The Fats of Life

The Role of Omega-3 Fatty Acids in the Prevention of Coronary Heart Disease

Charles R. Harper, MD; Terry A. Jacobson, MD

Epidemiological and clinical trial evidence suggests that ω-3 polyunsaturated fatty acids (PUFAs) might have a significant role in the prevention of coronary heart disease. Dietary sources of ω-3 PUFA include fish oils rich in eicosapentaenoic acid and docosahexaenoic acid along with plants rich in α-linolenic acid. Randomized clinical trials with fish oils (eicosapentaenoic acid and docosahexaenoic acid) and α-linolenic acid have demonstrated reductions in risk that compare favorably with those seen in landmark secondary prevention trials with lipid-lowering drugs. Several mechanisms explaining the cardioprotective effect of ω-3 PUFAs have been suggested, including antiarrhythmic, hypolipidemic, and antithrombotic roles. Although official US guidelines for the dietary intake of ω-3 PUFAs are not available, several international guidelines have been published. Fish is an important source of ω-3 PUFAs in the US diet; however, vegetable sources, including grains and oils, offer an alternative source for those who are unable to regularly consume fish.

The past 3 decades have been a period of rapid expansion in the scientific knowledge of ω-3 polyunsaturated fatty acids (PUFAs). Beginning with the study by Dyerberg et al1 involving Greenland Eskimos in the late 1970s, the body of evidence supporting a role for ω-3 PUFAs in the prevention of coronary heart disease (CHD) has continued to grow. Evidence from recent randomized trials2-5 in patients with CHD suggests that intake of ω-3 PUFAs from marine sources (eicosapentaenoic acid [EPA]) and plant sources (α-linolenic acid [ALA]) prevents cardiac death and nonfatal myocardial infarction (MI). This article reviews the available epidemiological evidence concerning ω-3 PUFAs and their inverse relationship with CHD. Review of their structure, nomenclature, and possible cardioprotective effects are then explored. Evidence from recent interventional clinical trials is reviewed, and clinical implications are discussed.

EPIDEMIOLOGICAL EVIDENCE

In the 1970s, Dyerberg et al1 evaluated the dietary habits of Greenland Eskimos, a population known to have a low mortality rate from CHD. This was one of the first epidemiological studies to explore the relationship between dietary ω-3 PUFA intake and the rate of CHD. Results of dietary surveys indicated that the Eskimo diet was not a low-fat diet and that approximately 39% of caloric (energy) intake was from fat. Further analysis revealed the intake of saturated fat to be low (9% of total calories), whereas the dietary intake of ω-3 polyunsaturated fat (ω-3 PUFA) was high (4.2% of total calories). These findings contrasted sharply with the dietary habits of an ethnically similar population in Denmark with much higher rates of CHD. The Danish diet had a comparable amount of total fat (42% of total calories) but a much lower intake of ω-3 polyunsaturated fat (ω-3 PUFA) was high (4.2% of total calories). These findings contrasted sharply with the dietary habits of an ethnically similar population in Denmark with much higher rates of CHD. The Danish diet had a comparable amount of total fat (42% of total calories) but a much lower intake of ω-3 polyunsaturated fat (<1% of total calories) and a much higher intake of saturated fat (22% of total calories). A second similar study6 followed inhabitants of Greenland and Denmark for 25 years; a 10-fold increase in MI was noted in the Danish Group.

In addition to cross-cultural epidemiological studies, results of various prospective observational cohort studies have suggested a cardioprotective effect of dietary ω-3 PUFAs. Early important cohort

From the Department of Medicine (Dr Harper) and the Office of Health Promotion and Disease Prevention (Dr Jacobson), Emory University, Atlanta, Ga.

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studies include the Zutphen and Western Electric studies, which demonstrated an inverse relation between fish consumption and mortality from CHD.

In a more recent prospective cohort study, the US Physicians Health Study, 20531 US male physicians aged 40 to 84 years who were free of cardiovascular disease were evaluated. These men were asked to complete food frequency questionnaires on fish consumption and were then followed up for 11 years. Consuming at least 1 fish meal per week reduced the risk of sudden cardiac death by 52% (P = .03) compared with those consuming fish only monthly. All levels of fish consumption up to 1 meal per week were associated with decreased risk of sudden death. At levels of consumption greater than 1 fish meal per week the risk reduction did not change, indicating a threshold effect.

The previously mentioned studies involved predominantly the ω-3 PUFAs eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which are derived from marine sources. However, cohort studies have also examined plant-based sources of ω-3 PUFA (ALA). In the usual care cohort (n = 6250 men) of the Multiple Risk Factor Intervention Trial (MRFIT), multivariate regression analysis was used to determine the effect that dietary PUFA intakes had on 10 1/2-year mortality rates. Intake of PUFA was calculated from 4 dietary recall interviews at baseline and 1-, 2-, and 3-year follow-up. Significant inverse associations were demonstrated for the intake of the ω-3 PUFA ALA on mortality rates from CHD (P < .04), total cardiovascular disease (P < .03), and all-cause mortality (P < .02).

Participants in the Nurses Health Study11 consisted of 76283 women aged 30 to 55 years who were free of cardiovascular disease. Intake of ALA was derived from a 116-item food frequency questionnaire. After adjustment for several possible confounding variables, a higher intake of ALA was associated with a lower relative risk of fatal CHD. The relative risks from lowest to highest quintiles ranged from 1.00 to 0.55 (P = .01 for trend). The finding that consumption of foods known to be rich dietary sources of ALA was associated with reduced CHD risk further substantiated this inverse association between ALA and fatal CHD. Specifically, women who consumed oil and vinegar salad dressing frequently were at lower risk for fatal CHD. Salad dressings are typically made with unhydrogenated soybean oil, which contains approximately 7% ALA.

Not all prospective cohort studies of the relationship between ω-3 PUFA consumption and cardiovascular mortality rates have reported inverse associations. Three negative studies involved cohorts with higher baseline intakes of ω-3 PUFAs than the earlier cohort studies. In addition, these studies had few participants who consumed less than 1 fish meal per week. A threshold effect, in which fish intake is cardioprotective in small amounts, could possibly explain these discordant results. In addition, each population studied was already at low risk for CHD.

Finally, a recent systematic review of 11 prospective cohort studies by Markmann and Gronbaek examined the relationship between fish intake and CHD mortality rates. Four of these studies were judged to be high quality in terms of study design. Two of the high-quality studies were performed on low-risk populations and demonstrated no cardioprotective effect from fish consumption. The other 2 high-quality studies were performed on populations at higher risk for CHD and found an inverse association between fish consumption and CHD death. It was suggested that in these higher-risk cohorts, consumption of 40 to 60 g of fish per day could reduce the risk of CHD death by 40% to 60%. To date, there has been no systematic review that examines ω-3 PUFA intake from both marine and plant sources.

STRUCTURE AND NOMENCLATURE

Fatty acids consist of a hydrocarbon chain with a hydrophilic methyl group at one end and a hydrophilic carboxyl group at the other end (Figure 1). The methyl end of the molecule is also referred to as the omega end, and the carboxyl group is located at the delta end. Biochemists describe fatty acids using the omega numbering system. In this system, carbon atoms are numbered in order starting from the methyl end. The length of the carbon chain and the number and location of the double bonds determine the properties of the different fatty acids. Fatty acids are also categorized by the number of double bonds present in the fatty acid mol-

Figure 1. Important fatty acids. The number of carbon atoms is indicated first and the number of double bonds is indicated after the colon. The position of the first double bond counted from the methyl end is listed after the comma.
A fatty acid can be saturated (no double bonds), monounsaturated (1 double bond), or polyunsaturated (≥2 double bonds).17

Polyunsaturated fatty acids can be divided into 2 subcategories: ω-3 and ω-6. The ω-3 PUFA s have their first double bond located at the third carbon molecule (C-3), whereas the ω-6 PUFA s have their first double bond located at C-6. The ω-6 and ω-3 PUFA s are considered essential fatty acids because humans cannot synthesize them and must be obtained through the diet. The ω-3 PUFA ALA and the ω-6 PUFA linoleic acid are the predominant essential fatty acids in humans. Linoleic acid can be elongated and desaturated to arachidonic acid, whereas ALA is elongated and desaturated into EPA and then into DHA (Figure 2). Eicosapentaenoic acid and DHA are the major ω-3 PUFA s found in fish and are thought to be responsible for the cardioprotective effect.18 It is thought that ALA conversion to EPA might depend on levels of the ω-6 PUFA linoleic acid because ALA and ω-6 PUFA s are competitive substrates for the rate-limiting enzyme Δ6 desaturase (Figure 2).19 Leukotrienes, prostaglandins, and thromboxanes are eicosanoids that are derived from the previously mentioned essential fatty acids. Eicosanoids derived from arachidonic acid are generally proinflammatory and proaggregatory agonists, whereas those derived from ω-3 PUFA s tend to inhibit platelet aggregation and be anti-inflammatory.20 Eicosapentaenoic acid and DHA are found predominantly in certain fish, whereas ALA is found in flaxseed grain, canola oil, and certain vegetables.

### CARDIOVASCULAR EFFECTS OF ω-3 PUFA S

Although many researchers have suggested that ω-3 PUFA s might be cardioprotective due to multiple mechanisms, their role as potential antiarrhythmics has recently received serious attention. It is thought that ω-3 PUFA s stabilize the electrical activity of cardiac myocytes by inhibiting sarcolemmal ion channels, resulting in a prolonged relative refractory period.21 This antiarrhythmic effect was demonstrated by Leaf and Kang22 in work with dogs. Ligating the left main coronary artery while an inflatable cuff was continuously inflated to prevent ventricular fibrillation when the cuff was inflated. Susceptible dogs (n=13) were then studied. Intravenous infusion of fish oil before the exercise ischemia test prevented ventricular fibrillation in 10 of 13 dogs. In the control exercise ischemia tests conducted 1 week before and 1 week after infusion of fish oil, animals were given a soybean oil infusion instead and developed ventricular fibrillation that required defibrillation. Using the same protocol, the dogs were also given an intravenous infusion of the plant-derived ω-3 PUFA ALA. Beneficial antiarrhythmic results similar to those in the fish oil group were obtained with ALA.

The ω-3 PUFA s also have significant antithrombotic properties. Eicosapentaenoic acid has been shown to inhibit the synthesis of thromboxane A2, a prostaglandin that causes platelet aggregation and vasoconstriction.23 Ingestion of EPA results in reduced platelet adhesion and reactivity, which manifests itself as increased bleeding time and decreased adhesion of platelets to glass beads.24 Other antithrombotic effects reported include reductions in fibrinogen and increases in tissue plasminogen activator (Table 1).20

Endothelial function is also favorably affected by ω-3 PUFA s because the vasodilatory effect of nitrous oxide is enhanced by EPA. Treating humans with fish oil has been shown to decrease oxygen-derived free radical production in...
neutrophils.25 It has been suggested that this reduction in free radicals increases the bioavailability of nitrous oxide. Studies20 using ultrasonic tracking of brachial artery flow-mediated vasodilation have demonstrated improved large artery endothelium-dependent dilation in patients treated with fish oil. Endothelial function can also be improved by reducing the endothelial expression of vascular cell adhesion molecules, thus resulting in a reduction in leukocyte binding to the endothelium.27

Ingestion of EPA and DHA also has been shown in animal studies to inhibit atherosclerotic plaque formation. Two important cells in the development of an atherosclerotic plaque are smooth muscle cells and macrophages. Platelet-derived growth factor is a key chemotactant and mitogen for smooth muscle cells and macrophages. Platelet-derived growth factor production and messenger RNA synthesis are decreased by the ingestion of ω-3 PUFAs.28

The effect of ω-3 PUFAs on lipid metabolism is predominantly antiatherogenic. Consuming fish oil (a rich source of EPA) has been shown to lower total cholesterol and triglyceride concentrations by inhibiting very low-density lipoprotein and triglyceride synthesis in the liver.29 Large doses of fish oil have been shown to have profound effects in reducing triglyceride levels in hypertriglyceridemic patients. Apolipoprotein B production is also reduced by fish oil consumption compared with vegetable oils not containing ω-3 PUFAs.20 Pretreatment with ω-3 PUFAs also markedly reduces postprandial lipemia, which typically occurs after consumption of a fatty meal, and postprandial lipoproteins are atherogenic. Postprandial lipemia is also thrombogenic because it increases levels of activated factor VII, a procoagulant. Ingesting olive oil results in the same degree of increase in factor VII as ingesting butter, whereas consuming fish oil prevents this postprandial increase.30

Unlike vegetable oils rich in ω-6 PUFAs, ω-3 PUFAs do not lower high-density lipoprotein (HDL) cholesterol levels. In contrast, they have been shown to result in a favorable change in HDL cholesterol metabolism. It seems that ω-3 PUFAs cause an increase in the large cholesterol-rich HDL2 subtype while decreasing the smaller triglycerol-rich HDL3 subtype.31,32 HDL2 is considered to be the most antiatherogenic HDL subtype.

Some concerns have been raised about potential atherogenic changes in lipid metabolism caused by ω-3 PUFAs. Low-density lipoprotein cholesterol levels have been shown to occasionally increase with ω-3 PUFA supplementation; however, this effect does not occur consistently.31 Also, some concern has been raised about in vitro studies that demonstrate that ω-3 PUFA supplementation might increase low-density lipoprotein cholesterol susceptibility to oxidation. It has been demonstrated, however, that this oxidation can be reduced by supplementation with the antioxidant vitamin E.29

In summary, ω-3 PUFAs have mostly antiatherogenic properties. Most of these antiatherogenic effects have been demonstrated with the marine-derived ω-3 PUFAs. Most studies with ALA have evaluated the efficiency with which it is converted to the longer chain ω-3 PUFAs EPA and DHA. More studies are needed to delineate the potential cardioprotective mechanisms of ALA.

**ANGIOGRAPHIC TRIALS**

Randomized, controlled trials with fish oils with angiographic end points have had mixed results. In a Norwegian study,33 610 patients undergoing coronary artery bypass grafting were randomly assigned either to a fish oil group (4 g/d) or to a control group. The primary end point was graft patency at 1 year, which was assessed by angiography. Vein graft occlusion rates were 27% in the fish oil group and 33% in the control group (odds ratio, 0.77; 95% confidence interval, 0.60-0.99; P = .034). It was also noted that there was an inverse relation between relative changes in serum ω-3 PUFA levels and vein graft occlusions.33

In another more recent angiographic, randomized, controlled trial,34 223 patients with angiographically proven CHD were randomized to receive fish oil capsules or to a control group receiving capsules containing PUFAs resembling those in the average European diet. Results showed that ingesting ω-3 PUFAs had a modest mitigating effect on the progression of CHD.

Clinical trials in patients undergoing angioplasty generally have not demonstrated a benefit from ω-3 PUFA supplementation. Although some trials are exceptions, the larger high-quality trials have not shown a benefit. A recent study35 with 500 patients undergoing elective coronary angioplasty randomized participants to treatment with fish-derived ω-3 PUFA capsules (5 g/d) or a control group receiving corn oil capsules (5 g/d). Treatment with ω-3 PUFA or corn oil was started 2 weeks before angioplasty and was continued until evaluation by angiography at 6 months. Restenosis occurred in 40.6% of the ω-3 PUFA group and 33.4% of the placebo group (odds ratio, 1.25; 95% confidence interval, 0.87-1.80; P = .21). Treatment with ω-3 PUFAs does not seem to prevent the high rate of restenosis experienced after angioplasty.35

**CLINICAL TRIALS**

Perhaps the most provocative studies concerning the role of dietary ω-3 PUFAs in CHD are the randomized, controlled, secondary prevention trials with hard clinical end points (CHD death and nonfatal MI). Trials with clinical end points have been completed recently with marine-based (EPA) and plant-based (ALA) sources of ω-3 PUFA (Table 2).

One of the first trials with clinical end points was the Diet and Reinfarction Trial (DART),3 which involved 2033 Welsh men who recovered from an MI. Participants were assigned to receive or not receive advice on each of 3 dietary components: a reduction in fat intake, an increase in fish intake, and an increase in cereal fiber intake. Total mortality was the primary end point, and participants were followed up for 2 years. The advice on fat or fiber intake was not associated with any change in the mortal-
ity rate. Participants in the fish advice group were instructed to eat mackerel 2 times per week or to take fish oil capsules if they could not tolerate the fish. Those advised to eat fish had a 29% reduction in 2-year all-cause mortality compared with the nonfish groups (P < .05). Consuming fish 2 times per week resulted in an absolute risk reduction of 3.5%, with a number needed to treat (NNT) to prevent 1 death of 28 during the 2-year trial.

In the Lyon Diet Heart Study, the plant-derived ω-3 PUFA ALA was supplemented in a canola oil margarine, along with a Mediterranean diet pattern. The rationale for this study was derived from a landmark dietary study, The Seven-Country Study, in which a cohort from Crete had a lower mortality rate from CHD compared with similar cohorts in other countries. Cretan participants had 3-fold higher serum concentrations of ALA compared with a similar cohort from the Netherlands. With this background, the Lyon Diet Heart Study was conducted to evaluate the effect of a Cretan Mediterranean diet—high in fruits and vegetables, rich in monounsaturated fatty acids (olive oil), and high in ALA—on CHD morbidity and mortality rates. The sources of ALA in the Cretan diet are thought to be leafy vegetables such as purslane, in addition to nuts and legumes. Because olive oil was not gastronomically acceptable to the study population in France, a customized margarine was used that had a fatty acid composition similar to olive oil in being rich in monounsaturated fat yet supplemented with ALA. The composition of the margarine included 4.8% ALA and 48% monounsaturated fat (oleic acid).

After a first MI, 605 patients were randomly assigned to receive the Mediterranean-style diet or to a control group receiving a diet similar to the National Cholesterol Education Program Step I diet. At 27 months, there was a 76% relative risk reduction in the major primary end points of cardiovascular death and nonfatal MI. The NNT was 23.

This level of risk reduction occurred without significant changes in low-density lipoprotein, HDL, or total cholesterol. The Lyon results compare favorably with those of other secondary prevention trials with lipid-lowering drugs such as the Scandinavian Simvastatin Survival Study (NNT = 12) and the Cholesterol and Recurrent Events (CARE) trial with pravastatin (NNT = 34). The risk reduction seen in the Lyon Diet Heart Study was also maintained at 46-month follow-up of the Lyon patients. Although these results are impressive, one limitation of the Lyon study is that there were numerous other changes made in the diet of the treatment group so as to resemble a Mediterranean-style dietary pattern. In addition to a 3-fold higher dietary intake of ALA, the treatment group had significantly higher oleic acid intake (olive oil), lower saturated fat intake, and decreased ω-6 PUFA (linoleic acid) intake. This makes it difficult to ascertain whether the cardioprotective effects were from the ALA-supplemented margarine or from other features of the Mediterranean diet. Although difficult to verify, the study investigators suggest that most of the risk reduction was from the ALA supplementation.

In another smaller secondary prevention trial, the Indian Experiment of Infarct Survival, 360 patients less than 1 day after MI were randomized to 1 of 3 arms: a group receiving fish oil capsules (EPA, 1.08 g/d, and DHA, 0.72 g/d), a group receiving mustand seed oil, 20 g/d (ALA, 2.9 g/d), and a control group (aluminum hydroxide, 100 mg/d). After 1 year, total cardiac events (total cardiac deaths and nonfatal MI) were significantly less in the fish oil and mustard oil groups compared with the placebo group (24.5% and 28.0%, respectively, vs 34.7%; P < .01).4

Finally, in a recent secondary prevention trial, the GISSI-Prevenzione Trial, 11,324 post-MI patients in Italy were followed up for 3½ years. Participants were randomized to 1 of 4 groups: one receiving a fish oil supplement, 1 g/d, containing 850 mg of EPA and DHA; a group receiving a vitamin E supplement (300 mg/d), a group receiving both; and a control group receiving neither. Use of vitamin E did not demonstrate any clinical benefit, whereas supplementation with EPA (850 mg/d) provided significant benefit. Supplementation with fish oil reduced cardiovascular events (cardiovascular death, nonfatal MI, and nonfatal stroke) by 20% (P = .008). That this degree of risk reduction could occur in Italian heart attack survivors practicing a prototype Mediterranean diet suggests

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Intervention</th>
<th>End Points</th>
<th>IER, %</th>
<th>CER, %</th>
<th>ARR, %</th>
<th>NNT</th>
</tr>
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<tbody>
<tr>
<td>Diet and Reinfarction Trial</td>
<td>Randomized controlled, 2033 men after MI, 24-mo follow-up</td>
<td>Fish meal twice weekly or fish oil (1500 mg/d)</td>
<td>Total mortality</td>
<td>9.3</td>
<td>12.8</td>
<td>3.5</td>
<td>28</td>
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<tr>
<td>Lyon Diet Heart Study</td>
<td>Randomized controlled, 605 patients after MI, 27-mo follow-up</td>
<td>ALA-enriched spread</td>
<td>CHD death and nonfatal MI</td>
<td>1.32</td>
<td>5.5</td>
<td>4.3</td>
<td>23</td>
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<tr>
<td>Indian Experiment of Infarct Survival</td>
<td>Randomized controlled, 360 patients after MI, 12-mo follow-up</td>
<td>Fish oil (EPA, 1 g/d) or mustard seed oil (ALA, 2.9 g/d)</td>
<td>CHD death and nonfatal MI</td>
<td>24.5</td>
<td>34.7</td>
<td>10.2</td>
<td>9.8</td>
</tr>
<tr>
<td>GISSI-Prevenzione Trial</td>
<td>Randomized controlled, 11,324 patients after MI, 42-mo follow-up</td>
<td>Fish oil (EPA + DHA, 850 mg/d)</td>
<td>CHD death and nonfatal MI</td>
<td>6.9</td>
<td>9.2</td>
<td>2.3</td>
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*IER indicates intervention event rate; CER, control event rate; ARR, absolute risk reduction; NNT, number needed to treat to prevent 1 cardiovascular event; MI, myocardial infarction; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; CHD, coronary heart disease; and ALA, α-linolenic acid.
that greater benefits might be seen with ω-3 PUFAs in a western-style diet typified by increased consumption of saturated fats and low intake of ω-3 PUFAs.

CURRENT US CONSUMPTION AND RECOMMENDATIONS

In the United States, the average intake of ω-3 PUFAs is about 1.6 g/d (about 0.7% of a 9240-kJ [2200 kcal] diet). The principal sources of ω-3 PUFA in the US diet are vegetable oils and fish. Vegetable oils (soybean and canola) are the primary source of ALA, and fish is the leading source of EPA and DHA. Recommending an optimal dietary intake is complicated by the fact that the rate at which ALA is elongated to EPA is determined by the intake of other dietary fats, notably ω-6 PUFAs (linoleic acid) and trans fatty acids. Although no official recommendations for ω-3 PUFA intake have been made in the United States, an expert panel of nutrition scientists recently suggested some guidelines (Table 3). The British Nutrition Foundation as well as several other international health organizations made similar recommendations. Based on these recommendations, ALA intake in the United States would have to increase from 1.4 g/d to 2.2 g/d (a 57% increase) and EPA and DHA intake would have to increase from 0.2 g/d to 0.65 g/d (a 400% increase) to comply with the previously mentioned recommendations.

DIETARY SOURCES OF ω-3 PUFAs

One challenge facing physicians and other primary care providers is recommending palatable sources of ALA, EPA, and DHA. Results of the Nationwide Food Consumption Survey suggest that Americans currently get the bulk of their ω-3 PUFA from 3 key food groups: (1) meat, poultry, and fish; (2) vegetable oils and salad dressings; and (3) grain products. Certain species of fatty cold water fish, such as halibut, mackerel, herring, and salmon, are good sources of EPA and DHA.
Table 6. Fatty Acid Composition of Common Oils*

<table>
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<tr>
<th>Oil</th>
<th>ω-3 PUFA, %</th>
<th>ω-6 PUFA, %</th>
<th>MUFA, %</th>
<th>Saturated Fat, %</th>
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<td>Flax</td>
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<td>Soybean</td>
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<td>13</td>
<td>85</td>
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*PUFA indicates polyunsaturated fatty acid; MUFA, monounsaturated fatty acid.

**REFERENCES**