Evidence Report

Chapter 1. Introduction

This report is one of a group of evidence reports prepared by three Agency for Healthcare Research and Quality (AHRQ)-funded Evidence-Based Practice Centers (EPCs) on the role of omega-3 fatty acids (both from food sources and from dietary supplements) in the prevention or treatment of a variety of diseases. These reports were requested and funded by the Office of Dietary Supplements, National Institutes of Health. The three EPCs – the Southern California EPC (SCEPC, based at RAND), the Tufts-New England Medical Center (NEMC) EPC, and the University of Ottawa EPC – have each produced evidence reports. To ensure consistency of approach, the three EPCs collaborated on selected methodological elements, including literature search strategies, rating of evidence, and data table design.

The aim of these reports is to summarize the current evidence on the effects of omega-3 fatty acids on prevention and treatment of cardiovascular diseases, cancer, child and maternal health, eye health, gastrointestinal/renal diseases, asthma, immune-mediated diseases, tissue/organ transplantation, mental health, and neurological diseases and conditions. In addition to informing the research community and the public on the effects of omega-3 fatty acids on various health conditions, it is anticipated that the findings of the reports will also be used to help define the agenda for future research.

This report focuses on the effects of omega-3 fatty acids on immune-mediated diseases, bone metabolism, and gastrointestinal/renal diseases. Subsequent reports from the SCEPC will focus on cancer and neurological diseases and conditions.

This chapter provides a brief review of the current state of knowledge about the metabolism, physiological functions, and sources of omega-3 fatty acids.

The Recognition of Essential Fatty Acids

Dietary fat has long been recognized as an important source of energy for mammals, but in the late 1920s, researchers demonstrated the dietary requirement for particular fatty acids, which came to be called essential fatty acids. It was not until the advent of intravenous feeding, however, that the importance of essential fatty acids was widely accepted: Clinical signs of essential fatty acid deficiency are generally observed only in patients on total parenteral nutrition who received mixtures devoid of essential fatty acids or in those with malabsorption syndromes. These signs include dermatitis and changes in visual and neural function. Over the past 40 years, an increasing number of physiological functions, such as immunomodulation, have been attributed to the essential fatty acids and their metabolites, and this area of research remains quite active.[1, 2]

Fatty Acid Nomenclature

The fat found in foods consists largely of a heterogeneous mixture of triacylglycerols (triglycerides)--glycerol molecules that are each combined with three fatty acids. The fatty acids can be divided into two categories, based on chemical properties: saturated fatty acids, which are usually solid at room temperature, and unsaturated fatty acids, which are liquid at room

Note: Appendixes and Evidence Tables are provided electronically at http://www.ahrq.gov/clinic/epcindex.htm

temperature. The term “saturation” refers to a chemical structure in which each carbon atom in the fatty acyl chain is bound to (saturated with) four other atoms, these carbons are linked by single bonds, and no other atoms or molecules can attach; unsaturated fatty acids contain at least one pair of carbon atoms linked by a double bond, which allows the attachment of additional atoms to those carbons (resulting in saturation). Despite their differences in structure, all fats contain approximately the same amount of energy (37 kilojoules/gram, or 9 kilocalories/gram).

The class of unsaturated fatty acids can be further divided into monounsaturated and polyunsaturated fatty acids. Monounsaturated fatty acids (the primary constituents of olive and canola oils) contain only one double bond. Polyunsaturated fatty acids (PUFAs) (the primary constituents of corn, sunflower, flax seed and many other vegetable oils) contain more than one double bond. Fatty acids are often referred to using the number of carbon atoms in the acyl chain, followed by a colon, followed by the number of double bonds in the chain (e.g., 18:1 refers to the 18-carbon monounsaturated fatty acid, oleic acid; 18:3 refers to any 18-carbon PUFA with three double bonds).

PUFAs are further categorized on the basis of the location of their double bonds. An omega or n notation indicates the number of carbon atoms from the methyl end of the acyl chain to the first double bond. Thus, for example, in the omega-3 (n-3) family of PUFAs, the first double bond is 3 carbons from the methyl end of the molecule. The trivial names, chemical names and abbreviations for the omega-3 fatty acids are detailed in Table 1.1.

Finally, PUFAs can be categorized according to their chain length. The 18-carbon n-3 and n-6 short-chain PUFAs are precursors to the longer 20- and 22-carbon PUFAs, called long-chain PUFAs (LCPUFAs).

Table 1.1. Nomenclature of omega-3 fatty acids.

Names		Abbreviations		
Trivial	IUPAC*	Carboxyl-reference	Omega-reference	Other
Linolenic acid	9,12,15-octadecenoic acid	$18{:}3\Delta^{9\ 12\ 15}$	18:3n-3 18:3 (ω-3)	ALA α-LA LNA α-LNA
Docosahexaenoic acid	4,8,12,15,19- docosahexaenoic acid	$22{:}6\Delta^{4\ 8\ 12\ 15\ 19}$	22:6n-3 22:6 (ω-3)	DHA
Docosapentaenoic acid	7,10,13,16,19-docosapentaenoic acid	$22{:}5\Delta^{7\ 10\ 13\ 16\ 19}$	22:5n-3 22:5 (ω-3)	DPA
Eicosapentaenoic acid Icosapentaenoic acid Timnodonic acid	5,8,11,14,17- eicosapentaenoic acid	$20{:}5\Delta^{5\ 8\ 11\ 14\ 17}$	20:5n-3 20:5 (ω-3)	EPA

*IUPAC=International Union of Pure and Applied Chemistry

Fatty Acid Metabolism

Mammalian cells can introduce double bonds into all positions on the fatty acid chain except the n-3 and n-6 position. Thus, the short-chain alpha-linolenic acid (ALA, chemical abbreviation: 18:3n-3) and linoleic acid (LA, chemical abbreviation: 18:2n-6) are essential fatty acids. No other fatty acids found in food are considered ‘essential’ for humans, because they can all be synthesized from the short chain fatty acids.

Following ingestion, ALA and LA can be converted in the liver to the long chain, more-unsaturated n-3 and n-6 LCPUFAs by a complex set of synthetic pathways that share several enzymes (Figure 1). LC PUFAs retain the original sites of desaturation (including n-3 or n-6).

The omega-6 fatty acid LA is converted to gamma-linolenic acid (GLA, 18:3n-6), an omega-6 fatty acid that is a positional isomer of ALA. GLA, in turn, can be converted to the longer-chain omega-6 fatty acid, arachidonic acid (AA, 20:4 n-6). AA is the precursor for certain classes of an important family of hormone-like substances called the eicosanoids (see below).

The omega-3 fatty acid ALA (18:3n-3) can be converted to the long-chain omega-3 fatty acid, eicosapentaenoic acid (EPA; 20:5 n-3). EPA can be elongated to docosapentaenoic acid (DPA 22:5n-3), which is further desaturated to docosahexaenoic acid (DHA; 22:6n-3). EPA and DHA are also precursors of several classes of eicosanoids and are known to play several other critical roles, some of which are discussed further below.

The conversion from parent fatty acids into the LC PUFAs - EPA, DHA, and AA - appears to occur slowly in humans. In addition, the regulation of conversion is not well understood, although it is known that ALA and LA compete for entry into the metabolic pathways.

Physiological Functions of EPA and AA

As stated earlier, fatty acids play a variety of physiological roles. The specific biological functions of a fatty acid are determined by the number and position of double bonds and the length of the acyl chain.

Both EPA (20:5n-3) and AA (20:4n-6) are precursors for the formation of a family of hormone-like agents called eicosanoids. Eicosanoids are rudimentary hormones or regulating - molecules that appear to occur in most forms of life. However, unlike endocrine hormones, which travel in the blood stream to exert their effects at distant sites, the eicosanoids are autocrine or paracrine factors, which exert their effects locally – in the cells that synthesize them or adjacent cells. Processes affected include the movement of calcium and other substances into and out of cells, relaxation and contraction of muscles, inhibition and promotion of clotting, regulation of secretions including digestive juices and hormones, and control of fertility, cell division, and growth.[3]

The eicosanoid family includes subgroups of substances known as prostaglandins, leukotrienes, and thromboxanes, among others. As shown in Figure 1.1, the long-chain omega-6 fatty acid, AA (20:4n-6), is the precursor of a group of eicosanoids that include series-2 prostaglandins and series-4 leukotrienes. The omega-3 fatty acid, EPA (20:5n-3), is the precursor to a group of eicosanoids that includes series-3 prostaglandins and series-5 leukotrienes. The AA-derived series-2 prostaglandins and series-4 leukotrienes are often synthesized in response to some emergency such as injury or stress, whereas the EPA-derived series-3 prostaglandins and series-5 leukotrienes appear to modulate the effects of the series-2 prostaglandins and series-4 leukotrienes (usually on the same target cells). More specifically, the series-3 prostaglandins are formed at a slower rate and work to attenuate the effects of excessive levels of series-2 prostaglandins. Thus, adequate production of the series-3 prostaglandins seems to protect against heart attack and stroke as well as certain inflammatory diseases like arthritis, lupus, and asthma.[3]

EPA (22:6 n-3) also affects lipoprotein metabolism and decreases the production of substances – including cytokines, interleukin 1ß (IL-1ß), and tumor necrosis factor a (TNF-a) –

that have pro-inflammatory effects (such as stimulation of collagenase synthesis and the expression of adhesion molecules necessary for leukocyte extravasation [movement from the circulatory system into tissues]).[2] The mechanism responsible for the suppression of cytokine production by omega-3 LC PUFAs remains unknown, although suppression of omega-6-derived eicosanoid production by omega-3 fatty acids may be involved, because the omega-3 and omega-6 fatty acids compete for a common enzyme in the eicosanoid synthetic pathway, delta-6 desaturase.

DPA (22:5n-3) (the elongation product of EPA) and its metabolite DHA (22:6n-3) are frequently referred to as very long chain n-3 fatty acids (VLCFA). Along with AA, DHA is the major PUFA found in the brain and is thought to be important for brain development and function. Recent research has focused on this role and the effect of supplementing infant formula with DHA (since DHA is naturally present in breast milk but not in formula).

Dietary Sources and Requirements

Both ALA and LA are present in a variety of foods. LA is present in high concentrations in many commonly used oils, including safflower, sunflower, soy, and corn oil. ALA is present in some commonly used oils, including canola and soybean oil, and in some leafy green vegetables.

Thus, the major dietary sources of ALA and LA are PUFA-rich vegetable oils. The proportion of LA to ALA as well as the proportion of those PUFAs to others varies considerably by the type of oil. With the exception of flaxseed, canola, and soybean oil, the ratio of LA to ALA in vegetable oils is at least 10 to 1. The ratios of LA to ALA for flaxseed, canola, and soy are approximately 1: 3.5, 2:1, and 8:1, respectively; however, flaxseed oil is not typically consumed in the North American diet. It is estimated that on average in the U.S., LA accounts for 89% of the total PUFAs consumed, and ALA accounts for 9%. Another estimate suggests that Americans consume 10 times more omega-6 than omega-3 fatty acids.[4] Table 1.2 shows the proportion of omega 3 fatty acids for a number of foods.

Figure 1.1. Classical omega-3 and omega-6 fatty acid synthesis pathways and the role of omega-3 fatty acid in regulating health/disease markers.

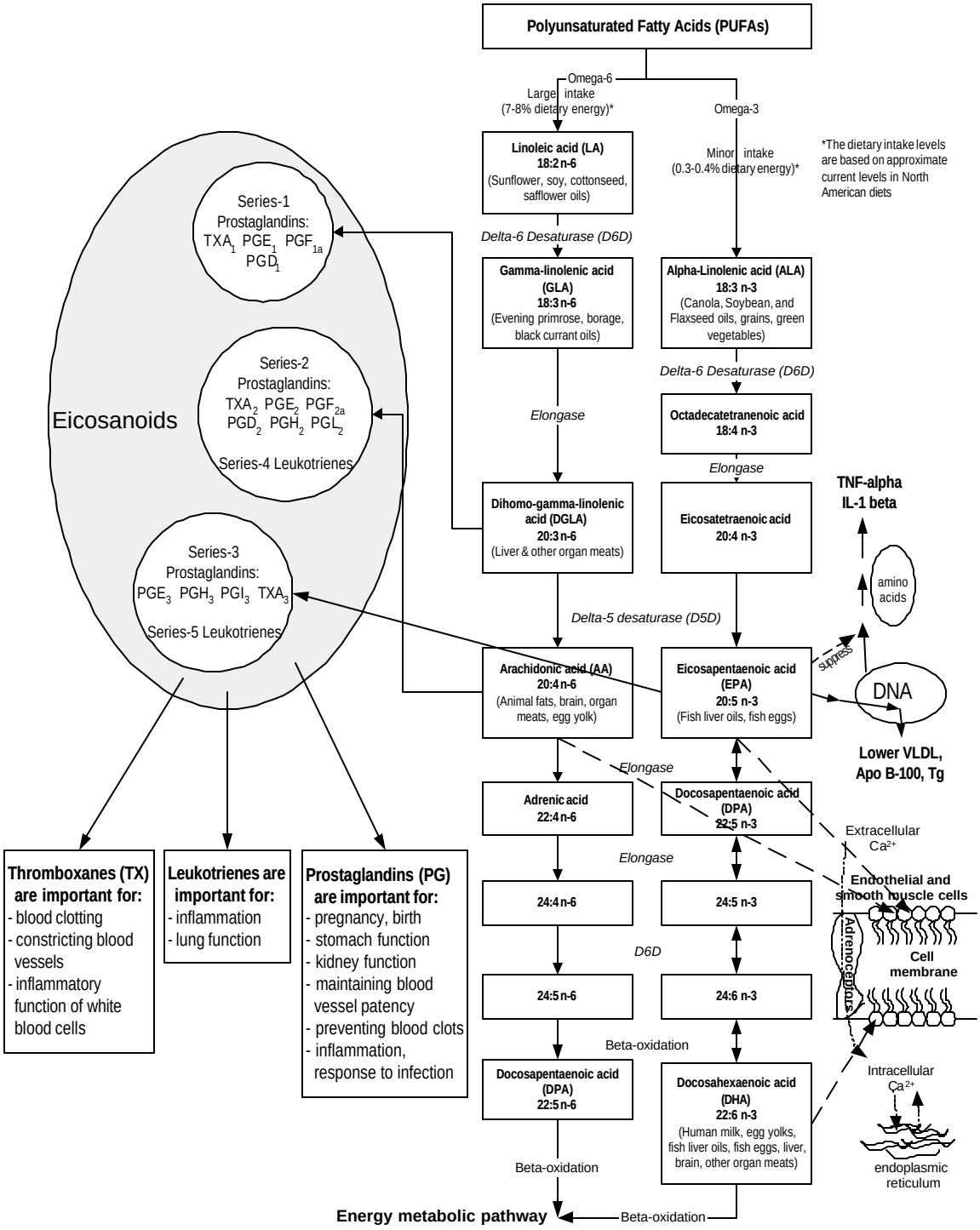

Table 1.2. Sources and proportions of omega-3 fatty acids in common foods and supplements.

Food/supplement	EPA 20:5n-3	DHA 22:6n-3	DPA 22:5n-3	ALA 18:3n-3
Foods in which Total Omega-3 Fatty Acids account for more than 50% of Total PUFA				
Fish				
Anchovy	v	v	v	
Halibut	v	v	v	
Herring	v	v	v	
Mackerel	v	v	v	
Salmon	v	v	v	
Sardine	v	v	v	
Tuna				
Canned, waterpacked	v	v	v	
Fresh Bluefin	v	v	v	
Oils/Supplements				
Cod liver oils	v	v	v	
Coromega *	v	v		
Fish oil capsules*	v	v		
*Flaxseed/linseed oil**				v
Herring oil	v	v	v	
MaxEPA*	v	v		
Menhaden oil	v	v	v	
Neuromins*		v		
Omacor*	v	v		
Ropufa*	v	v	v	
Salmon oil	v	v	v	
Sardine oil	v	v	v	
Seeds				
Flaxseeds/Linseeds				v
Foods/Supplements in which total Omega 3 fatty acids are 10-50% of total PUFA				
Oils				
Black currant oil				v
Canola oil**				v
Mustard seed oils				v
Soybean oil				v
Walnut oil				v
Wheat germ oil				v
Other foods				
Wheat germ				v
Human milk				v
Foods/Supplements in which total Omega 3 fatty acids are less than 10% of total PUFA				
Efamol Marine*	v	v		
Soybeans				v
Walnuts				v

* Dietary Supplement
** Also called rapeseed oil

Several lines of research have suggested that the high ratio of omega 6s to omega 3s currently consumed in the U.S. promotes a number of chronic diseases.[4] Because of the slow rate of elongation and further desaturation of the essential FA, the importance of LC PUFAs to many physiological processes, and the overwhelming ratio of omega 6s to omega 3s in the average U.S. diet, nutrition experts are increasingly recognizing the need for humans to augment the body's synthesis of omega 3 LC PUFAs by consuming foods that are rich in these compounds. According to data from two population-based surveys, the major dietary sources of LC omega-3 fatty acids in the U.S. population are fish, fish oil, vegetable oils (principally canola and soybean), walnuts, wheat germ, and some dietary supplements, and the primary dietary sources of omega-6 LC PUFAs are meats and dairy products. These surveys, the Continuing Food Survey of Intakes by Individuals 1994-98 (CSFII) and the third National Health and Nutrition Examination (NHANES III) 1988-94 surveys, are the main sources of dietary intake data for the U.S. population. The CSFII has the advantage of collecting dietary recall data over a period of several days, which may permit estimates of omega-3 intake that more accurately reflect individual intakes than do those of NHANES. However, NHANES intake data have the advantage of being able to be linked to health outcomes. Table 1.3 provides a list of food sources of omega-3 fatty acids.

Table 1.3. Good food sources* of omega 3 fatty acids.

	EPA+DHA	ALA		EPA+DHA	ALA
Fish (3oz. Cooked)			**Oils (1 Tbs.)**		
Anchovy	v		Canola		v
Halibut	v		Cod liver	v	
Herring, Atlantic	v		Flaxseed/linseed		v
Pacific	v		Herring	v	
Mackerel, Atlantic	v		Menhaden	v	
Pacific	v		Salmon	v	
Salmon, Atlantic**	v		Sardine	v	
Sardines	v		Soybean		v
Trout, Rainbow	v		Walnut		v
Tuna, Albacore	v		Wheat germ		v
Canned light, water-packed	v				
Canned white, water-packed	v				
Fresh Bluefin	v				
Organ Meats (3 oz. Cooked)			**Seeds**		
Brain, lamb	v		Flaxseeds/linseeds (1 Tbs.)		v
Brain, pork	v				
Thymus, calf		v			
Other Foods					
Caviar (1 oz.)#	v				
Human breast milk (1c)#		v			
Soybeans, cooked (1/2c)		v			
Tofu, regular (1/2c)		v			
Walnuts (1/4c)		v			
Wheat germ (1/4c)#		v			

Source: Figures adapted from USDA, 2003; *Foods that provide (per serving) 10% or more of the Adequate Intake (AI) for ALA or the Acceptable Macronutrient Distribution Range (AMDR) for EPA and DHA (10% of the AMDR for ALA); an AI is a recommended average daily intake level based on observed or experimentally determined estimates of nutrient intake by a group of apparently healthy people (thus, assumed to be adequate) when an RDA cannot be determined; an AMDR is defined as "a range of intakes for a particular energy source that is associated with reduced risk of chronic disease while providing adequate intake of essential nutrients."[5]

Standard serving size not established; **Farm-raised Atlantic salmon have nearly identical omega-3 fatty acid levels to wild Atlantic salmon and significantly more omega-3 fatty acids than wild Pacific salmon.

Table 1.4 shows the mean and median intakes of omega-3 and omega-6 fatty acids reported by NHANES III.[1] Table 1.5 shows the mean and median intakes of omega-3 and omega-6 fatty acids reported by CSFII.

Table 1.4. Estimates of the mean intake of LA, ALA, EPA, and DHA in the U.S. Population from analysis of NHANES III data.*

	Grams/day		Percent energy intake/day	
	Mean ± SEM	Median (range)**	Mean ± SEM	Median (range)**
LA (18:2n-6)	14.1 ± 0.2	9.9 (0 - 168)	5.79 ± 0.05	5.30 (0 - 39.4)
ALA (18:3n-3)	1.33 ± 0.02	0.90 (0 - 17)	0.55 ± 0.004	0.48 (0 - 4.98)
EPA (20:5n-3)	0.04 ± 0.003	0.00 (0 - 4.1)	0.02 ± 0.001	0.00 (0 - 0.61)
DHA (22:6n-3)	0.07 ± 0.004	0.00 (0 - 7.8)	0.03 ± 0.002	0.00 (0 - 2.86)

*Based on analysis of a single 24-hour dietary recall from NHANES III data; **Distributions are not adjusted for the over-sampling of Mexican –Americans, non-Hispanic African Americans, children 5 years old and under, and adults 60 years and over in the NHANES III dataset.

Table 1.5. Mean, range, and median usual daily Intakes (ranges) of n-6 and n-3 PUFAs, in the U.S. population, from analysis of CSFII data (1994 to 1998).*

	Mean (gms/d) (± SEM)**	Range of Means (gms/d) (±SEM)	Median (gms/d) (± SEM)**
LA (18:2n-6)	13.0 ± 0.1	6.7 ± 0.1-17.6 ± 0.5	12.0 ± 0.1
Total n-3 FA	1.40 ± 0.01	0.72 ± 0.02 - 1.86 ± 0.04	1.30 ± 0.01
ALA (18:3n-3)	1.30 ± 0.01	0.72 ± 0.02 - 1.73 ± 0.04	1.21 ± 0.01
EPA (20:5n-3)	0.028	0.002 - 0.049	0.004
DPA (22:5n-3)	0.013	0.001 - 0.019	0.005
DHA (22:6n-3)	0.057 ± 0.018	< 0.0005 ± 0.001	0.046 ± 0.013

Source: Adapted from Dietary Reference Intakes Report;[5] *Estimates are based on respondents' intakes on the first day of survey and were adjusted using the Iowa State University method; **For all individuals.

Lacking sufficient evidence from research on the effects or correction of dietary deficiencies to establish Recommended Dietary Allowances (RDAs) for the essential fatty acids, the Food and Nutrition Board (FNB) of the Institute of Medicine[5] has set adequate intakes[2] (AI) for the essential fatty acids, based on the average intakes of healthy CSFII participants. The AIs for the essential fatty acids vary by age group and sex, as well as for particular conditions such as pregnancy and breastfeeding. For ALA, the AI for men 19 and older, is 1.6 grams/day and the AI for (non-pregnant, non-breastfeeding) women is 1.1 grams/day. The AI for LA is 17 grams/day for men and 11 grams/day for women.

Based on evidence suggesting a role in prevention or treatment of some chronic diseases, the FNB has also established Acceptable Macronutrient Distribution Ranges (AMDR) for the essential fatty acids. An AMDR is defined as "a range of intakes for a particular energy source that is associated with reduced risk of chronic disease while providing adequate intake of

[1] The population represented by NHANES III includes individuals ages 2 months and older. Mexican Americans and non-Hispanic African-Americans, children 5 years old and younger, and adults 60 years of age and over were over-sampled to produce more precise estimates for these population groups. There were no imputations for missing 24-hour dietary recall data. A total of 29,105 participants had complete and reliable dietary recall data. The NHANES III also included a physical examination and health survey of each participant.

[2] An Adequate Intake (AI) is defined as "the recommended average daily intake level based on observed or experimentally determined approximations or estimates of nutrient intake, by a group (or groups) of apparently healthy people, that are assumed to be adequate – used when a recommended dietary allowance cannot be determined."[5] An AI is set when data are insufficient or inadequate to establish an Estimated Average Requirement, on which the RDA is based, and indicate the need for more and better research. The EAR is "the average daily nutrient intake level estimated to meet the requirement of half the healthy individuals in a particular life stage and gender group," based on a specific indicator or criterion of adequacy.

essential nutrients."[5] The AMDR is expressed as a percentage of total energy intake: The AMDR for LA is set at 5 to 10 percent of usual energy intake, and the AMDR for ALA is 0.6 to 1.2 percent of energy intake. Of this amount, up to 10 percent can be consumed as EPA and/or DHA, the omega-3 LC PUFAs. For a person who consumes 2000 kcal/day, ALA intake should range from 1.3 to 2.6 grams/day, and EPA/DHA intake can substitute for 0.13 to 0.26 of that quantity. Table 1.3 lists foods that provide 10 percent or more of these recommended intakes per serving, which may be referred to as "good sources." [3] Table 1.6 provides the actual omega-3 content per 100 gm for a variety of foods.

Rationale for and Organization of this Report

Studies show that tissue levels of AA and EPA-derived eicosanoids influence many physiological processes, including platelet aggregation, vessel wall constriction, and immune cell function (IOM), resulting in protection against heart attack and stroke as well as certain inflammatory diseases like arthritis, systemic lupus erythematosus, and asthma. Epidemiological studies have suggested that groups of people who consume diets high in omega 3 FAs may experience a lower prevalence of these conditions, and many small trials have attempted to assess the effects of adding omega 3 fatty acids to the diet, either as omega-3 FA-rich foods or as dietary supplements (primarily fish oils). In addition, dietary omega 3FA have been found to increase calcium absorption, rates of bone formation, and bone strength in rodents and birds. In response to this evidence, a number of omega-3 FA-containing dietary supplements that claim to protect against a variety of conditions have appeared on the market. Thus, AHRQ and the NIH Office of Dietary Supplements have requested a synthesis of the research to date on the health effects of diets rich in omega-3 FA.

The remainder of this report is organized into four chapters. Chapter Two describes the methods we used to identify and review studies related to the role of omega-3 fatty acids in immune-mediated diseases, bone metabolism, and gastrointestinal/renal diseases. Chapter Three presents our findings related to the effects of omega-3 fatty acids on those diseases/conditions. Chapter Four presents our conclusions and recommendations for future research in this area.

[3] Identifying a food as a "good source" of a nutrient strictly means that one standard serving of the food supplies 10 to 19 percent of the Daily Value for that nutrient. The Daily Values are based on the FDA's Daily Reference Values, standards for the macronutrients (fats, protein, carbohydrates, and dietary fiber), which are similar, although not identical to the DRIs (RDAs) and are based on the amount of energy consumed per day (2000 kcal/d is the reference for calculating DVs). In the case of the PUFAs, no DVs have been established: For this report, the FNB's AIs and AMDRs, have been used instead.

Table 1.6. The omega-3 fatty acid content, in grams per 100 g food serving, of a representative sample of commonly consumed fish, shellfish, and fish oils, and nuts and seeds, and plant oils that contain at least 5 g omega-3 fatty acids per 100 g.

Food item	EPA	DHA	ALA	Food item	EPA	DHA	ALA
Fish (Raw [a])				**Fish, continued**			
Anchovy, European	0.6	0.9	-	Tuna, Fresh, Yellowfin	trace	0.2	trace
Bass, Freshwater, Mixed Sp.	0.2	0.4	0.1	Tuna, Light, Canned in Oil [e]	trace	0.1	trace
Bass, Striped	0.2	0.6	trace	Tuna, Light, Canned in Water [e]	trace	0.2	trace
Bluefish	0.2	0.5	-	Tuna, White, Canned in Oil [e]	trace	0.2	0.2
Carp	0.2	0.1	0.3	Tuna, White, Canned in Water [e]	0.2	0.6	trace
Catfish, Channel	trace	0.2	0.1	Whitefish, Mixed Sp.	0.3	0.9	0.2
Cod, Atlantic	trace	0.1	trace	Whitefish, Mixed Sp., Smoked	trace	0.2	-
Cod, Pacific	trace	0.1	trace	Wolf fish, Atlantic	0.4	0.3	trace
Eel, Mixed Sp.	trace	trace	0.4				
Flounder & Sole Sp.	trace	0.1	trace				
Grouper, Mixed Sp.	trace	0.2	trace	**Shellfish (Raw)**			
Haddock	trace	0.1	trace	Abalone, Mixed Sp.	trace	-	-
Halibut, Atlantic and Pacific	trace	0.3	trace	Clam, Mixed Sp.	trace	trace	trace
Halibut, Greenland	0.5	0.4	trace	Crab, Blue	0.2	0.2	-
Herring, Atlantic	0.7	0.9	0.1	Crayfish, Mixed Sp., Farmed	trace	0.1	trace
Herring, Pacific	1.0	0.7	trace	Lobster, Northern	-	-	-
Mackerel, Atlantic	0.9	1.4	0.2	Mussel, Blue	0.2	0.3	trace
Mackerel, Pacific and Jack	0.6	0.9	trace	Oyster, Eastern, Farmed	0.2	0.2	trace
Mullet, Striped	0.2	0.1	trace	Oyster, Eastern, Wild	0.3	0.3	trace
Ocean Perch, Atlantic	trace	0.2	trace	Oyster, Pacific	0.4	0.3	trace