disease (low to moderate certainty evidence).

▶ Consuming 1% or less of total energy intake as TFA reduces LDL cholesterol (high certainty evidence), is associated with reduced risk of CVDs and coronary heart disease (low certainty evidence), and may be associated with reduced risk of all-cause mortality (very low certainty evidence).

▶ Replacing TFA with unsaturated fatty acids and carbohydrates lowers LDL cholesterol (high certainty evidence) and is associated with reduced risk of all-cause mortality. Replacing TFA with monounsaturated fatty acids from plant-based foods is associated with reduced risk of coronary heart disease (low certainty evidence).

▶ Replacing TFA with either carbohydrates or polyunsaturated fatty acids is associated with reduced risk of type 2 diabetes (moderate and very low certainty evidence, respectively).

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1 Based on the grades of evidence set by the GRADE Working Group. High certainty means that we are very confident that the true effect lies close to that of the estimate of the effect; moderate certainty means that we are moderately confident in the effect estimate – the true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different from the estimate of the effect; low certainty means that our confidence in the effect estimate is limited – the true effect may be substantially different from the estimate of the effect; and very low certainty means that we have very little confidence in the effect estimate – the true effect is likely to be substantially different from the estimate of the effect (54).
There were no indications of any adverse effects from reduced TFA intake. No studies were identified that met the inclusion criteria established for the systematic review of TFA intake in children (58).

**Additional evidence reviewed**

Additional evidence was reviewed and is summarized below, although the NUGAG Subgroup on Diet and Health did not use it to support the formulation of recommendations for the reasons given in the section *Interpreting the evidence for SFA*.

**Tissue measurements of SFA intake**

In addition to studies in which SFA intake was self-reported (via 24-hour recall, food diaries, food frequency questionnaires, etc.), the systematic review of observational studies (56) also identified studies in which intake was assessed by measuring total SFA in tissues of the body (e.g. plasma phospholipids, red blood cells, fat biopsies). Meta-analyses of observational studies assessing total SFA intake via tissue measurements found that lower intake is associated with a 31% reduction in the risk of coronary heart disease (95% CI: 0.51 to 0.91) and a 23% reduction in the risk of type 2 diabetes (95% CI: 0.63 to 0.94). Several studies also assessed the intake of individual SFA via tissue measurement, as described below.

**Individual SFA intake**

The systematic review and meta-analyses of observational studies included studies that assessed individual SFA intake via tissue measurement (56), including lauric acid (12:0), myristic acid (14:0), pentadecanoic acid (15:0), palmitic acid (16:0), heptadecanoic acid (17:0), stearic acid (18:0) and very long chain fatty acids (i.e. longer than 18 carbons) of different length. Data for all-cause mortality and CVDs were limited, and consistent associations were not observed between individual SFA and these outcomes. Increased intake of pentadecanoic acid, heptadecanoic acid and very long chain SFA were strongly associated with reduced risk of type 2 diabetes, whereas increased intake of palmitic acid was strongly associated with increased risk of type 2 diabetes.

The effects of individual SFA (lauric acid, myristic acid, palmitic acid and stearic acid) on blood lipids were also assessed in the systematic review and multiple regression analyses of blood lipids, as the effects of isocalorically replacing a mixture of carbohydrates with these individual SFA (58). Replacement of carbohydrates with lauric acid, myristic acid or palmitic acid all significantly raised total, LDL and HDL cholesterol (with the magnitude of effect decreasing in the order myristic > palmitic > lauric), and lowered triglyceride levels and the triglyceride to HDL cholesterol ratio. Lauric acid lowered the total cholesterol to HDL cholesterol ratio and the LDL cholesterol to HDL cholesterol ratio. Stearic acid did not have a significant effect on any outcome assessed. Although differences were observed in effects of the individual SFA on the lipid profile, reported intakes of lauric acid and myristic acid in the individual trials included in the regression analysis were low (mean of 1.2% of total energy intake), which may have influenced the results.

**Interpreting the evidence**

Several observations were made in interpreting the results of the systematic reviews, some based directly on data from the review and others supported by background questions and information that helps to establish the context for the recommendation (54). They are summarized below.

**SFA**

*Replacement nutrients.* The NUGAG Subgroup on Diet and Health reaffirmed what has been previously noted in the literature, that is, associations between lower SFA intake and relevant health outcomes are limited or generally not observed when the nutrients replacing SFA are not specified. In the systematic reviews assessed for this guideline, the only effect observed when replacement nutrients were not accounted for was on CVDs as assessed in RCTs; no effects were seen for other disease outcomes. Only when specific replacement nutrients were assessed were significant associations observed. This suggests that, in studies where no association is observed between lower SFA intake and reduced risk of disease, the nutrients replacing SFA may themselves increase the risk of disease and therefore may mask any benefit of
reducing SFA intake. Consequently, choice of replacement nutrient is key to obtaining a health benefit from reducing SFA intake.

**Extrapolating results from adults to children.** Although ample evidence was available from studies in children, the NUGAG Subgroup on Diet and Health considered the available evidence for cardiovascular, mortality and blood lipid outcomes from adults to also be relevant to children, given that preclinical signs of atherosclerosis in the form of atherosclerotic lesions in the aorta and coronary arteries can begin to appear in childhood (34, 35); these changes are positively associated with abnormal blood lipid levels and other CVD risk factors (36, 37). Therefore, in formulating recommendations for children, the NUGAG Subgroup on Diet and Health considered not only the evidence from direct assessments in children but also the evidence for adults, without downgrading for indirectness.

**Tissue measurement of SFA intake.** Although data were available for both self-reported intakes of SFA and estimates of intake based on tissue levels of SFA, there were more studies that included self-reported data on total SFA intake, and these data were generally more robust. In addition, although assessment of SFA in tissues can be a fairly reliable indicator of dietary intake, the potential contribution of endogenous synthesis cannot be consistently estimated. Therefore, using a conservative approach, the evidence from tissue levels was not included in the evidence base supporting the recommendations for SFA intake, even though these results are in line with the results and conclusions from self-reported intakes, as well as the other reviews assessed for these guidelines.

**Individual SFA.** Significant associations were observed between certain individual SFA (as assessed by tissue levels) and type 2 diabetes, and differences were observed between individual SFA with respect to their effects on blood lipids, with the exception of stearic acid (which showed little effect on blood lipids). However, results observed for associations between individual SFA and disease outcomes were consistent with the results observed for total SFA – that is, none of the statistically non-significant effects observed for individual SFA suggested benefit with increased intake, but some suggested harm. In addition, as noted above, the NUGAG Subgroup on Diet and Health had concerns with tissue measurements because of the inability to ensure consistent measurement of endogenous synthesis of SFA, as well as with the low reported intakes of lauric acid and myristic acid in the blood lipids analyses. Finally, there was no evidence available from RCTs assessing the effects of consuming individual SFA on disease outcomes. It was therefore concluded that further research is needed before recommendations on the intake of individual SFA can be made.

**TFA**

**Total, industrially produced and ruminant TFA.** As per the original PICO questions, results were generated for total TFA intake,¹ and separately for industrially produced and ruminant TFA 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 TFA intake were similar for risk of coronary heart disease, but not for all-cause mortality or CVDs, for which only total TFA intake demonstrated a significant association between reduced intake and reduced risk. No associations were observed for the analysis of studies reporting effects of ruminant TFA intake.² In the regression analysis of RCTs, reduced intake of total TFA or industrially produced TFA 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 TFA intake on lowering LDL cholesterol was only observed when ruminant TFA were replaced with polyunsaturated fatty acids. For all other blood lipid outcomes, results were not statistically significant; however, they were similar to those for total and industrially produced TFA in both direction and magnitude.³

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

² Results for effects of industrially produced and ruminant TFA on all-cause mortality and CVDs came 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 SFA via tissue measurements, the correlation between TFA measured in tissue and dietary intake has generally been shown to be stronger (60). Results for effects of industrially produced and ruminant TFA on coronary heart disease came from studies in which dietary intake was self-reported.

³ The two trials that reported ruminant TFA intakes at levels more similar to the intakes reported for industrially produced TFA (i.e. >2% of total energy intake) reported greater reductions in LDL cholesterol (61, 62).
Intake of ruminant TFA in the studies included in the analyses of both prospective observational studies and RCTs was very low relative to intake of industrially produced TFA, and the difference between lower and higher intakes was very small. The available evidence suggests that differences in effects on health outcomes between ruminant, industrially produced and total TFA observed in many studies may be due to differences in the amount of TFA being consumed rather than differences between types of TFA. To further assess the nature of the observed differences, post hoc analyses were conducted in which the intakes observed in the studies of ruminant TFA were approximated in the studies of total TFA, such that the highest intakes of total TFA were limited to 0.7–1.3% of total energy intake and then compared with the lowest intakes. When total TFA intake was assessed in this manner, the associations and dose–response relationships originally observed between lower TFA intake and reduced risk of all-cause mortality remained, but those for CVDs 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 TFA intake in the small number of studies, the difference between the lowest and highest intakes was not large enough to allow reliable comparisons. It was further noted that in the very few studies assessing LDL cholesterol in which the highest levels of ruminant TFA intake were closer to those observed for industrially produced and total TFA, the effects of ruminant TFA intake were similar to, or more pronounced than, those observed for industrially produced and total TFA.

It was therefore determined that the available evidence did not support making a distinction between industrially produced and ruminant TFA. Because the vast majority of studies included in the systematic reviews reported results for total TFA intake (which includes intake from industrially produced and ruminant sources), it was considered appropriate to consider only evidence from total TFA when formulating the recommendations on TFA intake.

**Conjugated linoleic acid (CLA).** CLA is found in fat from ruminant animals and represents several isomers of linoleic acid in which the two double bonds are conjugated (i.e. separated by a single bond), resulting in a three-dimensional shape that is different from most other TFA isomers. As CLA contains both cis and trans 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, although the number of such trials was limited and intakes of CLA were very low. Nevertheless, results of these trials provided no indication that CLA had an effect on blood lipids that was significantly different from other TFA when consumed at similar levels. The NUGAG Subgroup on Diet and Health therefore concluded that, because CLA contributes to total TFA intake, it should be included in the definition of TFA as used in the recommendations on TFA.

### Recommendations and supporting information

All recommendations for SFA and TFA should be considered in the context of other WHO guidelines on healthy diets, including those on total fat (63), polyunsaturated fatty acids (3), sugars (64), sodium (65), potassium (66) and carbohydrates (67). An explanation of the strength of WHO recommendations can be found in Box 1.

#### SFA recommendations

1. WHO recommends that adults and children reduce saturated fatty acid intake to 10% of total energy intake (strong recommendation).
2. WHO suggests further reducing saturated fatty acid intake to less than 10% of total energy intake (conditional recommendation).
3. WHO recommends replacing saturated fatty acids in the diet with polyunsaturated fatty acids (strong recommendation); monounsaturated fatty acids from plant sources (conditional recommendation); or carbohydrates from foods containing naturally occurring dietary fibre, such as whole grains, vegetables, fruits and pulses (conditional recommendation).

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1. WHO guidance on polyunsaturated fatty acids is currently being updated.
Rationale and remarks

The following provides the reasoning (rationale) behind the formulation of the recommendations, as well as remarks designed to provide context for the recommendations and facilitate their interpretation and implementation. This information is also provided for the recommendations on TFA.

Rationale for SFA recommendations 1 and 2

 Recommendations 1 and 2 are based on evidence from four systematic reviews that assessed the effects of lower compared with higher SFA intake. These systematic reviews found that lower SFA intake reduced the risk of all-cause mortality and CVDs. The overall certainty in the evidence for recommendation 1 was moderate, and for recommendation 2 was very low.

 Specific findings from the reviews supporting these recommendations include the following.

- As assessed in RCTs in the systematic review by Hooper et al. (55), reducing SFA intake reduced the risk of CVDs in adults (moderate certainty evidence); greater reductions in SFA intake resulted in greater reduction in risk. No effect, or effects that trended towards reduced risk of CVDs, were observed for other critical outcomes; none suggested increased risk. All but one of the trials included in the analyses reported SFA intakes of more than 10% of total energy intake at baseline, and although stepwise testing of thresholds of intake did not find a clear effect on any cardiovascular or mortality outcome at SFA intakes of less than 10% of total energy intake, significant reductions in risk of CVDs and CVD mortality were observed with SFA intakes of less than 9% of total energy intake. Consequently, there is ample evidence supporting reduction of SFA intake to 10% of total energy, but only limited evidence supporting a reduction to below 10% of total energy intake.

- As assessed in prospective observational studies in the systematic review by Reynolds et al. (56), lower SFA intake compared with higher intake (very low certainty evidence) and consuming SFA at a level of less than 10% of total energy intake compared with intakes greater than 10% (low certainty evidence) are associated with reduced risk of all-cause mortality in adults.

- As assessed in RCTs and strictly controlled feeding trials in the systematic review by Mensink (57), replacing SFA with polyunsaturated fatty acids, monounsaturated fatty acids and carbohydrates all resulted in reductions in low-density lipoprotein (LDL) cholesterol in adults (high certainty evidence). The LDL cholesterol-lowering effects of replacing saturated fatty acids with other nutrients are cumulative – that is, the more SFA intake is reduced, the more LDL cholesterol is lowered. The effects...
were observed down to SFA intakes of 2% of total energy intake (effects were observed across a wide range of SFA intakes, from 2% to 24% of total energy intake).

- Reducing SFA intake, as assessed in RCTs conducted in children (58), resulted in reduced LDL cholesterol and blood pressure (both high certainty evidence). All but one of the trials included in the analyses reported SFA intakes of more than 10% of total energy intake at baseline and very limited evidence suggests that reducing SFA intake to less than 10% of total energy intake reduces LDL cholesterol to a greater extent than reducing intake to a level higher than 10% of total energy intake (moderate certainty evidence).

Evidence from the systematic review by Hooper et al. (55) did not suggest undesirable effects in adults from reduced SFA intake with respect to any of the critical outcomes, cancer incidence or mortality, serum lipids, blood pressure, measures of body fatness, or quality of life. Rather, the evidence suggested small benefits or no effect. Evidence from the systematic review by Mensink (57) suggested a slight increase in triglycerides and a reduction in high-density lipoprotein (HDL) cholesterol when SFA are replaced by carbohydrates of mixed composition. However, the clinical relevance of such changes is not clear (68). This finding was therefore not an influential consideration in the balance of desirable and undesirable effects, given the evidence for disease and mortality outcomes, and taking into account recommendation 3 on replacement nutrients for SFA. Evidence from the systematic review conducted in children indicates that reducing SFA intake does not compromise children’s linear growth, micronutrient status, cognitive development or sexual development (58). No other data on undesirable effects in adults or children were identified.

Recommendation 1 was assessed as strong because evidence of moderate certainty overall from different study types assessing both risk factors and incidence of CVDs suggested reduced risk of CVDs with lower SFA intake. No undesirable effects or other mitigating factors were identified that would argue against a lower SFA intake.

Recommendation 2 was assessed as conditional because, although evidence from different study types from each of the systematic reviews suggested reduced risk of CVDs with SFA intakes of less than 10% of total energy intake, the evidence is much more limited than for intakes greater than 10% of total energy intake and therefore there is less confidence in it (very low certainty evidence overall). No undesirable effects or other mitigating factors were identified that would argue against reducing SFA intake to less than 10% of total energy intake. A conservative approach was therefore taken, leading to a conditional recommendation.

Rationale for SFA recommendation 3

Recommendation 3 is based on moderate certainty evidence overall for replacing SFA with polyunsaturated fatty acids and low certainty evidence overall for replacing SFA with monounsaturated fatty acids or carbohydrates. Evidence comes from four systematic reviews that assessed the effects of lower compared with higher SFA intake via replacement nutrient analysis. These reviews found that lower SFA intake reduced the risk of all-cause mortality, CVDs and coronary heart disease. Specific findings from the reviews supporting this recommendation include the following.

- Subgroup analysis of RCTs in the systematic review by Hooper et al. (55) showed a reduction in risk of CVDs and coronary heart disease when SFA were replaced with polyunsaturated fatty acids (moderate certainty evidence), but not when SFA were replaced by carbohydrates, monounsaturated fatty acids (for which there was insufficient evidence to allow an adequate assessment) or protein.¹

- As assessed in prospective observational studies in the systematic review by Reynolds et al. (56), replacing SFA with polyunsaturated fatty acids (low certainty evidence overall) or plant-based monounsaturated fatty acids (moderate certainty evidence overall) was associated with reductions in risk of CVDs, coronary heart disease and all-cause mortality. More limited evidence shows that replacing SFA with carbohydrates, particularly those from whole grains and foods described by

¹ In this review, polyunsaturated fatty acids were primarily from plant-based oils, rich in linoleic acid; carbohydrates were of largely unknown, and likely mixed, composition; and little to no data were available for nature of the protein.
the authors of the individual studies as having a low glycaemic index, was associated with small reductions in risk of CVDs and all-cause mortality (very low certainty evidence).

- As assessed in RCTs and strictly controlled feeding studies in the systematic review by Mensink (57), replacing SFA with polyunsaturated fatty acids, monounsaturated fatty acids or carbohydrates\(^1\) all resulted in reductions in LDL cholesterol (high certainty evidence). The greatest reduction in LDL cholesterol was observed for polyunsaturated fatty acids, followed by monounsaturated fatty acids and then carbohydrates.

- Very limited evidence from RCTs conducted in children (58) suggests that replacing SFA with polyunsaturated fatty acids or monounsaturated fatty acids reduces LDL cholesterol to a greater extent than replacing SFA with other nutrients (moderate certainty evidence).

The evidence for the health benefits of replacing SFA with carbohydrates from whole grains, vegetables, fruits and pulses is based on studies in which the composition of the carbohydrates was either unspecified and therefore likely a mixture, or were reported as coming from whole grains or foods described by the authors of the individual studies as having a low glycaemic index. Although the evidence from the systematic reviews that informed the development of this recommendation did not specifically assess the replacement of SFA with carbohydrates from vegetables, fruits or pulses (whole grains were assessed directly), robust evidence from systematic reviews informing WHO recommendations on carbohydrate intake (69–74) indicates that consuming whole grains, vegetables, fruits and pulses is associated with health benefits, and therefore that carbohydrates in the diet should primarily come from these foods (67).

The recommendation for replacing SFA with polyunsaturated fatty acids from plant sources was assessed as strong because evidence of moderate certainty overall from different study types that assessed both risk factors and disease incidence suggested that such replacement reduces the risk of CVDs and all-cause mortality.

The recommendations for replacing SFA with monounsaturated fatty acids from plant sources or carbohydrates from whole grains, vegetables, fruits and pulses was assessed as conditional because they are primarily based on evidence from observational studies, and also because vegetables, fruits and pulses were not directly assessed in the prospective cohort studies assessing replacement (whole grains were assessed directly).

**Remarks for Recommendation 3**

- To facilitate implementing this recommendation, replacing SFA can be achieved via a single recommended nutrient or a combination of nutrients.

- For further guidance on consumption of whole grains, vegetables, fruits and pulses, see the WHO guideline on carbohydrate intake (67).

- The guidance on replacement nutrients is relevant for a state of energy balance, in which total energy consumed is balanced by total energy expended. For energy balance, when the intake of one nutrient is reduced, the resulting energy deficit must be compensated for by intake of another nutrient. In cases of positive energy balance, and where a reduction in total energy intake is desired, SFA intake may be reduced in part or entirely without the need for a replacement nutrient.

**Remarks for all SFA recommendations**

- The recommendations as they apply to children are based on the totality of evidence, including both results of the review conducted in children and extrapolation of the results obtained from the reviews conducted in adults.

- The systematic review of prospective observational studies by Reynolds et al. (56) identified studies in which SFA exposures were assessed either by self-reported dietary intakes or measurement

\(^1\) In this review, polyunsaturated fatty acids were predominantly linoleic acid and α-linolenic acid; monounsaturated fatty acids were predominantly oleic acid; and carbohydrates were of largely unknown, and likely mixed, composition.
of SFA in tissues (e.g. plasma phospholipids, red blood cells, fat biopsies). The results for some outcomes differed between the two methods of exposure assessment: significant reductions in risk were observed for coronary heart disease and type 2 diabetes in studies where SFA intake was assessed by measuring SFA content of tissues, whereas no or non-significant results were observed for all outcomes in studies where SFA intake was assessed by self-reported dietary intakes, when replacement is not considered. Although assessment of SFA in tissues can be a relatively reliable indicator of dietary intake, the potential contribution of endogenous synthesis cannot be consistently estimated. Therefore, although the results for SFA tissue levels in the systematic review provide evidence of benefit of lower SFA tissue levels and generally support the evidence from other studies and analyses, the evidence from tissue levels was not formally assessed or included in the evidence base supporting the recommendations for SFA intake.

- Although there is evidence for differential effects of individual SFA, it is insufficient to inform the development of specific recommendations. SFA found naturally in foods are generally mixtures; consequently, intakes of individual SFA tend to be highly correlated with one another (75). Therefore, recommendations for individual SFA may be of limited utility to end users and difficult to implement – for example, in developing food-based dietary guidelines. Before recommendations can be made for individual SFA, further research is needed into their health effects and how such recommendations might be effectively used.

- These recommendations do not preclude consumption of particular foods. However, foods containing high levels of SFA should be consumed sparingly to meet the recommended level of intake.

### TFA recommendations

1. **WHO recommends that adults and children reduce trans-fatty acid intake to 1% of total energy intake** (strong recommendation).
2. **WHO suggests further reducing trans-fatty acid intake to less than 1% of total energy intake** (conditional recommendation).
3. **WHO recommends replacing trans-fatty acids in the diet with polyunsaturated fatty acids or monounsaturated fatty acids primarily from plant sources** (conditional recommendation).

### Rationale for TFA recommendations 1 and 2

- Recommendations 1 and 2 are based on evidence from two systematic reviews that assessed the effects of lower compared with higher TFA intake. These systematic reviews found that lower TFA intake reduced the risk of CVDs. The overall certainty in the evidence for recommendation 1 was moderate and for recommendation 2 was low.

- Specific findings from the reviews supporting these recommendations include the following.
  - As assessed in prospective observational studies in the systematic review by Reynolds et al. (56), lower TFA intake compared with higher intake (moderate certainty evidence overall) and consuming TFA at a level of less than 1% of total energy intake compared with intakes greater than 1% (low certainty evidence overall) were associated with reduced risk of all-cause mortality, CVDs and coronary heart disease. Greater reductions in TFA intake resulted in greater reductions in risk of all-cause mortality and coronary heart disease (i.e. dose–response relationships).
  - As assessed in RCTs in the systematic review by Brouwer (59), replacing TFA with polyunsaturated fatty acids, monounsaturated fatty acids and carbohydrates all resulted in reductions in LDL cholesterol (high certainty evidence) and overall improvements in blood lipid profile. The LDL cholesterol–lowering effects of replacing TFA with other nutrients are cumulative – that is, the more TFA intake is reduced, the more LDL cholesterol is lowered. These effects were observed across a wide range of TFA intakes, from 0% to 10.9% of total energy intake.
Recommendation 1 was assessed as strong because evidence of overall moderate certainty from different study types assessing both risk factors and incidence of CVDs suggested reduced risk of all-cause mortality, CVDs and coronary heart disease with lower TFA intake (in a dose-dependent manner with respect to all-cause mortality and coronary heart disease). No undesirable effects or other mitigating factors were identified that would argue against a lower TFA intake.

Recommendation 2 was assessed as conditional because, although there is evidence from different study types from each of the systematic reviews suggesting reduced risk of all-cause mortality, CVDs and coronary heart disease with TFA intakes of less than 1% of total energy intake, the evidence is more limited than for intakes greater than 1% of total energy intake and therefore there is less confidence in it (low certainty evidence overall). No undesirable effects or other mitigating factors were identified that would argue against reducing TFA intake to less than 1% of total energy intake. A conservative approach was therefore taken, leading to a conditional recommendation.

Rationale for TFA recommendation 3

Recommendation 3 is based on very low certainty evidence overall for replacing TFA with polyunsaturated fatty acids and moderate certainty evidence overall for replacing TFA with monounsaturated fatty acids from plant sources. Evidence comes from two systematic reviews that assessed the effects of lower compared with higher TFA intake via replacement nutrient analysis. These reviews found that lower TFA intake reduced the risk of all-cause mortality, CVDs, coronary heart disease and type 2 diabetes.

Specific findings from the reviews supporting this recommendation include the following.

- As assessed in prospective observational studies in the systematic review by Reynolds et al. (56), replacing TFA with polyunsaturated fatty acids was associated with reduced risk of type 2 diabetes (very low certainty evidence), and replacing TFA with monounsaturated fatty acids from plant sources was associated with reduced risk of all-cause mortality, CVDs and coronary heart disease (moderate certainty evidence overall).

- As assessed in RCTs in the systematic review by Brouwer (59), replacing TFA with polyunsaturated fatty acids, monounsaturated fatty acids or carbohydrates resulted in reductions in LDL cholesterol (high certainty evidence) and overall improvements in blood lipid profile. The greatest reduction in LDL cholesterol was observed for polyunsaturated fatty acids, followed by monounsaturated fatty acids and then carbohydrates.

- Recommendation 3 was assessed as conditional because evidence for disease outcomes comes only from a limited number of observational studies; most of the evidence is from RCTs with LDL cholesterol as an outcome. The evidence for LDL cholesterol is of high certainty. However, although LDL cholesterol is a well-established biomarker for measuring the effects of interventions on CVD risk, and is 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, leading to a conditional recommendation.

Remarks for TFA recommendation 3

The recommendation to replace TFA with polyunsaturated fatty acids or monounsaturated fatty acids from plant sources does not preclude replacing TFA with carbohydrates, as replacement with carbohydrates significantly lowered LDL cholesterol in the analysis of RCTs that assessed blood lipids. However, polyunsaturated fatty acids and monounsaturated fatty acids had greater effects on LDL cholesterol when used as replacements for TFA, and replacement of TFA with monounsaturated fatty acids from plant sources reduced the risk of coronary heart disease and all-cause mortality in prospective observational studies. Limited evidence suggests that replacing TFA with carbohydrates of unspecified composition also reduces the risk of type 2 diabetes, but that replacing TFA with free sugars or carbohydrates described by study authors as refined carbohydrates has little effect on risk of coronary heart disease. Therefore, a conclusive interpretation of the results for carbohydrate replacement of TFA in the analyses supporting the recommendations in this guideline was not possible.
Replacement of TFA with saturated fatty acids did not improve disease outcomes or blood lipids in the two systematic reviews. Saturated fatty acids are therefore not a preferred replacement for TFA.

To facilitate implementing this recommendation, replacing TFA can be achieved via polyunsaturated fatty acids or monounsaturated fatty acids alone, or a combination of the two.

This guidance on replacement nutrients is relevant for a state of energy balance, in which total energy consumed is balanced by total energy expended. For energy balance, when the intake of one nutrient is reduced, the resulting energy deficit must be compensated for by intake of another nutrient. In cases of positive energy balance, and where a reduction in total energy intake is desired, TFA intake may be reduced in part or entirely without the need for a replacement nutrient.

Remarks for all TFA recommendations

Because there weren't any relevant studies identified in a systematic review of TFA intake in children, the recommendations as they apply to children are based on extrapolation of the results obtained from the reviews conducted in adults.

For the purposes of these recommendations, TFA includes all fatty acids with a double bond in the trans configuration, regardless of whether the TFA come from ruminant sources or are produced industrially. This definition includes conjugated linoleic acid.

These recommendations do not preclude consumption of particular foods. However, foods containing high levels of industrially produced TFA should largely be avoided.

Translation and implementation

The recommendations in this guideline should be considered in conjunction with other WHO guidance on healthy diets to guide effective policy actions and intervention programmes to promote healthy diets and nutrition, and prevent diet-related NCDs.

The recommendations in this and related WHO guidelines acknowledge that both quantity and quality of fat consumed are important for maintaining health. Public health interventions should therefore aim to reduce total fat intake where necessary, while reducing SFA and TFA intake, through replacement with unsaturated fatty acids and/or carbohydrates, without increasing free sugars intake.

A detailed discussion of how the recommendations on SFA and TFA intake might be implemented is beyond the scope of this guideline, however they can be considered by policy-makers and programme managers when discussing possible measures, including:

- assessing current intake of SFA and TFA in their populations relative to benchmarks;
- developing policy measures to reduce intake of SFA and/or TFA, where necessary, through a range of public health interventions, many of which are already being implemented by countries, including:
  - nutrition labelling (i.e. mandatory nutrient declaration) and front-of-pack labelling systems
  - regulation of marketing of foods and non-alcoholic beverages that are high in SFA and/or TFA, including bans on marketing of foods that contain industrially produced TFA
  - restriction of the sale and promotion of foods and beverages that are high in SFA and/or TFA in and around schools
  - implementation of fiscal policies targeting foods and beverages that are high in SFA and/or TFA
  - consumer education;
- developing strategies to reformulate food products; and
- translating the recommendations at the country-level into culturally and contextually specific food-based dietary guidelines that take into account locally available foods and dietary customs.

Elimination of industrially produced TFA is among the priority actions identified by WHO in its 13th General Programme of Work, which will guide the work of WHO in 2019–2023. Industrially produced TFA are the
predominant source of dietary TFA in many populations. They can be found in baked and fried foods (e.g. doughnuts, cookies, crackers, pies), pre-packaged snacks and food, and partially hydrogenated cooking oils and fats, which are often used in homes, in restaurants and in the informal sector (e.g. by street vendors). Therefore, removing industrially produced TFA from the food supply through legislation or regulatory action represents a well-defined mechanism for translating the recommendations in this guideline into action and achieving significant reductions in TFA intake at the population level.

In 2018, WHO released the REPLACE action package, which provides support for implementing the WHO recommendations on TFA and is a roadmap for countries to achieve prompt, complete and sustained elimination of industrially produced TFA from the food supply. In 2019, 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 TFA from the food supply. Before the release of REPLACE, industrially produced TFA had already largely been removed or were in the process of being removed from the food supply at the national and subnational levels in many countries (9, 76, 77). As of September 2022, 60 countries had implemented mandatory TFA limits; of these, 43 countries had implemented a best-practice TFA policy that either virtually eliminates industrially produced TFA or bans partially hydrogenated oils (78), demonstrating that global reduction in TFA intake may be an achievable goal.

Providing comprehensive dietary guidance is beyond the scope of these guidelines, because such guidance should be based on overall dietary goals that consider all required nutrients. However, it is feasible to achieve the recommendations in this guideline while respecting national dietary customs, because a wide variety of fresh foods are naturally low in SFA and TFA, and reduced-fat versions of whole foods (e.g. reduced-fat dairy foods, lean cuts of meat) are available.
References

References for which a URL is listed were last accessed on 1 January 2023.


