Achieving optimal essential fatty acid status in vegetarians: current knowledge and practical implications[1–3]

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ABSTRACT Although vegetarian diets are generally lower in total fat, saturated fat, and cholesterol than are nonvegetarian diets, they provide comparable levels of essential fatty acids. Vegetarian, especially vegan, diets are relatively low in α-linolenic acid (ALA) compared with linoleic acid (LA) and provide little, if any, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Clinical studies suggest that tissue levels of long-chain $n-3$ fatty acids are depressed in vegetarians, particularly in vegans. $n-3$ Fatty acids have numerous physiologic benefits, including potent cardioprotective effects. These effects have been demonstrated for ALA as well as EPA and DHA, although the response is generally less for ALA than for EPA and DHA. Conversion of ALA by the body to the more active longer-chain metabolites is inefficient: <5–10% for EPA and 2–5% for DHA. Thus, total $n-3$ requirements may be higher for vegetarians than for nonvegetarians, as vegetarians must rely on conversion of ALA to EPA and DHA. Because of the beneficial effects of $n-3$ fatty acids, it is recommended that vegetarians make dietary changes to optimize $n-3$ fatty acid status. *Am J Clin Nutr* 2003;78(suppl):640S–6S.

KEY WORDS Essential fatty acids (EFAs), vegetarians, vegans, eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), α-linolenic acid (ALA), cardiovascular disease

INTRODUCTION

It is widely recognized that overall morbidity and mortality are lower in vegetarians compared with omnivores (1). The dietary patterns of vegetarians as well as their healthful lifestyle practices are thought to at least partly explain these differences. One notable difference relates to the type and amount of fat in the diet. Vegetarian diets are slightly lower in total fat than omnivorous diets (28–32% for vegans, 30–34% for lactoovovegetarians, and 34–36% for omnivores) (2). However, vegetarians eat about one-third less saturated fat (vegans about one half) and about one-half as much cholesterol (vegans consume none) as omnivores (3–5). Intake of *trans* fatty acids is highly dependent on the amount of processed foods in the diet. Limited research comparing *trans* fatty acid intake of omnivores and vegetarians suggests that vegetarians consume slightly smaller amounts, with more pronounced differences for vegans eating whole-foods diets (3). One study found that vegetarians had a markedly lower serum *trans* fatty acid level than nonvegetarians (0.03% compared with 0.50% of total fatty acids), a finding that presumably reflects differences in intake (6).

With respect to essential fatty acid (EFA) intake and balance, vegetarian diets appear to offer no advantages over omnivorous dietary patterns. Some have suggested that vegetarians could be at a significant disadvantage, as consumption of α-linolenic acid (ALA) is low relative to linoleic acid (LA), resulting in limited conversion of ALA to eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (6). In addition, vegetarians consume very little EPA and DHA (7).

The purpose of this paper is to review the EFA intake and status of vegetarians; compare the biological effects of ALA and its long-chain metabolites, EPA and DHA; and explore the practical implications of this knowledge as it relates to optimal EFA status in this population. In addition, the scientific justification for ensuring adequate EFA status with respect to cardiovascular disease (CVD) will be reviewed.

OPTIMIZING ESSENTIAL FATTY ACID STATUS

Essential fatty acid conversion

There are 2 EFAs, both polyunsaturated fats: LA (the parent $n-6$ fatty acid) and ALA (the parent $n-3$ fatty acid). Humans are able to convert LA and ALA to more physiologically active fatty acids through a series of elongation and desaturation reactions [LA to arachidonic acid (AA) and ALA to EPA and DHA]. The resulting highly unsaturated fatty acids are necessary for cell membrane function, the proper development and functioning of the brain and nervous system, and the production of eicosanoids (thromboxanes, leukotrienes, prostaglandins, and prostacyclins). While conversion of LA to AA is typically very efficient, conversion of ALA to EPA and DHA is much less so. In healthy individuals, ≈5–10% of ALA is converted to EPA, and ≈2–5% to DHA (8–10). In a recent study conducted with women, estimated net fractional conversion of ALA to EPA was 21% and 9% for DHA (11). The long-chain members of the $n-6$ and $n-3$ families generally are not considered essential because humans have the capacity to make them from "parent" fatty acids. $n-6$ And $n-3$ fatty acids compete for enzymes responsible for their conversion;

because they are not interchangeable, humans must consume both. This takes on special importance in people with diabetes or other metabolic disorders where conversion enzymes may be compromised, as well as in those who have inherited a limited ability to produce these enzymes (12).

Recommended essential fatty acid intake

Recently, the National Academies released the Dietary Reference Intakes Report for Energy and Macronutrients (13). Adequate intakes (AIs) have been set for LA and ALA.

The AI for LA is 17 and 12 g/d for men and women aged 19–50 y, respectively. The AI for ALA is 1.6 and 1.1 g/d for men and women aged 19 to > 70 y, respectively. In 1999, the National Institutes of Health (NIH) sponsored an international workshop on the essentiality and recommended dietary intakes for $n-6$ and $n-3$ fatty acids. The NIH Working Group proposed AIs of 2–3% of total calories for LA, 1% of total calories for ALA, and 0.3% of total calories for EPA and DHA. The Working Group further recommended intakes of EPA and DHA of ≥ 650 mg/d and a minimum of 300 mg DHA/d during pregnancy and lactation (14). Health Canada suggests a minimum of 3% of energy from $n-6$ fatty acids and 0.5% from $n-3$ fatty acids or 1% for infants who do not receive a preformed source of EPA and DHA (15). The United Kingdom recommends that 1% of energy be from ALA and 0.5% from EPA and DHA combined (16). While there are no official recommendations for vegetarians and vegans, it is not possible for this population to achieve the NIH Working Group's proposed AIs for EPA and DHA. Even with the use of DHA-enriched eggs, some seaweed, and/or DHA supplements, the best vegetarians could do is to meet the recommended intakes for DHA. Some experts suggest that vegetarians (and others receiving no direct sources of EPA and DHA) at least double the recommended intakes for ALA (S Innis, A Simopoulos, and B Holub, personal communication, 2002). This would suggest an intake of ALA in the range of 1–2%.

The ratio of $n-6$ to $n-3$ fatty acids is often used to assess the balance between EFA in the diet, although there is some controversy as to its practical significance. For vegetarians and others who consume little, if any, EPA and DHA, the $n-6$-to-$n-3$ ratio is of greater relevance than for individuals who consume significant daily sources of EPA and DHA. A number of recommendations have been made on the basis of the ratio of $n-6$ to $n-3$ fatty acids. The World Health Organization/Food and Agriculture Organization suggests a ratio of 5:1–10:1 (17), Sweden recommends a ratio of 5:1 (18), Canada recommends 4:1–10:1 (15), and Japan recently changed its recommendation from 4:1 to 2:1 (19). On the basis of the proposed AIs, the NIH suggests a ratio of 2:1–3:1 (14). One study found that a ratio of 4:1 allows for adequate conversion to DHA in healthy vegetarians (20). Another research group suggested that the optimal ratio to maximize the conversion of ALA to DHA is 2.3:1 (21). Given the rate of conversion of ALA to EPA and DHA, it has been suggested that a safe and adequate ratio for the vegetarian and vegan populations would be in the range of 2:1–4:1 (22). This can best be achieved by increasing ALA in the diet and decreasing LA, if indicated (see below).

Essential fatty acid intake and status of vegetarians

Total $n-3$ fatty acid intakes are similar for vegans, vegetarians, and omnivores (< 1–3 g/d, with the current average being ≈ 1.1–1.6 g/d) (2, 18). However, the intakes of very-long-chain $n-3$ fatty acids (EPA and DHA) vary appreciably. Vegans consume negligible amounts of EPA and DHA, while vegetarians consume minimal EPA (< 5 mg/d) and varying amounts of DHA depending on egg consumption (a source of DHA, averaging < 33 mg/d) (7). Consumption of EPA and DHA in omnivores varies according to fish and egg intake, with average intakes in the 100–150-mg/d range. $n-6$ Intakes are significantly higher in vegan and vegetarian populations than in omnivorous populations, ranging from a low of about 5–7% of calories in omnivores to a high of about 10–12% of calories in vegans. As a result, the $n-6$-to-$n-3$ ratio is generally considered to be elevated in vegans (< 14:1–20:1) and lactoovovegetarians (< 10:1–16:1) compared with omnivores (< 10:1).

Because the consumption of long-chain $n-3$ fatty acids is low in vegetarian populations and LA intakes are relatively high, one would expect decreased levels of EPA and DHA in body stores. Reports to date suggest that this is indeed the case but that it is significantly more pronounced in vegans than in vegetarians. In 1978 Sanders et al (23) first noted that the plasma and total erythrocyte lipid levels of vegans were significantly decreased. EPA levels were only 12–15% and DHA levels were 32–35% of those of nonvegetarians (23). Another study found that vegan plasma EPA levels were only 22% of those of omnivores and DHA levels were 38% of those of omnivores, although AA levels were similar (24). In 1994, Reddy et al (25) again demonstrated that vegetarians (who appeared to be vegan, based on zero intake of EPA and DHA) had reduced EPA in plasma phospholipids (37% of nonvegetarians), and DHA (52% of nonvegetarians). Krajcovicova et al (26) compared the fatty acid status of vegans, lactoovovegetarians, semivegetarians, and omnivores between the ages of 11 and 15 y. While the levels of EPA and DHA were similar in vegetarians and nonvegetarians, they were significantly lower in vegans (62–65% of nonvegetarians' values). Interestingly, while long-chain $n-3$ fatty acids were reduced in vegan children, they were not as severely decreased as reported in vegan adults. The higher EPA and DHA levels in vegan children may be the result of reserves from extended breast-feeding, or from better conversion in this age group (26).

Ågren et al (27) compared the serum lipid levels of vegans eating uncooked food with those of nonvegetarians. The proportions of EPA and DHA in vegans were only 29–36% and 49–52% those of nonvegetarian controls, respectively, while the levels of AA were similar, indicating no difficulty with the $n-6$ conversion (27). Sanders and Reddy (28) compared the EFA content of vegan, vegetarian, and nonvegetarian human milk. Milk from vegan mothers had over double the LA and ALA of the nonvegetarian mothers' milk but less than half of the DHA. The EFA status of the infants (as determined by erythrocyte lipids) reflected the levels in the milk they received. Vegan infants had < 30% of the EPA and DHA of omnivorous infants (28). Melchert et al (29) examined the fatty acid levels in the serum of vegetarians and nonvegetarians and found that DHA levels were < 40% lower in vegetarians than nonvegetarians. Two other groups comparing EPA and DHA status of vegetarians and nonvegetarians found little difference in their levels (30, 31). Conquer and Holub (31) suggest that it is possible that Canadian vegetarians have elevated ALA intakes because of the high consumption of canola oil in their diets and, thus, better DHA status. These studies did not assess EFA status in vegans.

Implications of reduced essential fatty acid status

Highly unsaturated fatty acids are essential for health (32). AA, dihommo-γ-linolenic acid (DGLA), and EPA all serve as

precursors for eicosanoids. Eicosanoids formed from AA are very potent, increasing blood pressure, inflammation, platelet aggregation, thrombosis, vasospasm, and cell proliferation, while those formed from EPA and DGLA are less potent in these responses. While the eicosanoids formed from AA are important, their overproduction relative to eicosanoids formed from EPA has been associated with elevated risk for numerous disease states, including heart disease, cancer, diabetes, osteoporosis, and numerous immune/inflammatory disorders.

Docosahexaenoic acid is not a precursor for eicosanoids but is an important structural component of the gray matter of the brain, the retina of the eye, and specific cell membranes and is found in high levels in the testes and sperm. Low levels of DHA have been associated with several neurological and behavioral disorders, such as depression, schizophrenia, Alzheimer's disease, and attention deficit hyperactivity disorder (7). In addition, low levels of DHA are linked to suboptimal visual acuity and reduced brain development in infants (33). Thus, while these long-chain fatty acids are not technically "essential" nutrients, it is important to ensure that there are sufficient levels by relying on conversion from "parent" fatty acids or by consuming them directly.

Scientific evidence for increasing $n-3$ fatty acids in vegetarian diets

There is a rapidly expanding database that demonstrates remarkable effects of $n-3$ fatty acids on primary and secondary prevention of CVD. Both epidemiologic and randomized controlled clinical studies have evaluated the effects of marine and plant sources of $n-3$ fatty acids from food sources as well as supplements. These studies have established that both marine- and plant-derived $n-3$ fatty acids have cardioprotective effects. These findings have prompted great interest in unraveling the biological mechanisms that explain these reported benefits. It is apparent that multiple mechanisms of action mediate the effects of $n-3$ fatty acids. Collectively, advances from epidemiologic, clinical, and biochemistry/molecular biology research will be useful in refining dietary guidance to further decrease the risk of CVD.

Epidemiologic studies

α-Linolenic acid. Recent epidemiologic studies have shown that dietary ALA is associated with a lower risk of CVD in men and women. In the EURAMIC Study, the relative risk for myocardial infarction (MI) was 0.42 (*P* for trend = 0.02) in individuals with the highest quintile of adipose tissue ALA, a long-term measure of ALA intake (34). However, after other CVD risk factors were adjusted for, this association was nonsignificant. In a 10-y follow-up of the Nurses' Health Study, Hu et al (35) reported a dose-response relationship between ALA intake and relative risk of fatal ischemic heart disease. Risk was reduced by 45% in the highest quintile of ALA intake (*P* for trend = 0.01). However, ALA was not related to nonfatal myocardial infarction. In the Health Professionals Follow-up Study, conducted with men, a 1% increase in ALA was associated with a 0.41 relative risk for acute MI (*P* for trend = 0.01); however, there was no association with fatal coronary artery disease (CAD) (36). The highest quintile of ALA intake was 1.4–1.5 g/d. In a recent cross-sectional study (the National Heart, Lung, and Blood Institute Family Heart Study) with 4584 men and women, ALA was inversely related to coronary artery disease (37). ALA intake in the top 3 quintiles was 0.65, 0.76, and 0.96 g/d, respectively; the prevalence odds ratio of coronary artery disease in these 3 quintiles was reduced < 40% for men and 50–70% for women.

In contrast to epidemiologic studies that have demonstrated a beneficial association between ALA intake and CVD, some studies have found the contrary. In the Zutphen Elderly Study, a prospective epidemiologic study with 667 men aged 64–84 y, there was no beneficial effect of ALA intake on incidence of 10-y coronary artery disease (38). The results of this study, however, were complicated by an association between ALA and *trans* fatty acid intake (37) as well as limitations in the collection of the dietary data (estimated on the basis of only food tables and dietary recollection) (39). ALA intake still had no effect on coronary artery disease risk even when foods that did not contain *trans* fatty acids were evaluated.

On the basis of available epidemiologic evidence from within population studies, ALA has been shown to have a protective effect on coronary disease. These findings provide strong support for conducting well-controlled intervention trials to assess the cause-and-effect relationship between ALA and incidence of CVD.

Marine-derived $n-3$ fatty acids. There has been controversy in the literature about whether fish consumption decreases coronary artery disease CAD mortality. To resolve this controversy, Marckmann and Gronbaek (40) systematically reviewed studies of fish or $n-3$ polyunsaturated fatty acid intake and CAD death (11 studies with 116 764 subjects). This analysis found that individuals at high risk for CAD appeared to benefit in a dose-responsive manner from increased fish consumption. The optimum dose for fatal CAD prevention in high-risk populations is 40–60 g/d. In contrast, increased fish consumption does not appear to protect against CAD in individuals at low risk of CAD and with healthy lifestyles.

There have been various explanations for the conflicting data from epidemiologic studies of fish consumption and CAD risk. These explanations reflect many differences in the experimental designs employed, including definition of sudden death (41), endpoint, experimental design, fish intake estimation method, and study population (42). Another consideration relates to the type of fish consumed; fatty fish but not lean fish consumption was associated with a lower CAD mortality (43). In addition, the possible adverse effects of the methylmercury content of fish may attenuate the health benefits of $n-3$ fatty acids (44).

The available evidence supports a conclusion that fish consumption is protective against coronary disease. Because of this, the American Heart Association Dietary Guidelines recommend 2 servings of fish per week, preferably fatty fish, to reduce the risk of coronary disease (45).

Randomized controlled clinical trials

The effects of $n-3$ fatty acids have been evaluated in clinical trials with subjects either with existing CVD or at high risk for CVD using both marine-derived EPA and DHA and plant-derived ALA. Collectively, these studies have shown a beneficial effect of both sources of $n-3$ fatty acids.

The Diet and Reinfarction Trial (DART) reported a 29% reduction in all-cause mortality after 2 y in male MI survivors who were counseled to increase their intake of oily fish (200–400 g fatty fish/wk, which provided an additional 500–800 mg $n-3$ fatty acids/d) (46). A fish oil supplement (900 mg EPA and DHA/d) was given to patients who would not eat fish, and a subsequent analysis demonstrated that $n-3$ fatty acids accounted for the protective effect (47). Singh et al (48) studied patients with suspected acute MI who were randomized to either fish oil capsules (containing 1.8 g EPA and DHA/d), mustard oil (20 g/d providing 2.9 g

ALA), or placebo. Total cardiac events were lower in the fish oil and mustard oil groups (25% and 28%, respectively) compared with the placebo group (ie, 35%; $P < 0.01$). In the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico-Prevenzione (GISSI-Prevention Study) (49), 11 324 patients with preexisting CAD who were receiving conventional cardiac pharmacotherapy were randomly assigned to receive 850 mg $n-3$ fatty acid ethyl esters (as EPA and DHA), 300 mg vitamin E, both, or neither. The $n-3$ fatty acid intervention group experienced a 20% reduction in all-cause mortality ($P = 0.01$) and a 45% reduction in sudden death ($P < 0.001$) compared with the control group. No additional benefit was conferred by vitamin E.

In contrast, a recent study did not find an effect of 3.5 g DHA and EPA/d compared with corn oil on cardiac events in post-MI patients ($n = 300$) after 1.5 y of intervention (50). The lack of response to marine-derived $n-3$ fatty acids may have been due to the high habitual fish intake in western Norway, beyond which supplemental $n-3$ fatty acids would not be of benefit.

The effects of ALA on coronary events have been evaluated in 4 studies. Three studies reported beneficial effects of ALA, and one did not. The Indian Experiment of Infarct Survival (48) (*see* above) reported a significant decrease in total cardiac events in the mustard seed oil (a source of ALA) group that was quite comparable to that of the EPA and DHA treatment group. The Lyon Diet Heart Study, a seminal study, was designed to evaluate the effects of a blood cholesterol–lowering, Mediterranean-type diet (including increased amounts of ALA: 1.5 g/d compared with 0.5 g/d for the control group) on recurrence rates of cardiac events compared with rates for a prudent Western diet (51, 52). Subjects in the experimental group were instructed to adopt a Mediterranean-type diet that contained more bread, more root and green vegetables, more fish, fruit at least once daily, less red meat (replaced with poultry), and less butter and cream (replaced with margarine high in ALA supplied by the study). This diet provided 30% of calories from fat, 8% from saturated fat, 13% from monounsaturated fat, 4.6% from polyunsaturated fat (0.84% ALA), and 203 mg cholesterol/d. Despite a similar coronary risk factor profile (plasma lipids and lipoproteins, systolic and diastolic blood pressure, body mass index, and smoking status), subjects following the Mediterranean-style diet had a 50–70% lower risk of recurrent heart disease. The Mediterranean α-Linolenic Enriched Groningen Dietary Intervention (MARGARIN) study reported a trend toward fewer CVD events in subjects ($n = 124$ men and 158 women) with multiple CVD risk factors who used a margarine high in ALA versus LA (1.8% compared with 5.7%; $P = 0.20$) (53). However, the 10-y estimated ischemic heart disease risk decreased similarly in both groups (2.1% and 2.5%, respectively).

The Norwegian Vegetable Oil Experiment (54) did not report a beneficial effect of ALA. In this study, over 13 000 men aged 50–59 y with no history of MI were randomly assigned to consume 5.5 g ALA/d (from 10 mL linseed oil) or 10 mL sunflower seed oil for 1 y. In each treatment group there were 27 cases of new CAD or sudden death, and 40 compared with 43 deaths from any cause in the control and linseed oil groups, respectively.

In summary, evidence from randomized controlled trials shows a beneficial effect of dietary and supplemental $n-3$ fatty acids, including both EPA/DHA and ALA, on CAD (55). Thus, there is scientific evidence that adequate intake of $n-3$ fatty acids will elicit cardioprotective effects. From this, it follows that $n-3$ fatty acids are an integral component of a diet designed to maximally reduce risk of CVD.

Achieving optimal essential fatty acid status in vegetarians

There are 2 important steps vegetarians can take to improve their EFA status. *1*) Maximize the conversion of ALA to EPA and DHA. *2*) Provide a direct source of EPA and DHA.

For those with increased needs for EPA and DHA (eg, pregnant and lactating women) or at greater risk for poor conversion (persons with diabetes, those with neurological disorders, premature infants, the elderly), it may be prudent to ensure that there is a direct source of EPA and DHA. While it is not common, it is possible to overconsume $n-3$ fatty acids. If a person minimizes $n-6$ fatty acids and uses large amounts of $n-3$ fatty acids [ie, > 2 tbsp (28 g) flax oil/d], resulting in an $n-6$-to-$n-3$ ratio of < 1:1, insufficient LA conversion to AA can occur. Elongase and desaturase enzymes preferentially convert $n-3$ fatty acids, when compared with $n-6$ fatty acids. A balance of 2:1–4:1 ($n-6$:$n-3$) appears optimal for vegetarians and others who do not receive preformed EPA and DHA.

Maximizing conversion of n−3 fatty acids

While conversion of EFAs to longer-chain fatty acids is, at least in part, dependent on genetics, age, and overall health, several dietary factors also have a significant impact on the conversion process. First, it is important to ensure that the diet is nutritionally adequate, as poorly designed diets can impair the conversion process. Insufficient energy or protein decreases the activity of conversion enzymes, as can deficiencies of pyridoxine, biotin, calcium, copper, magnesium, and zinc (56, 57). Excessive intakes of trans fatty acids can also depress conversion enzymes. In addition, alcohol inhibits the activity of Δ-5 and Δ-6 desaturase and depletes tissues of long-chain $n-3$ fatty acids (58). High $n-6$ fatty acid content can have a profound effect on $n-3$ fatty acid conversion, reducing it as much as 40% (59).

In the context of current guidelines, total polyunsaturated fatty acid intake should be < 7% of calories, with 10% being the maximum. Vegetarians currently consume about 8–12% of their calories from polyunsaturated fatty acid. Given a total polyunsaturated fatty acid intake of 7–10% of calories, to achieve an $n-6$-to-$n-3$ ratio of 4:1, < 1.5–2% of calories should be obtained from $n-3$ fatty acids and, as a result, 5.5–8% of calories from $n-6$ fatty acids. Alternatively, LA can be decreased; however, given the recognized health benefits of LA, a marked reduction is not recommended. To achieve reduced $n-6$ intakes, oils rich in $n-6$ fatty acids should not be used as primary cooking oils. Cooking oils with the greatest $n-6$ fatty acid content include safflower oil (75% $n-6$), grapeseed oil (70% $n-6$), sunflower oil (65% $n-6$), corn oil (57% $n-6$), cottonseed oil (52% $n-6$), and soybean oil (51% $n-6$). Processed foods, convenience foods, and snack foods also are significant contributors to $n-6$ intake; thus, their use should be moderate. $n-6$-Rich whole foods such as sunflower seeds, pumpkin seeds, sesame seeds, walnuts, wheat germ, and soy foods need not be avoided, as they tend to be relatively minor contributors to overall $n-6$ intake. These foods also provide a myriad of beneficial dietary components, including phytochemicals, fiber, B vitamins, vitamin E, and trace minerals.

The primary fat in the diet should come from foods and oils rich in monounsaturated fat. When monounsaturated fats predominate, saturated fats, trans fatty acids, and $n-6$ fatty acids are kept in check and the ratio of $n-6$ to $n-3$ fatty acids improves. Monounsaturated fats are high in nuts (except for

TABLE 1
α-Linolenic acid (ALA) and linoleic acid (LA) content of selected plant foods[1]

Food and serving size	ALA	ALA	LA	n−6:n−3	Comments
	%	*g/serving*	*%*		
Flaxseed oil, 1 tbsp (14 g)	57	8.0	16	0.28:1	Richest known source of ALA, highly unstable, should not be heated.
Flaxseed, whole, 2 tbsp (24 g)	57	5.2	16	0.28:1	Keeps well at room temperature.
Flaxseed, ground, 2 tbsp (24 g)	57	3.8	16	0.28:1	Best kept refrigerated or frozen.
Greens (mixed), 1 cup (56 g)	56	0.1	11	0.19:1	Fat in greens is >50% ALA; however, because total fat is so low, they are not significant contributors to intake for most people.
Hempseed oil, 1 tbsp (14 g)	19	2.7	57	3:1	One of the few foods that contains GLA (1.7% GLA).
Walnuts, 1 oz (¼ cup; 28 g)	14	2.6	58	4:1	Highest n−3 content of any common nut; only the candlenut has more (30% ALA).
Canola oil, 1 tbsp (14 g)	11	1.6	21	2:1	Excellent n−6-to-n−3 ratio. To avoid the genetically engineered canola, buy certified organic.
Soybean oil, 1 tbsp (14 g)	7	0.9	51	7:1	Not the best choice for general use because of high n−6 content.
Soybeans, 1 cup cooked (172 g)	7	1.0	50	7:1	Can make a significant contribution to total ALA intake.
Tofu, firm, ½ cup (4.5 oz; 126 g)	7	0.7	50	7:1	Same as soybeans.

[1] Adapted from reference 60. GLA, γ-linolenic acid.

walnuts and butternuts), peanuts (a legume), olive oil, olives, avocados, canola oil, high-oleic sunflower oil, and high-oleic safflower oil. Whole foods rather than oils are better sources of monounsaturated fats because they contribute many other nutrients to the diet.

Finally, it is important to ensure that there are sufficient amounts of ALA, which is necessary for the production of EPA and DHA. Most healthy vegetarians would be well advised to double their intake of ALA, providing ≥1% of energy from n−3 fatty acids or 1.1 g/1000 cal. For those with increased needs or decreased capacity to convert, an intake of 2% of energy or 2.2 g/1000 cal may be necessary. The primary sources of ALA are selected seeds, nuts, and legumes (flaxseed, hempseed, canola, walnuts, and soy) and the green leaves of plants, including phytoplankton and algae. **Table 1** provides a list of common ALA-rich plant foods, the percentage of fat as ALA, the n−6-to-n−3 ratio, and the total grams of ALA per serving.

Providing a direct source of eicosapentaenoic and docosahexaenoic acids

The primary sources of EPA and DHA are fish and seafood. Thus, for vegetarians, increasing consumption of these long-chain n−3 fatty acids can be a challenge. For lactoovovegetarians, eggs provide a reasonable amount of DHA (<50 mg/egg) but very little EPA. Most supermarkets also sell DHA-rich eggs, providing 2–3 times the DHA of conventional eggs. Eggs from chickens fed flax generally provide 60–100 mg DHA/egg, while those from chickens fed microalgae contain 100–150 mg DHA/egg.

The only plant sources of long-chain n−3 fatty acids are plants of the sea—microalgae and seaweed. There is a great deal of confusion about the EFA content of plants. While they are the original sources of EPA and DHA (fish do not produce long-chain n−3 fatty acids), most are not concentrated sources because of their extremely low total fat content. An important exception is a DHA-rich microalgae that provides 10–40% DHA by dry weight and is currently available in supplement form. When supplementing with a direct DHA source, 100–300 mg/d is recommended. Blue-green algae (spirulina and *Aphanizomenon flos-aquae*) are

low in long-chain n−3 fatty acids. Spirulina is rich in γ-linolenic acid (GLA, n−6), while *A. flos-aquae* is more concentrated in ALA. Though blue-green algae is not a significant source of EPA or DHA, some research indicates that it has a very high conversion rate in comparison to other plants (R Kushak et al, unpublished observations, 1999). Macroalgae, otherwise known as seaweed, is even lower in fat than most vegetables (<1–14% of calories from fat), although it does contain small amounts of long-chain fatty acids. A 100-g serving provides, on average, ≈100 mg EPA but little DHA. Seaweeds do not contribute significantly to EPA intakes in the Western world but are important sources where people use large quantities of seaweed on a daily basis (eg, Japan and other parts of Asia). Thus, while vegetarians can rely on eggs and/or microalgae supplements for DHA, most consume little if any EPA. However, <10–11% of DHA is retroconverted to EPA; thus, if sufficient ALA and DHA are consumed, total EPA production would be expected to be adequate (61).

Practical guidelines

Practical guidelines for achieving optimal EFA intake in vegetarians are as follows. *1*) Make a wide variety of whole plant foods the foundation of the diet. *2*) Get most fat from whole foods— nuts, seeds, olives, avocados, and soy foods. *3*) If using concentrated fats and oils, select those rich in monounsaturated fats, such as olive, canola, or nut oils. n−3-Rich oils can also be used but should not be heated. Moderate use of n−6-rich oils is recommended. *4*) Limit intake of processed foods and deep-fried foods rich in *trans* and n−6 fatty acids. *5*) Reduce intake of foods rich in saturated fat and cholesterol. *6*) Include foods rich in n−3 fatty acids in the daily diet. Aim for 2–4 g ALA/d. *7*) Consider using a direct source of DHA. Aim for 100–300 mg/d.

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REFERENCES
1. American Dietetic Association. Position of the American Dietetic Association: vegetarian diets. J Am Diet Assoc 1997;97:1317–21.

2. Messina MJ, Messina VL. The dietitian's guide to vegetarian diets: issues and applications. Gaithersburg, MD: Aspen Publishers, 1996.

3. Draper A, Lewis J, Malhotra N, Wheeler E. The energy and nutrient intakes of different types of vegetarian: a case for supplements? Br J Nutr 1993;69:3–19. (Published erratum appears in Br J Nutr 1993; 70:812.)

4. Janelle KC, Barr SI. Nutrient intakes and eating behavior scores of vegetarian and non-vegetarian women. J Am Diet Assoc 1995;95:180–8.

5. Melby CL, Foldflies DG, Toohey ML. Blood pressure differences in older black and white long-term vegetarians and non-vegetarians. J Am Coll Nutr 1993;12:262–9. (Published erratum appears in J Am Coll Nutr 1993;12:after the table of contents.)

6. Lee HY, Woo J, Chen ZY, Leung SF, Peng XH. Serum fatty acid, lipid profile and dietary intake of Hong Kong Chinese omnivores and vegetarians. Eur J Clin Nutr 2000;54:768–73.

7. Conquer J, Holub B. Docosahexaenoic acid ($n-3$) and vegetarian nutrition. Veg Nutr Int J 1997;1:42–9.

8. Ghafoorunissa SA. Requirements of dietary fats to meet nutritional needs and prevent the risk of atherosclerosis—an Indian perspective. Indian J Med Res 1998;108:191–202.

9. Emken EA, Adolf RO, Gulley RM. Dietary linoleic acid influences desaturation and acylation of deuterium-labeled linoleic and linolenic acids in young adult males. Biochim Biophys Acta 1994;1213: 277–88.

10. Gerster H. Can adults adequately convert α-linolenic acid (18:3 $n-3$) to eicosapentaenoic acid (20:5 $n-3$) and docosahexaenoic acid (22: 6 $n-3$)? Int J Vitam Nutr Res 1998;68:159–73.

11. Burdge GC, Wootton SA. Conversion of alpha-linolenic acid to eicosapentaenoic, docosapentaenoic and docosahexaenoic acids in young women. Br J Nutr 2002;88:411–20.

12. Simopoulos AP. Essential fatty acids in health and chronic disease. Am J Clin Nutr 1999;70(suppl):560S–9S.

13. Institute of Medicine of the National Academies. Dietary Reference Intakes: energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. Washington, DC: National Academies Press, 2002.

14. Simopoulos AP, Leaf A, Salem N. Workshop on the essentiality of and recommended dietary intakes for $n-6$ and $n-3$ fatty acids. Bethesda, MD: National Institutes of Health, 1999.

15. Scientific Review Committee. Nutrition recommendations. Ottawa: Canadian Government Publishing Centre, Supply and Services Canada, 1990.

16. COMA Cardiovascular Review Group. Report on health and social subjects: nutritional aspects of cardiovascular disease. London: Department of Health, 1994:46.

17. WHO and FAO Joint Consultation: fats and oils in human nutrition. Nutr Rev 1995;53:202–5.

18. Nordic Working Group on Diet and Nutrition within Nordic Committee of Senior Officials for Food Issues, the Nordic Council of Ministers. Nordic nutrition recommendations. Scand J Nutr 1996;40:161–5.

19. Kris-Etherton PM, Taylor DS, Yu-Poth S, et al. Polyunsaturated fatty acids in the food chain in the United States. Am J Clin Nutr 2000; 71(suppl):179S–88S.

20. Indu M, Ghafoorunissa SA. $n-3$ Fatty acids in Indian diets: comparison of the effects of precursor (alpha-linolenic acid) vs. product (long chain $n-3$ polyunsaturated fatty acids). Nutr Res 1992;12: 569–82.

21. Masters C. $n-3$ Fatty acids and the peroxisome. Mol Cell Biochem 1996;165:83–93.

22. Davis B, Melina V. Becoming vegan. Summertown, TN: Book Publishing Company, 2000.

23. Sanders TAB, Ellis FR, Dickerson JWT. Studies of vegans: the fatty acid composition of plasma choline phosphoglycerides, erythrocytes, adipose tissue, and breast milk, and some indicators of susceptibility to ischemic heart disease in vegans and omnivore controls. Am J Clin Nutr 1978;31:805–13.

24. Sanders TAB, Roshanai F. Platelet phospholipid fatty acid composition and function in vegan compared with age- and sex-matched omnivore controls. Euro J Clin Nutr 1992;46:823–31.

25. Reddy S, Sanders TAB, Obeid O. The influence of maternal vegetarian diet on the essential fatty acid status of the newborn. Eur J Clin Nutr 1994;48:358–68.

26. Krajcovicova-Kudlackova M, Simoncic R, Bederova A, Klvanova J. Plasma fatty acid profile and alternative nutrition. Ann Nutr Metab 1997;41:365–70.

27. Ågren J, Törmalä M, Nenonen M, Hänninen O. Fatty acid composition of erythrocyte, platelet, and serum lipids in strict vegans. Lipids 1995;30:365–9.

28. Sanders TBA, Reddy S. The influence of a vegetarian diet on the fatty acid composition of human milk and the essential fatty acid status of the infant. J Pediatr 1992;120:S71–7.

29. Melchert HU, Limsathayourat N, Mibajlovic H, Eichber J, Thefeld W, Rottkea H. Fatty acid patterns in triglycerides, diglycerides, free fatty acids, cholesterol esters and phosphatidylcholine in serum from vegetarians and non-vegetarians. Atherosclerosis 1987;65:159–66.

30. Kirkeby K, Bjerkedal I. The fatty acid composition in serum of Norwegian vegetarians. Acta Med Scand 1968;183:143–8.

31. Conquer JA, Holub BJ. Dietary docosahexaenoic acid as a source of eicosapentaenoic acid in vegetarians and omnivores. Lipids 1997; 21:341–5.

32. Holman RT, Johnson SB, Hatch TF. A case of human linolenic acid deficiency involving neurological abnormalities. Am J Clin Nutr 1982;35:617–23.

33. Uauy R, Hoffman DR, Peirano P, Birch DG, Birch EE. Essential fatty acids in visual and brain development. Lipids 2001;36:885–95.

34. Guallar E, Aro A, Jimenez J, et al. $N-3$ Fatty acids in adipose tissue and risk of myocardial infarction. The EURAMIC Study. Arterioscler Thromb Vasc Biol 1999;19:1111–8.

35. Hu FB, Stampfer MJ, Manson JE, et al. Dietary intake of α-linolenic acid and risk of fatal ischemic heart disease among women. Am J Clin Nutr 1999;69:890–7.

36. Ascherio A, Rimm EB, Giovannucci EL, Spiegelman D, Stampfer M, Willett WC. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. BMJ 1996;313:84–90.

37. Djousse L, Pankow JS, Eckfeldt JH, et al. Relation between dietary linolenic acid and coronary artery disease in the National Heart, Lung, and Blood Institute Family Heart Study. Am J Clin Nutr 2001; 74:612–9.

38. Oomen CM, Ocke MC, Feskens EJ, Kok FJ, Kromhout D. α-Linolenic acid intake is not beneficially associated with 10-y risk of coronary artery disease incidence: the Zutphen Elderly Study. Am J Clin Nutr 2001;74:457–63.

39. Visioli F, Galli C. α-Linolenic acid and cardiovascular disease. Am J Clin Nutr 2002;75:1121 (letter).

40. Marckmann P, Gronbaek M. Fish consumption and coronary heart disease mortality: a systematic review of prospective cohort studies. Eur J Clin Nutr 1999;53:585–90.

41. Kromhout D. Fish consumption and sudden cardiac death. JAMA 1998;279:65–6.

42. Sheard NF. Fish consumption and risk of sudden cardiac death. Nutr Rev 1998;56:177–9.

43. Oomen CM, Feskens EJ, Rasanen L, et al. Fish consumption and coronary heart disease mortality in Finland, Italy, and the Netherlands. Am J Epidemiol 2000;151:999–1006.

44. Rissanen T, Voutilainen S, Nyyssonen K, Lakka TA, Salonen JT. Fish-oil derived fatty acids, docosahexaenoic acid and docosapentaenoic acids, and the risk of acute coronary events. The Kuopio Ischaemic Heart Disease Risk Factor Study. Circulation 2000;102:2677–9.

45. Krauss RM, Eckel RH, Howard B, et al. AHA dietary guidelines: revision 2000: a statement for healthcare professionals from the nutrition committee of the American Heart Association. Circulation 2000; 102:2284–99.

46. Burr ML, Fehily AM, Gilbert JF, et al. Effects of change in fat, fish

and fibre intakes on death and myocardial reinfarction: Diet and Rein-farction Trial (DART). Lancet 1989;2:757–61.

47. Burr ML, Sweetham PM, Fehily AM. Diet and reinfarction. Eur Heart J 1994;15:1152–3.
48. Singh RB, Niaz NIA, Sharma JP, Kumar R, Rastogi V, Moshiri M. Randomized, double-blind, placebo-controlled trial of fish oil and mustard oil in patients with suspected acute myocardial infarction: the Indian Experiment of Infarct Survival, IV. Cardiovasc Drug Ther 1997;11:485–91.
49. GISSI-Prevenzione Investigators. Dietary supplementation with $n-3$ polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. Lancet 1999;354:447–55.
50. Nilsen DW, Albrektsen G, Landmark K, Moen S, Aarsland T, Woie L. Effects of a high-dose concentrate of $n-3$ fatty acids or corn oil introduced early after an acute myocardial infarction on serum tria-cylglycerol and HDL cholesterol. Am J Clin Nutr 2001;74:50–6.
51. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardio-vascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. Circulation 1999;99:779–85.
52. de Lorgeril M, Renaud S, Mamelle N, et al. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart dis-ease. Lancet 1994;343:1454–9.
53. Bemelmans WJ, Broer J, Feskens EJ, et al. Effect of an increased intake of α-linolenic acid and group nutritional education on cardio-vascular risk factors: the Mediterranean α-Linolenic Enriched

Groningen Dietary Intervention (MARGARIN) study. Am J Clin Nutr 2002;75:221–7.
54. Natvig H, Borchgrevink CF, Dedichen J, Owren PA, Schiotz EH, Westlund K. A controlled trial of the effect of linolenic acid on the incidence of coronary heart disease. Scand J Clin Lab Med 1968; 105(suppl):1–20.
55. Bucher HC, Hengstler P, Schindler C, Meier G. $n-3$ Polyunsaturated fatty acids in coronary heart disease: a meta-analysis of randomized controlled trials. Am J Med 2002;112:298–304.
56. Siguel EN, Lerman RH. Altered fatty acid metabolism in patients with angiographically documented coronary artery disease. Metabo-lism 1994;43:982–93.
57. Horrobin DF. Nutritional and medical importance of gamma-linolenic acid. Prog Lipid Res 1992;31:163–94.
58. Nervi AM, Peluffo RO, Brenner RR. Effects of ethanol administra-tion on fatty acid desaturation. Lipids 1980;15:263–8.
59. Emken EA, Adlof RO, Gulley RM. Dietary linoleic acid influ-ences desaturation and acylation of deuterium-labeled linoleic and ALAs in young adult males. Biochim Biophys Acta 1994;1213: 277–88.
60. US Department of Agriculture. Nutrient Data Lab. Internet: http:// www.nal.usda.gov/fnic/foodcomp (accessed December 2002).
61. Conquer JA, Holub BJ. Supplementation with an algae source of docosahexanoic acid increases $(n-3)$ fatty acid status and alters selected risk factors for heart disease in vegetarian subjects. J Nutr 1996;126:3032–9.