The Omega-3 Fatty Acid Story

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Introduction

The discovery of the apparent health benefits of omega-3 fatty acids represents perhaps one of the most exciting chapters in nutrition research. Numerous areas of omega-3 research are being explored including chemistry, biochemistry, sources, health aspects, and employment in animal agriculture, as a feed ingredient, and in food processing, as a food additive or supplement.

The pioneering studies of Dyerberg et al. (1978) and Dyerberg and Bang (1979) have catalyzed an incredible amount of research over the past decade because the results suggested that omega-3 oils protect against coronary thrombosis in Greenland Eskimos in spite of a large intake of fat and cholesterol. Numerous review articles have been written on the subject of omega-3 fatty acids. Extensive reviews are recommended to the reader, including those by Barlow and Stansby (1982) and Lands (1986). A good but very brief review on health aspects has been prepared by Glomset (1985). An excellent clinically-oriented review has been written by von Schacky (1987).

Structure, Nomenclature and Sources

Chemistry, structure and nomenclature of selected fatty acids are summarized in Table 1. Omega-3 (N-3) fatty acids possess the first methylene interrupted double bond at the third carbon atom from the methyl end of the molecule. N-6 fatty acids have the first double bond at the sixth carbon atom. The principal N-3 fatty acids are α-LA, EPA and DHA, the latter two found in fish and seafood. The principal N-6 fatty acids are LA and AA; LA is abundant in vegetable oils whereas AA is found in the muscle membranes (phospholipids), accounting for perhaps 20% of the phospholipids (which in turn accounts for less that 1% of meat on a wet weight basis). Animals, including fish, are unable to convert N-6 to N-3.

Marine fish and seafood serve as sources of EPA and DHA (Lands, 1986). However, a common misconception is that only marine (saltwater) fish contain EPA and DHA or are significant sources of these fatty acids. Recently, Wang et al. (1989) demonstrated generally high levels of N-3 fatty acids, including EPA and DHA, in fish from Lake Superior. Therefore, it appears salinity is not as important as water temperature in stimulating the accumulation of N-3 fatty acids in the tissue. Cold water favors high levels of EPA and DHA. Some rough fish (Carp) from northern areas of the U.S. have reasonably high levels of EPA and DHA.

Possible Health Benefits

Although much research remains to be done, N-3 fatty acids appear to have potential in the prevention and treatment of a number of diseases. Recent research has made a case for N-3 fatty acids as being essential fatty acids (Neuringer and Connor, 1986). Currently under intense investigation are the following: cancer, stroke, rheumatoid arthritis, hypertension, systemic lupus erythematosus and psoriasis, to name but a few. The present review will confine itself to coronary heart disease (CHD) because the data available on CHD are extensive and results are the most well-established with regard to effects of N-3 oils.

To begin to understand how N-3 fatty acids, in particular EPA and DHA (Table 1), might help prevent or treat CHD, it is important to understand certain essential aspects of the disease itself.

CHD may be most easily understood if viewed as a three-step process: arterial endothelial injury, atherosclerosis and arterial thrombosis/spasm (Addis and Park, 1989). CHD, specifically atherosclerosis, is not simply the "clogging" of blood vessels by cholesterol but is rather an exceedingly complex pathological process. Atherosclerosis, the plaque build-up process, begins with an injury to the inner lining of arterial cells, the endothelial cells, and involves the actions and interactions of monocytes, macrophages, platelets, low-density lipoprotein (LDL) particles, cellular growth and chemotactic factors, arterial smooth muscle cells and foam cells. The process is roughly as follows: arterial injury (endothelial) causes monocytes and possibly other white cells to attach to the injured area. Platelets join in the "repair" process, which is a "response to injury" as discussed by Ross (1986). Subendothelial migration of monocytes aid in their conversion into macrophages (scavenger cells) which engulf oxidatively modified LDL particles and the cholesterol and fat contained by them and, by this process, become foam cells. Subendothelial fatty streaks are formed by the extensive production of foam cells from macrophages. These fatty streaks, if extensive enough, may disrupt the overlying endothelium and cause even larger injury than before. Thus, the "response to injury" cycle repeats with the utlimate production of extensive raised plaques which narrow the lumen of the artery and increase the possibility of a serious complication should a coronary thrombosis or an arterial spasm occur. Either one or both thrombosis or spasm may result in a damaged heart muscle (myocardial infarction or MI).

The aggregation of platelets are of obvious importance. Prevention of the platelets from aggregating and forming a

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Table 1. Structures, Symbols and Nomenclature of Important Omega-3 (N-3) and Omega-6 (N-6) Fatty Acids.

Name	*Type*[a]	*Abbreviation*[b]	*Symbol*[c]	*Systematic name*	*Structure*[d]
α-linolenic	N-3	α-LA	18:3N-3	9,12,15-octatrienoic	
eicosapentaenoic	N-3	EPA	20:5N-3	5,8,11,14,17-eicosapentaenoic	
docasahexaenoic	N-3	DHA	22:6N-3	4,7,10,13,16,19-docosahexaenoic	
linoleic	N-6	LA	18:2N-6	9,12-octadienoic	
arachidonic	N-6	AA	20:4N-6	5,8,11,14-eicosatetraenoic	

[a] N-3 refers to "omega-3" in which the first methylene interrupted double bond begins at the third carbon atom from the methyl carbon atom at the end of the fatty acid; N-6 refers to the double bond starting at the sixth carbon.
[b] Abbreviations used commonly and in this report.
[c] 18:3N-3 translates to α-linolenic acid, 18 carbons in length, three double bonds, the first starting at the third carbon (N-3) from the methyl end.
[d] These structures are not stereochemically correct. The reader should consult Lands (1986) for the correct stereochemistry of these fatty acids.

blood clot (or prevention of arterial spasm) can do much to prevent MI, even if extensive plaques have formed. This finding has been known for some time. What has not been known until more recently is that by reducing the activity of platelets and monocytes it may be possible to slow the *early* stages of atherosclerosis (Ross, 1986; Leaf and Weber, 1987, 1988; Addis and Park, 1989). To understand why, it is necessary to learn some facts about platelets, growth factors and chemotactic factors.

Platelets and other cells secrete chemotactic factors and growth factors during atherosclerosis. The result is that arterial smooth muscle cells, normally found in the media of the vessel, are recruited to migrate into the intimal layer (chemotaxis) and multiply under the influence of platelet-derived growth factor (PDGF). The arterial smooth muscle cells, when filled with fat and cholesterol (from LDL), become the foam cells which are the hallmark of atherosclerosis (Ross, 1986) and provide much additional plaque material in a mature atherosclerotic lesion.

Table 2. Proposed Benefits from Consumption of Fish Oil N-3 Fatty Acids for CHD[a].

N-3 Oil Appears to Reduce:

(1) Platelet aggregation
  (a) Decrease propensity to form thrombi (clots)
  (b) Slow or interrupt atherosclerosis
(2) Propensity for spasm of artery which would "pinch-off" blood flow
(3) Blood pressure-hypertension is important factor accelerating atherosclerosis
(4) Serum triglycerides — recently shown to be an independent risk factor in CHD
(5) Serum cholesterol — long known to be a serious risk factor for CHD
(6) Ventricular arrhythmias — after an MI
(7) Viscosity of blood (increased deformability of blood cells)
(8) Reduced rate of re-stenosis following coronary artery angioplasty

[a] Adapted from Leaf and Weber (1987, 1988)

Fish Oils and CHD

Consumption of oily fish, cod liver oil or fish body oil appears to have a number of benefits with regard to the prevention and treatment of CHD (Kromhout et al., 1985; Phillipson et al., 1985; Glomset, 1985). Why do fish oils appear to be so effective and what biochemical mechanisms are involved? Table 2 presents a brief summary of the multiplicity of hypothesized benefits of N-3 fatty acids from fish oils (with respect to CHD). Several general references are available, including Barlow and Stansby (1982), Herold and Kinsella (1986), Lands (1986) and Addis and Park (1989), but three very key papers are Leaf and Weber (1987, 1988) and von Schacky (1987).

As Table 2 indicates, there are numerous benefits to be derived from fish oils (or oily fish) in the diet. Virtually every major phase of CHD appears to be lessened by fish oil. For the middle-aged person, the possibility of preventing MI, even in partly occluded arteries, by preventing spasms and clots, is obviously very appealing. For the young person, the possibility of slowing or interrupting atherosclerosis is equally exciting. It is the opinion of the author that some time in the future fish oils will be widely used to treat and prevent CHD. However, not all scientists and medical authorities agree with this view and, indeed, there are some potential risks associated with high-level consumption of fish oils. These are summarized in Table 3.

Most of the risks outlined in Table 3 are familiar but (4) and (5), those dealing with lipid oxidation products, deserve special attention. The possible effects of dietary and *in vivo*-

Table 3. Possible Adverse Effects of High-Level Consumption of Fish Oils in Humans.

(1) Depletion of tissue vitamin E
(2) Environmental contaminants (polychlorinated biphenyls, mercury)
(3) Increased propensity to bleed because of reduced platelet activity
(4) Increased consumption of lipid oxidation products
(5) Increased *in vivo* production of lipid oxidation products

produced lipid oxidation products on CHD have been reviewed (Addis et al., 1983; Addis, 1986; Addis and Park, 1989). Fish oils are notoriously susceptible to autoxidation and few if any reports on fish oil feeding specify methods for controlling lipid autoxidation (Fritsche and Johnston, 1988). The lack of consensus regarding effects of dietary N-3 fatty acids may stem in part from the rapid autoxidation which occurs after fish oil is mixed with feed, which can elevate to six-fold or higher levels (over day 0 values) after only two days of storage without antioxidant (Fritsche and Johnston, 1988). Autoxidation of EPA and DHA may also occur after oral administration by gavage, although the tissue location of autoxidation is unknown. Theiry and Seidel (1987) noted that fish oil feeding by gavage actually accelerated atherosclerosis in cholesterol-fed (1.5% of diet) rabbits; fish oil-treated rabbits also displayed a 4-fold increase in serum peroxides over rabbits which did not receive fish oils. The results of Fritsche and Johnston (1988) and Thiery and Seidel (1987) emphasize the importance of lipid peroxides in atherosclerosis and also the critical need for controlling lipid autoxidation in fish oils, whether for research or clinical purposes. Lipid peroxides have been associated with all three phases of CHD: injury, plaque formation and thrombosis/spasm (Addis, 1986; Addis and Park, 1989).

Mechanisms of Action of EPA and DHA

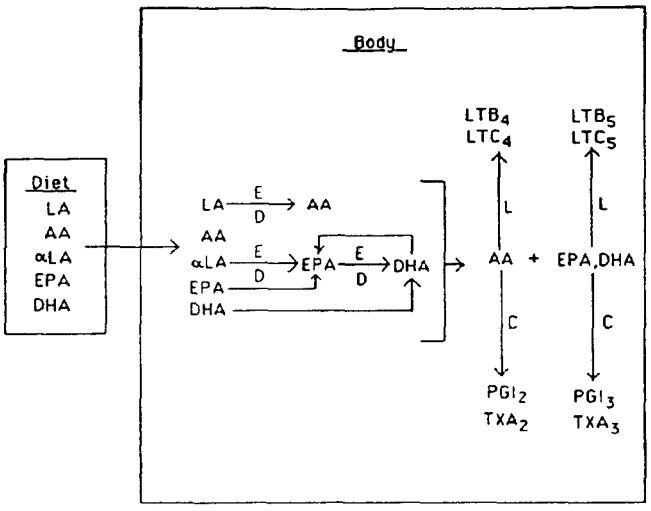
Two primary mechanisms of N-3 fatty acids involve influence on eicosanoids and plasma lipids.

Eicosanoids

Several excellent reviews are available (Barlow and Stansby, 1982; Lands, 1986; von Schacky, 1987; Kinsella, 1988). The present article will briefly outline the biochemical mechanisms by which N-3 fatty acids exert biological effects of clinical importance.

With regard to the critical thrombosis/spasm aspect of CHD, fish oils appear to alter the ratio of prostacyclin/thromboxane so that platelet activity is reduced and coronary arteries are less likely to spasm shut. To understand how this effect occurs, one must understand the essential aspects of eicosanoid biochemistry. Eicosanoids are prostaglandins and leukotrienes, both potent chemicals formed from arachidonic acid (AA). Aspirin can inhibit the conversion of AA to prostaglandins but not the AA conversion to leukotrienes. Fish oils inhibit both conversions by a number of mechanisms. A general scheme has been developed to illustrate the mechanisms in Figure 1. It is interesting to note that the high dietary intake of LA (18:2N-6), promoted by the American Heart Association and the National Institutes of Health as a means of lowering serum cholesterol, may be part of the problem with respect to thrombosis/spasm and monocyte activity insofar as LA is easily converted to AA.

Figure 1



Pathways for eicosanoid synthesis which may be significant in coronary heart disease. Abbreviations used: E = elongase; D = desaturase; C = cyclooxygenase; L = 5'lipoxygenase.

As Figure 1 indicates, N-3 fatty acids (notably EPA and DHA) compete with N-6 in a number of systems to influence ultimate ratios of the various types of eicosanoids produced. N-3 fatty acids inhibit the conversion of LA to AA to EPA. N-3 and N-6 fatty acids compete directly for the same desaturase and elongase enzymes and for the 2-position on all membrane phospholipids which serve as immediate cellular sources of AA, EPA and DHA. EPA competes with AA for cyclooxygenase, slowing the production of the thromboxane A_2 (TXA_2), a potent stimulation of vasoconstriction (spasm) and platelet aggregation. Instead, more thromboxane A_3 (TXA_3) is formed, which is a much less potent form of thromboxane. In endothelial cells, EPA (DHA) does not markedly inhibit cyclooxygenase—a factor which favors production of prostaglandin I_2 (PGI_2 or prostacyclin), a chemical which promotes vasodilation and inhibits platelet aggregation. Also, EPA is converted to prostaglandin I_3 which augments the activity of prostaglandin I_2 (Leaf and Weber, 1988).

Leukotrienes may be important in early atherosclerosis because leukotrienes help to control behavior of monocytes, macrophages and platelets. Generally, N-3 fatty acids tend to produce lower quantities of or less active forms (LTB_5, LTC_5) of leukotrienes (Lands, 1986). The retroconversion of DHA to EPA has been demonstrated (Figure 1).

Plasma Lipids

Plasma lipid lowering by dietary N-3 fatty acids is less clear than effects on eicosanoids. However, evidence exists that LDL and very-low-density-lipoprotein (VLDL) in plasma are reduced by fish oils (von Schacky, 1987). Inhibition of LDL apoprotein B synthesis appears to be the mechanism of LDL and VLDL lowering by dietary fish oils. However, contrary results for LDL have been reported (Zucker et al., 1988).

It is generally agreed that humans can convert α-LA to EPA, in turn, to DHA. The rate of these conversions is generally believed to be very slow, suggesting the importance of including EPA and DHA in the diet. However, recent studies by Emken et al. (1989) suggest that, in fact, conversion of α-LA to EPA and DHA proceeds at a rapid rate in young men. If true, these results are significant in that it may be possible to use α-LA as the chief N-3 source. It is interesting to note that Owren, Hellem and Odegaard (1964) demonstrated that a reduction in thromobotic tendency was

stimulated in humans by the consumption of linseed oil and α-LA. Both the results of Emken et al. (1989) and Owren, Hellem and Odegaard (1964) were conducted on subjects consuming low-fat diets. α-LA is far less susceptible to rancidity than EPA or DHA. Obviously, further research is needed but the practical implications of these studies could be great.

Applications to Agriculture and Food Processing

Both Kinsella (1988) and Addis and Park (1989) have discussed the "engineering" of foods to reduce the potential risk of CHD. With regard to N-3 fatty acids, the benefits and potential problems would both appear to be great. There currently is a great deal of interest in direct or indirect incorporation of fish oil N-3 fatty acids into non-traditional foods including eggs, meat, poultry and even surimi, a gel-type product made from fish in which all the fish oil has been removed in processing. The relative abundance and low prices of fish oil will make it economically viable to incorporate fish oils but the rancidity problem (fishy flavor) will need to be solved.

Indirect Incorporation

Several avenues of N-3 incorporation are possible. Feeding fish meal as a protein source results in chicken meat which is comparable to cod in N-3 content (Ackman et al., 1988). Feeding chickens fish meal also results in deposition of N-3 fatty acids in egg yolk. Some farmers in Minnesota are exploring the possibility of feeding rough fish such as carp to pigs. In feeding fish to farm animals, an important consideration is the role that fish tissue lipoxygenase plays in fish rancidity. Wang, Miller and Addis (submitted) recently demonstrated that a brief heat treatment (80°C., 90 sec.) improved tissue stability by destroying lipoxygenase activity in Lake Superior lake herring. Such results may be useful for operations planning to use fish as a protein source for animals.

Certainly any monogastric animal will deposit N-3 fatty acids. Ruminants will not deposit N-3 fatty acids, due to the extensive saturation activity of rumen microorganisms, unless some mechanism of encapsulation could be used to protect the N-3 fatty acids.

Susceptibility to rancidity, fishy flavor development and accelerated color loss may accompany the deposition of N-3 fatty acids in eggs and meat, as discussed below.

Direct Incorporation

Direct incorporation into foods is being studied in several laboratories. At least one company is in the primary business of selling technology and assisting other companies in the area of direct incorporation of EPA and DHA into foods (Table 4). The company claims to have an inexpensive method for removal of peroxides, reduction of cholesterol and the stabilization of N-3 fatty acids in foods (Table 4).

Table 4. Commercial Activity of Omega Source Corporation[a].

Activities
Provides purified oils
Provides stabilized oils
Provides cholesterol reduction technology (low cost)
Provides technology to stabilize N-3 oils in foods
Practical Accomplishments
Removal of 70% of cholesterol from fish oil
Removal of 90% of cholesterol from lard and butterfat
Successfully incorporated into fresh pork sausage, Polish sausage, liver sausage and bologna, 5, 3, 2 and 3% N-3 oil respectively and maintained flavor stability for expected shelf-life

[a] The Omega Source Corp.
12241 Nicollet Avenue South
Burnsville, MN 55337

Other meat applications include incorporating N-3 oils into sausage, mixing meat and N-3 containing fish and producing a comminuted red-meat, fish combination and producing extra lean sausage with N-3 fatty acids incorporated. In all such applications, the deodorization and stabilization of fish oil are key factors in the potential success of the product. Two recent articles on the subjects of deodorization and stabilization of fish oils are helpful (Nawar and Hultin, 1988; Chang, 1988).

One possible mechanism of rancidity inhibition in an N-3 incorporated meat product would be to process meat prerigor. Research has consistently demonstrated the efficacy of prerigor processing of pork with respect to resistance to rancidity (Judge and Aberle, 1980; Drerup et al., 1981; Yaskoski et al., 1984). An excellent reference with respect to the inhibition of lipid oxidation by various types of synthetic and natural antioxidants is the article by Peng (1985). It is obvious that some excellent possibilities exist for development of N-3 meat and poultry products. Much research needs to be done to successfully market such products.

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Discussion

J. Keeton: I noticed that some of these omega three's will present fishy flavors in meat products. Do you know what levels actually cause the fishy flavors that sometimes develop?

P. Addis: That's a very good question and unfortunately the answer to that is an extremely small amount of omega-3 oxidation will produce off-flavors or odors. Work done at the University of Massachusetts by Herb Hultin presented at last fall's American Chemical Society meeting indicated that fish will develop a fishy flavor due to rancidity of these fats, even in extremely small quantities — that is, not enough to see a decline in nutritional value. You cannot measure a loss in omega-3 content. It's just that there is enough hydroperoxide formed that it smells and tastes fishy. It's a really difficult technical accomplishment to prevent the rancidity but not an impossible one.

Keeton: So is it the hydroperoxide?

Addis: In this type of fishiness, yes. There's another type of fishiness that is related to trimethylamine oxide conversion into trimethylamine. That's not really related to this at all because trimethylamine production results from bacterial reduction of trimethylamine oxide.

F. McKeith: Paul, is there any advantage to using alternate sources of omega threes? Are you looking at full fat soybeans? You're going to have omega sixes as well as omega threes. Will that have a negative effect when people consume pork or other meat products as well?

Addis: You mean in terms of platelet function?

McKeith: If you wanted to boost the level of omega threes in meat products, could one use something like a full fat soybean where you will get some elevation?

Addis: You would get some. There is already so much omega six there and any omega three that you put in the product will materially alter that ratio. But whether or not the person would get the health benefits, that's something that still needs to be figured out. John, you've done some work with some flaxseed, which is probably the best of all the oils for linolenic acid content. That's another oil where you have a