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May 27, 2015

Tina Namian, Branch Chief
Policy and Program Development, USDA
RE: Docket ID FNS-2011-0029

Dear Ms. Namian:

The board of the Weston A. Price Foundation (WAPF), representing over fifteen thousand active members, respectfully submits the following comments on the Proposed Rules for the Child and Adult Care Feeding Program (CACFP or Program). These comments have been prepared by registered dietitians in conjunction with the WAPF board. CACFP regulates meals served to 3.67 million children and adults, and while older adults are also a vulnerable population in regards to nutritional intake, our comments will concern only proposed rule changes to the infant and childcare feeding programs.

According to the USDA's Proposed Rules (Federal Register, pp. 2037-2060) (herein referred to as "the proposed rules"), "[i]mplementation of this proposed rule would *serve as a step towards more nutritious meals that improve the dietary habits of participants in day care* (emphasis ours) (Federal Register, p. 2037). The Healthy Hunger-Free Kids Act of 2010 (HHFKA) "redefined the purpose of the CACFP...for provision of nutritious foods that *contribute to the wellness, healthy growth, and development of young children...*(emphasis ours) (Federal Register, p. 2038). We wholeheartedly concur that the fundamental purpose for providing guidelines for feeding young children must be to promote growth, development, and overall wellness. However, we do not agree with the rule that only 1 percent or fat-free milk, or fat-free flavored milk, be served to children two and older. This unscientific rule cannot and will not support those nutrition objectives and represents a step backwards.

The proposed CACFP meal pattern revision will align the CACFP with the following 2010 Dietary Guidelines, guidelines that apply to healthy individuals age 2 years and older:

1. Consume less than 300 mg per day of dietary cholesterol;
2. Consume less than 10 percent of calories from saturated fatty acids by replacing them with monounsaturated and polyunsaturated fatty acids;
3. Reduce the intake of calories from solid fats;
4. Increase intake of fat-free or low-fat milk and milk products.

In addition, the rule may be based on the *belief* that reducing the fat content of the milk provided will assist with overall caloric reduction and healthy weight management in the children served. We will show why none of these reasons stands up to scrutiny when the evidence base is examined. We will also show why whole milk is nutritionally superior to 1 percent and fat-free milk.

To understand the rationale behind the rule to serve only fat-free or 1 percent milk, an understanding of the Congressional mandate for the CACFP along with the actions preceding its enactment is needed. In 1984, a Consensus Conference by the National Heart, Lung, and Blood Institute “adopted the McGovern dietary goals for Americans and unexpectedly extended coverage from adults to all persons older than 2 years. This was the first time children were included *on the basis of studies conducted in adults*, and it was done *without any new scientific data*. This announcement by the Consensus Conference provoked a storm of criticism to the effect that children are not at risk for atherosclerosis and *should be exempted from this advice for adults*” (emphasis ours) (Olsen, 2000). Criticism came from the American Academy of Pediatrics (AAP) which argued that “dietary guidelines developed for adults should not be applied to children,” (Olsen, 2000) as well as from independent researchers.

Despite the scientifically unsupported extension of adult dietary goals to children, federal law requires the USDA to “update the CACFP meal patterns to ensure that meals are consistent with the goals of the most recent Dietary Guidelines for Americans (DGA) and promote the health of the populations served by the most recent and relevant nutrition science and appropriate authoritative scientific agency and organizations recommendations” (Federal Register, pp. 2039-2040). For the current update, the USDA utilized a report generated by the Institute of Medicine in November 2010 (Institute of Medicine, 2010) following the issuance of the 2010 DGAC Report, but prior to the issuance of the 2010 Dietary Guidelines for Americans. In the IOM Report, the IOM cited “[e]xamples of food specifications that show alignment with the *Dietary Guidelines* include the following: *Low-fat milk products*: Milk and yogurt will be nonfat or 1 percent fat; the use of low-fat cheese is encouraged; *Decreasing the intakes of saturated fat and added sugars*: Specifications limit milk fat and flavored milk” (IOM, 2010, pp. 172-173).

The recently issued 2015 Dietary Guidelines Advisory Committee Report (DGAC Report) (DGAC, 2015), while not making any definitive recommendations on the overall intake of fat, acknowledged that “low-fat diets induce dyslipidemia” (DGAC 2015 meeting 5), stating in the final DGAC Report that “dietary advice should put emphasis on optimizing types of dietary fat and not reducing total fat” (DGAC Report, p. Part D. Ch 6. line 460). Yet, in a summary statement, the DGAC Report stated: “the overall body of evidence examined by the 2015 DGAC identifies that a healthy dietary pattern is higher in...low- or non-fat dairy...” The DGAC also took the step of removing limits on dietary cholesterol, limits that the data have never supported and which have contributed to inadequate intakes of choline, among other nutrients; it is reasonable to expect that the final 2015 DGA will reflect this change.

If the final 2015 DGA reflect the DGAC Report recommendations (as previous DGA editions have), neither an overall reduction of fat nor limits on dietary cholesterol should be a requirement of the CACFP in order to align this program with the upcoming DGA. Therefore, the goal of reducing dietary cholesterol or reducing the overall intake of fat can be eliminated as reasons for serving fat-free or 1 percent milk. Reliance on an outdated IOM report that almost certainly will not be in complete alignment with the next iteration of the DGA is unwise and inopportune. We urge caution in doing so, and advise waiting for the release of the 2015 DGA at minimum to issue a final rule on the CACFP.

If adherence to the DGA is the foundation of the CACFP rules, the only potential concern that could remain about serving whole milk would be exceeding the 10 percent of calories from saturated fat – a result of not reducing the intake of “solid fats.” Aside from this, it may be that an unfounded belief persists that the addi-

tional fat in the whole milk potentially contributes to overweight/obesity in children (Scharf, et al., 2013). We will demonstrate why neither saturated fat nor additional calories from whole milk are valid concerns, and why whole milk is nutritionally superior for children.

WHOLE MILK IS NOT ASSOCIATED WITH OVERWEIGHT IN CHILDREN

According to USDA educational materials directed to parents for their children: “[d]rinking lowfat milk is one way to get less fat, especially saturated fat.” (USDA FNS) Presumably, the directive to provide only lowfat or nonfat milk to CACFP participants is following similar faulty reasoning. Guidance to reduce total fat is not evidenced-based and in fact, it is now widely recommended that refined carbohydrates be replaced with calories from fat at all ages (DGAC, 2015, Part D: Ch 6, p 13). Because dietary fat consumption by American children actually declined between 1984 and 2004, “it seems unlikely that increased fat consumption is responsible for the large increase in pediatric obesity” (Slyper, 2004). Furthermore, the Bogalusa Heart Study demonstrated that “children who occupied the lowest percentiles of fat consumption had increased their intake of simple sugars” (Slyper, 2004).

According to Ludwig and Willet, “[I]acking high-quality interventional data, beverage guidelines presume that the lower calorie count of reduced-fat milk will decrease total caloric intake and excessive weight gain. However, a primary focus on reducing fat intake does not facilitate weight loss compared with other dietary strategies, as shown in observational studies and clinical trials, perhaps because reduced-fat foods tend to have lower satiety value” (Ludwig S, Willett WC, 2013). In an interview for *Time Magazine*, Dr. Ludwig, the John Fielding Crigler, Jr. & Mary Adele Sippel Crigler Chair in Pediatric Endocrinology and Professor of Nutrition Harvard School of Public Health, summarized their commentary, “[s]omehow this low-fat milk has become so entrenched in the nutritional psyche, it persists despite the absence of evidence. To the contrary, the evidence that now exists suggests an adverse effect of reduced-fat milk” (Sifferlin, 2013).

Dr. Ludwig was referring to evidence from prospective studies that show that children (Scharf, et al., 2013) and adolescents (Berkey, et al., 2005) experience greater rates of weight gain with the consumption of reduced-fat compared to whole milk. Using data from the Early Childhood Longitudinal Study, a representative sample of 10,700 US preschoolers, Scharf, et al. found that: “children drinking 1% skim milk at both 2 and 4 years were more likely to become overweight/obese between [those] 2 time points,” and reviewing the totality of the evidence concluded that there is a need for “weight-targeted recommendations with a stronger evidence base.” Using data from the Growing Up Today Study, a cohort study of more than 10,000 US children ages 9 through 14 years, Berkey, et al. found that “skim and 1% milk appeared more strongly linked to weight gain than whole or 2% milk.”

In conclusion, any concerns about the increased fat (and/or calories) and the contribution to increased body weight from the consumption whole milk are without merit.

SATURATED FAT IS NOT A CONCERN

The WAPF recently submitted comments regarding the 2015 DGAC Report in which we detailed the evidence conclusively dismissing animal foods high in saturated fat as contributors to cardiovascular disease. We reiterate that evidence at the end of this document, but first want to make you aware of comments on the DGAC Report submitted by the Academy of Nutrition and Dietetics (The Academy). The Academy states: “In the spirit of the 2015 DGAC’s commendable revision of previous DGAC recommendations to limit dietary cholesterol, the Academy suggests that the HHS and USDA support a similar revision *deemphasizing saturated fat as a nutrient of concern*. While the body of research linking saturated fat intake to the modulation of LDL and other circulating lipoprotein concentrations is significant, this evidence is *essentially irrelevant* to the question of the relationship between diet and heart disease” (emphasis ours) (Academy of Nutrition and Dietetics, 2015). We concur with the Academy’s “suggestion” and urge that you give it your full consideration. Saturated fat is

not a nutrient of concern and as we will explain, foods rich in saturated fat, including whole milk, are sources of valuable and often shortfall nutrients.

BENEFITS OF WHOLE MILK – CENTRAL NERVOUS SYSTEM DEVELOPMENT

Wisely, the CACFP in alliance with the American Academy of Pediatrics and the IOM continues to recommend whole milk for children ages 1-2 years, following cessation of bottle or breast feeding. According to Frank R. Greer, chairman of the AAP's nutrition committee, “[e]ighty percent of brain development occurs in the first two years of life, so you want the fat at that point” (Hannon K, 2009). However, most physicians and researchers now recognize the fact that the brain continues to develop into the adolescent years, and in fact, accumulating data demonstrate continued brain development well into the 3rd decade of life (Lebel C, Beaulieu C, 2011). The corpus callosum, the major connection between the cerebral hemispheres, undergoes its most rapid expansion of cortical capabilities in childhood and early adolescence (Keshavan, et al., 2002). It is common for psychiatric disorders to emerge during late adolescence and young adulthood, presumably due to abnormal brain development (Lebel C, Beaulieu C, 2011; Paus T, Keshavan M, Giedd JN, 2008). Thus, optimal nutrition is critical through the preschool, adolescent, teen and early adult years to ensure normal brain development.

BENEFITS OF WHOLE MILK – GASTROINTESTINAL HEALTH

Butterfat contains glycosphingolipids, a special category of fatty acids that protect against gastro-intestinal infection, especially in the very young and the elderly. For this reason, children who drink nonfat milk have diarrhea at rates three to five times greater than children who drink whole milk (Koopman, et al., 1984).

BENEFITS OF WHOLE MILK – THE ROLE OF CHOLESTEROL IN THE BODY

As discussed above, limits on dietary cholesterol are not evidence-based and it is expected that these limits will be formally rescinded in the upcoming 2015 DGA. Cholesterol is a vital substance for growth and development in children; cholesterol is essential for the normal structure of sterol-rich membranes, notably myelin; proper function of serotonin, oxytocin, and other brain neurotransmitter transporters and receptors; and for the synthesis and metabolism of sterols, including neuroactive steroids, a deficit of which is associated with anxiety and mood disorders (Aneja A, Tierney E, 2008). Clearly, cholesterol is not a substance to be vilified, but a nutrient critical to optimal central nervous system functioning. Of note, there is a high prevalence of abnormally low serum cholesterol in the autism spectrum population (Aneja A, Tierney E, 2008). These authors and others have suggested that cholesterol supplementation should be considered as a “helpful treatment approach while awaiting an improved understanding of cholesterol metabolism and ASD” (Aneja A, Tierney E, 2008). Whole milk, of course, is a rich source of cholesterol.

BENEFITS OF WHOLE MILK – VITAMIN E

Vitamin E is a shortfall nutrient for Americans (DGAC, 2015). Data included in the IOM Report (IOM, 2010) indicates that vitamin E (α -tocopherol) targets are not being met for children in CACFP; for example, for children 2-4 years of age, the target is 1.4 mg, the amount supplied by meals (averaged over a 5-day week) is 0.40 mg, a 72 percent shortfall; two 8-ounce servings of whole milk supply 0.30 mg of α -tocopherol, compared to only 0.04 supplied by non-fat or 1% milk; the 0.30 mg in 16 ounces of whole milk would almost double the vitamin E intake. Other sources of full-fat dairy such as butter and whole-milk cheese could close the gap. According to the USDA Nutrient database (USDA), butter supplies 0.12 mg α -tocopherol per pat, and 1 slice of full-fat cheddar cheese supplies 0.15 mg α -tocopherol. Thus, if a preschooler's diet included 2 glasses of whole milk (vs. skim or non-fat), 2 pats of butter, and 1 slice of natural cheddar cheese, this would supply an additional 0.69 mg of α -tocopherol in the daily diet, bringing the total α -tocopherol to 1.09 mg, much closer to the target of 1.4 mg. One whole egg served daily could provide another 0.60 mg, now exceeding the target at 1.69 mg.

ALLOWING FLAVORED (I.E. SWEETENED) FAT-FREE ONLY MILK TO BE SERVED WITHOUT RESTRICTION IS INADVISABLE

This is not advisable for three important reasons:

1. As demonstrated above, full-fat milk is nutritionally superior to low-fat or nonfat milk;
2. “Intake of calorically-sweetened beverages is positively related to adiposity in children” (Academy of Nutrition and Dietetics, 2006);
3. The 2015 DGAC Report recommends an overall reduction in sugar including in beverages: “[i]mproved beverage selections that limit or remove sugar-sweetened beverages and place limits on sweets and desserts would help lower intakes of the food component, added sugars” (DGAC, 2015). Flavored milk contains sugar, and is an unacceptable substitution for whole unflavored milk. The American Academy of Pediatrics (AAP) recommends that pediatricians “educate parents about flavored milk and that it contains sugar and is not equal to plain lowfat milk” (American Academy of Pediatrics, 2015).

According to Ludwig and Willett, noted pediatric obesity and nutrition researchers, “there has been a marked increase in the proportion of sweetened milk consumption in recent years. This trend may reflect, to some degree, compensation for the lower palatability and satiety value of fat-reduced milk. However, the substitution of sweetened reduced-fat milk for unsweetened whole milk – which lowers saturated fat by 3 g but increases sugar by 13 g per cup – clearly undermines diet quality, especially in a population with excessive sugar consumption.” According to the USDA Nutrient Database, 1% lowfat sweetened milk contains 28 g of sugar per 8 ounces, while whole milk (3.25% fat) contains 12 grams of naturally occurring sugars per 8 ounces, a difference of 16 g per cup (USDA). Even with the proposed rules’ limit of 22 grams of sugar per 8 ounces for flavored milk, this is still an increase of 10 grams of added sugar per cup.

The caloric difference between whole milk and fat-free flavored/sweetened milk is estimated to be about *20 calories more in the fat-free flavored than in the whole milk*. It was difficult to locate a fat-free flavored milk, and in fact the USDA Nutrient Database did not include it; therefore, we computed the caloric difference between unflavored lowfat and unflavored nonfat milk and subtracted that difference (19 calories) from flavored lowfat milk containing 188 calories per cup, to yield an estimate of 169 calories in flavored nonfat milk as compared to 149 calories in whole milk.

In conclusion, the evidence does not support the serving of 1 percent, nonfat, or flavored milk - nonfat or otherwise. Whole (unflavored) milk should be served to all children 1 year of age and older in the CACFP, unless medically contraindicated.

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ADDENDUM: WAPF COMMENTS ON THE 2015 DGAC REPORT

The following is excerpted from the Weston A. Price Foundation Comments re: the 2015 DGAC Report submitted electronically (#28781) on May 8th, 2015, to the secretaries of Agriculture and Health and Human Services (see <http://www.westonaprice.org/wp-content/uploads/DGAC2015GuidelinesWAPFComments.pdf> for the full report and references):

While acknowledging that replacing SFA with carbohydrate or MUFA is “not effective in reducing risk of CVD,”⁷ the DGAC attempts to summarize the studies on replacing SFA with PUFAs in order to answer the question: “What is the relationship between intake of saturated fat and risk of cardiovascular disease?” In their summary, they draw two conclusions:

1. “Strong and consistent evidence from RCTs shows that replacing SFA with unsaturated fats, especially PUFA, significantly reduced total and LDL-cholesterol.”
2. “For every 1 percent of energy intake from SFA replaced with PUFA, incidence of CHD is reduced by 2 to 3 percent.”

Then the DGAC makes the recommendation that: “Sources of saturated fat should be replaced with unsaturated fat, particularly polyunsaturated fatty acids.”

CONCLUSION 1

RCTs show increased PUFA intake reduces LDL-C, but

- Does this reduction result in improved outcomes?
- Is an increase in PUFA intake proven to be safe?

EFFECT ON OUTCOMES – DOES INCREASED PUFA TRANSLATE TO BETTER HEALTH?

The effect of replacing SFA with PUFA to lower total and LDL-cholesterol is assumed to be beneficial overall; the Report cites a meta-analysis of trials that found that 1% replacement of SFA with an equal amount of PUFA resulted in a reduction of 1.8 mg/dL of LDL-cholesterol, while at the same time lowering HDL-cholesterol by 0.2 mg/dL. But do these surrogate outcomes from dietary changes translate into actual reductions in morbidity or mortality? Not according to a 2010 Institute of Medicine report, which found that “data supports use of LDL as a surrogate endpoint for some cardiovascular outcomes for statin drug interventions, but not for all cardiovascular outcomes or other cardiovascular interventions, *foods*, or supplements” (emphasis ours). In their 2014 position paper on dietary fats, the Academy of Nutrition and Dietetics stated “despite documented influence of saturated fat on surrogate disease markers, the effect of saturated fat intake on disease end points is not clear.”

RECOMMENDING AN INCREASED PUFA INTAKE IS DANGEROUS.

While very small amounts of unsaturated omega-3 and omega-6 fatty acids are essential for health, the numerous adverse effects of higher intakes of PUFAs are now widely recognized.

It has been established that in atherogenesis, PUFAs are the components in circulating LDL-cholesterol that are oxidized, and as a result, generate antigenic substances that are recognized by immune cells for clearance of oxidized LDL. In fact, a direct association between PUFA intake and luminal narrowing in women with CHD has been observed.

Commentary published in 2014 in the Mayo Clinic Proceedings by Ravnskov et al. summarized the numerous studies warning against increased omega-6 PUFA intakes in humans. We urge you to review this commentary. The authors cite evidence going back as far as the early 1960s that replacement of SFAs by higher intakes of

omega-6 PUFAs (largely coming from industrial seed oils) is associated with increased risks of stroke, cancer (especially breast cancer) and overall mortality; and with suppression of HDL-cholesterol and immune system function.

In summary, Ravnskov et al. conclude “[t]o exchange SFAs with PUFAs is not a wise decision” while reasonably hypothesizing that “the current epidemics of obesity, metabolic syndrome, and type 2 diabetes that started shortly afterward [the recommendations 35 years ago to exchange carbohydrates for dietary SFAs] may be an effect of this diet.” Clearly, benefit has not been realized and it is very probable that the American public has suffered great harm from this recommendation.

NEW CONCLUSIONS FROM AN UPDATED META-ANALYSIS ON INCREASED PUFA INTAKE

Ramsden et al. recovered and re-evaluated data from the Sydney Diet Heart Study where men with recent coronary events were randomized to an intervention diet that replaced saturated fats with linoleic acid from safflower oil, or to a control diet with no specific instruction. Using intention-to-treat survival analysis, they showed that in the Sydney Study, compared with the control group, the intervention group had significantly *increased* risks in all-cause mortality (62%), cardiovascular mortality (70%), and mortality from CHD (74%). When the recovered data were used to update their ongoing meta-analysis of selectively increased linoleic acid intervention trials, the updated ME showed nonsignificant trends toward increased risks for death from CHD (+33%) and cardiovascular disease (+27%), *despite a significant reduction in total cholesterol*. In their 2013 publication, the authors point out that there is currently no clinical trial evidence indicating that replacing SFAs with n-6 linoleic acid, without a concurrent increase in n-3 PUFAs, lowers the risk of CVD or death. In a 2012 study, these same authors showed that lowering n-6 LA in human diets for twelve weeks reduced oxidized LA metabolites, the most abundant oxidized fatty acids in oxidized LDL.

The 2015 DGAC Report itself raises questions about the known safety and efficacy of its recommendation to replace SFA with PUFAs. It states as two “Needs for Future Research:”

1. Examine lipid and metabolic effects of specific oils modified to have different fatty acid profiles (e.g. commodity soy oil [high linoleic acid] vs. high oleic soy oil).
Rationale: As more modified vegetable oils become commercially available, it is important to assess their long-term health effects. In addition, future studies should examine lipid and metabolic effects of plant oils that contain a mix of n-9, n-6, and n-3 fatty acids, as a replacement for animal fat, on cardiovascular disease risk factors.
2. Examine the effects of saturated fat from different sources, including animal products (e.g. butter, lard), plant (e.g., palm vs. coconut oils), and production systems (e.g. refined deodorized bleached vs. virgin coconut oil) on blood lipids and cardiovascular disease risk.
Rationale: Different sources of saturated fat contain different fatty acid profiles and thus, may result in different lipid and metabolic effects. In addition, virgin and refined coconut oils have different effects in animal models, but human data are lacking.

By recommending Americans simultaneously reduce their refined carbohydrate intake, reduce their intake of foods containing saturated fat, and increase their intake of foods high in unsaturated fats including processed vegetable oils, the result will be an increasingly greater intake of linoleic acid. Although unintended, the inevitable consequence will be even higher rates of CVD and other chronic diseases. Significantly, no population studied has consumed large quantities of PUFAs for extended periods of time, therefore we lack any demonstration that high intakes of linoleic acid are safe.

CONCLUSION 2

Replacing SFA with PUFA reduces incidence of CHD, but

- What does the literature actually say?
- What are the reasons for the continued debate among scientists?

The literature relied upon in the Report to draw this conclusion is incomplete and misinterpreted, and as we stated previously, the DGAC did not conduct their own evidence analysis for this Report. Before addressing these deficiencies, we must reconsider the mistakes of the past that have led us in the wrong direction. The earliest editions of the DGAs recommended an increased consumption of carbohydrates and a lower intake of fat, saturated fatty acids (SFA) and cholesterol. The 2015 Report has wisely acknowledged: “simply reducing SFA or total fat in the diet by replacing it with any type of carbohydrates is not effective in reducing risk of CVD.” And yet, knowing the abject failure of that dietary experiment, the DGAC chooses to ignore conflicting data on the impact of increased omega-6 PUFA intake on incidence of CHD. We offer a more in-depth analysis of the literature cited in the Report, as follows:

- According to the Report, “Hooper et al.’s 2012 Cochrane MA of trials involving SFA reduction/modification found that reducing SFA by reducing and/or modifying dietary fat reduced the risk of cardiovascular events by 14 percent.”²² Yet, according to Hooper et al.: “*removing studies with a systematic difference in care between the intervention and control arms, or removing studies with dietary differences other than dietary fat differences both removed the statistical significance of the effect*” (emphasis ours), and further: “Dietary fat intervention reduced cardiovascular events in men, but not in women or in combined studies of men and women, and studies in community settings reduced events, but those in residential institutions did not. Studies published in the 1960s, and in the 1990s, reduced cardiovascular events significantly, but not studies published in other decades.” Clearly, the DGAC cherry-picked from the results to reinforce their pre-drawn conclusions on SFA reduction.
- According to the Report: “Mozaffarian et al., 2010 found in a MA of eight trials (13,614 participants with 1,042 CHD events) that modifying fat reduced the risk of myocardial infarction or coronary heart disease death (combined) by 19 percent, ...corresponding to 10 percent reduced CHD risk...for each 5 percent energy of increased PUFA”. However, Mozaffarian et al. “excluded the trial of Rose et al. and the Sydney trial (reviewed above), both of which resulted in a higher mortality in the treatment group. Mozaffarian’s meta-analysis is “also in conflict with the results from a recent report of 4 unsuccessful trials in which SFAs were exchanged with omega-6 PUFAs only.”
- According to the Report: “Farvid et al., 2014 conducted an SR and MA of prospective cohort studies of dietary linoleic acid (LA), which included 13 studies with 310,602 individuals and 12,479 total CHD events (5,882 CHD deaths). Farvid et al. found dietary LA intake is inversely associated with CHD risk in a dose-response manner: when comparing the highest to the lowest category of intake, LA was associated with a 15 percent lower risk of CHD and a 21% lower risk of CHD deaths... A 5 percent of energy increment in LA intake replacing energy from SFA intake was associated with a 9 percent lower risk of CHD events ...and a 13 percent lower risk of CHD deaths.” However, in the Farvid study over half of the data is from unpublished sources; there is no way to guarantee the quality of data that has not undergone the rigors of the peer-review process. Out of the 14 cohorts analyzed, in the data analysis of 11 of the cohorts, comparing highest to lowest intakes of LA, the confidence interval crosses 1, implying that there is no difference between the two groups being compared. The only reason an effect is seen overall is due to the fact that the three studies which show an effect are given over 62% of the weight in the weighted analysis. In other words, instead of conducting their own NEL analysis, the DGAC chose a highly flawed and inconclusive study upon which to base their conclusion.
- Curiously, the Report dismissed the findings of a meta-analysis conducted by Chowdhury et al., stating, “there was no significant association between LA intake and CHD risk, but the analysis was based on a limited number of prospective cohort studies.” This seems to contradict the methodology utilized to include the Mozaffarian and the Farvid meta-analyses, which also did not consider the full breadth of the literature.

- According to the Report: “In Jakobsen et al.’s 2009 pooled analysis of 11 cohorts (344,696 persons with 5,249 coronary events and 2,155 coronary deaths), a 5 percent lower energy intake from SFAs and a concomitant higher energy intake from PUFAs reduced risk of coronary events by 13 percent...and coronary deaths by 16 percent.”²⁵ The overall association is carried solely by the associations found for women under the age of 60. Among women over 60, no significant association was found between substitution of PUFA for SFA with regard to coronary events or coronary deaths. Among adult men of any age, there was no significant association between substitution of PUFAs and risk of coronary events or coronary deaths. For women under the age of 60 a slim inverse association was found between risk of coronary events and the substitution of PUFAs for SFA. Only in this group was there an inverse association between risk of coronary death and the substitution of PUFAs for SFA. They also found a positive association between substitution of MUFA or CHO for SFA and risk of coronary events, but not risk of coronary deaths. In addition, Jakobsen et al.. excluded more than a dozen cohort studies which reported no difference in SFA intake between people with and without CHD, and they ignored the cohort studies of patients with stroke.

In their 2013 review of observational studies, Schwingshackl and Hoffman concluded, and we concur: “The observational literature is not consistent regarding self-reported SFA intakes and risk of CVD/coronary heart disease. Recent studies have shown that SFA intakes were not associated with changes in coronary heart disease, stroke or CVD frequencies although in one of these trials, increased MUFA intake was associated with a significantly lower risk of coronary heart disease. The interventional literature likewise is inconsistent and therefore, no reliable conclusions can be drawn at this time.

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