Position of the American Dietetic Association and Dietitians of Canada: Dietary Fatty Acids

ABSTRACT

It is the position of the American Dietetic Association (ADA) and Dietitians of Canada (DC) that dietary fat for the adult population should provide 20% to 35% of energy and emphasize a reduction in saturated fatty acids and *trans*-fatty acids and an increase in n-3 polyunsaturated fatty acids. ADA and DC recommend a food-based approach for achieving these fatty acid recommendations; that is, a dietary pattern high in fruits and vegetables, whole grains, legumes, nuts and seeds, lean protein (ie, lean meats, poultry, and low-fat dairy products), fish (especially fatty fish high in n-3 fatty acids), and use of nonhydrogenated margarines and oils. Implicit to these recommendations for dietary fatty acids is that unsaturated fatty acids are the predominant fat source in the diet. These fatty acid recommendations are made in the context of a diet consistent with energy needs (ie, to promote a healthful body weight). ADA and DC recognize that scientific knowledge about the effects of dietary fats on human health is incomplete and take a prudent approach in recommending a reduction in those fatty acids that increase risk of disease, while promoting intake of those fatty acids that benefit health. Registered dietitians play a pivotal role in translating dietary recommendations for fat and fatty acids into healthful dietary patterns for different population groups.
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POSITION STATEMENT

It is the position of the American Dietetic Association and the Dietitians of Canada that dietary fat for the adult population should provide 20% to 35% of energy and emphasize a reduction in saturated fatty acids and trans-*fatty acids, and an increase in n-3 polyunsaturated fatty acids. The American Dietetic Association and Dietitians of Canada recommend a food-based approach for achieving these fatty acid recommendations; that is, a diet high in fruits and vegetables, whole grains, legumes, nuts, lean protein (ie, lean meats, poultry, and low-fat dairy products), fish (especially fatty fish high in n-3 fatty acids), together with the use of nonhydrogenated margarines and oils.*

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Recommendations for the intake of dietary fat and fatty acids have been made for healthy populations as well as for prevention and treatment of chronic disease (1-12). The guidance issued generally is consistent in recommending a decrease in the intake of saturated fatty acids (SFA) and *trans*-fatty acids (TFA), and in recommending 20% or 25% to 35% of energy from fat. This can be accomplished by a reduction in SFA and TFA, which results in a decrease in energy intake, or by partial or complete replacement of SFA and TFA with unsaturated fatty acids and carbohydrates or to a lesser extent with protein within the recommendations made for macronutrients. Recent recommendations also recognize the importance of n-3 fatty acids. An increase in n-3 polyunsaturated fatty acids (PUFA) can be attained by choosing fats and oils high in α-linolenic acid (ALA), and increasing consumption of fish, particularly fatty fish. This paper evaluates the evidence of benefits and adverse effects (or lack thereof) of dietary fatty acids to issue dietary recommendations for total fat, SFA, TFA, monounsaturated fatty acids (MUFA), and n-6 and n-3 PUFA for healthy individuals. The endpoints used to determine risks and benefits are based largely on lipid and lipoprotein responses because they respond to changes in dietary fatty acids and because they are important risk factors for cardiovascular disease (CVD). Moreover, elevated biomarkers of inflammation are associated with CVD and numerous metabolic disorders that are responsive to dietary fat (13). Research relating the role of dietary fatty acids to inflammatory and immune disorders does not suggest that among healthy populations, different recommendations are needed for fat and fatty acid.

Recommendations for dietary fat and fatty acids for treatment of disease or clinical practice are outside the scope of this paper. However, a brief discussion of the relationship of specific fatty acids to different diseases/conditions is provided when appropriate. For those with hyperlipidemia, the American Dietetic Association's (ADA's) updated Hyperlipidemia Evidence Analysis Library (14) provides a useful "Guide for Practice," which includes recommendations for modifying dietary fatty acid intakes for managing hyperlipidemia/dyslipidemia. In addition, Dietitians of Canada's (DC's) Practice-based Evidence in Nutrition service provides practice guidance (15).

FATS IN THE FOOD SUPPLY

Fatty acids are the major form of dietary fat (mainly as triglycerides). Fatty acids are 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), and the configuration of the double bonds (*cis* or *trans*). In addition, PUFA are further classified based on the position of the first double bond from the methyl terminus of the fatty acid as n-6 or n-3 fatty acids. MUFA found in the diet are largely n-9 fatty acids, with small amounts of n-7 fatty acids. The Figure presents the struc-

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Fatty acid classification	Structure	Biological actions	Common food sources
Saturated fatty acids			
Lauric acid	C12:0	Raises total, LDL[a], and HDL[b] cholesterol and increases some hemostatic/thrombotic factors that promote thrombosis	Coconut oil
Myristic acid	C14:0		Butter fat, coconut oil
Palmitic acid	C16:0		Most fats and oils
Stearic acid	C18:0	Does not increase total, LDL, and HDL cholesterol	Most fats and oils, cocoa butter, fully hydrogenated vegetable oils
Monounsaturated fatty acids			
cis-configuration			
Palmitoleic acid	C16:1	Decreases total and LDL cholesterol when substituted for saturated fat and decreases total cholesterol compared with dietary carbohydrate	Some fish oils, beef fat
Oleic acid	C18:1		Most fats and oils, nuts, seeds, avocados
trans-configuration			
Elaidic acid	C18:1	Raises total and LDL cholesterol similar to saturated fat, decreases HDL vs saturated fat, and raises the total-to-HDL ratio more so than saturated fat; induces systemic inflammation and endothelial dysfunction	Partially hydrogenated vegetable oils
Vaccenic acid	C18:1	Not established	Butterfat, meat
Polyunsaturated fatty acids			
n-6 fatty acids			
Linoleic acid	C18:2	Decreases total and LDL cholesterol	Liquid vegetable oils, nuts, seeds
Arachidonic acid	C20:4	Precursor for eicosanoids (prostaglandins, thromboxanes, leukotrienes)	Meat, poultry, fish, eggs
Conjugated linoleic acid	C18:2 (variants)	Anti-cancer properties, decreases body fat in growing animals	Butterfat, meat
n-3 fatty acids			
α-linolenic acid	C18:3	Decreases cardiovascular risk through multiple mechanisms including platelet function, inflammation endothelial cell function, arterial compliance, arrhythmia	Flaxseed, canola oil, soybean oil, walnuts
Eicosapentaenoic acid	C20:5	Decreases risk of sudden death through multiple mechanisms including platelet function, endothelial cell function, arterial compliance and arrhythmia and has beneficial effects on nervous system development and health	Fish oil, algae
Docosapenteonoic acid	C22:5		Fish oil, algae
Docosahexaenoic acid	C22:6		Fish oil, algae

Figure. Fatty acids in the food supply—structure, function, and common food sources. LDL=low-density lipoprotein; HDL=high-density lipoprotein.

ture of different fatty acids, their biological actions, and common food sources. Humans are unable to synthesize n-6 or n-3 fatty acids, thus these fatty acids are essential dietary nutrients.

SFA are present in relatively high amounts in animal fats and tropical vegetable oils. Meat and dairy products contribute approximately 60% of SFA in the diet in the United States and Canada. Animal fats contain predominantly palmitic acid (16:0) and stearic acid (18:0). Tropical vegetable oils, such as palm kernel and coconut oils, contain high amounts of lauric and myristic acid. SFA are formed by industrial hydrogenation (addition of hydrogen atoms to unsaturated bonds creating saturated bonds) of vegetable oils. Fully hydrogenated fats are high in stearic acid.

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 (t9-18:1), 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 in the rumen. The major TFA in ruminant meat and milk is vaccenic acid (t11-18:1), with smaller amounts of other TFA.

MUFA are present in vegetable, nut, and seed oils, as well as in meats and dairy products. The major dietary MUFA is oleic acid (18:1n-9). Oleic acid is present in high amounts in olive oil, canola oil, mid-oleic sunflower oil, and other mid- and high-oleic vegetable oils, peanuts, pistachios, almonds, and avocados. PUFA are found in vegetable, nut, and seed oils with the amounts of n-6 and n-3 fatty acids varying greatly. The major

dietary PUFA are the 18-carbon n-6 linoleic acid (LA) and the n-3 ALA. LA (18:2n-6) is the parent fatty acid of the n-6 fatty acids and is present in high amounts in soybean, corn, safflower, and sunflower oils. ALA (18:3n-3) is the parent fatty acid of the n-3 fatty acids and is highest in flaxseed, canola and soybean oils, and walnuts. γ-Linoleic acid (18:3n-6) is present in foods in very small amounts, except for evening primrose and borage oils. The desaturation (addition of double bonds) and elongation (addition of carbon atoms) of LA and ALA to longer carbon chain PUFA occurs in phytoplankton and animal cells. LA is elongated to arachidonic acid (ARA), and ALA is converted to eicosapentaenoic acid (EPA; 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-3) animal cells. Fish and seafood, particularly fatty fish such as mackerel, herring, salmon, tuna, and trout, as well as oysters, are the richest dietary sources of the n-3 longer carbon chain PUFA, EPA, and DHA. The major dietary sources of ARA are meat, poultry, and eggs.

Conjugated linoleic acids (CLA) are PUFA in which the double bonds occur on adjacent carbons. Small amounts of CLA are present in the milk and meat of ruminants. A major CLA is *cis*-9, *trans*-11 18:2, termed c9, t11 18:2, which has one *trans*- and one *cis*-bond, compared to the all *cis*-LA, which is c9,c12 18:2. CLA are not classified as TFA for the purpose of food labeling, or in regulations relating to TFA in Canada. The *trans*-fat labeling rules in the United States and Canada do not differentiate among TFA from ruminant animals and industrial hydrogenation, but do exempt conjugated dienes (CLA) from labeling. Thus, all *trans* fats, including ruminant *trans* fats, but excluding CLAs, are included on the Nutrition Facts label, regardless of origin.

The fatty acid composition of common dietary fats and oils are presented in Table 1 (available at www.adajournal.org). The US Department of Agriculture's Nutrient Data Laboratory (16) and the Canadian Nutrient File (17) provide excellent resources on the nutrient content of foods, including fatty acids. The Keep It Managed database (18) is an interactive software program that provides information about the n-3 and n-6 fatty acid content of 9,000 food servings.

FATTY ACID RECOMMENDATIONS

The Dietary Reference Intake (DRI) report on macronutrients recommended that SFA and TFA be as low as possible while consuming a diet that provides an adequate intake of all essential nutrients (12). The Dietary Guidelines for Americans 2005 (9) recommended <10% of calories from SFA, and that TFA consumption be as low as possible. The American Heart Association's (AHA) Diet and Lifestyle Recommendations recommended that SFA intake be <7% of calories, and that TFA be <1% of calories (5). Likewise, the Nutrition Recommendations and Interventions for Diabetes 2007 (1) recommend that SFA be <7% of calories and that TFA be minimized. The Canadian Diabetes Association recommended that SFA be <10% of calories and that use of processed foods containing SFA and TFA be limited (http://www.diabetes.ca/Files/nutritional_guide_eng.pdf). The upper limit of 35% calories from fat was based on data to show that higher fat intakes are associated with a greater intake of energy and SFA. The lower limit for fat intake was set to minimize the increase in plasma triglyceride and decrease in high-density lipoprotein (HDL) cholesterol levels that occurs with high intakes of carbohydrates.

The DRI report and the *Dietary Guidelines for Americans 2005* recommend an acceptable macronutrient distribution range of 5% to 10% dietary energy from n-6 PUFA, and 0.6% to 1.2% of energy from n-3 PUFA, but did not set a Recommended Dietary Allowance or Estimated Average Requirement for individual fatty acids. The DRI report on macronutrients (12) provided an Adequate Intake (AI) for n-6 and n-3 fatty acids, which is an intake equivalent to the observed median intake in the United States. The AI for n-6 fatty acids is 17 g/day for men 19 to 50 years, 12 g/day for women 19 to 50 years. The AI for ALA is 1.6 and 1.1 g/day for men and women 19 to older than 70 years of age, respectively (12). These intakes are equivalent to about 5% to 6% energy from LA and 0.5% energy from ALA. The DRI report on macronutrients also noted that EPA and DHA can provide up to 10% of total dietary n-3 fatty acids, which is based on median consumption patterns for these fatty acids in the United States. Mean intakes of LA and ALA are about 5% and 0.5% of dietary energy in Canada (19).

This position paper emphasizes that the AI are the observed median intakes of n-6 and n-3 fatty acids for the US population, and should not be confused with the Recommended Dietary Allowance or with those intakes of fatty acids that confer the lowest risk of disease. For primary prevention of coronary heart disease (CHD), the US Dietary Guidelines Advisory Committee (20), AHA (5,6), the National Heart Foundation of Australia (21), and the United Kingdom Scientific Advisory Committee (10) all recommend two servings of fish per week, preferably fatty fish, providing about 450 to 500 mg EPA and DHA per day. The National Health and Medical Research Council (2006) Nutrient Reference Values for Australia and New Zealand recommend 610 mg/day for men and 430 mg/day for women for chronic disease risk reduction (http://www.nhmrc.gov.au/publications/synopses/_files/n35.pdf). The National Academies (22) recently recommended that adolescent males, adult males, and females who will not become pregnant, as well as adult males and females who are at risk of CVD consume two 3-oz servings of fish per week. The report acknowledged that females who are or may become pregnant or who are breastfeeding, and children up to age 12 may benefit from consuming two 3-oz servings of seafood, especially those with higher concentrations of EPA and DHA.

ADA and DC Position on Fat and Fatty Acids

ADA and DC concur with other expert groups in recommending a dietary fat intake for adults in the range of 20% to 35% energy, emphasizing a reduction of SFA and TFA, and an increase in n-3 PUFA. These recommendations for specific fatty acids are applicable to children over 2 years of age, but should be followed with age-appropriate total fat and energy intakes to support normal growth and development. For children 1 to 3 years of age, a total fat intake of 30% to 40% of energy is recommended, and for children 4 to 18 years of age, a total fat intake of 25% to 35% of energy is recommended. For children under 2 years of age, cow's milk, if fed, should

be full-fat milk. The recommendations for total fat for children are based on a gradual transition from high-fat intakes during infancy to the total fat recommendation for adults.

Decreasing SFA and TFA is a strategy for reducing the energy content of the diet. Alternatively, SFA and TFA can be replaced with unsaturated fatty acids, and/or carbohydrate, protein. Replacing SFA with unsaturated fat avoids the triglyceride-raising effect when carbohydrates are substituted for SFA. Complex and unrefined carbohydrates result in a higher fiber intake and can attenuate, and even prevent, the triglyceride-raising effect of low-fat diets (23-25). Replacing SFA with protein also results in lower triglycerides than when carbohydrates are used as the substitute (26). MUFA avoid the HDL-lowering effects of diets high in n-6 LA. However, uncertainty surrounds the overall risk-to-benefit of high intakes of MUFA vs LA; consensus on the optimal intake of LA has not been reached. The recommended range for n-6 PUFA in the United States is 5% to 10% of energy (7-9,12) based on evidence of beneficial effects of PUFA on CVD and diabetes (27-31). Other international groups have recommended lower intakes of n-6 PUFA of 4% to 8% [European Commission (32)], 5% to 8% (Food and Agriculture Organization of the United Nations/World Health Organization [11]), 3% to 4% [The Japan Society for Lipid Nutrition (33)], and 2% to 3% [International Society for the Study of Fatty Acids and Lipids (34)] of energy to meet essential fatty acid requirements.

Recommendations for lowering the intake of n-6 PUFA have centered around concerns that high intakes may antagonize n-3 PUFA metabolism, contribute to an increased risk of inflammatory, immune, and other disorders associated with an excess production of n-6 fatty acid–derived eicosanoids, and may increase susceptibility of tissue and plasma lipids to oxidative modification (35,36). Thus, ALA, EPA, and DHA, which favorably affect risk of CVD and other diseases (37), could be affected adversely by high n-6 fatty acid intakes. Consensus on the beneficial effect of reducing LA intakes on human health has not been reached (38). Higher LA intakes can play an important role in cholesterol-lowering (39), may have beneficial effects on insulin sensitivity (40), and may reduce plasma triglycerides in some individuals (41). Some n-6 fatty acid–derived eicosanoids also have important effects in the resolution of inflammation and markers of CVD and CVD events are also lowest in individuals with high levels of both n-6 and n-3 fatty acids (28,42).

ADA and DC concur with the acceptable macronutrient distribution range of the DRI report on macronutrients (12) for ALA, which is 0.6% to 1.2% of energy. The median intake of ALA in the United States and Canada is 0.5% energy. The intake of TFA should be reduced to as low as possible. SFA should be replaced with *cis*-unsaturated fat or complex carbohydrates to maintain a total fat intake of 20% to 35% of energy. To a limited extent, SFA calories can be replaced with protein.

Fish and Shellfish. ADA and DC consider that n-3 PUFA from fish are an important part of a healthful diet, and recommend two servings per week, preferably fatty fish. Approximately 8 oz of cooked fish per week provides about 500 mg/day EPA and DHA. For vegans who do not consume any preformed sources of EPA and DHA, additional research is needed before recommendations can be made for these fatty acids, including supplements. It is important to note the absence of reported adverse health effects in this population that consumes no fish.

The presence of some contaminants in fish, including dioxins and methyl mercury, has raised concern. Federal guidelines for avoiding environmental contaminants in fish should be followed (the US Food and Drug Administration and the Environmental Protection Agency (43); Canadian Food Inspection Agency (44)]. The Environmental Protection Agency and the US Food and Drug Administration have issued a joint consumer advisory for pregnant women, women who could become pregnant and young children not to eat shark, swordfish, king mackerel, or tilefish because of their higher mercury levels. Up to 12 oz per week of low-mercury fish are recommended, such as shrimp, canned light tuna, salmon, pollock, and catfish. As albacore (white) tuna has more mercury than canned light tuna, these risk groups may eat up to 6 oz of albacore tuna per week. These same recommendations apply to young children, but smaller portions should be served. The Canadian Food Inspection Agency, different from the US Food and Drug Administration and Environmental Protection Agency, recommends a maximum intake of 0.20 μg/kg/day for pregnant women, women who could become pregnant, and young children. A report from the National Academies (22), which states that pregnant women or women who may become pregnant should stay within Federal advisories for consumption of specific fish and seafood types, and state/regional advisories for locally caught fish.

Many of the fish noted in federal advisories as containing high levels of mercury are not commonly consumed by Canadian women and children, and other fish not noted in the reports may contain methyl mercury. The EPA and DHA content of commonly consumed fish and amounts of environmental contaminants has been reported by Mozaffarian and Rimm (45). Adherence with federal (and regional) advisories on fish consumption will help achieve the benefits associated with fish and seafood consumption without increasing exposure to environmental contaminants.

Breast Milk. Human milk is the preferred source of nutrition for all infants under 6 months of age, unless clinically contraindicated. Infants less than 1 year of age who are not breastfed should be fed human milk substitutes that contain at least 4% energy from LA and 0.75% energy from ALA, which are the minimum amounts of LA and ALA recommended in formulas for term infants (46), and both DHA and ARA. For women who wish to use a human milk substitute, DHA should be at least 0.2% of total fatty acids and the level of ARA should not be lower than DHA.

FATTY ACIDS AND HEALTH

While many factors contribute to the risk of CHD, the important lipid and lipoprotein risk factors include plasma (or serum) total cholesterol, low-density lipoprotein (LDL) cholesterol, HDL cholesterol, triglycerides,

and the ratio of total to HDL cholesterol. Despite the significance of lipid/lipoprotein risk factors, many nonlipid factors, including inflammatory markers, are also important for assessing CHD risk (47). Thus, targeting multiple risk factors holds the most promise for attaining the greatest reduction in disease risk. Moreover, because inflammation plays a central role in many diseases, strategies to reduce elevated inflammatory mediators may have beneficial effects on many other diseases.

SFA

Epidemiologic studies have shown a positive association between the intake of SFA and the incidence of CHD (27,48,49), and clinical studies have shown that SFA raise total and LDL cholesterol (40,50). For every 1% increase in energy from SFA, LDL cholesterol levels increase by 1.3 to 1.7 mg/dL (0.034 to 0.044 mmol/L) (51-53), and HDL cholesterol levels increase by 0.4 to 0.5 mg/dL (0.010 to 0.013 mmol/L) (54). Individual SFA differ in their effects (55,56). Lauric acid (12:0) and myristic acid (14:0) have a greater total cholesterol raising effect than palmitic acid (16:0), while stearic acid (18:0) has a neutral effect on total, LDL, or HDL cholesterol (53,54,57). Lauric acid, but not myristic or palmitic acid, decreases the total-to-HDL cholesterol ratio because of an increase in HDL cholesterol (54). Foods contain mixtures of SFA, thus, selecting foods based on individual SFA content is not recommended.

MUFA

Oleic acid lowers total and LDL cholesterol when it replaces SFA (7,8,12,58). When compared to carbohydrate, MUFA decrease triglycerides, increase HDL cholesterol, and is inversely related to total-to-HDL cholesterol ratio. A meta-analysis of studies with individuals with diabetes showed that high-fat diets with 22% to 33% energy from MUFA resulted in lower plasma total cholesterol, very–low-density-lipoprotein cholesterol, and triglyceride levels than did low-fat, high-carbohydrate (49% to 60% energy) diets (59).

The association between MUFA and risk of cancer is inconsistent (60). In some studies, food sources of oleic acid were associated with a lower risk of breast cancer (61,62), but meta-analysis found that tissue oleic acid levels were positively associated with breast cancer (63). Because oleic acid can be synthesized in vivo, tissue oleic acid is not necessarily of dietary origin.

Current recommendations for MUFA were derived by considering recommendations for SFA, TFA, and n-6 and n-3 PUFA, then deriving a recommendation for MUFA based on the amount needed to obtain the recommended intake of total fat. National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) recommends that MUFA not exceed 20% of calories. There is an increasing trend for higher MUFA in many foods, due in part to use of fats with higher MUFA in place of TFA. Some evidence suggests an adverse effect of high MUFA diets on atherosclerosis in animals (64), but this has not been observed in humans.

TFA

High intakes of TFA have been associated with an increase in the relative risk of coronary artery disease, CHD death rate, and risk of fatal and nonfatal myocardial infarction, and sudden death (27,65-70). Clinical trials have shown a dose-dependent effect of TFA over the range of 0.5% to 10% of energy in increasing LDL cholesterol levels (12). Relative to SFA, TFA decrease HDL cholesterol and increase the TC-to-HDL cholesterol and LDL-to-HDL cholesterol ratios (54). TFA also increases other coronary artery disease risk factors, such as small dense LDL, postprandial lipids, endothelial function, and systemic inflammatory mediators (71-73), all of which may contribute to the effect of TFA on CHD mortality and morbidity (74,75).

Evidence of no relationship and a positive association between industrially produced TFA and cancer (76-79) and diabetes (80) has been reported. Clinical studies with the major *trans* MUFA (vaccenic acid) in ruminant meats and milks have not been done; thus, evidence to support a recommendation for the intake of fat from ruminant meats and dairy products that differs from those based on SFA is insufficient.

CLA, in ruminant meat and dairy fats, have unique biological effects, including decreased fat deposition, and anticarcinogenic, antiatherogenic, and immune-modulating properties (81). There is some evidence that CLA supplements (3 g/day) impair insulin sensitivity (82), increase C-reactive protein [t10,c12 CLA, 3.4 g/day (83)], and increase oxidative stress and insulin resistance [c9,t11 CLA, 3 g/day (84)] in those with obesity and metabolic syndrome. Supplementation with about 2 g CLA per day may reduce milk fat secretion during lactation (85), although other studies have not confirmed this (86). These doses are above those attainable from usual foods; thus, whether dietary CLA provides any health benefit is not clear. Consumption of higher-fat dairy and red meats as a means to increase CLA intake is not recommended because of the accompanying increase in SFA.

Foods containing industrially derived TFA should be minimized. Reducing TFA in some foods, for example, pastry that requires solid fat, without increasing SFA is complex. In some foods, TFA have been removed by replacement with fats higher in SFA. The current recommendations advise that TFA replacement strategies not result in a higher TFA and SFA (87).

PUFA

The n-6 (LA and ARA) and the n-3 (ALA, EPA, and DHA) fatty acids are important in many aspects of health. Early studies focused on diets rich in LA in reducing plasma lipid risk factors and CVD morbidity and mortality (31). More recently, there has been increased understanding of the importance of n-3 fatty acids in reducing CVD risk, in neurological function, and in inflammatory and immune disorders. ALA, EPA, and DHA differ in their metabolic and physiological roles, and the relative importance of the different n-3 fatty acids remains incompletely understood. Concern has been raised that a high dietary n-6:n-3 fatty acid ratio may contribute to many diseases associated with Western diets. Because the intake of EPA and DHA are much lower than

for LA and ALA, increases in EPA and DHA intake that have physiological effects do not substantively alter the dietary n-6:n-3 fatty acid ratio. Scientific consensus has not been reached on whether current intakes of LA are too high, and it is unclear whether associations between an increased risk of disease and high dietary n-6:n-3 fatty acid ratios are explained by high n-6, low n-3 fatty acid intakes, or effects of their ratio. Recent studies have suggested that conversion of ALA into EPA is not determined by the ratio of LA to ALA, but by the absolute amounts of ALA or LA in the diet (88). Use of n-6/n-3 fatty acid ratios is also problematic because an identical ratio can be achieved with very different amounts of each fatty acid class (89). Therefore, because of the difficulty in applying one ratio across diets varying in ALA, EPA, and DHA, the recommendations in this paper focus on the absolute intakes of n-6 and n-3 fatty acids.

n-6 Fatty Acids. Early clinical trials found that 13% to 21% dietary energy from PUFA decreased total plasma cholesterol by 13% to 15%, and decreased CHD events by 25% to 43%, although there was a lack of effect on all-cause mortality (90-92). Predictive equations based on changes in blood cholesterol estimated that an increase of 1% energy from PUFA reduces total cholesterol by 0.9 mg/dL (0.023 mmol/L), while a similar intake of SFA raises total cholesterol by approximately twice as much (51,52). PUFA increase HDL cholesterol when substituted for carbohydrate, although less than SFA and MUFA (54). Because PUFA lower total cholesterol and increase HDL cholesterol, the net effect is a decrease in the total-to-HDL and LDL-to-HDL cholesterol ratios. PUFA may also have beneficial effects on glucose metabolism and insulin resistance and, hence, on type 2 diabetes (80,93), as suggested by the inverse association of vegetable fat and PUFA with type 2 diabetes in the Nurses' Health Study (80) and the inverse association between PUFA and fasting blood glucose levels in the Italian Nine Communities Study (94). However, the Canadian Diabetes Association gave a D-level evidence rating to the nutrition recommendation for PUFA intake of ≈10% of energy because of insufficient clinical evidence that high LA has beneficial effects in diabetes (95).

The possibility that high intakes of n-6 PUFA may increase the risk of cancer, gallstones, CHD mortality, cerebral infarction, hyperinsulinemia, and subsequent insulin resistance, or immune and inflammatory disorders has been raised (33,35,96-99). Meta-analysis of 16 case-control studies and seven major cohort studies concluded that it is unlikely that a high intake of LA substantially raises risk of breast, prostate, or colorectal cancer (96). The Health Professionals' Follow-Up Study showed that men in the highest vs lowest quintile of PUFA intake had a lower risk of gallstone formation (100). In contrast, increased LA intake from 1950 to 2000 in Japan has been associated with an increased morbidity from cerebral infarction and ischemic heart disease (33,97,98). Similarly, there is a linear relationship between n-6 PUFA and CHD mortality among populations in different countries (101).

High intakes of n-6 PUFA have been proposed to increase production of proinflammatory and proaggregatory eicosanoids derived from ARA (102), and increase susceptibility of LDL and tissue lipids high in LA to oxidative modification (103). LDL from subjects consuming LA-rich vs MUFA-rich diets is more susceptible to oxidative modification (35). Consumption of oils high in LA has increased in the United States, Canada, and other countries following Westernized diets (104), with an increase in LA intakes from about 3% dietary energy in the 1930s to current intakes of about 5% to 6% dietary energy in the United States and Canada. Current evidence does not support a need to increase LA intakes in individuals consuming LA at the lower end of the acceptable macronutrient distribution range. However, whether benefit is attained by recommending a decrease in LA intakes for individuals with intakes above the median of 5% to 6% energy is unclear. One position favors a decrease in the intake of LA in favor of MUFA and ALA. A decrease in LA would also reduce the dietary ratio of LA to ALA, which has been suggested to be too high in Western diets (105). An alternate position is that current intake of LA be maintained, and that n-3 fatty acids increased. In support of the latter position, recent studies have suggested that n-6 and n-3 fatty acids together are associated with the lowest level of inflammation (28,42).

n-3 Fatty Acids. ALA, the predominant dietary n-3 fatty acid, is converted to EPA and DHA, although the conversion of ALA to EPA and especially to DHA is very low in humans (106-110). Epidemiologic studies in the United States reported that ALA intakes of 0.53 to 2.8 g per day were associated with a reduced risk of CVD events, fatal ischemic heart disease, and all-cause mortality (111-113). The Health Professionals' Follow-Up Study reported that a 1% energy increase in ALA intake was associated with a 40% lower risk of myocardial infarction, after adjustment for total fat intake (69), while results from the Nurses' Health Study (114) found that women in the lowest quintile of ALA intake had a 38% to 40% lower risk of sudden cardiac death than those in the highest two quintiles. In the Lyon Diet Heart Study, postmyocardial infarction patients who consumed a Mediterranean-style diet with 0.81% energy from ALA, 8.3% energy from SFA and 217 mg/day cholesterol had a 50% to 70% lower risk of recurrent heart disease than patients consuming an AHA step 1 diet (115). Recent epidemiologic studies continue to support a beneficial effect of dietary ALA on CVD, particularly in the presence of a low fish intake (116).

Meta-analysis have raised concern that ALA may increase the risk of prostate cancer (117). Nine observational studies (four prospective studies and five nonprospective studies) assessed the relationship between prostate cancer incidence or prevalence, and intake or blood levels of ALA, and reported an increased risk of prostate cancer (1.70; 95% confidence interval: 1.12 to 2.58). However, in prospective studies, the combined estimate of relative risk for prostate cancer incidence was 1.32 (95% confidence interval: 0.80 to 2.18). A subsequent study also reported no association between ALA intake and prostate cancer risk (118). In addition, subjects in the Lyon Diet Heart Study who consumed a Mediterranean diet with 0.8% of energy from ALA did not have increased risk of prostate cancer (119). Most evi-

dence suggests no adverse effect of ALA and risk of prostate cancer.

EPA and DHA reduce risk of sudden cardiac death, possibly by increasing the threshold for ventricular fibrillation, which is a leading cause of sudden death (120,121). Recent studies in patients with implanted defibrillators, however, caution that this effect may not be present in this patient population (122). The intake of EPA and DHA from fish in five epidemiologic studies in the United States associated with the lowest risk of coronary events, including CHD death, primary cardiac arrest, and ischemic heart disease death was 496 mg per day (123-127). A daily intake of approximately 500 mg EPA and DHA is equivalent to about 8 oz of fatty fish per week. Other recent meta-analyses reported that five or more servings of fish per week was associated with a lower CHD mortality (128) and a lower incidence of stroke (129) when compared with no fish or fish less than once per month. A recent systematic review of the literature of primary and secondary prevention studies with ≥1 year duration with fish or fish oils also reported reduced rates of all-cause mortality, cardiac and sudden death, and possibly stroke (130).

Epidemiologic studies have reported that high fish intakes are associated with a reduced risk of breast and colorectal cancer (131), which is consistent with evidence that EPA and DHA may reduce markers of colorectal cancer (60,131,132) and reduce expression of genes involved in colorectal cancer cell growth (133). However, a recent review of 20 cohorts from seven countries that evaluated 11 different types of cancer found no substantial association between plant and marine-derived n-3 fatty acids and incidence of cancer (134).

DHA is important in the nervous system, including the retina. Clinical and epidemiologic studies have shown that low dietary intakes of n-3 fatty acids and low plasma or red blood cell DHA are associated with several neurological and visual system problems (135-137). Three recent population studies found an association between higher intakes of DHA or EPA and DHA and lower risk of cognitive decline or verbal fluency (138-140). Men and women in the Framingham Heart Study who were in the top quartile of plasma phosphatidylcholine DHA levels had a 47% reduction in risk of developing all-cause dementia (138). Likewise, in the Atherosclerosis Risk in Community Study (140), higher plasma cholesteryl ester levels of EPA and DHA were associated with a lower decline in verbal fluency. Similarly, in the Zutphen Elderly Study (139), individuals who consumed fish had considerably less 5-year cognitive decline than nonconsumers.

While research in these areas is rapidly increasing, sufficient information is as yet unavailable to suggest recommendations for dietary n-3 fatty acids with respect to mental health or visual problems that are different from those based on CVD.

PREGNANCY, LACTATION, AND INFANCY

The importance of DHA in neural development and function has focused attention on the importance of n-3 fatty acids during pregnancy, lactation, and infancy. Observational and controlled clinical studies show that dietary PUFA during pregnancy and lactation influences the transfer of PUFA across the placenta and through breast milk (141,142). Important questions are whether dietary ALA can provide sufficient amounts by conversion through the elongation-desaturation pathway, and whether TFA from hydrogenated n-6 may have adverse effects on the mother or her infant. An intake of 2% energy from the n-6 LA prevents clinical and biochemical signs of essential fatty acid deficiency in infants (143), and no evidence is available to suggest that LA intakes are inadequate in the United States or Canada. On the other hand, the lower blood lipid and brain levels of DHA in infants fed some formulas lacking DHA than in breast-fed infants (144,145) raises the possibility that inadequate dietary supplies of n-3 fatty acids can adversely influence infant development.

An inverse association of TFA with DHA in newborn infant blood and with growth has been reported, and TFA may be deposited in infant tissues or converted to other unusual metabolites (146-148). TFA levels as high as 18% of milk fatty acids have been reported in the United States and Canada (149-151), which is higher than in Europe, where dietary intakes of TFA are lower (148). Levels of TFA in human milk have decreased since the introduction of *trans*-fat food labeling (148,152).

The conversion of ALA to DHA appears to be very low in humans (107-109). Newborn infants can convert ALA to EPA and DHA, and LA to ARA, and there is no evidence of metabolic immaturity in this pathway in infancy (106,153,154). Although the fractional conversion of ALA to DHA appears to be higher in women than men, and increases during pregnancy (155), intervention studies have shown that increased intakes of ALA do not increase levels of DHA in pregnant women or infants (156). On the other hand, higher maternal DHA increases the transfer of DHA to the infant both before birth and via breast milk after birth (142,157). A positive association between blood levels of DHA in infants and higher scores on measures of neural and visual maturation has been reported (158-162). The Avon Longitudinal Study of Parents and Children (163) reported that the verbal intelligence quotients were higher among children 6 months to 8 years of age of mothers who consumed more than 349 g seafood per week during pregnancy than among children of mothers who reported no seafood consumption.

The long-term benefits of early exposure to n-3 fatty acids (EPA and DHA) on later child development are uncertain (76,164-166). The Harvard Center for Risk Analysis extrapolated the results of studies on cognitive development of infants fed formula with DHA to suggest that increasing maternal DHA intake by 100 mg/day would increase child IQ by 0.13 points (167). However, a linear relation between DHA intake and IQ is unlikely, and nutrient requirements cannot be extrapolated from formula to lactating women. Studies in Denmark in which women with low fish intakes were supplemented with 4.5 g/day fish oil (equivalent to 1.3 g/day n 3 longer carbon chain PUFA) for 4 months after delivery found that despite higher blood lipid DHA, there were no advantages to visual maturation, but passive vocabulary and word comprehension were lower in the infants at 1 year of age (168,169). In other studies, maternal supplementation with 200 mg/day DHA for the

Table 2. Recommendations by the American Dietetic Association and Dietitians of Canada for total fat (for adults) and saturated, monounsaturated, n-6, n-3, and polyunsaturated fatty acids based on a 2,000 calorie diet[ab]

Total fat[cd]	Amount
20% of energy	44 g
25% of energy	56 g
30% of energy	67 g
35% of energy	78 g
Fatty acid/fatty acid class % of energy	
SFA[e] (as low as possible)	
3% of energy	7 g
7% of energy	16 g
10% of energy	22 g
MUFA[f] (provides remaining fatty acids to meet total fat)	
8% of energy	18 g
14% of energy	31 g
20% of energy	44 g
25% of energy	56 g
n-6 PUFA[g] (3% to 10% of energy)	
3% of energy	7 g
5% of energy	11 g
7% of energy	16 g
10% of energy	22 g
n-3 PUFA-ALA[h] (0.6% to 1.2% of energy)	
0.6% of energy	1.3 g
0.9% of energy	2.0 g
1.2% of energy	2.7 g
n-3 PUFA—long-chain PUFA	500 mg

[a] *Trans*-fatty acids should be as low as possible.
[b] Numbers are rounded to nearest whole number.
[c] 27 g oils recommended—US Department of Agriculture Food Guide.
[d] The Acceptable Macronutrient Distribution Range for total fat is 30% to 40% of energy for children 1 to 3 years and 25% to 35% of energy for children 4 to 18 years. The Acceptable Macronutrient Distribution Range provided by the 2002 Dietary Reference Intake is 5% to 10% energy from n-6 PUFA. Intakes of 3% energy from n-6 fatty acids prevents signs of deficiency, and intakes of n-6 fatty acids in the range of 3% to 5% energy will support a n-6:n-3 ratio of about 4:1 when the intake of n-3 fatty acids is at least 0.7% energy.
[e] SFA=saturated fatty acids.
[f] MUFA=monounsaturated fatty acids.
[g] PUFA=polyunsaturated fatty acids.
[h] ALA=α-linolenic acid.

first 4 months of lactation also had no effect on infant visual maturation, but psychomotor, not mental, development test scores were higher in infants at 30 months of age (170). Similarly, supplementation of pregnant women in Norway with fish oil had no effect on early measures of infant neural maturation (158), but cognitive development at 4 years of age was increased (166). Fish oil supplementation in pregnancy may reduce ARA in both mothers and their newborn infants (171). Reduced growth was reported in three studies with preterm infants fed formula containing DHA from fish oil without ARA (172-175). The risks and benefits of fish oil supplementation during pregnancy and lactation, or in infants as a means to increase infant DHA are thus unclear.

Although some epidemiologic and intervention studies have suggested that higher intakes of EPA and DHA are associated with a small increase in gestational length and reduced risk of some pregnancy-associated complications (158,176,177), other studies have not confirmed a benefit on length of gestation, preeclampsia, eclampsia, or gestational hypertension (158,171,178-181). Thus, current evidence does not support a recommendation to increase EPA and DHA to reduce risk of preterm delivery or pregnancy-associated complications.

Since 1990, the impact of formulas containing DHA or DHA plus ARA on visual maturation and neurodevelopment in term and preterm infants has been extensively studied. Some investigators have shown benefits in term infants fed formulas with DHA and ARA, or DHA from birth, or after initial breastfeeding (182,183), but others have not (184). However, most studies assessed neurodevelopment between 6 and 24 months, an age during which there is latency in expression of minor neurological dysfunction (185). Evidence that preterm infants benefit from inclusion of DHA and ARA in formula fed throughout the first year after birth is more consistent (184,186,187), and formula for preterm infants now includes both DHA and ARA. Formulas for term infants with ARA and DHA are also widely available. One review of 6 of 10 randomized controlled trials of addition of DHA and ARA to formula concluded no substantial effect on infant development, and that more-expensive formula with added DHA and ARA is not necessary (188).

The preferred and recommended source of nutrition for infants under 6 months is human milk, and the availability of infant formulas containing ARA and DHA does not change this recommendation. Some studies have found benefits of including DHA and ARA in formulas for term infants, and no adverse effects of feeding marketed infant formula containing both ARA and DHA in amounts found in human milk are known. Because of possible benefits and lack of adverse effects, it is recommended that all infants who are not breastfed be fed a formula containing both ARA and DHA through at least the first year of corrected age.

ROLE OF FOOD AND NUTRITION PROFESSIONALS IN IMPLEMENTING FATTY ACID RECOMMENDATIONS FOR HEALTH

The special expertise of registered dietitians (RDs) is important to individualize dietary recommendations to achieve optimal food-based dietary patterns. The complexity of translating fat and fatty acid recommendations requires the knowledge and skills of food and nutrition professionals. Once specific targets have been set for total fat and fatty acids (Table 2), then the type and amount of different fats (Table 1, available at www.adajournal.org) to be included

to meet the total fat and fatty acid goals in an energy-controlled diet can be determined. For all fatty acids, the guiding philosophy is a food-based approach. For individuals who do not eat fish, other options may be pursued, such as "designer" foods high in these fatty acids, foods fortified with these fatty acids, or even supplements (189). The RD's expertise is also needed to translate recommendations for fatty acids to the appropriate energy intake to achieve a healthful dietary pattern.

Consumers lack knowledge on how to use the Nutrition Facts labels, or ingredient lists to determine fatty acid contents, RDs must also be aware that individual's vary in their response to dietary fat and must be able to recommend appropriate dietary fat changes when necessary. My Fats Translator, a calculator that translates fat/fatty acid recommendations into daily limits, is available from the AHA at www.myfatstranslator.com. Implicit in this is the need to also monitor adherence to food-based dietary recommendations.

As RDs stay abreast of current dietary recommendations, it is important that they effectively respond to topics where there is not yet scientific agreement, as is the case with n-6 PUFA. Consequently, RDs will be called upon to provide guidance in areas where the science is still emerging. As always, the evolution of the science-base brings clarity and forms the basis for ongoing revisions in dietary recommendations and consequent dietary patterns.

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Table 1. Fatty acid profiles of vegetable and animal fats and oils

Fat	Amount	Total FA[a]	Lauric 12:0	Myristic 14:0	Palmitic 16:0	Stearic 18:0	Total MUFA[b]	Oleic 18:1	Total PUFA[c]	Linoleic[d] 18:2	Linolenic[e] 18:3 (n-3)	EPA[f]+DHA[g] +DPA[h] 20:5+22:6+22:5
Almond oil	100 g	8.2	0	0	6.5	1.7	69.9	69.4	17.4	17.4	0	0
	1 Tbsp	1.1	0	0	0.9	0.2	9.5	9.4	2.4	2.4	0	0
Apricot oil	100 g	6.3	0	0	5.8	0.5	60	58.5	29.3	29.3	0	0
	1 Tbsp	0.9	0	0	0.8	0.1	8.2	8.0	4.0	4.0	0	0
Avocado oil	100 g	11.6	0	0	10.9	0.7	70.5	67.9	13.5	12.6	1.0	0
	1 Tbsp	1.62	0	0	1.53	0.09	9.88	9.5	1.89	1.75	0.13	0
Beef tallow	100 g	49.8	0.9	3.7	24.9	18.9	41.8	36	4	3.1	0.6	0
	1 Tbsp	6.4	0.1	0.5	3.2	2.4	5.4	4.6	0.5	0.4	0.1	0
Butter[i]	100 g	51.4	2.6	7.4	21.7	10	21.0	20.0	3.0	2.7	0.3	0
	1 Tbsp	7.3	0.4	1.1	3.1	1.4	3.0	2.8	0.4	0.4	0.0	0
Canola oil	100 g	7.1	0	0	4	1.8	58.9	56.1	29.6	20.3	9.3	0
	1 Tbsp	1.0	0	0	0.6	0.2	8.2	7.8	4.1	2.8	1.3	0
Canola oil, high oleic	100 g	6.5	0	0	3.4	1.9	72.0	70.0	17.1	14.5	2.6	0
	1 Tbsp	0.9	0	0	0.5	0.3	10.1	9.8	2.4	2.0	0.4	0
Cocoa butter	100 g	59.7	0	0.1	25.4	33.2	32.9	32.6	3	2.8	0.1	0
	1 Tbsp	8.1	0	0	3.4	4.5	4.5	4.4	0.4	0.3	0	0
Coconut oil	100 g	86.5	44.6	16.8	8.2	2.8	5.8	5.8	1.8	1.8	0	0
	1 Tbsp	11.8	6.1	2.3	1.1	0.4	0.8	0.8	0.2	0.2	0	0
Corn oil	100 g	12.9	0	0	10.6	1.8	27.6	27.3	54.7	53.5	1.2	0
	1 Tbsp	1.8	0	0	1.4	0.2	3.7	3.7	7.4	7.3	0.2	0
Cottonseed oil	100 g	25.9	0	0.8	22.7	2.3	17.8	17	51.9	51.5	0.2	0
	1 Tbsp	3.5	0	0.1	3.1	0.3	2.4	2.3	7.1	7	0	0
Flaxseed oil	100 g	9.4	0	0	5.3	4.1	20.2	20.2	66	12.7	53.3	0
	1 Tbsp	1.3	0	0	0.7	0.6	2.7	2.7	9.0	1.7	7.2	0
Grapeseed oil	100 g	9.6	0	0.1	6.7	2.7	16.1	15.8	69.9	69.6	0.1	0
	1 Tbsp	1.3	0	0	0.9	0.4	2.2	2.1	9.5	9.5	0	0
Hazelnut oil	100 g	7.4	0	0.1	5.2	2	78	77.8	10.2	10.1	0	0
	1 Tbsp	1.0	0	0	0.7	0.3	10.6	10.6	1.4	1.4	0	0
Herring oil	100 g	21.3	0.2	7.2	11.7	0.8	56.6	11.9	15.6	1.1	0.8	11.1
	1 Tbsp	2.9	0	1.0	1.6	0.1	7.7	1.6	2.1	0.2	0.1	1.5
Lard	100 g	39.2	0.2	1.3	23.8	13.5	45.1	41.2	11.2	10.2	1	0
	1 Tbsp	5.0	0	0.2	3.0	1.7	5.8	5.3	1.4	1.3	0.1	0
Mustard oil	100 g	11.6	0	1.4	3.8	1.1	59.2	11.6	21.2	15.3	5.9	0
	1 Tbsp	1.6	0	0.2	0.5	0.2	8.3	1.6	29	2.1	0.8	0
Olive oil	100 g	13.5	0	0	11	2.2	73.7	72.5	8.4	7.9	0.6	0
	1 Tbsp	1.8	0	0	1.5	0.3	9.9	9.8	1.1	1.1	0.1	0
Palm oil	100 g	49.3	0.1	1	43.5	4.3	37	36.6	9.3	9.1	0.2	0
	1 Tbsp	6.7	0	0.1	5.9	0.6	5.0	4.9	1.3	1.2	0	0
Palm kernel oil	100 g	81.5	47	16.4	8.1	2.8	11.4	11.4	1.6	1.6	0	0
	1 Tbsp	11.1	6.4	2.2	1.1	0.4	1.5	1.5	0.2	0.2	0	0
Pistachio oil	100 g	13.8	0	0	4.9	0.5	49.3	22.7	32.5	13.2	0	0
	1 Tbsp	1.9	0	0	0.7	0.1	6.9	3.2	4.5	1.8	0	0
Peanut oil	100 g	16.9	0	0.1	9.5	2.2	46.2	44.8	32	32	0	0
	1 Tbsp	2.3	0	0	1.3	0.3	6.2	6.0	4.3	4.3	0	0
Poppyseed oil	100 g	13.5	0	0	10.6	2.9	19.7	19.7	62.4	62.4	0	0
	1 Tbsp	1.8	0	0	1.4	0.4	2.7	2.7	8.5	8.5	0	0
Rice bran oil	100 g	19.7	0	0.7	16.9	1.6	39.3	39.1	35	33.4	1.6	0
	1 Tbsp	2.7	0	0.1	2.3	0.2	5.3	5.3	4.8	4.5	0.2	0
Safflower oil	100 g	6.2	0	0	4.3	1.9	14.4	14.4	74.6	74.6	0	0
	1 Tbsp	0.8	0	0	0.6	0.3	1.9	1.9	10.1	10.1	0	0
Salmon oil	100 g	19.9	0	3.3	9.8	4.2	29.0	17.0	40.3	1.5	1.1	34.2
	1 Tbsp	2.7	0	0.4	1.3	0.6	4.0	2.3	5.5	0.2	0.1	4.7
Sesame oil	100 g	14.2	0	0	8.9	4.8	39.7	39.3	41.7	41.3	0.3	0
	1 Tbsp	1.9	0	0	1.2	0.7	5.4	5.3	5.7	5.6	0	0

(continued)

Table 1. Fatty acid profiles of vegetable and animal fats and oils (continued)

Fat	Amount	Total FA[a]	Lauric 12:0	Myristic 14:0	Palmitic 16:0	Stearic 18:0	Total MUFA[b]	Oleic 18:1	Total PUFA[c]	Linoleic[d] 18:2	Linolenic[e] 18:3 (n-3)	EPA[f]+DHA[g] +DPA[h] 20:5+22:6+22:5
Sheanut oil	100 g	46.6	1.3	0.1	4.4	38.8	44	43.5	5.2	4.9	0.3	0
	1 Tbsp	6.3	0.2	0	0.6	5.3	6.0	5.9	0.7	0.7	0	0
Soybean oil	100 g	14.4	0	0.1	10.3	3.8	23.3	22.8	57.9	51	6.8	0
	1 Tbsp	2.0	0	0	1.4	0.5	3.2	3.1	7.9	7.0	0.9	0
Sunflower oil, low oleic	100 g	10.3	0	0	5.9	4.5	19.5	19.5	65.7	65.7	0	0
	1 Tbsp	1.4	0	0	0.8	0.6	2.6	2.6	8.9	8.9	0	0
Sunflower oil, mid-oleic	100 g	9.0	0	0.1	4.2	3.6	57.3	57.0	28.9	28.9	0	0
	1 Tbsp	1.2	0	0	0.6	0.5	7.8	7.8	3.9	3.9	0.0	0
Sunflower oil, high oleic	100 g	9.7	0	0	3.7	4.3	83.6	82.6	3.8	3.6	0.2	0
	1 Tbsp	1.4	0	0	0.5	0.6	11.7	11.6	0.5	0.5	0	0
Tomatoseed oil	100 g	19.7	0	0.2	15	4.4	22.8	21.9	53.1	50.8	2.3	0
	1 Tbsp	2.7	0	0	2.0	0.6	3.1	3.0	7.2	6.9	0.3	0
Teaseed oil	100 g	21.1	0.1	0.1	17.5	3.1	51.5	49.9	23	22.2	0.7	0
	1 Tbsp	2.9	0	0	2.4	0.4	7	6.8	3.1	3.0	0.1	0
Vegetable oil	100 g	11.6	0	1.4	3.8	1.1	59.2	11.6	21.2	0	0	0
	1 Tbsp	1.6	0	0.2	0.5	0.2	8.3	1.6	3.0	0	0	0
Walnut oil	100 g	9.1	0	0	7	2	22.8	22.2	63.3	52.9	10.4	0
	1 Tbsp	1.2	0	0	0.9	0.3	3.1	3.0	8.6	7.2	1.4	0
Wheat germ oil	100 g	18.8	0	0.1	16.6	0.5	15.1	14.6	61.7	54.8	6.9	0
	1 Tbsp	2.6	0	0	2.3	0.1	2.0	2.0	8.4	7.4	0.9	0

[a]SFA=saturated fatty acids.
[b]MUFA=monounsaturated fatty acids.
[c]PUFA=polyunsaturated fatty acids.
[d]LA=linoleic acid
[e]ALA=α-linolenic acid.
[f]EPA=eicosapentaenoic acid.
[g]DHA=docosahexaenoic acid.
[h]DPA=docosapentaenoic acid.
[i]The sum of the fatty acids in butter is less than the mass of butter because of the water content.