

# THE QUEEN OF FATS

**WHY OMEGA-3s WERE REMOVED FROM THE WESTERN DIET AND  
WHAT WE CAN DO TO REPLACE THEM**



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S U S A N   A L L P O R T

## ONE

# WHAT'S FOR DINNER?

I look upon it, that he who does not mind  
his belly will hardly mind anything else.

SAMUEL JOHNSON, 1763

THE YEAR 2003 WILL BE REMEMBERED AS A TIME WHEN AMERICA LOST its dietary senses. Overnight, it seemed, this country switched from a low-fat regime, in which people shunned every form of visible fat, to the Atkins regime, in which fat consumption was encouraged but carbohydrates were to be avoided. Jack Sprat, who could eat no fat, suddenly became Sprat's wife and could eat no lean.

The accumulated nutritional advice from decades of research was tossed aside like an old blanket, and grocery stores were suddenly filled with such gastronomical oxymora as low-carb bread and beer. Thin women in tight jeans were overheard saying that they loved beets and apples but had to stay away from them because of all their carbs. Large men in business suits ordered bunless burgers dripping with bacon grease and raved about their diets. Anyone coming back to the United States after time spent in Europe or Asia had an Alice-in-Wonderland experience, as several returnees told me: black had become white and carbohydrates, the food that feeds most of the world's peoples, including the world's leanest peoples, were suddenly the bad guys.

But 2003 should be remembered not only as the year that America lost its dietary senses (which it did) but also as the year that the center would no longer hold. By 2003, the nutritional advice given out to Americans by government agencies like the United States Department of Agriculture and medical organizations like the American Heart Association had become so out of sync with current research and biological understanding that schisms and confusion became inevitable.

It is unfortunate that those schisms took the form of total rejection, on the part of many Americans, of all the acquired wisdom about what constitutes a healthy diet. But that's what happens when the center doesn't hold, when the marketplace is full of such absurdities as overly sweetened breakfast cereals, such as Cocoa Puffs and Lucky Charms, being endorsed by the American Heart Association (because they have no cholesterol or saturated fat)—when the oversimplistic, low-fat mantra of the 1980s and 1990s made the Atkins craze almost inescapable. As a dieter in Texas confides, “Eating low-fat guarantees that I will binge on fried foods. Eating low-carb guarantees that I will binge on a bag of chips.”

Much of the country is now on that fried-food, high-fat binge (or has binged out on Atkins and moved on). Many of us are more confused than ever about the simplest, most fundamental of questions: What should we have for dinner?

In the midst of this confusion, I'd like to throw my hat into the ring of nutritional advice with a tribute to one food, or family of foods: the fatty acids popularly known as the omega-3s. Because these fats were not recognized as being essential to human health until the 1980s, most current recommendations and nutritional advice took shape without them. At the same time, they were being eliminated from many foods because their presence caused problems with product stability and shelf life. Their absence,

from our foods and our guidelines, is a key, a large and growing number of scientists believe, to many of our health problems—and even our befuddlement about food.

I have none of the usual qualifications to write this homage. I am neither a physician who treats the diseases to which people who are deficient in these fats are prone nor a scientist who has spent a lifetime researching the membranes that these fats call home. But that may be an advantage, since scientists and physicians tend to focus on the one piece of the puzzle they are looking at and these fats, as it turns out, affect the entire body in many different ways.

Rather, I am a science writer, a curious denizen of twenty-first-century America with a long-standing interest in food and the difficulties of being a human omnivore, and I will try to present the big picture. Quite simply, trying to understand health and diet without an appreciation of these fats is like trying to understand earthquakes without knowledge of plate tectonics, or motion without knowledge of physics. Until we revise our foods and guidelines to incorporate all that has been learned about omega-3 fatty acids in the past fifty years, our diet will be lacking in a very important way.

After I introduce these fats, I think you will begin to see why they deserve this book of their own. This introduction will involve some chemistry, but only what is necessary and most of which will be familiar to cooks, shoppers, and nutrition-conscious readers. Further explanations and diagrams can be found in the glossary, which begins on page 159. All that readers need to know from the get-go is that fatty acids, the components of fats and cell membranes, are chains of carbons and hydrogens with an acidic group at one end. The first of the omega-3 fatty acids is alpha linolenic acid, or ALA, the single parent of this family of fats. Found primarily in the leaves and other green parts of plants, alpha linolenic

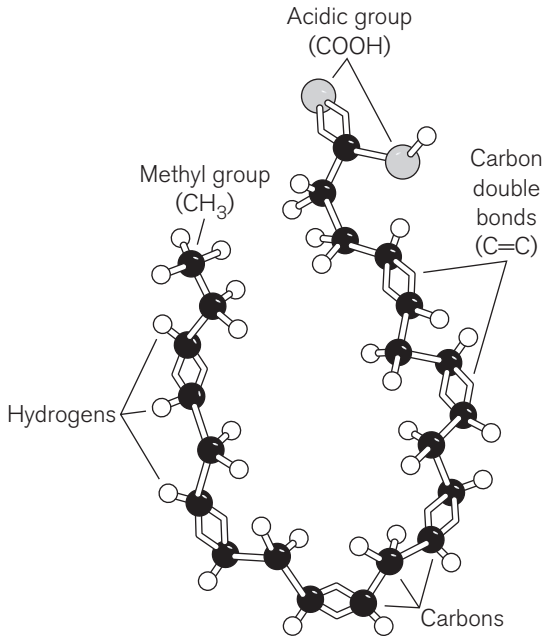
acid is the fat associated with the complex photosynthetic machinery of plants, the fat that enables plants to capture single photons of light and turn them into sugars, the basis of all life on earth. Alpha linolenic acid doesn't play a significant role in animals, for reasons I will soon discuss, but it does give rise to offspring who do work that is every bit as important to animals as photosynthesis is to plants.

Like all fatty acids, alpha linolenic acid is a weak acid—that is, it has a slight tendency to lose a hydrogen ion and develop a negative charge. It has the same strength as a very familiar acid, vinegar, which is not surprising since vinegar, or acetic acid, is also a fatty acid that is common in living tissues but too short (just two carbons long) to be of use in storing energy or building structures.

Fatty acids lose their acidic leaning when they team up with a molecule of glycerol to make triglycerides, the substances we commonly call fats (the substances we cook with and that don't mix with water). In most contexts, we can think of *fatty acids* and *fats* as equivalent terms. And we can think of the acidic end of a fatty acid as the hook, or the coupling, that enables our bodies to move these long, sticky chains of carbons and hydrogens around. It's also helpful to understand that all triglycerides have an identical glycerol backbone attached to three, often different, fatty acids, sixteen to twenty-two carbons in length.



Whether these triglycerides take the shape of butter, vegetable oil, lard, or suet depends entirely on which fatty acids are involved. Some fatty acids have straight, saturated chains (saturated with hydrogens, that is) and produce solid fats; others have kinky, unsaturated chains (where some of the hydrogens have been replaced by double bonds between the carbons) and produce liquids.



**FIGURE 1** DHA in one of its many conformations. Sometimes this fatty acid curls up in a ball; sometimes it is as straight as a ruler. Its six double bonds keep it constantly on the move.

Alpha linolenic acid has a markedly kinky tail, and the fats in which it is abundant—linseed, canola, and soybean oils—are liquids, even at very low temperatures. But alpha linolenic acid is not kinky enough for animals, which are faster (more mobile) than plants, and animals lengthen and add double bonds to this eighteen-carbon fatty acid before they put it to work in their tissues.

Docosahexaenoic acid, or DHA, is one of several offspring of alpha linolenic acid and it is the longest, most desaturated fatty

acid in animal tissues. It is the fat that permits animals to think and see. DHA is found in its highest concentrations in the membranes of the cells of the brain and eyes, where its ability to flip-flop between hundreds of different shapes, billions of times per second—the result of an extremely kinky chain with six carbon double bonds (twice as many as in alpha linolenic acid)—enables nerve cells to send their rapid signals. DHA is a quick-change artist, scientists have recently learned, and its concentrated presence in cell membranes, the thin envelopes surrounding cells, transforms those barriers from orderly guards into dancers at an all-night rave. Its dilute presence in cells throughout the body is like oil added to an engine.

Animals make very different use of a second, somewhat shorter, offspring of alpha linolenic acid: eicosapentaenoic acid. Eicosapentaenoic acid, or EPA, is one of several fatty acids, all twenty carbons long, that animal cells release from their membranes in order to communicate with each other and affect each other's behavior (fat signals instead of smoke signals). I'll talk more about these cell messengers later—how they were discovered and what kinds of reactions they produce—but the reader should know that this kind of communication is necessary in any organism with more than one cell and that eicosapentaenoic stands out as the mediator or peacemaker of these fat messengers. When this omega-3 fatty acid is released from a cell, it produces just the kind of measured reaction in its neighbors that is desirable in most family or neighborly interactions. It does not elicit the extreme reactions of other fat messengers—say, arachidonic acid, which enters the scene like a SWAT team. Sending in a SWAT team can be useful in some situations (in fighting infection, for example), but not to coordinate everyday disagreements.

The omega-3 fats are not rare in nature, as their remarkable behaviors might lead us to think. In fact, alpha linolenic acid,

found in the chloroplasts of green leaves, is the most abundant fat on earth. Green leaves are not known for being fatty, high-calorie foods; but the planet has more green vegetation on it than anything else, and the small amount of fat in each leaf adds up. DHA and eicosapentaenoic acid\* are also common, since these offspring of alpha linolenic acid accumulate in the tissues of animals that eat green leaves, as well as in the tissues of animals that eat the animals that eat green leaves. Both DHA and eicosapentaenoic acid are also made by some aquatic plants.

But these fats have become rare in most Americans' diets, which are short on leafy greens and long on seeds and the oil

\*To those wondering about my inconsistency in referring to these fatty acids, using the acronym for docosahexaenoic acid, DHA, but writing out alpha linolenic acid and eicosapentaenoic acid—it is intentional. I didn't want the reader thinking of the American Library Association (ALA) or the Environmental Protection Agency (EPA) every time I used their accepted acronyms. But DHA, as far as I know, doesn't stand for any familiar organization, and the use of at least one acronym may be helpful in keeping these fats straight. When I introduce the members of the omega-6 family of fats, I will be more consistent and spell each of them out. The abbreviation for arachidonic acid used by the scientific community is AA, and I don't want anyone's mind going to Alcoholics Anonymous. Or to Los Angeles, since LA is the abbreviation of linoleic acid, the parent of this second family. I will be inconsistent throughout the book, however, in using a combination of common and scientific names for these fatty acids, whichever seems to be the most reader-friendly. The common names reflect the discovery of these compounds—oleic acid was first isolated from olive oil; linolenic acid, from linseed oil—and are sometimes less cumbersome than their scientific equivalents (here, octadecenoic and octadecatrienoic acid, respectively). But the names derived from Greek or Latin—eicosapentaenoic acid, for example—have the advantage of giving the number of carbons and double bonds and are sometimes easier to remember.



from seeds, including soybeans and corn. And this rarity—this deficiency or insufficiency, as people have been calling it since the 1980s—is now being linked to a whole host of human ills. These include diseases of the brain, because of the high concentration of DHA in healthy nervous tissue, as well as heart disease, arthritis and other inflammatory diseases, certain kinds of cancers, and metabolic diseases such as obesity and diabetes, the diseases that tend to specifically plague Western populations—the diseases of civilization, as they have been called, without irony.

Scientists do not know everything there is to know about this family of fats and how their absence from the human diet causes disease—far from it. But what they do know should make physicians and government agencies sit up and take notice before uttering another word of dietary advice. It should cause a thorough reevaluation of our guidelines about fats and health. Why it hasn't is a good question and has something to do with resistance on the part of food industries (which have been removing omega-3s from foods because the many double bonds in omega-3 fats make them more easily oxidized than other fats, resulting in a shorter shelf life for the products that contain them), as well as with the complexity of the science that is involved. (Who would have ever thought that something as lumpish as fat could be so complicated?) It may also have something to do with the slow, meandering history of our understanding of these fats and with our very gradual realization that a balance of the different fats is essential for health.

Which brings me to the reason I have written this book: that the telling of this history may help us to see how omega-3s came to be eliminated from both our diets and our nutritional thinking and to discover how to put them back. A recounting of the ideas that shaped research and dominated medicine may reveal where the advice given us went wrong and give us the courage to

make amends. This book is a tribute to the missing fats in our diet. It is also the history of how researchers discovered that these fats were missing—a nutritional whodunit that plays out in Greenland, Africa, and the many Western countries whose inhabitants first experienced this absence in the form of an epidemic of heart disease.

For many reasons, we have arrived at a critical time for this history. Though for decades we have been advised to consume diets that are low in cholesterol and saturated fat (avoiding foods such as butter and lard, which have a high percentage of straight, saturated chains), and though cholesterol and saturated fat have been reduced in the American diet, heart disease continues to afflict just as many Americans—and we're now facing epidemics of obesity and diabetes. Saturated fat and cholesterol were supposed to be the problem, so where did we go wrong? Why are our health woes multiplying instead of going away? Many explanations for this unhealthy trend have been proposed, including larger portion sizes, excess calories, an increase in the consumption of processed carbohydrates and trans fats, and a decrease in exercise, all of which may share some of the responsibility. But it's time we learned that certain fats—the fats in most of our foods—slow down metabolism, as researchers in Australia are finding. It's time we learned that many companies, in processing food, routinely eliminate the omega-3 fats that are important to both maintaining energy balance and protecting the heart.

A new labeling policy instituted in the United States, effective January 1, 2006, requires food producers to state the amount of trans fats in their products. Such labels are a good thing, since they will enable consumers to avoid these altered fats, which result from a hydrogenation process that makes vegetable oils more solid and stable (that is, less susceptible to oxidation). But the labels won't do much if food producers substitute fats that are

just as unhealthy as trans fats, which is how the food industry seems to be handling the trans hysteria.

Recognizing that the public usually focuses on one bad guy at a time, food manufacturers have replaced trans fats with saturated or monounsaturated fats, or with polyunsaturated fats of the omega-6 family. These polyunsaturates also have multiple double bonds (though somewhat fewer than omega-3s, thereby making them more stable) and are also essential for health, but they compete with omega-3s for places in our cells and membranes and have very different effects on human health, as research has clearly shown. It is time we stopped thinking about good guys and bad guys, good fats and bad fats, and developed a more nuanced outlook toward these underappreciated and frequently maligned nutrients, understanding that all fats play a role in human nutrition and that disease is caused by imbalances between them, not by their mere presence in our diet.

At the same time, biologists are now able to genetically modify plants to produce many different kinds of fats. They are close to being able to insert into spinach the genes used by marine plants in making DHA, for example. Some of these engineered plants will be a boon to health, since they will be able to restore fats that are missing in the diet. Others will be new to human bodies, or available in amounts never seen before, and their effect on the body (over time) is anyone's guess. Scientists are also altering the cooking and salad oils that are familiar to us, using conventional hybridizing techniques as well as genetic modification, and some of the new oils do not live up to their old reputations.

Take canola oil, for instance, an oil that is associated with good health because it is high in alpha linolenic acid, the parent omega-3. Canola oil, which was introduced into the United States in 1985, has been single-handedly responsible for a small increase in the omega-3 content of our diet in recent decades.

Meanwhile plant biologists have been developing low alpha linolenic strains of rapeseed, the plant whose seeds are used to produce canola oil, just as they have developed low alpha linolenic strains of soybeans. The new strains produce oils that are less susceptible to oxidation and might not require hydrogenation, but they are also less beneficial.

Another pressing reason for this history is the confusion and concern about contamination of fish with mercury and PCBs (polychlorinated biphenyls). Pregnant and nursing women are in a terrible quandary. They are told that the fats in fish will help their infant's brain, eyes, and heart to develop properly; yet mercury is known to cause neurological damage. They are advised to consume two meals of fish a week but warned that they should avoid albacore tuna or eat no more than 6 ounces of it a week, advice that is off-putting to even the biggest fans of fish. The issue of PCBs is just as perplexing, with the Food and Drug Administration saying that they pose no threat to most fish supplies and environmental groups urging caution.

Those who are neither pregnant nor nursing worry about contaminants as well, and any thinking person must be concerned about the health of our seas and the sustainability of our fish resources (especially if the advice to eat fish twice a week is taken seriously). At a time when more than 70 percent of commercial fish stocks are said to be "fully exploited, overfished or collapsed," it's critical for us to understand that eating fish is not the only way to increase our intake of omega-3s. Nor is it the most effective way, as I will explain. Protecting our seas and waters is important for many reasons, but simply eating fish cannot solve this nutritional problem.

This history is also timely because many of the researchers who first reported the problem associated with omega-3s are retired from their laboratories, or close to retiring. But they were

still very much alive and able to tell me their stories when I visited them in Minnesota, Copenhagen, London, and Washington and when they spoke to me by phone from Ohio, Sweden, Italy, and Canada. They know firsthand how different fats behave in the cells of our bodies. They collected data showing that people in America and other Western countries have greatly reduced the amounts of omega-3s in their bodies, and they began linking those reductions to illnesses like heart disease. Others have expanded on that work to reveal how omega-3s operate in healthy, well-nourished people and to describe the special role of DHA in dynamic, fast-acting cells like neurons and heart muscle.

I end this introduction, then, with a few more examples of where DHA is found in humans and other animals, examples that speak to the new and growing understanding that DHA, the longest and most unsaturated omega-3, is required for life's speediest tasks.

In human bodies, as I've already noted, the tissues with the most DHA are the tissues of the brain and the eyes, including the eyes that are focused on this page. The second-highest concentration of DHA is found in sperm, which have to swim the fastest, most competitive race of all; next is heart muscle, which beats seventy times per minute, some two billion times in a lifetime.

DHA is found in lesser amounts throughout the rest of the human body, and there its concentrations are influenced by exercise and genetics, as well as diet. Athletes trained for endurance have much more DHA in their skeletal muscle than do less-active individuals. The Pima Indians of Arizona, a population with the highest reported incidence of type 2, or non-insulin-dependent, diabetes, have much *less* DHA in their skeletal muscle than other populations, a finding that scientists attribute to genetic differences.

In hummingbirds, some of life's speediest creatures, the flight muscles, which beat *fifty-two times per second*, are extremely rich in DHA; the leg muscles are not.\* In rattlesnakes, the high-frequency rattle muscle has much more DHA than the low-frequency stomach muscle.

Reptiles, in general, have lower levels of DHA in their tissues than do mammals and birds (and lower metabolic rates); and fish have higher levels, which is understandable since fish live under pressure, in cold, dim environments. They need greater flexibility in their membranes, *and* they have ready access to algae and other high omega-3 foods.

Finally, caribou, which walk the frozen tundra of the far north, have more DHA in their hooves than in their upper legs, which improves the circulation of those parts in direct contact with the permafrost. And hibernating animals, like the yellow-bellied marmot from Colorado, have much *less* DHA in their tissues when they are hibernating than when they are awake. It's not that they lose DHA as they sleep; rather, their bodies do not slow down and go into hibernation when their diet is rich in omega-3s.

\*Hummingbirds eat more than nectar, in case the reader is wondering where they get all this DHA. With their beaks agape, they nab insects (a source of DHA) in the air.