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May 8, 2015

The Honorable Tom Vilsack
Secretary of Agriculture
1400 Independence Avenue, SW
Washington, DC 20250

The Honorable Sylvia Mathews Burwell
Secretary of Health and Human Services
200 Independence Avenue, SW
Washington, DC 20201

Dear Sirs/Madams:

The board and the members of the Weston A. Price Foundation enthusiastically support the United States Government's ambitious goal of creating an American "Culture of Health." Over the past fifteen years, our now almost 15,000 members have dedicated themselves to improving not only their own health, but the health of their families, their communities, and the environment through the acquisition and dissemination of accurate nutrition information.

Our exhaustive review of the Scientific Report of the 2015 Dietary Guidelines Committee (the Report) has revealed several limitations in the recommendations, recommendations that will be used to inform and direct the upcoming revised Dietary Guidelines for Americans. These limitations are a result of focusing on the goal of reducing the risk of chronic disease, while relegating the goal of meeting nutrient requirements to a distant second. Our commentary and analysis will show that neither goal will be met, due to the lack of evidence that the recommended dietary approaches actually reduce rates of chronic disease and the fact that these approaches lead to a compromised intake of several essential nutrients.

As concerned citizens and as health and nutrition professionals, we have the obligation to make you aware of these limitations by providing scientific evidence that may have been overlooked or misinterpreted. We offer this in the spirit of assisting your agencies with the monumental task of directing Americans in making better dietary choices for themselves and their children. We trust you will act upon this information and use it to the benefit of the American public.

We are focusing on six general areas of concern in the Report:

1. The implied limits on dietary fat with specific limits on dietary saturated fat;
2. The failure to recommend land animal proteins and the specific recommendation to limit red meat;
3. The prevalent underconsumption of several nutrients and their best dietary sources;
4. The nutritional needs of vulnerable populations that will continue to remain unmet;
5. The reversal on limits of dietary cholesterol while failing to recommend nutrient-dense foods that contain cholesterol;
6. The failure to consider the variety of dietary patterns in our multi-cultural nation and their importance both socially and nutritionally.

We will elaborate on each of these points in the order above with supporting research cited. We will show that the DGAC's noble objective:

An overarching premise of the DGAC is that that the Dietary Guidelines for Americans should provide food-based guidance for obtaining the nutrients needed for optimal reproductive health, growth and development, healthy aging, and well-being across the lifespan (ages two years and older).

can not and will not be met if their recommended dietary patterns are followed:

The overall body of evidence examined by the 2015 DGAC identifies that a healthy dietary pattern is higher in vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes, and nuts; moderate in alcohol (among adults); lower in red and processed meats; and low in sugar-sweetened foods and drinks and refined grains.

We will provide substantial evidence that a healthy dietary pattern includes full-fat dairy, whole eggs and land animal proteins including red meat and/or poultry, and does not place limits on saturated fat intake nor encourages higher intakes of polyunsaturated fat. Nourishing full-fat animal foods are needed for all stages of the lifespan and are especially important for optimal reproduction, growth, and development.

1. LIMITS ON TOTAL FAT AND SATURATED FAT

Historically, the DGAs have recommended diets lower in total fat, while specifically targeting saturated fats and industrial *trans* fatty acids as contributors to chronic disease. To their credit, the 2015 DGAC has acknowledged that “low-fat diets induce dyslipidemia,”¹ stating in the final Report that “dietary advice should put emphasis on optimizing types of dietary fat and not reducing total fat.”² They 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.

Unfortunately, the Report fails to make any definitive recommendations on the overall intake of fat, and it continues to recommend a low-fat dietary pattern, erroneously leading the American public to conclude that limits on fat remain unchanged. The 2010 DGAC Report gives a clue why the messages on fat intake have not changed: “The ability to maintain intake of SFA less than 10% of energy is typically only achievable *when total fat intakes are less than 30% of energy*, and this is even more relevant for those attempting to adhere to levels of SFA less than 7% of energy as recommended for everyone” (emphasis ours).³ Furthermore, the Report fails

to acknowledge the leading role that the DGA low-fat recommendations played in promoting low-fat diets since the earliest release in 1980.

We strongly agree that optimizing the types of dietary fat would improve the health of Americans. We strongly *disagree* with the conclusions drawn in the Report on what constitutes optimal dietary fats. Although the evidence for limiting industrial *trans* fats is conclusive, the evidence for limiting saturated fats (to less than 10% of calories) while increasing the intake of unsaturated fats is at best weak and controversial. Saturated fats are erroneously identified as “an overconsumed nutrient of concern,” when in actuality they play important roles in numerous physiological processes.

Saturated fats from animals are rich sources of all four fat soluble vitamins: A, D, E, and K, and even more so when fats are obtained from pastured livestock. The Report has identified vitamins A, D, and E as “shortfall nutrients,” and notes that for vitamin K, only 34% of the population has usual intakes above the AI.⁴ Vitamin D is further identified as an “underconsumed nutrient of public health concern” and as such, the Report emphasizes the importance of ensuring that Americans meet their DRI. Dietary vitamin D is best supplied by animal foods; traditionally consumed foods such as pork/lard, butter, cheese and egg yolks are good sources, along with the more widely recognized contributions from cod liver oil, fatty fish and fluid milk.⁵ The synergistic actions of vitamins A and vitamin K2 with vitamin D have been shown to strongly protect against both CVD and osteoporosis and will be discussed below. These partners to vitamin D are also concentrated in the very same animal fats.

Recent meta-analyses of observational studies showing that saturated fat intake has no association with CVD events have, according to the Report, “re-ignited the debate regarding the current recommendation to limit saturated fat intake.”⁶ Despite this ongoing controversy, the DGAC chose to rely on “existing reports” to draw the Report’s conclusions, rather than conducting their own NEL analysis of the literature. The existing reports have limitations, and we will provide some insights into why controversy exists, and why we must challenge the Report’s conclusions.

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.”⁸

Then the DGAC makes the recommendation that: “Sources of saturated fat should be replaced with unsaturated fat, particularly polyunsaturated fatty acids.”⁹

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).¹⁰ 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.”¹¹

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.^{12,13} In fact, a direct association between PUFA intake and luminal narrowing in women with CHD has been observed.^{14,15}

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.¹⁶ 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.¹⁶

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.¹⁷ 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.¹⁸

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.”¹⁹

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 as-

sess 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.²⁰

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.”²¹ 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.”²³ 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” (24). 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."^{16,17}

- 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."²⁵ 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.²⁶ 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."²⁷ 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.¹⁶

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."²⁸ The interventional literature likewise is inconsistent and therefore, no reliable conclusions can be drawn at this time.

WHY SATURATED FATS ARE BENEFICIAL

First it must be pointed out that the main dietary sources of saturated fats (found in foods omitted from the list of recommended foods and given the designation "solid fats" that provide "empty calories")²⁹ such as tallow,

butter, lard and tropical oils, contain a mixture of different fatty acids, both saturated and unsaturated. For example, lard contains 44% SFA, 45% MUFA, and 11% PUFA; olive oil is 15% SFA, 73% MUFA, and 10% PUFA; and while butter fat is 66% SFA, 14% of that is short- and medium-chain length fatty acids that have unique functions in the body.³⁰ Mounting data indicates the positive roles SFAs play in the diet. “[C]ompositional analyses have shown remarkable specificities for particular saturated fatty acids in cellular compartments...most of which [SFAs] have been examined solely for their tendency to alter lipoprotein metabolism and to influence the concentrations of lipoproteins that carry cholesterol in the blood.”³¹ Recent findings suggest that individual SFA have specific properties associated with important biological functions.³²

Stearic acid has a neutral effect on LDL-cholesterol levels; beef and mutton tallow contain up to 16% of total fat as stearic acid. A research need identified by the 2010 DGAC states: “Examine stearic acid for its benefits as a solid fat, in contrast to liquid oils high in MUFA and PUFA; include other potential metabolic effects of stearic acid, such as inflammation and coagulation.”³³

High-fat dairy products also exert beneficial or neutral effects.³⁴ Intake of dairy fat is associated with a lower incidence of type 2 diabetes,³⁵ a lower risk of heart attacks in both men and women,³⁶ a reduced risk of adiposity in children,³⁷ and a lower rate of anovulatory infertility in adult females.³⁸

It is clear that the risk of CVD is not increased by the consumption of SFA in nutrient-dense foods such as meat, eggs and dairy. And in fact, according to Usual Intake data used in the Report,³⁹ Americans are currently consuming 11.2% of total calories as saturated fats, only 2.9 grams (the amount found in 1 pat of butter) over the Report’s recommended limit of 10%, and down from a mean intake of about 13% in 1971.⁴⁰ Food sources of saturated fat include 18% obtained from snacks and sweets.⁴¹ These types of food should not be defined as saturated fats because they are composed substantially of carbohydrates.⁴²

By contrast, the mean daily intake of linoleic acid intake between 1960 and 2012 has increased from about 5% of calories to 7% of calories, and the current average intake in adults is over 17 g/d.⁴³ While the AI for n-6 is 17 g/d for men and 12 g/d for women (ages 19-50), “[i]t is important to recall that AI recommendations are observed median intakes for the US population, *not an RDA or an intake of fatty acids shown to confer lower risk of disease*” (emphasis ours).¹¹ From 1909 to 1999, the availability of linoleic acid in the US food supply increased from 2.79% to 7.21% of calories, largely due to a greater than 1000-fold increase in per capita consumption of soybean oil.⁴⁴ Soybean oil is often consumed from foods fried in it and has replaced chemically stable SFA-containing fats such as tallow and lard for that purpose. The unforeseen consequence of this shift is that Americans are now unknowingly consuming a variety of harmful oxidative products from this thermal processing (which also depletes vitamin E). Although repeated thermal exposure amplifies the harmful effects of high-omega 6 oils like soybean oil on cardiovascular health, meals rich in even unheated PUFA vegetable oils “may cause injury to endothelial cells.”⁴⁵

Carbohydrates, according to the Report, are overconsumed by 126 g/day, or the equivalent of 8 slices of bread. The report then recommends 3 ounces of refined grains and 3 ounces of whole grains per day, identical to previous DGA guidance and contradicting the Report’s revised recommendation for lower intakes of refined grains which are identified as “detrimental.” We emphatically agree with the Report that Americans should limit their intake of nutrient-poor foods high in added sugars, and we go further to advise Americans to limit all refined grains; both adversely impact serum triglycerides. Reducing triglyceride levels is now considered a much more effective strategy in reducing risk for CVD, in part because LDL particles involved in atherogenesis contain less cholesterol and phospholipid, but more triglyceride.¹² Diets low in fat and high in carbohydrates will raise serum triglycerides and induce dyslipidemia, a fact the DGAC has acknowledged (citation above). Lipoprotein particle size is becoming a widely recognized marker of CVD risk, with smaller, denser LDL-cholesterol particles being a predictor of the onset of CVD.⁴⁶

Clearly there remain serious questions about the best dietary practices currently utilized for prevention of CVD. It is imperative that we end this population-wide experiment until the data is satisfactorily conclusive to make evidence-based recommendations on fat consumption.

2. THE FAILURE TO RECOMMEND LAND ANIMAL PROTEINS AND THE SPECIFIC RECOMMENDATION TO LIMIT RED MEAT.

In contrast to the 2015 DGAC Report, the 2010 DGAC Report included lean meats, poultry and eggs in their list of “nutrient-dense foods.” While the 2010 Report’s definition of nutrient density did imply that the “solid fats naturally present in the food” reduce the nutrient density of a food, these foods were not left off the list nor did they carry a specific recommendation for limitation in the diet. And yet, the 2015 Report contradicts itself by excluding meat from foods included in a “healthy dietary pattern,” while (in a footnote) suggesting “lean meats can be part of a healthy dietary pattern.”⁴⁷

The rationale behind the 2015 Report’s revised list is not entirely clear to us. We suspect it may have something to do with a new emphasis on sustainability of the diet. Sustainability is an important issue of today; however, the DGAC members clearly do not have expertise in this area, not even to evaluate the input of a special subcommittee. The statutory authority conferred by the United States Congress to the USDA and HHS regarding the final 2015 Dietary Guidelines must not be exceeded. We add our voices to Congressional admonition and urge that your departments “carefully consider [only] the most relevant nutrition scientific literature and reject the DGAC’s inconsistent conclusions regarding the role of meat in Americans’ diets as you finalize the Dietary Guidelines.”⁴⁸

The 2015 Report makes contradictory statements regarding the roles of protein and red meat in the diets of Americans. There is no mention of the importance of protein in the summary statements. These issues need to be re-evaluated by nutrition experts who do not have an underlying sustainability bias.

According to data cited in the Report, 40% of boys and girls ages 4 to 8, 60% of boys and 55% of girls ages 9 to 13, and 40% of boys and 75% of girls ages 14 to 18 do not meet the recommended intake of protein for their respective age and gender groups. Notably, “[a]cross all age groups and in both males and females, nearly 60 percent of the U.S. population meets the protein foods intake recommendation.”⁴⁹ That leaves over 40% of Americans not meeting the intake recommendation, and yet protein is not classified as a shortfall nutrient; we can only guess that this is because of the DGAC’s assumption that grains (a poor source of high biological value protein) and dairy intake may make up the shortfall.⁵⁰ This is a poor assumption as the intake of dairy among children drops after the age of 3 to less than 30% of boys and girls meeting the recommended intakes, to as low as 10% of adolescent girls meeting the recommendations. This shortfall persists at very low levels of intake among adult females across all ages. “Overall, more than 80 percent of the entire U.S. population does not meet the daily dairy intake recommendation.”⁵⁰

Each of the DGAC’s three healthy food patterns recommends protein intakes between 155% and 198% of the RDA. The Report clearly recognizes where the majority of Americans are getting their protein: “[m]ost of the protein foods intake across all age groups and for both males and females comes from meat, poultry, and eggs.”⁵¹ The result of removing meat, poultry and eggs from the recommended foods list will be an even greater number of Americans, especially children under 18, failing to meet their protein requirements. The numerous roles of protein in the body do not need to be restated here, however, it is important to emphasize the essential role of protein in growth and development, and in the maintenance of appropriate energy intakes. We do not disagree with the Report’s recommendation that sustainable seafood should be a part of a healthy diet, but that does not and must not preclude the intake of terrestrial animal foods.

Concerning red meat specifically, a population-wide recommendation to limit its intake is wholly irresponsible of the DGAC, and in fact contradicts the findings in their Report: “In addition to providing essential amino acids, some protein foods are important sources of iron, and iron is a shortfall nutrient and nutrient of public health concern among adolescent and adult females. Meat foods in the protein group provide heme iron, which is more bioavailable than non-heme plant-derived iron; according to the Report, “heme iron from lean meats is highly bioavailable, hence, an *excellent source* (emphasis ours).⁵² Heme iron is especially important for young children and women who are pregnant.”⁵³ We respectfully point out an error in the Report: “Excellent sources of heme iron include red meats, enriched cereal grains, and fortified breakfast cereals.”^{54,55} As any good first-year nutrition major should know, of those foods only red meat is a source of heme iron.

The report at times seems to be more interested in allowing “vegetarian options to be accommodated”⁵⁶ than respecting the diversity of diets among the American population. And yet, the recommended “Healthy Mediterranean-style dietary pattern” that the Report states has “positive health benefits” contains a higher intake of red meat than the amount contained in the recommended DASH-style diet or USDA Food Patterns.⁵⁷ Any possible concern over excess iron/heme iron intake should remain a matter of discussion between at-risk individuals and their healthcare providers.

Zinc is another mineral that may be underconsumed when red meat and animal proteins are limited. This is of particular concern for young children, who have high physiologic requirements for iron and zinc to support rapid growth and brain development; zinc is also essential for immune function.⁵⁸ Phytates in whole grains and legumes have an inhibitory effect on zinc absorption, which may be greater than previously estimated. According to Gibson et al, “[w]ithout a doubt, the poorer bioavailability of iron and zinc in vegetarian diets is likely to increase the risk of inadequate consumption of these trace minerals.”⁵⁹ Animal proteins are not only rich sources of zinc, their consumption improves the bioavailability of zinc from plant food sources.⁵⁸

Finally, when and if the topic of agricultural sustainability is considered by a Congressionally appointed department, we will be following that discussion closely. Optimal human nutrition requires the cultivation of crops and husbandry of livestock on fertile and sustainable farm and pasture lands. Our members seek out foods directly from farmers who use regenerative methodologies that reduce greenhouse gas emissions, topsoil erosion, water usage and water run-off. In this way, we help ensure our own good health and that of our planet.

3. THE PREVALENT UNDERCONSUMPTION OF SEVERAL NUTRIENTS AND THEIR BEST DIETARY SOURCES.

The Committee characterized these as shortfall nutrients: “vitamin A, vitamin D, vitamin E, vitamin C, folate, calcium, magnesium, fiber, and potassium. For adolescent and premenopausal females iron also is a shortfall nutrient. Of the shortfall nutrients, calcium, vitamin D, fiber, and potassium also are classified as nutrients of public health concern because their underconsumption has been linked in the scientific literature to adverse health outcomes. Iron is included as a shortfall nutrient of public health concern for adolescent females and adult females who are premenopausal due to the increased risk of iron-deficiency in these groups.”⁶⁰ Vitamin K and choline are not identified as shortfall nutrients by the DGAC, but we will show why they need to be considered as such. We will focus our attention on vitamin A and vitamin K in this section; iron and choline will be addressed in points number 4 and number 5 respectively.

Vitamin D has received considerable attention in the past several years, and like the DGAC, the FDA and multiple national and international organizations have recommended strategies to improve vitamin D intake across the population.⁶¹ As stated earlier, vitamin D works in concert with vitamins A and K. Yet few organizations,

with the notable exception of The Weston A. Price Foundation, have concerned themselves with low intakes of these two important fat-soluble vitamins. Dr. Weston A. Price was an early pioneer in the nutrition field and his in-depth population and clinical research helped elucidate the roles of the fat-soluble vitamins in growth, development and health throughout the lifespan.^{62,63} Since then research has confirmed the importance of these nutrients.

VITAMIN A

According to the data cited by the Report, 40% of Americans have intakes of vitamin A below their EAR.⁶⁴ At the same time, the estimated percent of persons below the recommended intake of orange and red vegetables is over 90% for all individuals ages 1 year or older.⁶⁵ One might conclude that by emphasizing an increase in these vegetables that owe their color to a variety of carotenes, some of which are considered effective precursor sources of true vitamin A, this shortfall could be reduced. Yet, vitamin A cannot be obtained from plant sources for a large percentage of the population due to poor absorption and/or conversion. Only about 12 percent of the carotenes in broccoli are absorbed, and only about 18 percent of carotenes in carrots are absorbed.⁶⁶ And, according to studies in men and women, conducted respectively by Hickenbottom et al. and Lin et al., for the USDA Agricultural Research Service, “[t]he vitamin A activity of β -carotene ... is “surprisingly low and variable” and the authors “confirmed [previous studies that demonstrated] the highly variable extent to which β -carotene provides vitamin A, even in identical diets. Some could achieve adequate vitamin A nutritional status from β -carotene alone, but 45% would not.”^{67,68} According to Lin et al., “Some individuals are characterized as responders and others as low- or nonresponders” which may be a stable characteristic and attributable in part to genetics. Thus, no amount of colorful vegetables or fruit would meet the vitamin A requirements of non-responders. While we would agree with encouraging the consumption of vegetables of all colors, this should not replace the recommendation to consume the only reliable sources of pre-formed vitamin A: animal foods and their fats.

Traditionally, liver has been considered a nourishing food to consume on a regular basis, along with other organ meats. Liver is the best dietary source of true vitamin A; another excellent source is cod liver oil, which is also a good source of vitamin D.⁶⁹ Other good sources include egg yolks and dairy fat such as butter and ghee. The reduction in the consumption of milk fortified with vitamin A is a related concern (over 85% of all individuals are below the recommendation for dairy intake, with 51% of dairy intake from fluid milk);⁷⁰ however we emphasize the importance of consuming foods with naturally occurring vitamin A. This would ensure that the recommendation made in the Report that “[t]he U.S. population should increase consumption of foods rich in vitamin A” be met.⁷¹

Vitamin A is necessary for vision, production of red blood cells, cell differentiation, bone remodeling, thyroid function, growth, immunity, steroid production, sperm production in males, prevention of spontaneous abortion in females, and proper prenatal development.⁷² According to Agarwal, newly discovered roles include: inhibiting adipogenesis (creation of new fat cells), enhancing apoptosis (breakdown) of fat cells, and regulating adipokines (hormones related to metabolism), thus playing important roles in the regulation of body weight. A recently published NHANES analysis showed that not only does 67% of the adult population have intakes of vitamin A below the EAR, but that 72% of overweight individuals and 76% of obese individuals have vitamin A intakes below the EAR.⁷³ Whether meeting the EAR for vitamin A will help reduce the prevalence of obesity is unclear; however, it is clear that Americans should at least be meeting the recommended daily amounts.

BETA-CAROTENE MAY HAVE A DARK SIDE

β -carotene can not only be converted into vitamin A, but can also be converted into a number of potentially harmful “eccentric cleavage products” within the cell.⁷⁴ When the *polyunsaturated fat, linoleic acid* (of which the Report recommends increased intake) is added to the mix, the production of these eccentric cleavage products dramatically increases; research suggests they can actually interfere with vitamin A activity (emphasis

ours).⁷⁵ Recent research suggests that common products of β -carotene metabolism are antagonists to pre-formed vitamin A at the retinoic acid receptor sites.⁷⁴

VITAMIN K

Vitamin K is not considered a shortfall nutrient in the Report, with 34% of population with usual intakes above AI,⁷⁶ representing only the intake of Vitamin K1 from plant sources as dietary recommendations are based on the current knowledge of the plant form phyloquinone (PK) and do not differentiate between PK and the animal form menaquinones (MK). The recommended intakes are sufficient for normal blood coagulation, but there is “emerging evidence that current dietary recommendations...may not be optimal for supporting vitamin K requirements in extrahepatic tissues...[o]bservational studies have reported favorable associations between MK (vitamin K2) intake and bone and cardiovascular health...randomized trials have provided some evidence to support the beneficial effects of MK on bone.”⁷⁷ A review commissioned by the International Life Sciences Institute of Europe concluded that knowledge gaps preclude setting a reference value for MK intake, but future recommendations should consider both MK and PK for vitamin K intakes. In the meantime “it may be preferable to recommend consumption of a wide variety of foods containing a combination of PK and MKs.”⁷⁷ Dairy products (especially bacterial fermented whole-milk cheeses such as Jarlsberg, emmental and Edam-type cheeses) are primary contributors to dietary MK intake, along with animal liver and kidney.^{77,78}

In children, suboptimal vitamin K status is most evident in puberty when bone metabolic activity is high.⁷⁹ Vitamin K2, including several MKs, is only found in animal fats (MK-4) and fermented foods (MKs with 5-13 prenyl units, known as long chain MKs); MK-4 present in poultry and pork products is the primary dietary source of MK-4 in the U.S. food supply.^{77,78} Neither of these foods is on the recommended list in the Report. Non-fat dairy is completely devoid of fat-soluble vitamins unless they are added back and K2 is currently not part of the fortification process; low-fat dairy contains less vitamin K2 than full-fat dairy.⁸¹ These data reflect only MK-4 content and do not include other MK forms.⁷⁷ The naturally occurring dairy fat in whole dairy products increases the absorption of all of the fat-soluble vitamins. When considered along with data showing other benefits for dairy fat (above), there is no rational reason to recommend only low-fat or nonfat dairy products, for children or at any age. Unfortunately, at the present time, only low-fat or nonfat milk is permitted to be sold in schools that participate with the National School Lunch or School Breakfast programs, and as of 2011, child-care sites that participate in the Child and Adult Care Food Program, yet flavored (i.e. sugar-sweetened) milks are not restricted.⁸² We find this particularly concerning because “[i]t is estimated that sugar-sweetened beverages account for one fifth of the weight gain by the US population since the 1970s.”⁸²

4. THE NUTRITIONAL NEEDS OF VULNERABLE POPULATIONS THAT WILL CONTINUE TO REMAIN UNMET

Children, pregnant women, the elderly, and minority populations are most likely to participate in Federal nutrition programs that adhere to the final DGA. However, research on diet-disease relationships in these groups is particularly limited, a fact acknowledged in the Report.

Children are the future of our country, and it is of the highest priority that we ensure that their diets provide all of the essential nutrients for healthy growth and development, beginning prenatally. It bears repeating: “An overarching premise of the DGAC is that the Dietary Guidelines for Americans should provide food-based guidance for obtaining the nutrients needed for optimal reproductive health, growth and development, healthy aging, and well-being across the lifespan (ages 2 years and older).”⁸³ Dietary interventions after the age of two “cannot undo the damage that was done because of the under-nutrition during the first 1000 days.”⁸⁴ Clearly, meeting essential nutrition should be the focus of dietary guidance for pregnant women and young children.

Of the nutrients that have been identified as population-wide shortfall nutrients, while all are essential, the fat-soluble vitamins A, D, E, and folate, vitamin C, and iron are paramount to healthy reproduction and growth. According to the Report: “Many of the shortfall nutrients in the general population also were shortfall nutrients among women who are pregnant. Among this group, 26 percent were below the EAR for vitamin A intake and 30 percent had vitamin C intakes below the EAR. For vitamin D, 90 percent had intakes below the EAR and for vitamin E, 94 percent had intakes below the EAR. Calcium intake was also low, where 24 percent had intakes below the EAR, and for folate, 29 percent had intakes below the EAR. *Notably, 96 percent of women who were pregnant had iron intakes below the EAR*” (emphasis ours).^{85,86}

Vitamin K, while not identified as a shortfall nutrient despite its often low intake, is also essential as explained above.

Vitamin A is vital for reproduction as it is required for differentiation and patterning of all the cells, tissues and organs within the developing body.⁸⁷ For example, during gestation, even a mild vitamin A deficiency results in a compromised number of nephrons, predisposing to reduced renal function in adulthood.⁸⁸ A deficit of vitamin A during lung formation can cause profound changes in the muscles surrounding the airways, causing adult lungs to respond to stimuli with excessive narrowing of the airways. According to research by Cardoso et al: “[o]ur study suggests that the presence of structural and functional abnormalities in the lungs due to vitamin A deficiency during development is an important and underappreciated factor in this susceptibility [to developing asthma symptoms].”⁸⁹

There is a widespread unwarranted recommendation that foods high in preformed vitamin A are to be limited or avoided during pregnancy;⁹⁰ the weight of the evidence supports the view that 20,000-25,000 IU of vitamin A during pregnancy is safe and may even reduce the risk of birth defects.^{91,92} Vitamin D deficiency during pregnancy is receiving attention,⁹³ but unfortunately warnings against preformed vitamin A consumption are now the norm. Most available prenatal vitamins now contain no preformed vitamin A and only low amounts of beta-carotene.

In regards to iron, as indicated earlier, “Iron is an essential mineral whose primary function is to transport oxygen in the blood. Inadequate iron status in the form of iron deficiency anemia leads to poor growth and development and the potential for cognitive deficits in children. Excellent sources of heme iron include red meats, enriched cereal grains, and fortified breakfast cereals [our correction: cereals are not sources of heme iron].”⁹⁴ Dietary intake estimates, together with the CDC nutritional biomarker data, indicate that iron is a nutrient of concern for children, premenopausal females and during pregnancy. Among women who are pregnant, 96 percent are below the EAR for iron intake.”⁹⁵

“Taken together, the DGAC concluded that iron was an underconsumed nutrient of public health concern for adolescent and premenopausal women and women who are pregnant.”⁹⁵ The guidance to limit red meat is very misguided and clearly not aligned with the DGAC’s overarching premise or its obligation to address nutrients of public health concern.

Although not designated as a shortfall nutrient, choline, discussed in more detail below, is important for fetal development and postnatally. Low gestational choline intake is associated with an increased risk of birth defects in the fetus⁹⁶ and large amounts of choline are present in human milk.⁹⁸ According to Zeisel, “[t]wo strategies could make more choline available for brain development: maternal diet could include more foods such as eggs and liver, a supplement. . . .”⁹⁷ Choline is critically needed for an infant’s growth and development; a mother’s intake of choline will influence the choline content of her breast milk, depending on her genotype.⁹⁶ Choline is not found in most prenatal or regular vitamins, therefore an increased consumption of choline-rich foods is needed to meet the high pre- and post-natal demands for choline.⁹⁸

Per the Report: “It is important to note that the sample size for women who were pregnant in WWEIA 2007-2010 is very small (n=133 respondents), so the estimates should be interpreted with caution and the generalizability of the data to all women in the United States who were pregnant is limited.”⁹⁹

Despite the available sample size being very small, until more data are available, excellent sources of foods containing these shortfall nutrients, including liver and other organ meats (one serving weekly), egg yolks and full fat dairy, should be strongly recommended for women of reproductive age.

Looking ahead, the USDA/HHS agencies are planning to include guidance for ages birth to 24 months, to be released in early 2018 and then provided to the 2020 DGAC. Upon release, federal agencies will be incorporating the B-24 Project guidance into federal nutrition and feeding programs.¹⁰⁰ We are greatly concerned that expansion of the government’s failed nutrition policy into the arena of the very young child will greatly increase the risk for undernutrition in this vulnerable population, having life-long adverse impacts. We will be following the B-24 Project very closely as it develops, and will provide our input.

5. THE REVERSAL ON LIMITS ON DIETARY CHOLESTEROL WHILE FAILING TO RECOMMEND NUTRIENT-DENSE FOODS THAT CONTAIN CHOLESTEROL.

The Report does not place any limits on dietary cholesterol as in previous Dietary Guidelines. In contradiction to this, however, the Report continues to suggest in many places that dietary cholesterol should be limited: “Additionally, research that includes specific nutrients in their description of dietary patterns indicate that patterns that are lower in saturated fat, cholesterol, and sodium...are beneficial for reducing cardiovascular disease risk.”¹⁰¹

The recommendation to limit dietary cholesterol was never based on the preponderance of the evidence, and we applaud the 2015 DGAC for taking the long-needed step to remove this recommendation. However, at the same time, foods typically higher in cholesterol such as whole eggs, meat/organ meat, and full-fat dairy, are still not recommended. These foods are good sources of several nutrients including choline, and eggs are actually considered by the Report to be a nutrient-dense food, “rich in vitamins, minerals, and other substances that may have positive health effects.”¹⁰²

CHOLINE

Choline plays numerous roles in the body. It is a critical component of cell membranes (phosphatidylcholine) and involved in neurotransmitter synthesis (acetylcholine), lipid transport from the liver (lipoproteins) and provision of methyl groups that regulate metabolic pathways and detoxify the body. Choline plays an important role in fetal brain development, influencing lifelong learning and memory.^{103,104} Endogenous synthesis of choline does not meet human requirements.¹⁰³

Only one out of ten American adults are meeting the Adequate Intake guidelines for choline, including pregnant women.¹⁰⁵ Although an RDA has not yet been determined, data derived from highly controlled feeding studies involving adult men suggests that the AI does not overestimate dietary requirements.⁹⁸

Ten percent of adults studied developed fatty liver and/or muscle damage even when consuming the AI of choline; these conditions resolved on high-choline diet.¹⁰³ There is genetic, gender and age variability on dietary choline needs, but that does not preclude the benefits of recommending foods that lead to a population-wide sufficient intake.

In the meetings of the 2010 DGAC, the Chairperson Van Horn stated "...the take-home message here is just the stunning number of Americans who still are well below the recommended intakes of calcium, potassium, fiber, and *choline*, and the vast majority that eat well beyond the recommended amounts of sodium and dietary cholesterol" (emphasis ours).¹⁰⁶ At that time, the Committee, despite a lack of evidence (they admit they did not have time to do an NEL analysis on this issue), chose to address what they considered an overconsumption of dietary cholesterol. Committee member Slavin commented: "I think for high quality protein I'm with kids and getting better diets into people with lower calories. I'm just really big on eggs and protein qualities. So making sure we don't let cholesterol make the decision here." And yet that DGAC did just that, they continued to recommend a limit on dietary cholesterol, effectively precluding the consumption of egg yolks and liver with the rationale they were attempting to "triage" the nutrient shortfalls. Slavin: "[I]t kind of comes back to the choline recommendations we heard about. We are not meeting that. Nobody is really thinking about how that's... how we're going to---." Committee member Nelson: "I think we have to focus on those nutrients, the shortfall nutrients that seem to have fairly profound health implication. I mean I think we have to triage some of those nutrients..."¹⁰⁷

Now that the 2015 DGAC has wisely decided to remove the recommendations for a limit on dietary cholesterol (a decision that considers the evidence available to previous DGACs), whole eggs containing 125 mg of choline per yolk, and liver containing 430 mg per 100 g in the beef variety, should be reinstated as nutrient-dense foods to be included in a health-promoting diet, not only for the choline they provide to help meet the shortfall, but for the myriad of other nutrients they contain. Eggs in particular can help Americans improve their protein intake at breakfast, which is associated with improved food intake regulation.¹⁰⁸

MEETING THE SHORTFALLS

"In comparison to recommended amounts in the USDA Food Patterns, the majority of the U.S. population has low intakes of key food groups that are important sources of the shortfall nutrients, including vegetables, fruits, whole grains, and dairy."¹⁰⁹ It is concerning that the Report fails to include meat, poultry, eggs, full-fat dairy, or organ meats on this list when they are the best sources of vitamins A, K2, choline and heme iron. The traditional foodways of every culture included a source of full-fat animal protein, and traditional cultures consumed all edible parts of land and sea animals they raised or caught.⁶²

6. THE FAILURE TO CONSIDER THE VARIETY OF DIETARY PATTERNS IN OUR MULTI-CULTURAL NATION AND THEIR IMPORTANCE BOTH SOCIALLY AND NUTRITIONALLY

The United States has been forged from a melting pot of ethnicities and cultures, each with its own dietary traditions, which have sustained health and promoted reproduction and growth. While these cultures each had unique dietary patterns, Dr. Weston A. Price discovered that all cultures valued animal foods in their dietary traditions.⁶² Unfortunately, the DGAC does not consider this diversity when deciding on the three recommended dietary patterns: the Healthy U.S.-style Pattern, the Healthy Mediterranean-style Pattern, and the Healthy Vegetarian Pattern.¹¹⁰

It is not uncommon for immigrants to become acculturated to American dietary patterns after one or two generations, and unfortunately this practice is not serving them well. According to the Report: "[a] large and growing body of research suggests that the extent of an individual or family's acculturation status may be a predictor of dietary intake and that together, diet and acculturation status may influence health status or disease risk."¹¹¹ A notable example is the fact that obesity rates are significantly higher among the Hispanic population; historically Hispanic and "less-educated" adults have not shifted away from lower fat diets as a strategy for weight management as have one-third of all American adults.¹¹²

We would urge the USDA and HHS to consider the foodways of our immigrant populations when making population-wide recommendations. This is especially necessary given the DGACs recognition of a need for future research to “[e]xpand WWEIA participation to include more respondents from race/ethnic minorities and non-U.S. born residents; while acknowledging that “[v]ery little is known about the dietary habits of many of the cultural subgroups in the United States. This knowledge is essential to moving forward any nutrition programs for first and second generation immigrants.”¹¹³ While formal studies may be limited, much is already known about which foods (beyond those recommended in the Report) are emphasized in well-balanced, cross-cultural traditional meal patterns: meat (livestock and game), poultry, sausage, offal, bones and joints especially via stocks, full-fat fermented dairy products and whole eggs are typical components of healthy traditional diets.¹¹⁴

A food widely consumed across traditional cultures is raw dairy, which has several unique nutritional properties. Beyond the benefits found in full-fat dairy, raw milk cheeses contain local bacterial strains that exert beneficial probiotic properties and potential food safety benefits, as compared to pasteurized cheeses manufactured with commercial bacterial strains.^{115,116} With the increasing knowledge about the effects of beneficial intestinal microbiota on all aspects of health, raw milk cheeses should be recommended as a valuable health-promoting food, a practice our own members find valuable.

CONCLUSION

All Americans are impacted by the DGA, either directly through federal food assistance, or indirectly through federal nutrition education and communication programs that are based on the DGA.¹¹⁷ The majority of Americans know neither the specific recommendations of the DGA or their far-reaching influence, which has expanded from the simple recommendations of the 1977 Dietary Goals to overarching standards that affect all of national nutrition policy and private sector practices.¹¹⁷ The present aim is for the DGAs to “be grounded in consensus science, methodical, and presumably without bias.”¹¹⁷ Our commentary clearly shows that this aim was not met.

There are considerable research gaps that remain in this 2015 Report, despite the DGAC’s attempts to address them. Evidence of bias and a lack of a methodical approach to consensus science is obvious. It is abundantly clear that the approach of the previous DGAs has not resulted in the outcomes predicted or hoped for. “[P]ersistent, prevalent, preventable health problems, notably overweight and obesity, cardiovascular disease, type 2 diabetes, and certain cancers, have adversely affected the health of the U.S. public for decades and raise the urgency for *immediate attention* and *bold action*” (emphasis ours).¹¹⁸ While not surprising, given the multiple nutritional shortcomings of the low-fat, low-animal fat approach, it is extremely distressing to anticipate a continuation of these failed policies, which are contributing to the very health problems cited in the Report.

Americans of all walks of life deserve better. The youth of America deserve better: “primary prevention in childhood [is] the single most potentially powerful method of halting and reversing America’s obesity epidemic.”¹¹⁹

We call for an immediate review of the Report’s recommendations by an independent and unbiased scientific panel that has the expertise to initiate bold action. Alternatively, at the minimum, we call for a postponement of the release of the 2015 DGAs until such a review can be completed. We trust you will give a full consideration to the facts presented here in an effort to provide a more complete understanding of the issues. We sincerely appreciate your mutual concern.

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REFERENCES

1. 2015 Dietary Guidelines Advisory Committee Meeting 5.
2. Scientific Report of the 2015 Dietary Guidelines Committee; herein referred to as “the Report.” (<http://www.health.gov/dietaryguidelines/2015-scientific-report/PDFs/Scientific-Report-of-the-2015-Dietary-Guidelines-Advisory-Committee.pdf>); Part D. Ch 6. line 460.
3. USDHHS and USDA. Dietary Guidelines for Americans, 7th edition.
4. Report: Figure D1.2
5. Report: Table D1.5
6. Report: Part D. Ch 6. Line 407
7. Report: Part D. Ch 6. Line 546
8. Report: Part D. Ch 6. p 12
9. Report: Executive Summary; Part A. line 347
10. Committee on Qualifications of Biomarkers and Surrogate Endpoints in Chronic Disease, Board on Health Care Services, Board on Health Sciences Policy, Food and Nutrition Board, Institute of Medicine. *Evaluation of Biomarkers and Surrogate Endpoints in Chronic Disease*. Washington, DC: National Academies Press; 2010;336. http://books.nap.edu/openbook.php?record_id=12869&page=133.
11. Vannice G, Rasmussen H. Position of the Academy of Nutrition and Dietetics: Dietary fatty acids for healthy adults. *J Acad Nutr Diet* 2014;114(1):136-153.
12. Lawrence GD. Dietary fats and health: Dietary recommendations in the context of scientific evidence. *Adv. Nutr.*2013;4:294-302.
13. Reaven P et al. Effects of oleate-rich and linoleate-rich on the susceptibility of low density lipoprotein to oxidative modification in mildly hypercholesterolemic subjects. *J Clin Invest.* 1993;91:668-76.
14. Mozzafarian et al. Dietary fats, carbohydrate, and progression of coronary atherosclerosis in postmenopausal women. *Am J Clin Nutr.* 2004;80(5):1175-84.
15. Silaste et al. Changes in dietary fat intake alter plasma levels of oxidized low-density lipoprotein and lipoprotein(a). *Arterioscler Thromb Vasc Bio.* 2004;24(3):498-503
16. Ravnskov U, DiNicolantonio JJ, Harcombe Z, Kummerow FA, Okuyama H, Worm N. The questionable benefits of exchanging saturated fat with polyunsaturated fat. *Mayo Clin Proc.* 2014; 89(4):451-53.
17. Ramsden et al. Use of dietary linoleic acid for secondary prevention of coronary heart disease and death. *BMJ.* 2013;4:346.
18. Ramsden et al. Lowering dietary linoleic acid reduces bioactive oxidized linoleic acid metabolites in humans. *Prostaglandins Leukot Essent Fatty Acids.* 2012;87:135-410.
19. Report: Part D. Ch 6. p 32.
20. Harcombe et al. Food for thought: have we been giving the wrong dietary advice? *Food Nutr Sci.* 2013;4:240-44.
21. Report: Part D. Ch 6. Line 545.
22. Report: Part D. Ch 6. Line 549.
23. Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore HJ, et al. Reduced or modified dietary fat for preventing cardiovascular disease. *Cochrane Database Syst Rev.* 2012;5:CD002137. PMID: 22592684.
24. Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. *Plos Med.* 2010;(7) (3):e1000252. PMID: 20351774.
25. Report: Part D. Ch 6. p 16.
26. Farvid MS, Ding M, Pan A, Sun Q, Chiuve SE, Steffen LM, et al. Dietary Linoleic Acid and Risk of Coronary Heart Disease: A Systematic Review and Meta-Analysis of Prospective Cohort Studies. *Circulation.* 2014;28;130(18):1568-1578.
27. Report: Part D, Ch 6, line 587.
28. Schwingshackl L, Hoffman G. Comparison of effects of long-term low-fat vs high-fat diets on blood lipid levels in overweight or obese patients: a systematic review and meta-analysis. *J Acad Nutr Diet* 2013;113:1640-1661.
29. Report: Appendix E-5: Glossary of Terms. p 4.
30. Enig M (2000). *Know Your Fats: The Complete Primer for Understanding the Nutrition of Fats, Oils, and Cholesterol*. Bethesda MD: Bethesda Press.
31. German JB, Dillard CJ. Saturated fats: what dietary intake? *Am J Clin Nutr.* 2004;80:550-555.
32. Legrand P, Rioux V. The complex and important cellular and metabolic functions of saturated fatty acids. *Lipids.* 2010;54:941-946.
33. Myers et al. A Critical Assessment of Research Needs Identified by the Dietary Guidelines Committees from 1980 to 2010. *J Acad Nutr Diet* 2013; 113:957-971.
34. Kratz M, Baars T, Guyenet S. The relationship between high-fat dairy consumption and obesity, cardiovascular, and metabolic disease. *Eur J Nutr.* 2013; 52:1–24.
35. Ericson et al. Food sources of fat may clarify the inconsistent role of dietary fat intake for incidence of type 2 diabetes. *Am J Clin Nutr.* 2015 May;101(5):1065-80.
36. Warensjö et al. Biomarkers of milk fat and the risk of myocardial infarction in men and women: a prospective, matched case-control study. *Am J Clin Nutr.* 2010;92(1):194-202.
37. Berkey et al. Milk, dairy fat, dietary calcium, and weight gain. *Arch Pediatr Adolesc Med.* 2005;159:543-550.
38. Chavarro et al. A prospective study of dairy foods intake and anovulatory infertility. *Hum Repr.* 2007; 22(5):1340–1347.
39. Report: Appendix E-2.1: Usual Intake Distributions, 2007-2010, by Age/Gender Groups, p. 8.
40. www.cdc.gov/mmwr/preview/mmwrhtml/mm5304a3.htm.
41. Report: Figure D1.34.
42. Harcombe et al.2013
43. Calder PC. The American Heart Association advisory on n-6 fatty acids: evidence based or biased evidence? *Br J Nutr.* 2010;104:1575-1576.
44. Blasbalg et al. Changes in consumption of omega-3 and omega-6 fatty acids in the United States in the 20th century. *Am J Clin Nutr.* May;93(5):950-962.
45. Chun-Yi et al. Heated vegetable oils and cardiovascular disease risk factors. *Vasc Pharm.* 2014;61:1-9.

46. Rizzo et al. Should we routinely measure low-density and high-density lipoprotein subclasses? *Clin Lab*. 2009;55(11-12):421-429.
47. Report: Part A: Executive Summary, p.4.
48. Bipartisan letter to the department Secretaries from Senator Thune and 29 colleagues, March 12, 2015; http://webcache.googleusercontent.com/search?q=cache:GFW_jOZF_qkJ:www.thune.senate.gov/public/index.cfm/2015/3/thune-leads-call-for-usda-hhs-to-include-lean-red-meat-in-2015-dietary-guidelines+&cd=1&hl=en&ct=clnk&gl=us.
49. Report: Part D. Ch 1. Line 33.
50. Report: Part D. Ch 1. Line 1179.
51. Report: Part D. Ch 1. Line 1249.
52. Report: Part D. Ch 1. p 9.
53. Report: Part D. Ch 1. Line 1227.
54. Report: Part D. Ch 1. Line 599.
55. Report: Table D1.9.
56. Report: Part D. Ch 1. Line 1224.
57. Report: Part D. Ch 1. Line 2607.
58. Roohani et al. Zinc and its importance for human health: an integrative review. *J Res Med Sci*. 2013;18(2):144-157.
59. Gibson RS et al. Is iron and zinc nutrition a concern for vegetarian infants and young children in industrialized countries? *Am J Clin Nutr*. 2014; Jul;100 Suppl 1:459S-68S.
60. Report: Executive Summary. Part A. p 2.
61. Report: Part D. Ch 1. Line 534.
62. Price WA & Price-Pottenger Nutrition Foundation (2003). Nutrition and Physical Degeneration. La Mesa, CA: Price-Pottenger Nutrition Foundation.
63. <http://www.westonaprice.org/health-topics/abcs-of-nutrition/on-the-trail-of-the-elusive-x-factor-a-sixty-two-year-old-mystery-finally-solved/>
64. Report: Figure D1.1.
65. Report: Figure D1.13.
66. West et al. Consequences of Revised Estimates of Carotenoid Bioefficacy for Dietary Control of Vitamin A Deficiency in Developing Countries," *J Nutr*, 132 (2002) 290S-292S.
67. Hickenbottom et al. Variability in conversion of beta-carotene to vitamin A in men as measured by using a double-tracer study design. *Am J Clin Nutr*. 2002;75:900-907.
68. Lin et al. Variability of the conversion of beta-carotene to vitamin A in women measured by using a double-tracer study design. *Am J Clin Nutr*. 2000 Jun;71(6):1545-1554.
69. Report: Table D1.5.
70. Report: Table D1.19.
71. Report: Part D. Ch 1. Line 300.
72. <http://www.westonaprice.org/health-topics/abcs-of-nutrition/vitamin-a-on-trial-does-vitamin-a-cause-osteoporosis/#astillvit>.
73. Agarwal et al. Comparison of the prevalence of inadequate nutrient intake on body weight status of adults in the United States: An analysis of NHANES 2001-2008. *J Am Coll Nutr*. 2015;34(2):126-134.
74. Harrison et al. Naturally occurring eccentric cleavage products of provitamin A: β -carotene function as antagonists of retinoic acid receptors. *J Biol Chem*. 2012;4;287(19):15886-95.
75. Russell, RM. The vitamin A spectrum: from deficiency to toxicity. *Am J Clin Nutr*. 2000; 71:878-884.
76. Report: Figure D1.2.
77. Walther B, Karl JP, Booth SL, Boyaval P. Menaquinones, bacteria, and the food supply: the relevance of dairy and fermented food products to vitamin K requirements. *Adv Nutr*. 2013;4:463-473.
78. Shearer J, Newman P. Recent trends in the metabolism and cell biology of vitamin K with special reference to vitamin K cycling and MK-4 biosynthesis. *J Lip Res*. 2014;55:345-362.
79. van Summeren et al. The effect of menaquinone-7 (vitamin K2) supplementation on osteocalcin carboxylation in healthy prepubertal children. *Br J Nutr*. 2009;102:1171-1178.
80. Beulens et al. The role of menaquinones (vitamin K2) in human health. *Br J Nutr*. 2013;110(8):1357-1368.
81. USDA National Nutrient Database for Standard Reference Release 27 Nutrients: Menaquinone-4 (μg)
82. Ritchie et al. Policy improves what beverages are served to young children in child care. *J Acad Nutr Diet* 2015;115:724-730.
83. Report: Part D. Ch 1. Line 246.
84. Clinton HR. 1,000 Days: change a life, change the future. US Department of State. 9/21/2010. <http://www.state.gov/secretary/20092013clinton/rm/2010/09/147512.htm>.
85. Report: Part D. Ch1. p 11.
86. Report: Table D1.2; Appendix E-2.4
87. Ross A. Vitamin A and Carotenoids. In: Shils M, Shike M, Ross A, Caballero B, Cousins R, eds. *Modern Nutrition in Health and Disease*. 10th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2006:351-75.
88. Gilbert T, Vitamin A and kidney development. *Nephrol. Dial Transplant*. 2002;17:78-80.
89. Cardoso et al. *J Clin Invest*. 2014;124(2):801-881.
90. One example: http://www.babycenter.com/0_vitamin-a-in-your-pregnancy-diet_675.bc?page=2.
91. Masterjohn C. Vitamins for Fetal Development: Conception to Birth. <http://www.westonaprice.org/health-topics/vitamins-for-fetal-development-conception-to-birth/>.
92. Mastroiacovo P, Mazzone T, Addis A, Elephant E, Carlier P, Vial T. High vitamin A intake in early pregnancy and major malformations: a multi-center prospective controlled study. *Teratology*. 1999;59(1):7-11.
93. <http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/Vitamin-D-Screening-and-Supplementation-During-Pregnancy>.
94. Report: Table D1.9.

95. Report: Part D. Ch 1. p 16.
96. Fischer et al. Choline intake and genetic polymorphisms influence choline metabolite concentrations in human breast milk and plasma. *Am J Clin Nutr.* 2010; 92(2): 336-346.
97. Zeisel S. Is maternal diet supplementation beneficial? Optimal development of infant depends on mother's diet. *Am J Clin Nutr.* 2009; 89:685S-7S.
98. Caudal MA. Pre- and postnatal health: evidence of increased choline needs. *J Am Diet Assoc.* 2010;110:1198-1206.
99. Report: Part D. Ch 1. Line 11.
100. Obbagy et al. USDA Nutrition Evidence Library: methodology used to identify topics and develop systematic review questions for birth-to-24 mo population. *Am J Clin Nutr.* 2014;99:692S-696S.
101. Report: Appendix E2.26.
102. Report: Appendix E-5: Glossary of Terms.
103. Zeisel SH, da Costa KA Choline: an essential nutrient for public health. *Nutr Rev.* 2009;67(11):615-623.
104. Sanders LM, Zeisel SH. *Nutr Today.* 2007;42(4):181-186.
105. Report: Figure D1.2.
106. 2010 DGAC 2nd meeting, Day 1: 1-29-09.
107. 2010 DGAC 2nd meeting, Day 2: 1-30-09.
108. O'Neil et al. The role of breakfast in health: definition and criteria for a quality breakfast. *J Acad Nutr Diet* 2014;114(12):S8-S28.
109. Report: Part A. p 2. line 61.
110. Report: Part D. Ch 1. Line 2827.
111. Report Part D. Ch 1. Line 422.
112. Andreyeva et al. Trying to lose weight: diet strategies among Americans with overweight or obesity in 1996 and 2003. *J Am Diet Assn.* 2010;110:535-542.
113. Report: Part D.Ch.1 Line 2838.
114. Fox M. Global food practices, cultural competency, and dietetics:parts 1, 2, and 3. *J Acad Nutr Diet* 2015;115(5 Suppl):S16-20.
115. Bilyk HT. Role of registered dietitian nutritionists in the research and promotion of cultural foods. *J Acad Nutr Diet* 2014;114(11):1736-38.
116. Montel et al. Traditional cheeses: rich and diverse microbiota with associated benefits. *Int J Food Microbiol.* 2014;177:136-154.
117. Rowe S. US evidence-based dietary guidelines: the history and the process. *Nutr Bull.* 2014;39:364-368.
118. Report: Executive Summary. Part A. Line 382.
119. Van Horn L. Development of the 2010 US Dietary Guidelines Advisory Committee Report: perspectives from a registered dietitian. *J Am Diet Assn.* 2010;110(11):1638-1645.