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CC:  
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Dear Dr. Schneeman and the Members of the 2020 Dietary Guidelines Advisory Committee:

CSPI submits the following comments to aid the 2020 Dietary Guidelines Advisory Committee (DGAC) in answering the following question: “What is the relationship between types of dietary fat consumed and risk of cardiovascular disease?”

The Center for Science in the Public Interest (CSPI) is a non-profit consumer education and advocacy organization that since 1971 has been working to improve the public’s health through better nutrition and food safety. CSPI helped to lead efforts to win passage of the Nutrition Labeling and Education Act, Healthy, Hunger-Free Kids Act (to improve school food), Food Safety Modernization Act, Menu Labeling, and Food Allergen Labeling and Consumer Protection Act. CSPI publishes Nutrition Action Healthletter (NAH) and is supported by the subscribers to NAH, individual donors, and foundation grants. CSPI is an independent organization that does not accept any government or corporate donations.

A key recommendation of the 2015 Dietary Guidelines for Americans (DGA) was to “[c]onsume less than 10 percent of calories per day from saturated fats.”\(^1\) The 2015 DGA also advised the public that “[s]trong and consistent evidence shows that replacing saturated fats with unsaturated fats, especially polyunsaturated fats, is associated with reduced blood levels of total cholesterol and of low-density lipoprotein-cholesterol (LDL-cholesterol). Additionally, strong and consistent evidence shows that replacing saturated fats with polyunsaturated fats is associated with a reduced risk of CVD events (heart attacks) and CVD-related deaths.”\(^2\)

The 2015 DGA translated that nutrient recommendation into advice about foods that are high in each type


of fat, and the DGA advised consumers that “the main sources of saturated fats in the U.S. diet include mixed dishes containing cheese, meat, or both, such as burgers, sandwiches, and tacos; pizza; rice, pasta, and grain dishes; and meat, poultry, and seafood dishes.” Moreover, the 2015 DGA encouraged the public to consume a healthy dietary pattern such as the DASH diet, which is not only low in saturated fat but “high in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, beans, and nuts and is low in sweets, sugar-sweetened beverages, and red meats.” We urge the 2020 DGAC to retain that sound advice, which is based on a body of evidence that has strengthened in recent years. Here we describe key elements of that evidence and note the flaws in some arguments against that advice.

RANDOMIZED CONTROLLED TRIALS (RCTs)

Replacing saturated fats with unsaturated fats lowers the risk of cardiovascular disease (CVD).

In 1962, an executive committee established with the support of the National Heart, Lung, and Blood Institute concluded that an RCT to test the impact of dietary fats on coronary heart disease (CHD) in middle-aged American men would require a population of up to 100,000 with a follow-up of 4 to 5 years to demonstrate a 20% change in rate. In 1971, a task force assembled at NHLBI’s request concluded that a National Diet-Heart Trial was not feasible, in part because intervention subjects might not adhere to the diet, control subjects might change their diets, and estimated costs ($500 million to $1 billion or more) would be formidable. Instead, the Institute decided to conduct a trial using diet, drugs, and/or other means to lower lipid levels.

As a result, researchers have conducted meta-analyses of smaller RCTs with CVD endpoints. Meta-analyses of these early trials have concluded that replacing saturated fats (SFA) with unsaturated fats, especially polyunsaturated fats (PUFA), lowers the risk of CVD. A 2015 review of 11 RCTs (including 13 comparisons) by the Cochrane Collaboration concluded that reducing dietary saturated fat significantly lowered the risk of cardiovascular events by 17% over a mean of 4.3 years. A subgroup of 7 trials that replaced SFA with PUFA showed a significant 27% reduction in the risk of CVD over 1.5 to 4.3 years. Although the reduction in mortality was not statistically significant for either reducing SFA or replacing SFA with PUFA, the Cochrane authors noted that “this perhaps was not surprising with mean trial durations of 4–5 years.” They concluded that “[l]ifestyle advice to all those with, or at risk of CVD and to lower-risk population groups should continue to include permanent reduction of dietary saturated fat and partial replacement by polyunsaturated fats.”

Similarly, a 2010 meta-analysis of 8 RCTs that replaced SFA with PUFA for at least 1 year reported a 19% reduced risk of CHD events. (On average, 10% of calories from SFA were replaced with PUFA.) CHD mortality dropped by 20%, but total mortality was not reduced. Likewise, a 2009 meta-analysis

6 NHLBI was called the National Heart Institute from 1948 to 1969 and the National Heart and Lung Institute from 1969 to 1976.
reported a 17% borderline significant reduction in total CHD events in 8 trials that replaced SFA with PUFA (Fig. 20).\textsuperscript{13} However, when the authors included only the 5 trials that reported a significant reduction in serum cholesterol levels, they found a 32% lower risk of CHD events and a 48% lower risk of fatal CHD, and a 20% lower risk of total mortality.

More recently, the expert panel that issued a Presidential Advisory from the American Heart Association examined four “core” RCTs that met six criteria: The trials compared high SFA with high PUFA intake; did not include trans unsaturated fat as a major component; controlled the dietary intake of the intervention and control groups; had at least 2 years of sustained intake of the assigned diets; proved adherence by objective biomarkers such as serum cholesterol or blood or tissue levels of polyunsaturated fatty acids; and collected and validated information on cardiovascular or coronary disease events.\textsuperscript{14} A meta-analysis of these core trials concluded that replacing SFA with PUFA over 4 to 8 years lowered the risk of CHD by 29%, similar to the reduction in risk achieved by treatment with statins.\textsuperscript{15} After considering the totality of the evidence—including prospective cohort studies, studies in nonhuman primates, and the impact of reducing SFA on LDL cholesterol—the Advisory stated that “we conclude strongly that lowering intake of saturated fat and replacing it with unsaturated fats, especially polyunsaturated fats, will lower the incidence of CVD.”

We note that some highly publicized meta-analyses have reached different conclusions concerning the impact of replacing SFA with PUFA on CHD and mortality. In particular, Chowdhury et al. concluded that “[c]urrent evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats.”\textsuperscript{16} However, that conclusion ignored, among other things, the results of its own meta-analysis (included in a supplement) showing a statistically significant 19 percent reduction in the risk of heart disease when the authors appropriately excluded the only trial that replaced SFA with a margarine high in trans fats. This and other flaws were noted in numerous letters to the editor.\textsuperscript{17} Other meta-analyses are not relevant, largely because they included RCTs that lowered all fats\textsuperscript{18} or because they only included RCTs that replaced SFA with n-6 PUFA\textsuperscript{19} (rather than n-6 and n-3 PUFA). Both also included the trial that replaced SFA with a margarine that was high in trans fats.

**LDL-cholesterol is a well-established surrogate for CVD risk.**

As the AHA advisory on dietary fats explains, wide-ranging and well-established evidence demonstrates that lowering LDL-cholesterol reduces the risk of atherosclerosis and CVD. As the advisory notes, that evidence includes “studies that compare populations that vary in LDL cholesterol; studies in single populations; genetic studies of high LDL cholesterol caused by mutations impairing the action of LDL receptors to remove LDL from the blood circulation and lower LDL cholesterol levels; studies of

\textsuperscript{15} Nine to 16 percent of calories from SFA were replaced with PUFA, depending on the trial.
mutations in numerous other genes that affect LDL cholesterol by other mechanisms; pharmacological studies that lower LDL cholesterol by decreasing cholesterol synthesis and increasing synthesis of LDL receptors by statins, decreasing cholesterol absorption, or inhibiting proprotein convertase subtilisin/kexin type 9 to increase LDL receptors; studies of mutations in genes that interfere with assembly of LDL and its precursor very-low-density lipoprotein (VLDL) in the liver that decrease the amounts that are secreted into the circulation; correlations between LDL cholesterol and CVD reduction in meta-analyses of randomized clinical trials of statin and other LDL cholesterol–lowering treatments; animal models that increase LDL cholesterol by diet or by genetic manipulation; and studies of the processes by which atherosclerosis starts, progresses, and regresses in arterial vessels and cells."^{20}

Similarly, in 2015, an expert panel of the National Lipid Association concluded that “(1) an elevated level of cholesterol carried by circulating apolipoprotein B-containing lipoproteins (non–high-density lipoprotein cholesterol and low-density lipoprotein cholesterol [LDL-C], termed atherogenic cholesterol) is a root cause of atherosclerosis, the key underlying process contributing to most clinical atherosclerotic cardiovascular disease (ASCVD) events; (2) reducing elevated levels of atherogenic cholesterol will lower ASCVD risk in proportion to the extent that atherogenic cholesterol is reduced."^{21} Similarly, in 2017 the European Atherosclerosis Society concluded that “[c]onsistent evidence from numerous and multiple different types of clinical and genetic studies unequivocally establishes that LDL causes ASCVD,“ or atherosclerotic cardiovascular disease.^{22}

The FDA accepts LDL-C lowering as a surrogate endpoint for cardiovascular events for health claims on foods and supplements.^{23} Lowering LDL-C with drugs and/or lifestyle is a prime target of clinical practice guidelines issued by the American Heart Association, American College of Cardiology, and other professional associations.^{24,25} In fact, the 2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk is based largely on RCTs with lipid and blood pressure endpoints. This advice has enormous public health importance given that one out of three U.S. adults have high LDL-C levels, and only one out of three adults with high LDL-C has the condition under control.^{26} Similarly, the DGAC should give considerable weight to the well-established evidence that replacing saturated fats with unsaturated fats lowers LDL-C.

HDL-cholesterol and triglycerides are not accepted as surrogates for heart disease risk.^{27} However, in the

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Sacks, 2017.


27 Food and Drug Administration, 2009.
RCTs cited below, replacing saturated fat with unsaturated fats either improved or did not worsen those components of the lipid profile. We did not consider LDL particle size, because it does not add information beyond that obtained by measuring LDL-C, triglyceride levels, and HDL-C.28 As the AHA advisory on dietary fats notes, “LDL size, per se, does not predict CVD in a multivariable analysis that includes triglycerides or LDL concentration” in a number of studies. Moreover, the advisory concludes that “this sparse set of findings suggests that replacement of saturated with monounsaturated or polyunsaturated fat reduces the concentration [of] all sizes of LDL.”29

Replacing saturated with unsaturated fats lowers LDL-cholesterol.

Based on the foregoing, the DGAC should give considerable weight to the well-established evidence that replacing SFA with unsaturated fats lowers LDL-C. Numerous randomized controlled feeding trials have tested the impact on LDL-C when SFA are replaced with PUFA, monounsaturated fats (MUFA), or carbohydrates. In 2016, the World Health Organization published a systematic review and regression analysis of 91 trials testing the effects of SFA on serum lipids and lipoproteins.30 Only studies with parallel or cross-over designs and with “thorough daily control of food intake” (as opposed to admonitions to follow a certain diet) were included. Dietary intervention periods had to last at least 13 days. The review concluded that LDL-C decreased by 2.1 mg/dL for each 1% of calories of SFA replaced with an equivalent amount of cis–PUFA and by 1.6 mg/dL for each 1% of calories of SFA replaced by an equivalent amount of cis-MUFA.31 Thus, replacing 5% of calories from SFA with PUFA would lower LDL by 10.6 mg/dL.

The 2013 Guideline on Lifestyle Management to Reduce Cardiovascular Risk issued by the American Heart Association and American College of Cardiology relied both on this extensive body of evidence and on key feeding trials that tested dietary patterns that are low in SFA.32 We will discuss that evidence in separate comments on dietary patterns. However, it is worth noting that the DASH (Dietary Approaches to Stop Hypertension) trial tested a “control diet” typically consumed by many Americans versus a “DASH diet,” which is rich in vegetables and fruits; includes whole grains, low-fat dairy products, poultry, fish, legumes, non-tropical vegetable oils, and nuts, and limits intake of sweets, sugar-sweetened beverages, and red meats.33 LDL-C was 10.7 mg/dL lower on the DASH diet (7% SFA) than on the control diet (14% SFA), although HDL-C was also 3.7 mg/dL lower on the DASH diet. As the OmniHeart trial demonstrated, replacing 10% of calories from carbohydrate in the original DASH diet with higher levels of PUFA and MUFA lowered triglycerides by 9.6 mg/dL and raised HDL by 1.1 mg/dL, leading to a 4% lower estimated 10-year coronary heart disease risk than the original DASH diet.34

In recent high-quality RCTs, SFA from dairy foods raise LDL-C.

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29 Sacks, 2017.
31 For each 1% of calories as SFA replaced with an equivalent amount of carbohydrates, LDL fell by 1.3 mg/dL. However, triglycerides increased by 1 mg/dL and HDL fell by 0.4 mg/dL, so the overall change is not favorable.
32 Eckel, 2014.
In recent years, numerous observational studies, largely funded by the dairy industry, have concluded that dairy fats do not increase the risk of heart disease. We address these observational studies below. However, we have identified several recent carefully controlled RCTs that report higher levels of LDL-cholesterol when people are fed higher levels of SFA from dairy foods rather than MUFA or PUFA. Unlike RCTs that give people dietary advice, these trials provided most of the food in the participants’ diets:

- **Bergeron et al., 2019.** Researchers randomly assigned 113 healthy men and women to 1 of 2 parallel arms (high-SFA or low-SFA) and within each arm, allocated them to diets with 12 percent of calories from one of three protein sources—red meat, white meat, or non-meat—for 4 weeks each. High SFA diets got 13-14% of calories from SFA, and low SFA diets got 7-8% of calories from SFA. In the low-SFA diets, SFA were largely replaced with MUFA. According to the authors, the “[d]ifferences in SFA content between the high- and low-SFA arms were achieved primarily by using high-fat dairy products and butter.” LDL-C was significantly higher on all three high-SFA diets (11.2 mg/dL higher on red meat, 8.9 mg/dL higher on white meat, 9.3 mg/dL higher on non-meat) than on the comparable low-SFA diets.

- **Chiu et al., 2017.** In a study on 53 men and women with predominantly small, dense LDL, participants were fed diets that were either high (18% of calories) or low (9% of calories) in SFA for 3 weeks. According to the authors, “[h]igh vs. low/non-fat dairy products were the major sources of differences in saturated fatty acid content,” and SFA were largely replaced by MUFA in the low-SFA diet. Compared to a baseline diet, LDL-C was 16.7% higher on the high-SFA diet and 8.7% lower on the low-SFA diet.

- **Mangravite et al., 2011.** Researchers fed 40 healthy men low-carbohydrate diets (31% of calories) that were either high (15% of calories) or low (8% of calories) in saturated fat for 3 weeks each in a cross-over design. Differences in saturated fat intake were achieved primarily through the use of full-fat dairy products,” and SFA were largely replaced by MUFA in the low-SFA diet. LDL-C was 13.9 mg/dL higher on the high-SFA than on the low-SFA diet. Further testing on a subset of 14 participants found that the high-SFA diet led to 33.5% higher levels of LDL apolipoprotein CIII, which may be particularly atherogenic.

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35 Studies by Chiu et al. (2017 and 2014) and Brassard et al. were partly funded by the dairy industry. The study by Mangravite et al. was partly funded by the beef industry. The study by Bergeron et al. was not industry funded.


37 All high-quality cross-over RCTs cited in this comment have adequate washout periods between diets.


39 Contrary to claims that high-SFA diets increase only large LDL particles but not small LDL particles, the high-SFA diet increased small LDL and led to non-significant differences in large or very small LDL particles. Moreover, most studies suggest that LDL size does not predict risk beyond predictions based on LDL-C, triglyceride levels, and HDL-C. See Institute of Medicine. *Evaluation of Biomarkers and Surrogate Endpoints in Chronic Disease.* Washington, DC: The National Academies Press. 2010:161. https://doi.org/10.17226/12869.


• **Chiu et al., 2014.** Unlike the studies discussed above, one study by the same group of researchers found no difference in LDL-C on low-SFA vs high-SFA diets. Researchers randomly assigned 158 overweight or obese men and women to either a baseline (control) diet or to 1 of 4 low-carbohydrate (35% of calories) diets that were either high or moderate in protein and high (15% of calories) or low (7% of calories) in SFA for 4 weeks. It is not clear why this study found no difference in LDL-C. A recent article suggested that SFA may have a greater impact on LDL-C in people who do not have obesity. The participants in Chiu et al., 2014 did have a higher mean BMI (33.9) than those in Chiu et al., 2017 (30.7 in low-SFA; 28.9 in high-SFA), Mangravite et al. (26.8), and Bergeron et al. (26.0). However, the recent study (which provided no food except butter or margarine) was based on a post-hoc analysis of subgroups, so it should be considered preliminary evidence that needs confirmation.

• **Brassard et al., 2017.** Researchers randomly assigned 92 men and women with abdominal obesity to diets rich in SFA (12.4-12.6% of calories) from either cheese or butter or to three diets that were low in SFA (5.8%) and rich in MUFA (19.6%), PUFA (11.5%), or carbs (58.9%) for 4 weeks each in a cross-over design. All foods were provided to the participants. The study reported a small difference in LDL-C on the butter (127.6 mg/dL) vs. cheese (123.4 mg/dL) diets. LDL-C was considerably higher on both butter and cheese than when the participants were given a diet high in PUFA (109.8 mg/dL) or MUFA (117.2 mg/dL). This large RCT is more informative than a meta-analysis funded by the dairy industry of 4 smaller RCTs, which reported an 8.5 mg/dL lower LDL-C on cheese than on butter. Note that only one of these RCTs had more than 22 subjects, and it did not control their diets beyond the provision of butter or cheese.

Overall, these studies make it clear that the SFA in full-fat dairy foods raise LDL-C, an atherogenic lipoprotein that is accepted as a surrogate for CVD risk. Although some dairy products may raise LDL more than others, those differences appear to be small, especially when compared to the far lower LDL levels seen in people who are fed diets high in PUFA or MUFA.

**Many RCTs testing dairy fat on LDL-C have serious limitations.**

Some RCTs, largely funded by the dairy industry, have suggested that SFA from dairy foods does not increase LDL-C. However, some of these studies have serious limitations. For example:

• **Raziani et al., 2016.** This study randomly assigned 164 men and women with at least two risk factors for the metabolic syndrome to consume regular cheese, reduced-fat cheese, or a no-cheese diet. However, unlike the studies mentioned above, Sundfør et al. did not provide most of the participants’ food, and the study still reported a higher LDL-C among obese participants who followed advice to eat a high-SFA diet (176.7 mg/dL) than those who followed advice to eat a low-SFA, high-PUFA diet (153.9 mg/dL).

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44 However, unlike the studies mentioned above, Sundfør et al. did not provide most of the participants’ food, and the study still reported a higher LDL-C among obese participants who followed advice to eat a high-SFA diet (176.7 mg/dL) than those who followed advice to eat a low-SFA, high-PUFA diet (153.9 mg/dL).
47 Studies by Raziani et al., Thorning et al., and Chiu et al. were partly or entirely funded by the dairy industry. The meta-analysis by Benatar et al., which had no funding to report, included studies with industry funding and studies with public funding.
carbohydrate control for 12 weeks using a parallel design.\textsuperscript{48} LDL was not significantly different among the three groups. However, as a letter to the editor noted, the study may have been underpowered to detect a difference.\textsuperscript{49}

- **Thorning et al., 2015.** Researchers randomly assigned 14 overweight postmenopausal women to diets that were high in cheese or meat, or to a high-carbohydrate control for 2 weeks each in a cross-over design.\textsuperscript{50} LDL-C did not differ on the three diets. However, as a letter to the editor pointed out, “[t]he cheese and meat diets were enriched with foods rich in PUFA such as nuts, canola oil, and sunflower oil, which are known to lower LDL cholesterol…”\textsuperscript{51}

- **Chiu et al., 2016.** Researchers randomly assigned 36 men and women to a “control diet” (16% of calories from SFA), a “standard DASH diet” (8% of calories from SFA), and a higher-fat, lower-carbohydrate version of the DASH diet with full-fat dairy products (14% of calories from SFA) for 3 weeks each in a cross-over design.\textsuperscript{52} LDL-C was significantly lower on the standard DASH diet (100.5 mg/dL) than on the control diet (108.7 mg/dL), while LDL on the higher-fat, lower-carb DASH diet (102.5 mg/dL) was not significantly different from the other two diets. The authors concluded that the higher-SFA DASH diet did not raise LDL-C. However, note that this study didn’t test the advice to replace SFA with MUFA or PUFA. It tested a lower-SFA, higher-carb diet against a higher-SFA, lower-carb diet. In other words, it tested advice to replace SFA with carbs (mostly from fruit juices), so it does not contradict the evidence behind advice to replace SFA with MUFA and PUFA.

- **Benatar et al., 2013.** This meta-analysis\textsuperscript{53} of nine RCTs is often cited\textsuperscript{54} as evidence that high-fat dairy foods do not raise LDL (or other CHD risk factors). The study concluded that “[t]here was no change in LDL cholesterol when whole fat dairy (+3.30, -4.30 to 10.90 mg/dL) or low-fat dairy (-1.42 , -4.74, to 1.91 mg/dL) food was increased.” However, the meta-analysis has numerous problems. For example, the results for “whole-fat dairy” included studies that encouraged people to consume either whole-fat or low-fat dairy. Of the six “whole fat” studies included in the LDL analysis (Fig. 6), one instructed participants to “consume four daily servings of dairy products (low-fat milk, low-fat cheese, and low-fat yogurt),”\textsuperscript{55} another “urged [participants] to use low-fat

alt="alternatives," and another used a test fat that was made of largely vaccenic acid-rich milk fat. Two of the other three studies did not specify fat levels. In addition, these RCTs tested advice to consume more dairy foods in self-selected diets, so they are not as well-controlled as RCTs that provide foods to participants.

These studies illustrate that many studies reporting no effect of full-fat dairy on LDL-C have limitations such as possible inadequate sample size and high-cheese diets enriched with LDL-lowering oils. Furthermore, some compare full-fat dairy foods with carbohydrates (rather than PUFA and MUFA), and others merely give advice (and sometimes imprecise advice) to consume full-fat dairy foods.

**Many RCTs are designed to find that red meat does not raise LDL-C.**

A number of RCTs, often funded by the meat industry, include design elements that make them more likely to find that red meats do not raise LDL-C.

For example, researchers fed 113 people in six groups diets rich in red meat, white meat, or non-meat protein—all with high-SFA or all with low-SFA levels—for 4 weeks each. As noted above, the high-SFA diets led to higher LDL-C levels than the low-SFA diets. However, for each level of SFA, LDL-C levels were no different between red or white meat diets, although they were lower after the non-meat diets. Unfortunately, a press release from the researchers’ university (and subsequent media reports) claimed that “red and white meats are equally bad for cholesterol.” In fact, it is no surprise that LDL-C levels did not differ, given that the red meat and white meat diets had equal levels of SFA. The researchers kept those levels equal by selecting only the leanest cuts of red meat. In reality, most varieties of red meat—especially ground beef—are higher in SFA than the cuts used in these studies.

Since the mid-1990s, studies have reported similar effects on LDL-C in RCTs that have pitted very lean cuts of red meat against white meat. These studies do not demonstrate that red meat is heart-healthy or

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60 Bergeron, 2019. This study was publicly funded.
61 Plant proteins may lead to lower levels of LDL-C due to soluble fiber, phytosterols, or other LDL-C lowering constituents. However, those findings do not negate the impact of SFAs on LDL-C.
exonerate the SFA in red meat as atherogenic. Furthermore, a growing body of evidence suggests that red meat may increase the risk of heart disease because it leads to an increase in TMAO in the body. (In addition, red meat is linked to a higher risk of certain cancers.)

PROSPECTIVE COHORT STUDIES

In prospective observational studies, replacing saturated with unsaturated fats is consistently associated with a lower risk of CVD.

Numerous meta-analyses of prospective cohort studies have examined the association between saturated fat and CVD. Unfortunately, some of the meta-analyses that have gotten the most publicity failed to compare diets high in saturated vs. unsaturated fat. This comparison is critical because studies that merely look at the association between various levels of SFA intake and CVD risk are in effect comparing a diet high in SFA to the typical U.S. diet, which is high in refined carbohydrates from grains, desserts, and sugar-sweetened beverages, which also may increase the risk of CVD.

In contrast, a pooled analysis that compared diets high in SFA to diets high in unsaturated fats reported that SFA are linked to an increased risk of CHD, consistent with findings from RCTs. The Presidential Advisory from the American Heart Association concluded that replacing 5% of calories from SFA with PUFA, MUFA, or carbohydrates from whole grains was associated with a 25%, 15%, and 9% lower risk of CHD, respectively, while replacing SFA with carbohydrates from refined starches and added sugars was not significantly associated with CHD risk. These estimates were based on a prospective cohort study that tracked roughly 84,00 women for 30 years and 43,000 men for 24 years. Furthermore, an analysis of the same cohorts reported that replacing 5% of calories from SFA with 5% of calories from PUFA was associated with significantly lower rates of total deaths as well as deaths from CVD, cancer, and neurodegenerative disease.

Prospective observational studies do not demonstrate that dairy fat has no impact on CVD.

Meta-analyses and systematic reviews of prospective cohort studies, often funded by the dairy industry, have reported that dairy foods or high-fat dairy foods are not associated with an increased risk of CVD.

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69 Sacks, 2017.
71 Wang DD, et al. Association of Specific Dietary Fats with Total and Cause-Specific Mortality. JAMA Intern Med. 2016;176(8):1134-45. Replacing 5% of calories from SFAs with MUFAs was associated with a significantly reduced risk of total mortality and neurodegenerative disease mortality.
However, few observational studies compare dairy foods with foods that are rich in unsaturated fat, which is the appropriate comparison to evaluate advice to replace SFA with unsaturated fats. Chen and colleagues made that appropriate comparison in three cohorts that tracked roughly 222,000 U.S. adults for 20 to 32 years.\textsuperscript{73} Every 5\% of calories from dairy fat that was replaced with 5\% of calories from PUFA was associated with a 24\% lower risk of CVD, a 26\% lower risk of CHD, and a 22\% lower risk of stroke. These findings are consistent with evidence from RCTs and observational studies on total SFA.

Some observational studies have used odd-chain fatty acids as biomarkers for dairy fat intake. However, the associations with CVD are inconsistent.\textsuperscript{74} Furthermore, 15:0 and 17:0 are not ideal biomarkers of dairy fat intake because they can be synthesized in humans;\textsuperscript{75} they occur in fish;\textsuperscript{76} and they may be raised by consuming some types of fiber.\textsuperscript{77} Moreover, like other observational studies, those using biomarkers are subject to residual confounding. Some studies suggest that higher levels of odd-chain SFA are associated with a higher intake of fruits, vegetables, and nuts, and a lower intake of red and processed meats and soft drinks.\textsuperscript{78}

In any case, the findings from observational studies do not negate the findings from RCTs demonstrating that dairy fats raise LDL-C. The DGAC should continue to recommend limits on SFA intake, including dairy fats, based on the totality of evidence from both RCTs and observational studies.

CONCLUSION

In summary, we urge the DGAC to retain the 2015 advice to limit SFA to less than 10 percent of calories and to replace foods that are high in SFA with foods that are high in PUFA and MUFA. That advice is backed by compelling evidence from RCTs with both CVD and LDL-C endpoints and from observational studies that make appropriate comparisons with foods rich in PUFA and MUFA. Recent well-controlled, adequately powered RCTs demonstrate that full-fat dairy products raise LDL-C when pitted against foods rich in PUFA and MUFA. RCTs that report no increase in LDL-C on diets rich in red meat often use very lean meats that are not typical. Reversing the long-standing DGA advice to limit SFA to less than 10 percent of calories would contradict the strongest scientific evidence on dietary fats and CVD.

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and Drouin-Chartier et al. were partly or entirely funded by the dairy industry. The systematic review and meta-analysis by Soedamah-Muthu et al. did not report any industry funding.


