Position of the Academy of Nutrition and Dietetics: Dietary Fatty Acids for Healthy Adults

ABSTRACT

It is the position of the Academy of Nutrition and Dietetics (the Academy) that dietary fat for the healthy adult population should provide 20% to 35% of energy, with an increased consumption of n-3 polyunsaturated fatty acids and limited intake of saturated and *trans* fats. The Academy recommends a food-based approach through a diet that includes regular consumption of fatty fish, nuts and seeds, lean meats and poultry, low-fat dairy products, vegetables, fruits, whole grains, and legumes. These recommendations are made within the context of rapidly evolving science delineating the influence of dietary fat and specific fatty acids on human health. In addition to fat as a valuable and calorically dense macronutrient with a central role in supplying essential nutrition and supporting healthy body weight, evidence on individual fatty acids and fatty acid groups is emerging as a key factor in nutrition and health. Small variations in the structure of fatty acids within broader categories of fatty acids, such as polyunsaturated and saturated, appear to elicit different physiological functions. The Academy recognizes that scientific knowledge about the effects of dietary fats on human health is young and takes a prudent approach in recommending an increase in fatty acids that benefit health and a reduction in fatty acids shown to increase risk of disease. Registered dietitian nutritionists are uniquely positioned to translate fat and fatty acid research into practical and effective dietary recommendations.
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POSITION STATEMENT

It is the position of the Academy of Nutrition and Dietetics that dietary fat for the healthy adult population should provide 20% to 35% of energy, with an increased consumption of n-3 polyunsaturated fatty acids and limited intake of saturated and *trans* fats. The Academy recommends a food-based approach through a diet that includes regular consumption of fatty fish, nuts and seeds, lean meats and poultry, low-fat dairy products, vegetables, fruits, whole grains, and legumes.

THIS IS AN UPDATE OF THE 2007 Dietary Fatty Acid Position Paper developed by the Academy of Nutrition and Dietetics (the Academy; formerly the American Dietetic Association) and the Dietitians of Canada.[1] This update and the associated position reflect the current opinions of the Academy and is based on the most current scientific literature with consideration of other academic or organizational body recommendations.[2-6] The objectives of the current position paper are to provide information on specific fatty acids including structure, current and recommended intakes, function, and impact on health. This position paper evaluates the evidence for both beneficial and adverse effects of dietary fatty acids for the purpose of providing a rationale for intake levels of total fat, n-3 and n-6 polyunsaturated fatty acids (PUFA), monounsaturated fatty acids (MUFA), saturated fatty acids (SFA), and *trans*-fatty acids (TFA) for healthy individuals. From this, a position has been developed that will guide registered dietitian nutritionists (RDNs) in their practice and dietetic technicians, registered (DTRs) who work under the supervision of the RDN in counseling healthy individuals. As the stated position reflects recommendations for fatty acid intakes in the context of healthy individuals, it will not provide dietary guidance for children, pregnant women, or specific disease states.

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Fatty acids are the major form of dietary fat and primarily exist in foods in the triglyceride form. Although fatty acids are often categorized by their saturation status (eg, monounsaturated, saturated), understanding the role of individual fatty acids on health, rather than as a group, is important. Because of this, fatty acids will be presented individually in the following order: polyunsaturated, monounsaturated, saturated, and *trans*. In addition, fatty acids are consumed as a part of foods that contain other nutrients and dietary compounds; these have both additive and synergistic effects on health and interact in complex ways that are difficult to delineate. Therefore, the roles of individual fatty acids within the broader context of food and dietary patterns will be discussed.

FATTY ACID CLASSIFICATION SYSTEM

Fatty acid structures vary considerably by both hydrocarbon chain length and saturation status. Although carbon chain length can vary from 2 to 40 carbons, most dietary fatty acids contain 12 to 22 carbons.[7] Fatty acids are often categorized into short chain (up to 6 carbons), medium chain (8 to 12 carbons), or long chain (>12 carbons). Although hydrocarbon chain length is an important determinant of function, fatty acids are often classified based on whether or not the fatty acid carbon chain contains no double bonds (SFA), one double bond (MUFA), or more than one double bond (PUFA), as well as the configuration of the double bonds (*cis* or *trans*). In addition, PUFAs are often further classified based on the position of the first double bond from the fatty acid methyl terminus, creating n-3 and n-6 fatty acids. In n-3s, the first double

This Academy of Nutrition and Dietetics position paper includes the authors' independent review of the literature in addition to a systematic review conducted using the Academy's Evidence Analysis Process and information from the Academy's Evidence Analysis Library. Topics from the Evidence Analysis Library are clearly delineated. The use of an evidence-based approach provides important added benefits to earlier review methods. The major advantage of the approach is the more rigorous standardization of review criteria, which minimizes the likelihood of reviewer bias and increases the ease with which disparate articles can be compared. For a detailed description of the methods used in the Evidence Analysis Process, go to www.andevidencelibrary.com/eaprocess.

Conclusion Statements are assigned a grade by an expert work group based on the systematic analysis and evaluation of the supporting research evidence:

- **Grade I=Good**
- **Grade II=Fair**
- **Grade III=Limited**
- **Grade IV=Expert Opinion Only**
- **Grade V=Not Assignable (because there is no evidence to support or refute the conclusion).**

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bond occurs at the third carbon of the fatty acid chain from the methyl (or omega) end, and in n-6s, the first double bond occurs at the sixth carbon in the fatty acid chain.[8] Figure 1 presents nomenclature and common food sources of dietary fatty acids. It is the differences in chain length and saturation status that dictate their performance in food and cooking, as well as their role in the body and impact on human health and disease risk.

TOTAL FAT VS INDIVIDUAL FATTY ACIDS

Total fat intake has been the primary focus of dietary recommendations, with increased emphasis on the health impact of individual fatty acids in recent years. Total fat intake of 20% to 35% of energy is recommended by the Institute of Medicine and the Food and Agriculture Organization of the United Nations (FAO) and is supported by the 2010 Dietary Guidelines for Americans (DGA).[5,9] The American Heart Association (AHA) and National Cholesterol Education Program recommend 25% to 35% of daily calories from fat.[3,4] These total fat intake recommendations are based on evidence that indicates consumption outside of these ranges is associated with a greater intake of energy and SFA (fat intake >35%) or greater intake in carbohydrate (fat intake <20%); higher intake of carbohydrate leads to increases in plasma triglyceride and reductions in high-density lipoprotein (HDL) cholesterol levels. According to the National Health and Nutrition Examination Survey (NHANES) 2009-2010, 33% of calories in the diets of both women and men came from fat, which is near the recommended upper limit of 35%.[10] It is important to note that individuals often eat more than their energy needs, and examining total fat intake as a percentage of calories might well reflect more fat in the diet than is recommended and necessary.

Although total fat recommendations have been emphasized, for example, low-fat diet (<20%); moderate-fat diet (20% to 35%); or high-fat diet (>35%), the importance of individual fatty acids has been overlooked. Achieving intake of total fat within the recommended range (20% to 35%) is an important goal, but the quality of fat in the diet is equally important. Altering fat consumption, for example, the unsaturated/saturated fat balance, instead of reducing total fat might be more advantageous to health and chronic-disease risk reduction.[11] In tissues, an increase in concentration of one fatty acid corresponds to the reduction of the same magnitude of another fatty acid.[12] The displacement or replacement of the quality of fatty acid is important to consider.

Recent recognition that individual fatty acids have differing effects on health, even within the conventional fat categories, has brought examination of these fatty acids into the forefront. Although this reductionist viewpoint allows for understanding specific fatty acid functions and is important in determining which fatty acids are best for health, examination of the foods containing these fatty acids is also important. Knowledge of the individual fatty acids in foods provides a foundation for the RDN to make whole food recommendations to their clients. The variety of fatty acids in common fats and oils is provided in Table 1. Additional information on individual fatty acids in foods can be found at the US Department of Agriculture's Nutrient Database (http://ndb.nal.usda.gov).[13]

DIFFERING, WIDE-RANGING IMPACTS ON HEALTH

The structure of each fatty acid differs, so it is reasonable that individual fatty acids might have unique impacts on health. The impact of specific fatty acids on disease incidence is difficult to elucidate, as chronic disease develops over many years and is the culmination of many genetic and lifestyle factors. This complexity makes randomized controlled trials of dietary interventions largely impractical, but these trials, coupled with observational, epidemiologic, and mechanistic studies, provide valuable evidence on the health effects of dietary fat and specific fatty acids. Because healthy individuals are the focus of this paper, the impact of fatty acid intake on those with chronic diseases is not discussed. However, despite not having established or diagnosed disease, "healthy" individuals can possess disease risk factors (eg, elevated low-density lipoprotein [LDL] cholesterol, glucose, obesity) that are influenced by specific fatty acid intake. Modulation of known and emerging risk factors, such as chronic low-grade inflammation, through dietary fatty acid intake can beneficially impact development of common diseases, such as cardiovascular disease, diabetes, and depression, as well as emerging diseases, such as Alzheimer's and dementia. For example, evidence from randomized clinical trials and observational studies provide convincing argument that inadequate intake of long-chain n-3 fatty acids is associated with an increased risk of sudden cardiac death.[14] Targeting multiple risk factors through modulation of diet, specifically fatty acids, holds promise for promoting public health and attaining meaningful reductions in disease risk in healthy individuals.

NEED FOR KNOWLEDGE

Fatty acids can no longer be viewed in general categories, such as saturated

Name	Common abbreviation[a]	Nomenclature	Common food sources[b]
Polyunsaturated fatty acids	PUFA		
n-3			
α-linolenic acid	ALA	C18:3	Flax, chia, walnuts, canola oil
Stearidonic acid	SDA	C18:4	GMO[c] soybean oil
Eicosapentaenoic acid	EPA	C20:5	Fish and seafood
Docosapentaenoic acid	DPA	C22:5	Fish and seafood
Docosahexaenoic acid	DHA	C22:6	Fish and seafood
n-6			
Linoleic acid	LA	C18:2	Soybean oil, corn oil, shortening
γ-linolenic acid	GLA	C18:3	Not commonly found in food
Arachidonic acid	ARA	C20:4	Meat, poultry, eggs
Conjugated linoleic acid	CLA	C18:2 (variants)	Ruminant meat and dairy
Monounsaturated fatty acids[d]	MUFA		
Palmitoleic acid		C16:1	Macadamia nuts, blue-green algae
Oleic acid		C18:1	Olive oil, canola oil, beef tallow, lard, avocado
Saturated fatty acids	SFA		
Carprylic acid	MCT[e]	C8:0	Coconut oil, palm kernel oil
Capric acid	MCT	C10:0	Coconut oil, palm kernel oil
Lauric acid	MCT	C12:0	Palm kernel oil, coconut oil
Myristic acid		C14:0	Beef tallow, cocoa butter
Palmitic acid		C16:0	Palm oil, most fats and oils
Stearic acid		C18:0	Meat (lard, beef tallow), fully hydrogenated vegetable oils
***Trans*-fatty acids**	TFA		
Elaidic acid		C18:1, t9	Partially hydrogenated vegetable oils
Vaccenic acid		C18:1, t11	Butterfat, meat

[a]Only fatty acids with common abbreviations are listed.
[b]Food sources of naturally occurring fatty acids are listed (with exception of GMO soybean oil); fortified food and supplement sources can be found in body of paper.
[c]GMO=genetically modified organism.
[d]Only monounsaturated fatty acids (MUFA) in the *cis* configuration are listed. MUFAs with *trans* configuration are listed under *trans* fatty acids.
[e]MCT=medium-chain triglyceride.

Figure 1. Nomenclature and common food sources of dietary fatty acids.

and unsaturated, because individual fatty acids within these categories have different influences on health status and disease risk. For example, both stearic acid (18:0) and palmitic acid (16:0) are saturated fats, differing only in chain length by two carbons, but they appear to have different effects on circulating LDL cholesterol.[15] Another example is the difference between the location of the first double bond on the fatty acid carbon chain. Whether the double bond is located on carbon number 3 or 6 (as in n-3 and n-6 PUFA) makes a

Table 1. Fatty acid profiles of select animal and vegetable fats and oils[a]

Lipid	Quantity	SFA[b]	8:0	10:0	12:0	14:0	16:0	18:0	MUFA[c]	18:1	PUFA[d]	18:2	18:3	EPA[e] DPA[f] DHA[g]	ARA[h]	TFA[i]
Avocado oil	1 Tbsp	11.9	0.0	0.0	0.0	0.0	11.3	0.7	72.6	69.9	13.9	12.9	1.0	0.0	0.0	0.0
Beef tallow	1 Tbsp	46.8	0.0	0.0	0.9	3.5	23.5	17.8	39.3	33.9	3.8	2.9	0.6	0.0	0.0	0.0
Butter[j]	1 Tbsp	53.6	5.1	2.6	2.7	7.8	22.6	10.4	22.0	20.8	3.2	2.9	0.4	0.0	0.0	0.0
Canola oil	1 Tbsp	7.6	0.0	0.0	0.0	0.0	4.4	6.8	65.1	63.5	29.0	19.6	9.4	0.0	0.0	0.4
Coconut oil	1 Tbsp	11.8	1.0	0.8	6.1	2.3	1.1	0.4	0.8	0.8	0.2	0.2	0.0	0.0	0.0	0.0
Corn oil	1 Tbsp	12.9	0.0	0.0	0.0	0.0	10.6	1.8	27.6	27.4	54.7	53.5	1.2	0.0	0.0	0.3
Flaxseed oil	1 Tbsp	9.0	0.0	0.0	0.0	0.1	5.1	3.4	18.5	18.3	67.9	14.3	53.4	0.0	0.0	0.1
Grapeseed oil	1 Tbsp	9.6	0.0	0.0	0.0	0.1	6.7	2.7	16.1	15.8	69.9	69.6	0.1	0.0	0.0	—
Lard	1 Tbsp	36.9	0.0	0.1	0.2	1.3	22.4	12.7	42.5	38.8	10.5	9.6	1.0	0.0	0.0	0.0
Olive oil	1 Tbsp	13.7	0.0	0.0	0.0	0.0	11.2	1.9	72.4	70.7	10.4	9.7	0.7	0.0	0.0	—
Palm oil	1 Tbsp	49.3	0.0	0.0	0.1	1.0	43.5	4.3	37.0	36.6	9.3	9.1	0.2	0.0	0.0	—
Palm kernel oil	1 Tbsp	81.5	3.3	3.7	47.1	16.4	8.1	2.8	11.4	11.4	1.6	1.6	0.0	0.0	0.0	—
Rice bran	1 Tbsp	19.7	0.0	0.0	0.0	0.7	16.9	1.6	39.3	39.1	35.0	33.4	1.6	0.0	0.0	—
Salmon oil	1 Tbsp	19.9	—	—	—	3.3	9.9	4.3	29.0	17.0	40.3	1.5	1.0	31.3	0.7	—
Soybean oil	1 Tbsp	15.7	0.0	0.0	0.0	0.0	10.4	4.4	22.8	22.6	57.7	51.0	7.1	0.0	0.0	0.5

[a]Listed as percent of total fatty acid content. Based on 13.6 g fatty acids/tablespoon (Tbsp). Cells without numbers did not have data in US Department of Agriculture Nutrient Database.
[b]SFA=saturated fatty acid.
[c]MUFA=monounsaturated fatty acid.
[d]PUFA=polyunsaturated fatty acid.
[e]EPA=eicosapentaenoic acid.
[f]DPA=docosapentaenoic acid.
[g]DHA=docosahexaenoic acid.
[h]ARA=arachadonic acid.
[i]TFA=*trans*-fatty acid.
[j]Butter contains 16% water and therefore the percentages are unable to be directly compared with percentages of the other fats/oils.

remarkable difference in biological function (eg, vasoconstriction vs vasodilation). Therefore, understanding the breadth of information while delineating specifics about individual dietary fatty acids is essential. RDNs have the opportunity and responsibility to translate research into practice for the population at large. National organizations specializing in evidence-based dietary recommendations are valuable resources. Table 2 summarizes intake recommendations for dietary fatty acids.

This section of the position paper includes the results of a systematic review of literature conducted using the Academy's Evidence Analysis Process and information from the Academy's Evidence Analysis Library. In this process, expert work groups identified dietetic practice–related topics, prioritized and selected questions, performed systematic reviews, and developed and rated a conclusion statement for each question. The workgroups used the Academy's process to answer a total of three questions related specifically to dietary fatty acids.

PUFAs

Specific Fatty Acids and Food Sources

PUFAs are triglycerides that contain fatty acids with two or more (poly) double bonds. PUFAs are liquid at room temperature and, because they are not solid fats, they are often referred to as oils. In recent decades, researchers have discovered that the function of PUFAs in human nutrition differ based on the fatty acid structure. The most common PUFAs are n-3 and n-6, and because humans cannot synthesize them, they are considered essential dietary nutrients.

Dietary fatty acids are oxidized as fuel; incorporated in plasma phospholipids, lipoprotein particles, and cell membranes; or stored as triglycerides. The most abundant PUFAs in the diet are α-linolenic acid (ALA; 18:3n-3) and linoleic acid (LA; 18:2n-6); both of these fatty acid hydrochains are 18 carbons in length and are considered the parent fatty acids for n-3 and n-6, respectively.

n-3 and n-6 fatty acids are long-chain fatty acids (18 carbons and longer) and are distinguished within each group by chain length. In the n-3 family, ALA and stearidonic acid (SDA; 18:4n-3) are considered the shorter-chain n-3s, while eicosapentaenoic acid (EPA; 20:5n-3), docosapentaenoic acid (DPA; 22:5n-3), and docosahexaenoic acid (DHA; 22:6n-3) are the longer-chain n-3s. ALA occurs in plant foods such as nuts, particularly walnuts and flax, chia and hemp seeds, and

Table 2. Dietary fatty acid intake recommendations[a]

Organization	Total fat (%)	PUFA[b] (n-3)	PUFA[c] (n-6)	MUFA[d]	SFA[e]	TFA[f]
2010 Dietary Guidelines for Americans	20-35	Use oils to replace solid fats where possible Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry	Use oils to replace solid fats where possible	Replace solid fats with MUFAs, possibly through adopting a Mediterranean dietary pattern	<10%	As low as possible
US Dietary Reference Intake	20-35	AI[g] for ALA[h] is 1.1-1.6 g/day or 0.6%-1.2% of intake; up to 10% can be EPA[i]+DHA[j]	5%-10% of intake; AI for LA[k] is 12-17 g/day		As low as possible	As low as possible
Academy of Nutrition and Dietetics	20-35	0.6%-1.2% of intake as ALA; 500 mg EPA+DHA/day	3%-10% of intake	15%-20% of intake	Goal of <7%, maximum intake of 10%	<1%
American Heart Association	25-35	Eat fish (particularly fatty fish) at least 2×/week	LA as 5%-10% of intake	Replace animal fats in the diet	<7%	<1%
WHO/FAO[l]	20-35	0.5%-2% of intake; minimum 0.5% from ALA; 250 mg EPA+DHA/day	AI for LA is 2%-3% of intake		Intake at no more than 10% of energy; should be replaced with PUFAs	<1%
EFSA[m]	20-35	AI for ALA is 0.5% of intake; 250 mg EPA+DHA/day	AI for LA is 4% of intake		As low as possible	As low as possible
ISSFAL[n]		ALA 0.7% of intake; minimum 500 mg EPA+DHA/day	AI for LA 2% of intake			

[a]Percentages based on total energy intake that meets the needs of the individual.
[b]PUFA=polyunsaturated fatty acids.
[c]Values reported for total n-6 PUFA unless indicated.
[d]MUFA=monounsaturated fatty acids.
[e]SFA=saturated fatty acids.
[f]TFA=*trans*-fatty acids.
[g]AI=Adequate Intake. AI is a recommended average daily nutrient intake level, based on experimentally derived intake levels or approximations of observed mean nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate. An AI is established when there is insufficient scientific evidence to determine an Estimated Average Requirement.
[h]ALA=α-linolenic acid.
[i]EPA=eicosapentaenoic acid.
[j]DHA=docosahexaenoic acid.
[k]LA=linoleic acid.
[l]WHO/FAO=World Health Organization/Food and Agriculture Organization.
[m]EFSA=European Food Safety Authority.
[n]ISSFAL=International Society for the Study of Fatty Acids and Lipids.

vegetable oils, such as canola and soybean oils. SDA is not typically found in food; very small amounts occur in fish and uncommon plant sources (eg, echium, black currant). In the United States, the soybean has been genetically modified to produce oil containing SDA n-3; the SDA-rich soybean oil has Generally Recognized as Safe approval in the United States.[16] EPA, DPA, and DHA occur in fatty fish and seafood, and the best sources are salmon, sardines, tuna, herring, and trout. An abundance of foods fortified with EPA and/or DHA from either marine or algal sources are now available; called *functional foods*, examples include soy milks and juices, cooking oils, spreads, snack foods and even fish sticks, where fortification is in the breading. Availability of functional foods varies by region. Eggs can contain shorter or longer-chain n-3s, as determined by the chicken feed. Most of the research on long-chain n-3s has used EPA and DHA. DPA is a lesser known n-3; it occurs in fatty fish along with EPA and DHA but is particularly rich in seal meat, a traditional food of Eskimos and other cultures. Interest in new or unique health contributions from DPA is growing. The predominant and best understood n-3 fatty acids are ALA from plants and EPA and DHA from fish and seafood.

In the n-6 family, LA and γ-linolenic acid (GLA; 18:3n-6) are shorter-chain fatty acids, and arachidonic acid (ARA; 20:4n-6) is a longer chain. Longer-chain n-3 and n-6 fatty acids ($\geq$20 carbons) are sometimes called highly unsaturated fatty acids. LA occurs in plant foods; the richest sources are soybean, corn, and safflower oils. GLA is not found to any great extent in foods and is available through dietary supplements from borage and evening primrose oil. The best sources of ARA are meat, poultry, and eggs.

Conjugated linoleic acid (CLA) is an 18-carbon n-6 PUFA that occurs in small amounts in the milk (about 0.3% to 0.6% total dairy fat) and meat of ruminants. A conjugated fatty acid is one in which the double bonds occur on adjacent carbons. The two major CLA forms are *cis*-9, *trans*-11 18:2 (c9, t11 18:2) and the *trans-10, cis-12* 18:2 (t10,c12 18:2). There is usually more c9, t11 18:2 in food, while supplements typically contain an equal mixture of both forms. For the purpose of food labeling, CLA is classified as an n-6 fatty acid and not a TFA. One pilot study reported intake of CLA from foods at 94.9 mg/day in healthy adults in North America.[17]

Within the n-3 and n-6 families, the shorter-chain and longer-chain fatty acids make different contributions to human nutrition. ALA n-3 is predominantly used as a fuel source for β-oxidation and only a small portion is converted to longer-chain fatty acids.[18] Desaturase and elongase enzymes are required for the conversion of shorter-chain to longer-chain fatty acids. It was originally believed that the shorter-chain fatty acids readily converted to the longer chains, but contemporary research indicates that n-3 conversion from short chain to long chain in humans is very limited; ALA converts to EPA at a rate of 5% to 15%, and $<$1% of ALA reliably converts to DHA.[19,20] Rate of conversion of these essential fatty acids is determined by the presence of and competition for the desaturase and elongase enzymes and is further influenced by other factors, including sex, diet, health status, and genetics.[21-24] This explains the interest in genetically engineered SDA; more SDA than ALA is converted to EPA because with SDA, the rate-limiting conversion step requiring delta-6 desaturase is bypassed. Conversion, however, is limited; two published human trials reported that about 17% of SDA converts to EPA and there is no conversion from SDA to DHA.[25,26] As a result, SDA is best considered an EPA-only precursor.[27]

Individuals who follow a vegetarian or vegan diet and include no marine foods in their diet will consume ALA because of its wide distribution in plant-sourced foods. Through conversion, ALA will provide some EPA but little, if any, DHA.[19] Emerging science suggests there is individual variation in conversion rate of fatty acids, influenced by genetics and dietary habits, including the presence of other fatty acids in the diet. Those who follow a vegetarian diet might be more efficient at n-3 conversion, but this has not been confirmed.[22,28]

As 20-carbon PUFAs, EPA n-3 and ARA n-6 function as eicosanoids. Eicosanoids are bioactive mediators and precursors for prostaglandins, thromboxanes, and leukotrienes. Eicosanoids are considered hormone-like substances because they are produced when stimulated, rapidly utilized and metabolized, and not stored in cells. Prostaglandins, thromboxanes, and leukotrienes are involved with a myriad of physiological and homeostatic activities, including inflammation modulation, platelet aggregation, cell growth and proliferation, smooth muscle contraction, and vasoconstriction and vasodilatation. EPA, ARA, and DHA are also involved with gene expression, cytokine activity, cell signaling, and immune modulation. The eicosanoids produced from EPA and ARA differ from each other in structure, and they function in complementary metabolic pathways. Eicosanoids produced from ARA have high biological activity and are considered more potent than EPA-derived eicosanoids. For example, prostaglandins produced from ARA promote inflammation, serve as vasoconstrictors, and stimulate platelet aggregation, and prostaglandins produced from EPA function as vasodilators and anti-aggregators. Eicosanoids derived from ARA are mostly pro-inflammatory but have some anti-inflammatory effects. A balance of eicosanoid synthesis in tissues is optimal. Resolvins and neuroprotectins, derived from EPA and DHA, respectively, are a new class of modulators that appear to have anti-inflammatory and neuroprotective activity.[8,29] Finally, DHA is a structural component of red blood cell membranes and exists in higher concentrations in retina tissue, neuronal cells, liver, and testes.[30]

PUFAs FROM SUPPLEMENTS

n-3 supplements are widespread in the marketplace. The most common supplement forms of ALA are flax and chia seed oils. SDA supplements from algae are being synthesized. EPA and DHA supplements are made from fish oil (typically anchovy, menhaden, and salmon oils), cod liver oil, krill oil, and squid (calamari) oil. Generally Regarded as Safe status of up to 3 g/day of EPA and DHA from fish oil in healthy people was obtained in 1997.[31] Vegetarian sources of EPA and DHA are available from algal sources, and genetically engineered supplements are under development. GLA n-6 supplements are most often sourced from

borage and evening primrose oils. CLA, the conjugated n-6 supplement, is synthesized from vegetable oils.

Current Intake of PUFAs

NHANES 2009-2010 provided information on dietary intake of n-3s and n-6s among adults.[10] NHANES reported mean daily intake of ALA among males was 1.77 g and 1.38 g among females. Mean daily intake of EPA among men was 40 mg and 30 mg for women. Mean daily intake of DHA was 80 mg for men and 60 mg for women. There is little difference between mean intakes by sex, race/ethnicity, and income. These values have remained quite stable as reported in the last NHANES data set (NHANES III 1988-94). For n-6s, the mean daily intake of LA among men was 17.84 g and among women, 13.33 g. This represents an increase as previous NHANES III data reported a mean daily consumption of LA at 14.1 g. Daily consumption of ARA was reported as 180 mg for men and 120 mg for women.

Economic disappearance data for each year from 1909 to 1999 was used to estimate consumption of food commodities per capita.[32] With regard to n-3, availability of ALA increased from 0.39% to 0.72% of energy. In 1909, the greatest dietary sources of ALA were fats, dairy, pork, beef, and grains; by 1999, sources were soybean oil, dairy, fats, other oils, beef, shortening, and nuts. Availability of EPA and DHA has remained virtually unchanged. Results for n-6, however, were strikingly different. The availability of LA increased 158%, from 2.79% of energy in 1909 to 7.21% of energy in 1999 (based on current fatty acid analysis). In 1909, the primary food sources of LA were fats, pork, grains, shortening, oils, and nuts. Ninety years later they were soybean oil, fats, shortening, other oils, poultry, and nuts. The availability of ARA has remained unchanged.

Dietary Recommendations for PUFAs

The 2005 Daily Reference Intakes recommend 5% to 10% energy from n-6 and 0.6% to 1.2% of energy from n-3, but it did not set a Recommended Dietary Allowance (RDA) or Estimated Average Requirement for either. The report did provide an Adequate Intake (AI) for n-6 and n-3. The AI for n-6 is 17 g/day and 12 g/day for men and women 19 to 50 years, respectively; the AI for ALA is 1.6 g/day and 1.1 g/day for men and women age 19 to older than 70 years, respectively.[9] These intakes are equivalent to 5% to 6% energy from LA and 0.5% energy from ALA. Recognizing that differences in metabolic function among n-3s existed, the Daily Reference Intake report noted that EPA and DHA could provide up to 10% of ALA intake. It 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.

The DGA recommend that adults consume 8 oz or more of seafood per week, or about 20% of the recommended intake of protein foods from a variety of seafood (fish and shellfish). For primary prevention of coronary heart disease (CHD), the AHA,[33] the National Heart Foundation of Australia,[34] and the United Kingdom Scientific Advisory Committee,[35] all recommend at least two servings of fish per week, preferably fatty fish, providing an average daily intake of 450 to 500 mg EPA and DHA. The same recommendation of two or more servings per week is suggested by the American Psychiatric Association[36] for support of mental health. An increased intake of nuts and seeds is also recommended. Because these foods have greater caloric density, they should replace servings of meat and poultry and/or be consumed in small portions.[5]

The World Health Organization/Food and Agricultural Organization established an upper Acceptable Macronutrient Distribution Range (AMDR) for total PUFAs at 11% energy. They recommend total n-3 intake of 0.5% to 2% energy, with a minimum requirement of >0.5% energy from ALA to prevent deficiency, and 250 mg EPA and DHA/day for men and nonpregnant women.[6] For LA n-6, they recommend an estimated average requirement of 2% energy, an AI of 2% to 3% energy, and an AMDR of 2.5% to 9%. The lower levels in the range are to prevent deficiency, and the upper level is suggested as part of healthy diet for long-term cardiovascular health. The European Food Safety Authority recommends an AI of 0.5% energy for ALA and an AI of 250 mg EPA and DHA for primary prevention in healthy adults. For n-6, the European Food Safety Authority recommends an AI of 4% energy for LA; they do not give a Dietary Reference Value for ARA.[37] The International Society for the Study of Fatty Acids and Lipids recommends a healthy intake of ALA n-3 as 0.7% energy and for cardiovascular health, a minimum of 500 mg/day of EPA and DHA n-3. They also recommended an AI of LA n-6 at 2% energy.[38]

In 2004, the Food and Drug Administration (FDA)[39] approved a Qualified Health Claim for n-3 fatty acids and heart disease, stating: "Supportive but not conclusive research shows that consumption of EPA and DHA n-3 fatty acids may reduce the risk of CHD. One serving of [name of food] provides [x] grams of EPA and DHA n-3 fatty acids." The FDA also approved a Qualified Health Claim for nuts and heart disease as well as walnuts and heart disease, stating: "Scientific evidence suggests but does not prove that eating 1.5 ounces per day of most nuts [such as *name of specific nut*] as part of a diet low in saturated fat and cholesterol may reduce the risk of heart disease." The FDA made the same statement for walnuts.[40]

Based on recent literature, increasing consumption of PUFAs with a particular focus on increasing n-3 intake (ie, striving to consume two or more servings of fatty fish per week to provide at least 500 mg EPA and DHA per day, and aiming toward an intake of at least 0.5% to 2% energy as n-3 fatty acids and 5% to 10% energy as n-6 fatty acids per day) is desirable.

PUFAs and Health

n-3 Fatty Acids. The role of n-3 fatty acids in heart health is one of the most studied areas of nutrition science. Observations specific to long-chain n-3 fatty acids were first made in the 1960s when epidemiologists observed that Greenland Eskimos and native Alaskans had a low incidence of CHD, although they consumed large amounts of fat. In the 1970s, death rates from heart disease among males aged 45 to 64 years were 40% in the United States and nearly 35% in Denmark, yet only 5% in Greenland.[41] Compared to the Danes, Greenland Eskimos had higher blood levels of saturated fat and lower levels of PUFAs, cholesterol, and triglycerides, even though both groups ate about the

same amount of total fat (Danes 40%; Eskimos 37%). The Greenland Eskimos also had considerably higher blood levels (up to 16%) of the n-3 fatty acid EPA. The researchers noted more of a qualitative than quantitative difference with respect to fatty acid composition in the diets.[42] Based on these findings, as well as observations in countries that consume large amounts of fish such as Japan, associations between fish consumption and overall cardiovascular mortality and sudden cardiac death have been noted. According to a 2011 meta-analysis, observational and randomized clinical trial evidence suggests that fish or fish oil consumption can also reduce inflammation, improve endothelial function, normalize heart rate variability, improve myocardial relaxation and efficiency, and, at higher doses, limit platelet aggregation.[43] In addition, inverse relationships between blood levels of n-3 and risk for sudden cardiac death have been observed.[44,45] Habitual fish consumption is associated with lower risk of CHD and ischemic stroke and, among the general healthy population, those who consume a modest amount of fish or fish oil providing ≥250 mg EPA and DHA/day have a 36% lower risk of fatal heart disease.[14] These benefits have not been shown among those who eat commercially fried fish or fish sandwiches; this might be a reflection of the type of fish commonly fried, as it tends to be lower in EPA and DHA content. Nonetheless, researchers have shown that consumption of nonfried fish is associated with higher EPA and DHA blood levels.[46,47]

In addition to providing long-chain n-3s, fish provides lean protein, vitamins, and minerals. Due to the concern of potential methylmercury contamination in fish, in 2004 the US Department of Agriculture[48] recommended pregnant women and children under 5 years old limit fish consumption and avoid the following types of fish: shark, swordfish, king mackerel, and tilefish. Since then, an extensive risk-to-benefit analysis concluded that, with the exception of select fish, such as those included in the US Department of Agriculture advisory, the health benefits of fish consumption outweigh potential risks.[49] At this time, however, the US Department of Agriculture advisory remains in effect. Although fish oil supplements have been shown to be equally effective as fish at increasing tissue levels of EPA and DHA,[50] it is unclear whether benefits observed in those with habitual fish consumption can be fully reproduced with refined fish oil supplements. Clinical research has shown that supplementing with refined fish oil can help reduce triglycerides and improve blood pressure and heart rate levels.[51-55]

The benefits of ALA independent of EPA and DHA are not well documented; but, because it is plant sourced, ALA is more readily available in the diet and is a particularly important source of PUFAs for vegetarians. Studies investigating cardiovascular benefits of ALA in the healthy population have shown mixed or inconsistent results.[56,57] Diets rich in ALA have been reported to lower lipid levels, reduce vascular inflammation, and reduce blood pressure in those with elevated cholesterol levels.[58,59] Interestingly, an 11-country study in Europe reported reductions in CHD in countries that consumed ALA-rich canola oil compared to countries that consumed primarily sunflower oil, which contains no ALA.[60] Walnuts are a particularly rich source of ALA and several studies have reported benefits from ALA by including walnuts and a variety of nuts in the diet.[61-63] Plant-sources of n-3s have also been shown to have a protective effect on bone metabolism.[64] However, a cardioprotective blood level of long-chain n-3s (EPA and DHA) cannot be achieved by consuming ALA alone.[65,66]

Health benefits of SDA independent of EPA and DHA are unclear. Although little is known about the biological effects of SDA, incorporating genetically engineered SDA into the food supply is motivated by barriers to fish consumption and global concerns for sustainability. Modulating lipid levels with EPA and DHA require relatively high doses; preliminary studies indicate that consuming SDA at a bioequivalent dose of EPA (approximately 4 g SDA= approximately 1 g EPA) has little impact on circulating lipids.[26,27]

Depression is an increasingly common mental health disorder. It can be chronic or recurrent, and the impact on individuals, families, caregivers, communities, and the work force is significant. It is the leading cause of disability worldwide and is predicted to be among the top three leading causes of burden of disease by 2030.[67] An association between fish consumption and incidence of major depression was first reported in 1998.[68] Since then, a link between low levels of long-chain n-3 fatty acids and depression has been observed in a number of trials.[69-73] The recommendation by the American Psychiatric Association to consume fatty fish at least twice a week reflects these findings.[36]

Some level of cognitive decline is considered normal with aging and lower levels of DHA have been observed in individuals with cognitive decline and Alzheimer's disease.[74-77] The results of studies examining the relationship between long-chain n-3s on cognitive decline have been mixed.[78-80] The Academy's Evidence Analysis Library recently examined this question (see Figure 2); 6 of the 14 studies analyzed reported positive associations between EPA, DHA, or fish consumption and decreased risk of cognitive decline. Results varied based on amount and source of EPA and DHA consumed and, perhaps more importantly, the state of cognitive health at the outset of the study. A study evaluating healthy middle-aged adults showed that DHA but not ALA or EPA was associated with better performance on cognitive tests, such as nonverbal reasoning, mental flexibility, memory, and vocabulary.[81]

Essential fatty acids are potentially potent anti-inflammatory agents and, as such, clinical evidence has shown a role for EPA and DHA in reducing symptoms of rheumatoid arthritis.[82,83] Currently, there is interest in the role of fatty acids in immune health, in part because long-chain fatty acids appear to influence proteins directly involved with immune cell activation.[29,84] Despite promising results from animal models and cultured tumor cell lines, no clear relationship between dietary intake of EPA and DHA and risk for cancer have been demonstrated.[65]

n-6 Fatty Acids. LA is the most highly consumed PUFA in the Western diet and is found in virtually all commonly consumed foods. LA is the metabolic precursor of ARA and there is concern that LA consumption can enrich tissues with ARA and contribute to overproduction of bioactive eicosanoids, thereby increasing inflammatory markers and/or chronic disease risk.

Question: What is the effect of eicosapentaenoic acid (EPA)/docosahexaenoic acid (DHA) from supplemental and dietary sources on cognitive decline in adults?

Conclusion: Based on 14 studies (1 case-control, 3 cross-sectional, 6 prospective cohort, and 4 randomized controlled trials) primarily of healthy elderly subjects or those matched for incident dementia, results are mixed. Six of the 14 studies found a positive association of EPA, DHA, or fish consumption and a decreased risk of cognitive decline. The majority of the studies included large populations of elderly subjects measuring either blood levels of EPA and DHA and fish consumption. Results vary based on dose of supplementation or dietary consumption of fish, EPA, or DHA. Further research is needed to determine whether EPA, DHA, and fish consumption has a protective effect for all-cause dementia. There is some evidence that ApoE e4 carrier status should be factored into future studies.

Grade[a] II=Fair

Question: What is the effect of linoleic acid (LA) on the risk of diabetes mellitus and/or insulin resistance in adults?

Conclusion: There is fair evidence that LA status is inversely associated with risk of type 2 diabetes mellitus. Limited studies have shown an inverse relationship between LA status and risk factors for type 2 diabetes mellitus including insulin resistance, metabolic syndrome, inflammation, nerve function/neuropathy, and the liver enzyme, alanine transaminase, as a marker of liver fat.

Grade II=Fair

Question: What is the effect of conjugated linoleic acid (CLA) on body composition and weight loss in healthy adults?

Conclusion: Fair evidence indicates that 3 to 6 months of CLA supplementation results in a decrease in fat mass and an increase in fat-free mass in healthy adults; however, fair evidence indicates that CLA does not affect body weight.

Grade II=Fair

[a]Academy of Nutrition and Dietetics Evidence Analysis Library Grade Definitions and Chart: http://andevidencelibrary.com/content.cfm?content_id=11.

Figure 2. Questions, conclusions, and grades from the Dietary Fatty Acids Evidence Analysis Project of Academy of Nutrition and Dietetics' Evidence Analysis Library.

However, a 2011 review reported that decreasing dietary LA up to 90% did not significantly correlate with change in ARA tissue levels and, similarly, increasing dietary LA levels did not increase ARA levels substantially. A 2012 systematic review of randomized controlled trials that assessed the impact of LA on biologic markers of chronic inflammation among healthy adults also reported no evidence that LA increased inflammatory markers.[85] Nevertheless, emerging evidence indicates significant racial differences in conversion rate of LA to ARA, particularly between those of European and African descent, making this an important area for further learning.[86] With regard to GLA, a linear relationship between dietary GLA and increases in plasma and serum phospholipid ARA levels has been measured.[87]

An AHA advisory published in 2009 summarized evidence on n-6 consumption, particularly LA and CHD risk. It reported that consuming 5% to 10% of energy from n-6 PUFAs reduced the risk of CHD relative to lower intakes.[88] A more recent meta-analysis, however, challenges this conclusion.[89]

Evidence from animal studies suggests a beneficial role for CLA for weight loss. However, results from supplementation trials using intake levels of CLA unattainable from food (1.8 to 4.5 g/day) have been mixed.[90-92] The Academy's Evidence Analysis Library recently reviewed this question (see Figure 2). Impaired insulin sensitivity has been reported in some but not all studies using CLA supplementation in those with obesity and metabolic syndrome.[91-93]

MUFAs

Specific Fatty Acids and Their Food Sources

MUFAs are present in a wide variety of foods, including vegetable, nut, and seed oils, as well as meats and dairy products. By definition, MUFAs contain one double bond; this double bond varies in location but is frequently located at carbon nine from the methyl end of the fatty acid hydrocarbon chain. MUFAs also differ based on chain length, and although these fatty acids can exist from 10 to 32 carbons, the majority exist as an 18-carbon fatty acid in the form of oleic acid.

Oleic acid contains one double bond at carbon nine, with the double bond existing in the *cis* position (18:1 c-9). One of the most abundant fatty acids found in foods, oleic acid is present in high amounts in olive and canola oils (see Table 1), as well as in avocados and almonds (9.8 g/avocado half and 8.8 g/1 oz almonds, respectively). Although these food sources are generally well known, at least 30% of the fatty acids in beef tallow, lard, and palm oil are oleic acid, and >20% of the fatty acids in both soybean and corn oil are oleic acid.[13] As a result, oleic acid is the most abundantly consumed fatty acid in the American diet at 12% energy intake.[10]

Several other MUFAs exist but are present in foods in low quantities. One MUFA that naturally exists in lower quantities in foods is erucic acid, a

22-carbon fatty acid that contains one double bond at carbon 9 (22:1, c-9). Food sources of erucic acid include rapeseed and other plants from the Brassicaceae family, including kale and broccoli. Canola oil is produced from rapeseed, but removal of erucic acid through genetic modification has essentially eliminated the erucic acid content in canola oil and subsequently from the diet.

In addition to n-9 MUFAs, an n-7 MUFA with a purported health benefit is palmitoleic acid, containing 16 carbons with one double bond at carbon 7 from the methyl end (16:1, c-7). Palmitoleic acid is not commonly found in foods, but is a product of palmitic acid (16:0) metabolism in the body. Foods that naturally contain palmitoleic acid include certain blue-green algae, macadamia nuts (3.7 g/oz; 17% of fat content), and sea buckthorn oil. Although the fatty acids mentioned have one double bond that exists in the *cis* formation, MUFAs can exist in the *trans* conformation as a result of industrial hydrogenation; most often these TFA exist as 18:1, t-9. TFAs will be discussed in the *trans*-fat section of this article. Although additional MUFAs exist, the quantities consumed through diet are negligible and therefore will not be discussed.

MUFAs from Supplements

Supplementation with MUFA is not common practice and is not supported by authoritative bodies. However, MUFAs are available in supplement form. Despite its abundance in various foods and oils, oleic acid from olive oil is marketed as a dietary supplement. Given the quantity of oleic acid in common food oils (9.6 g per Tbsp olive oil; 8.3 g per Tbsp canola oil; 3 g per Tbsp soybean oil), supplementing with olive oil does not provide appreciable quantities compared to the common daily diet. Oleic acid from olive oil can also be found in supplements containing the combination of n-3, n-6, and n-9 fatty acids; given the abundant supply of both n-6 (18:2) and n-9 (oleic acid; 18:1) in the diet, the supplemental use of this combination is not supported. In addition to oleic acid, palmitoleic acid is currently being marketed as an n-7 fatty acid supplement with claims of preventing or reducing heart disease.

Current Dietary Intake of MUFAs

NHANES 2009-2010 reports the dietary intake of MUFA for both men and women was 12% of total energy. This rivals SFA intake (11%) and is nearly double that of PUFA intake (7%).[10] Of all the fatty acid categories, MUFAs are consumed the most, comprising 36% of total fat intake. The majority (93%) of MUFA consumption is oleic acid at approximately 27 g/day; second to this is palmitoleic acid at 1.2 g/day. MUFA intake has remained relatively stable with an average daily intake of 30.9 g in 2001-2002 vs 28.7 g in 2009-2010. No differences in MUFA intake currently exist between sex, race, and income. Global consumption of MUFA is variable but approximates intake in the United States.[94]

MUFA Recommendations

MUFA intake recommendations by authoritative bodies exist as general guidelines. There is no AMDR for MUFAs. Although the DGA do not recommend a specific amount of MUFA to consume each day, they do recommend replacing solid fats with oils rich in PUFAs and MUFAs, possibly through adopting a Mediterranean dietary pattern.[5] Similarly, the AHA's 2006 Diet and Lifestyle Recommendations for cardiovascular disease (CVD) risk reduction in the general population stated PUFAs and MUFAs should replace animal fats in the diet, with a total fat intake of 25% to 35% of energy intake.[3] To date, the most specific recommendation for MUFAs has been the Adult Treatment Panel III (2004) recommendation, which states up to 20% of energy intake as MUFA, thereby comprising a majority of the recommended 25% to 35% of total fat intake.[4] No specific recommendations exist for MUFA intake with regard to cancer or diabetes prevention.

Most US authoritative bodies have not provided percentage intake recommendations for MUFA; instead, appropriate consumption levels need to be extrapolated from the quantity of fat remaining from total fat intake recommendations after PUFA and SFA recommendations are met. Indeed, a recent FAO report indicated that MUFA intake be calculated by difference (Total fat [%E]−SFA [%E]−PUFA [%E]−TFA [%E]).[6] This quantity determined through calculation by difference allows for up to 15% to 20% of total energy. While considering intake recommendations for total fat and fatty acids (discussed in their respective sections), one can extrapolate that MUFAs can comprise 9% to 29% of energy in our diet, assuming current intake levels of saturated and *trans* fat. For example, assuming no change from current consumption of saturated and *trans* fat (approximately 12% combined), then MUFA intake should not be more than 17% of energy in order to avoid consumption above intake recommendations for total fat. This is important, as excess consumption of energy-dense nutrients, such as fatty acids, can lead to weight gain and, depending on type of fatty acid, be detrimental to health. Based on currently available information, consumption of MUFA at a moderate level (15% to 20%) to account for appropriate PUFA intake, while keeping within 20% to 35% of energy as fat is desirable.

MUFAs and Health

Although current MUFA intake at 12% of energy falls within total fat recommendations and supports recommended PUFA intake, investigating the impact of MUFA in the diet is warranted. This is important because specific intake recommendations for MUFA are limited, specific health benefits of MUFAs are unclear, and recent emphasis on olive oil consumption by health professionals and the media can result in increases in MUFA intake. Understanding the role of MUFAs within the context of substitutions for other macronutrients is also important. With consumption of oleic acid at 93% of total MUFA intake, oleic acid is the primary focus in this section.

Oleic acid can be synthesized in vivo, so measuring serum oleic acid as a marker of health does not reflect intake. Although there is an inability to assess MUFA status, MUFA intake has been linked to alterations in markers of health and disease, such as reducing LDL cholesterol, triglycerides, total cholesterol to HDL ratio, and increasing HDL cholesterol.[95] In the context of macronutrient replacement, oleic acid lowers total and LDL cholesterol when it replaces SFA (12:0 through 16:0 SFAs).[96] Compared to carbohydrate, MUFA decreases

triglycerides, increases HDL cholesterol, and is inversely related to total-to-HDL cholesterol ratio. In addition, compared to diets with ≤12% MUFA, dietary regimens with high amounts of MUFA (>12%) resulted in lower fat mass, systolic blood pressure, and diastolic blood pressure.[97]

Despite evidence reporting health benefits from MUFA consumption, some recent studies have questioned these benefits. From a pooled analysis of 11 cohort studies, Jakobsen and colleagues reported in 2009 that PUFAs are a preferred energy source over both MUFA and carbohydrate; the analysis did not show that MUFA provided cardioprotection.[98] In addition, when coronary mortality was assessed over 30 years, serum MUFA levels were positively associated with coronary death.[99] In contrast, however, there is a large body of evidence reporting health benefits from consuming a Mediterranean diet (ie, a dietary pattern that includes olive oil at approximately 20% of energy intake).[100-102] Recent evidence also suggests benefits from olive oil consumption for obesity, according to a 14-point screener for adherence to the Mediterranean diet (PREDIMED [Prevención con Dieta Mediterránea] trial).[103] The finding that, compared to a low-fat diet, a Mediterranean diet enriched with either olive oil or nuts reduced the incidence of major cardiovascular events[104] supports potential health benefits from olive oil; it should be noted that although the subjects in this study were free of heart disease, they were at high cardiovascular risk and might not be considered healthy adults, which is the focus of this article. Finally, and as recognized in the previously mentioned PREDIMED trial,[103] olive oil is only one component of the Mediterranean diet. Because the dietary pattern also emphasizes vegetables and fruits, n-3 fatty acid-rich foods, nuts, low-fat dairy, and moderate red wine intake, reported benefits of a Mediterranean diet on CVD risk factors cannot be attributed solely to the MUFA content. Indeed, nut consumption has been shown to reduce total cholesterol, LDL cholesterol, and postprandial hyperglycemia compared to meals high in carbohydrates.[61,105]

In summary, MUFA consumption can be beneficial when replacing carbohydrate and saturated fat, but not as beneficial when replacing PUFAs. Although MUFA is shown to have positive impact on surrogate markers, the potential impact of MUFA intake alone on disease outcomes, such as CVD or diabetes, remains unclear. Further understanding of the precise role of MUFAs on health and disease when consumed within the context of an eating pattern (eg, Mediterranean diet) is warranted. Although most of the research on MUFAs has focused on risk of CVD and associated biomarkers, the relationship between MUFAs and cancer has been investigated. The American Cancer Society guidelines state that olive oil is not associated with an increased risk of cancer and likely has a neutral effect on cancer risk.[106]

SFAs

Specific Fatty Acids and Food Sources

SFAs are fatty acids that are fully hydrogenated. SFAs do not contain double bonds between carbon atoms in the fatty acid hydrocarbon chain and therefore have a linear chain, a structural property that allows the individual fatty acids to tightly pack and exist at a solid state at room temperature. SFAs vary in length, but most commonly exist in the food supply between 12 and 18 carbons: lauric acid (12:0), myristic acid (14:0), palmitic acid (16:0), and stearic acid (18:0). Less common in the food supply are shorter-chain SFAs, specifically caprylic (8:0) and capric (10:0). These are medium-chain triglycerides (MCTs), in the category of fatty acids that are 8-12 carbons in length.

SFAs originate primarily from animal sources, including meats, eggs, and butter, or from processed food products containing naturally saturated vegetable oils. Animal fats such as butter, lard, and beef tallow predominantly contain palmitic acid (16:0) and stearic acid (18:0) (see Table 1). Specifically, food fats high in stearic acid (18:0) include beef tallow (19% of fat as 18:0) and cocoa butter (33% of fat as 18:0). It should be noted that stearic acid does not negatively impact serum cholesterol levels; as a result, foods high in stearic acid can affect health in ways different from other SFAs.[107] Although the primary sources of SFAs are animal products, tropical vegetable oils such as palm and palm kernel oil are commonly used in processed foods, principally because of their physical properties. Although palm oil and palm kernel oil both originate from the oil palm tree, these oils have different fatty acid profiles; palm oil contains 49% of fat as SFA (primarily 16:0) and palm kernel oil contains 82% of fat as SFA (primarily 12:0). In comparison, 87% of the fatty acids in coconut oil (from the coconut palm) are saturated, with 12:0 in the highest concentration. Although coconut oil is not commonly used in processed foods, new food products on the market (eg, milk, spreads, yogurt) containing coconut oil are touting the purported health benefits of MCTs (see section on SFAs and Health). In addition to SFA that occur naturally in foods, vegetable oils are industrially hydrogenated (the process of adding hydrogen atoms to unsaturated bonds to create saturated bonds) to produce fats that have properties ideal for food production. In this process of creating fully hydrogenated fatty acids (SFA), partially hydrogenated fatty acids (TFAs) are also produced.

SFAs from Supplements

SFAs are not commonly marketed in supplement form; MCTs are an exception. MCT supplements contain primarily 8:0 and 10:0 fatty acids in capsule or liquid form. However, some MCT supplements are derived from coconut oil. More than 50% of the fatty acids in coconut oil are MCTs (58.7% are 6 to 12 carbons in length) and due to this are marketed as having health-promoting properties. It is important to note that supplemental MCT oil is used for medical nutrition therapy in patients who lack the ability to properly metabolize longer-chain lipids. Although the MCT oil used for patient care often originates from coconut or palm oil and might contain primarily 12:0 (rather than 8:0 or 10:0) as its fatty acid source, individuals with a medical indication for MCTs differ from the needs of the healthy individual. Caution should be taken when considering MCT supplements as the impact of the different MCT fatty acids on human health are not fully elucidated and many over-the-counter supplements do not identify which fatty acids they contain (see section on SFAs and Health).

Current Dietary Intake of SFAs

On average, both men and women consume 11% of energy from SFAs; this is higher than the recommendation from most authoritative bodies to consume <10% of energy as SFA.[10] No differences in saturated fat intake exist based on race/ethnicity (range of 10.0% to 11.3% of intake) or income. The majority of saturated fat consumed is from palmitic acid (16:0) and then stearic acid (18:0) (54% and 25% of saturated fat intake, respectively).[10] Food sources of SFA in the American diet, listed as greatest to least, are cheese, pizza, grain-based desserts, and dairy-based desserts; together these foods comprise 31% of SFA intake.[5] Most stearic acid is consumed from grain-based desserts, cheese, and various meats.[2] From a global perspective, saturated fat intake (based on food availability) varies by continent and country, with lower intakes in Asia (3.1% to 10.6% of kilocalories) to higher intakes in Europe (8.9% to 16.5%). With the diversifying population of the United States, identification of global SFA intake and the specific food sources is important.[94] Finally, because energy intake is often greater than energy need, evaluating SFA intake solely as a percentage, rather gram quantities, provides a somewhat limited view.

SFA Recommendations

No current AI or RDA exist, in part because SFA is synthesized in the body to meet physiological needs and because of the recognized role of SFA in CVD.[9] The AMDR states that SFA should be as low as possible while consuming a nutritionally adequate diet. The DGA recommend that <10% of calories come from saturated fat and should be replaced with MUFA and PUFA; <7% SFA intake was suggested for further reduction of CVD risk.[5] The guidelines also recommend limiting foods that contain solid fats (SFA and TFA), sugars, and sodium; this is especially important as these foods comprise 19% of total energy intake.

The AHA's Diet and Lifestyle Recommendations (2006) recommended SFA intake be <7% of calories (approximately 16 g based on a 2,000 kcal diet) for the general population for CVD risk reduction.[3] In addition, the AHA and American College of Cardiology recently released a report indicating a dietary pattern that achieves 5% to 6% of calories from saturated fat best for those that want to achieve lipid lowering.[108] In 2010, the FAO reported that SFA intake should be no more than 10% of energy and replaced with PUFAs.[6] Although the American Diabetes Association has a recent position on fat in diabetes management, no recommendations exist for SFA in primary prevention of either diabetes or cancer. A current American Cancer Society guideline, however, recommends limiting consumption of red and processed meats, recognizing their role as major contributors to total and saturated fat intake. Based on recent literature and consensus, the goal for SFA intake for the population should be 7% to 10% of total energy. The current intake of SFA at 11% exceeds the amount recommended for healthy individuals.

SFAs and Health

The linear structure of SFA and its ability to tightly pack in cell membranes, along with its signaling properties, have consequences often considered detrimental to health. As with the other fatty acid groups, individual SFAs have differing impacts on health. The SFAs 12:0, 14:0, and 16:0 have similar effects on serum lipoproteins; specifically, they increase LDL cholesterol.[15] In contrast, stearic acid (18:0) has a neutral impact on LDL cholesterol.[107] However, because stearic acid is consumed in foods that should otherwise be limited (eg, grain-based desserts, cheese, processed meat), it is prudent to make the same intake recommendations for stearic acid as the other SFAs. When evaluating the impact of replacing SFA with carbohydrates on CVD risk, SFA increases LDL cholesterol but lowers triglycerides and raises HDL cholesterol, and apolipoprotein B is not changed. Inclusion of 12:0, 14:0, or 16:0 increases LDL cholesterol and HDL cholesterol when replacing carbohydrate at 5% of the diet; however, a recent pooled analysis of large cohort studies indicate there is a significantly greater relative risk for CHD with carbohydrate intake vs SFA intake.[98] When stearic acid is replaced by carbohydrate, there is a nonsignificant change in these CVD risk factors.[95] In addition, replacing SFA with PUFA lowers CVD risk by approximately 10% (5% energy substitution).[109] There is limited evidence about any impact of saturated fat on inflammation. Despite documented influence of saturated fat on surrogate disease markers, the effect of saturated fat intake on disease end points is not clear. A recent systematic review reported insufficient evidence to link saturated fat intake with CHD.[110] Several studies show that reducing fat consumption, especially saturated fat, can reduce risk for diabetes with improvements in weight[111,112]; however, this is not supported by other trials.

Although known to be an important component of breast milk, MCTs are gaining in popularity among healthy adults. Unlike longer chain SFAs, MCTs are transported in portal circulation and more readily oxidized through the β-oxidation pathway. That these fatty acids are oxidized rather than stored as triglyceride in the body could be advantageous. The oxidation rate of MCTs, as well as their impact on thermogenesis, has been shown to be beneficial in decreasing adiposity and improving weight loss when compared to olive oil.[113,114] However, these results were from supplementation with oil containing 8:0 and 10:0 and not 12:0 fatty acids. New food products containing coconut oil and other palm oils (eg, milk, spreads, yogurt) are touting health benefits of MCTs. Given that 44% of coconut oil is 12:0 and 16% is 14:0, and these fatty acids are hypercholesterolemic, consumption of coconut products is not currently recommended.[115] There is, however, cause to focus on the impact of different MCT fatty acids in human health. As research is completed, the Academy will disseminate findings to RDNs with appropriate recommendations.

Dietary patterns that are high in saturated fat include the Western dietary pattern, characterized as high in total fat, saturated and *trans* fat, refined carbohydrate, and sodium. Although distinguished in part by its saturated fat content, saturated fat is only one component of the Western dietary pattern. The saturated fat content of red meat can contribute to disease risk, but other components such as iron, sodium, and compounds created when cooking red meat can also contribute. Compared to red meat, consumption of fish, poultry, dairy products, and

nuts is associated with a lower risk of CHD when the same servings are consumed.[116] Although this risk reduction could be due to a reduction in heme iron, sodium, or saturated fat, or an increase in PUFA, examining whole foods rather than individual components is important. In addition, although evidence on the impact of SFAs on disease end points such as heart disease, diabetes, and cancer is mixed, replacing of SFA with PUFA instead of refined carbohydrate appears to be beneficial. Finally, decreasing SFA without caloric replacement is an effective strategy for reducing total energy content of the diet and promoting healthy body weight.

TFAs

Specific Fatty Acids and Food Sources

Partial hydrogenation results in the formation of a large number of positional and geometric isomers of the naturally occurring *cis*-fatty acids. A major TFA in industrially hydrogenated oils is elaidic acid (18:1, t9), although many other *trans* isomers are formed. TFA are present in ruminant meat and milk fats as a result of biohydrogenation of unsaturated fatty acids (18:2 and 18:3) in the rumen. The major TFA in ruminant meat and dairy products are c9,t11-CLA, with vaccenic acid (18:1, t11), predominating at 50% to 80% of total ruminant TFA (rTFA) produced. Recent efforts to increase c9,t11-CLA content in ruminants has increased the presence of rTFA in both meat and dairy products.[117] As previously stated, CLA is classified as an n-6 fatty acid and not a TFA and, therefore, will not be discussed here. Sources of commercially partially hydrogenated TFA include hydrogenated vegetable and marine oils; these oils have been commonly used in commercial baked goods. Although the TFA content in foods has decreased recently (through food reformulation), it is important to monitor the type of fat used to replace TFA, as it might be SFA.

An FDA labeling requirement that went into effect in 2006 required food labels to list TFA on their Nutrition Facts panel (on a separate line under the saturated fat listing) when present at 0.5 g or more per serving. Dietary supplements were also required to list TFA in the Supplement Facts panel. If the TFA content is <0.5 g per serving, then TFA does not need to be listed. Consumption of foods that contain TFA but at <0.5 g per serving contribute to overall TFA intake, so foods commonly containing TFA should continue to be limited (eg, commercial pastries, cookies, fast food). In addition, the FDA has recently issued a Federal Register notice preliminarily determining that *trans* fats (partially hydrogenated oils) are no longer "generally recognized as safe," or GRAS. RDNs should monitor the determination of the FDA regarding GRAS status of *trans* fats and what this means to the availability of *trans* fats to the consumer.

TFA from Supplements

No TFA supplements currently exist, as their negative health impacts are widely known. However, this excludes CLA, considered a TFA, as the *cis-9, trans-11* CLA isomers contain a trans bond at *cis*-11. Although nutritional supplements in liquid or powder form can contain *trans* fats, and must declare it on the label if ≥0.5 g per serving, the majority of the products do not contain appreciable quantities.

Current Dietary Intake of TFAs

Recent intake data estimates that TFA intake is 3 to 4 g/day in North America; intake is similar in northern European countries and less in eastern Asian countries (<1 g/day). This is a reduction from previous consumption, which was estimated to be 10 g/day worldwide.[118] Data from the European TRANSFAIR study indicate that in the United States, 1.2 g/day of TFA originates from ruminants. This is comparable with other countries, as consumption of rTFA in Europe ranges from 0.8 to 1.7 g/day.[119] More recent evidence (2007) reports the consumption of rTFA is 20% of total TFA intake, with most (85%) of this coming from milk fat. Recent analysis of NHANES 2003-2006 dietary data indicate industrially produced TFA intake at 1.3 g per person per day.[120] This is a reduction from 4.6 g per day, as stated in the FDA ruling (2003), which established labeling requirements for TFA intake; this reflects a reduction of TFAs in processed foods. TFA intake is declining; a recent comparison using NHANES data indicated a 58% decrease in serum TFA levels from 2000-2009.[121]

TFA Recommendations

The 2005 Daily Reference Intake has not set an AI or RDA for TFAs. No upper limit is set, as any TFA intake increases CHD risk; in light of this, intake of TFA should be kept as low as possible.[9] The DGA also recommend TFA consumption to be as low as possible, especially by limiting foods that contain synthetic sources of TFA, such as partially hydrogenated oils, and by limiting other solid fats.[5] The AHA's Diet and Lifestyle Recommendations advocate that TFA be <1% of calories.[3] In 2008, the FAO/World Health Organization recommended a TFA upper limit (both ruminant and industrially produced) to be <1% of energy; a 2010 report acknowledged that the <1% recommendation might need to be revisited due to unknown distribution of intake and potential negative impact on subgroups with higher consumption.[6]

TFAs and Health

The impact of TFA intake is generally undisputed. There is convincing evidence that consumption of commercial partially hydrogenated vegetable oils increases CHD risk factors, as well as in metabolic syndrome and diabetes risk. For example, trans-C18:1 increased serum levels of lipoprotein(a), an atherogenic protein associated with apolipoprotein B–containing lipoproteins; as this is the most commonly consumed TFA, this finding may be important with regard to risk of disease in healthy individuals.[122] Limited research has examined the different impacts of industrially produced TFA and rTFA on CHD risk. The amount of TFA is also relevant when considering the impact on disease risk markers. At 3.7% of energy, both ruminant and industrial TFA have been shown to have adverse effects on blood lipids, specifically increases in LDL cholesterol and decreases in HDL cholesterol. However, when examined at lower doses (1.5% of energy from rTFA or 0.8% total TFA), no significant differences in blood lipids or lipoproteins were seen.[123] A relationship between *trans* fats and cancer has not been determined.[106] In summary, foods containing industrially derived TFA should be minimized. Due to recent changes in commercial food composition, there is less TFA in foods. Nonetheless, consumers should be cognizant of potential TFA in processed

foods and limit fast food in the diet in order to reduce TFA intake, ideally to <1% of energy. Replacing TFA with other fatty acids or carbohydrate is an improvement, but replacing TFA with PUFA is most beneficial for health.

Emerging Population Research

Nutrition research is the basis for ongoing revisions in dietary recommendations. Large and long-term investigations into the impact of specific fatty acid groups on public health are emerging. In a recent study, associations between type of dietary fat intake—PUFA, MUFA and SFA—and CHD risk were assessed using data from 11 American and European cohort studies to address the question of the best macronutrient substitution for SFA in the diet. From follow-up data over 4 to 10 years including 344,696 persons, researchers identified that replacing SFA with PUFA was preferable to MUFA and carbohydrate for reducing CHD risk; a 5% decrease in SFA that was replaced with PUFA showed an inverse and significant relationship with CHD.[98] The EPIC (European Prospective Investigation of Cancer)-Norfolk study involving 25,639 individuals reported blood levels of SFA were positively associated with CHD risk, while an inverse association was seen with PUFA levels.[11] In addition, a 2012 study from a prospective cohort among 91,981 women in the Nurses' Health Study reported that consumption of PUFAs as a proportion of fat was inversely associated with risk of sudden cardiac death, independent of traditional CHD risk factors.[12] Together, these data support dietary guidelines to replace intake of SFAs with n-3 and n-6 PUFAs.

American and international health and government organizations agree with recommendations to consume 7% to 10% of total calories as SFA and restrict consumption of *trans* fat. The optimal intake of MUFA is undetermined, and the optimal amount of n-3 and n-6 fats to consume within the PUFA category also remains unanswered. Many but not all fats and oils that contain n-6 fatty acids also contain some plant-based n-3s. Physiological differences between plant-based and marine-source n-3s exist and experts agree that increasing intake of n-3 is warranted. An upper intake level of 2% energy as n-3 and 10% energy as n-6 is recommended, but it might also be time to review these recommendations.[124,125]

Role of the RDN and DTR Team

Fat is a valuable macronutrient in human health. RDNs and DTRs, under the supervision of the RDN, can help clients set and maintain realistic goals for intake of total fat and fatty acids, thereby influencing lifelong health and weight. This requires providing guidance on the quantity and quality of dietary fat in addition to meal planning and food preparation. The guiding philosophy is to encourage food-based consumption first and supplements second. Monitoring total fat is important because it is abundant in the diet and contributes to excessive energy intake; however, it aids absorption of fat-soluble nutrients and provides desirable cooking and sensory qualities. Monitoring type of fatty acid is important because they each have different biological effects in the body and some are essential in human nutrition. Recommendations for specific foods (eg, avocados) or fatty acids (eg, more n-3) might be more practical than general statements. The concept of caloric density is relevant because fat contains 9 calories per gram, more than double the calories per gram of protein and carbohydrates. Replacing fat with less calorically dense foods or without caloric replacement, within the context of adequate nutrition, are effective strategies for limiting excess energy intake; helping patients make appropriate substitutions is equally important. Understanding, for example, that individuals who consume fried fish tend to consume higher amounts of SFA and TFA is useful when offering guidance.[47] Providing clients with helpful tools and resources, for example, how to choose snack foods with less SFA or fish that is recommended by environmental organizations, will support ongoing healthful choices. Teaching consumers how to understand and apply Nutrition Facts labels and ingredient lists has lasting results.

In addition to current knowledge, information on how fatty acids influence health through gene expression is forthcoming. For example, both the type and amount of PUFA influence peroxisome proliferator-activated receptor activity and its regulation of lipid metabolism, including lipogenesis, oxidation, and transport.[126] In addition, research on how an individual's genetic variation influences fatty acid utilization is underway; this work will provide rationale and direction for future individualized dietary prescriptions. For example, individuals carrying >2 signal transducer and activator of transcription 3 (STAT3) risk alleles have increased risk of obesity with a SFA intake of >15.5% of energy, compared with those with ≤1 risk allele.[127] Knowledge gained in this area of research will enable RDNs to customize diets for optimal health outcomes.

Nutrition science has moved beyond fat as a macronutrient; a commanding knowledge of food sources and health impact of individual fatty acids is a vital piece of RDN training and continuing education. It is essential that RDNs understand the current literature on dietary fat and fatty acids and build knowledge as science expands. Translating fat and fatty acid literature into dietary recommendations is a complex process yet highly suited for the skills and training of RDNs. New technologies are changing the types of fatty acids in the food supply: new sources of fatty acids are being discovered, structural changes within the fatty acids and how they are positioned within the triglyceride molecule are being made, and genetically engineered fatty acids are being incorporated into new foods. As modified fatty acids and their sources become available, RDNs will be called on to provide leadership and guidance in areas where the science is still emerging.

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