A R T I C L E

Fats and degeneration

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50 years ago, in the first phase of marketing the polyunsaturated fatty acids (PUFA), linoleic acid was “heart protective,” and the saturated fats raised cholesterol and caused heart disease.

In the second phase, the other “essential fatty acid,” linolenic acid, was said to be even better than linoleic acid.

In the third phase, the longer chain omega -3 (omega minus three, or n minus three) fatty acids, DHA and EPA, are said to be even better than linolenic acid.

Along the way, the highly unsaturated arachidonic acid, which we and other animals make out of the linoleic acid in foods, was coming to be identified with the “harmful animal fats.” But we just didn't hear much about how the amount of arachidonic acid in the tissues depended on the amount of linoleic acid in the diet.

U.S. marketing dominates the world economy, including of course the communication media, so we shouldn't expect to hear much about the role of PUFA in causing cancer, diabetes, obesity, aging, thrombosis, arthritis and immunodeficiency, or to hear about the benefits of the saturated fats.

The saturated fats include the “tropical fats,” because they are synthesized in very warm organisms, and are very stable at those temperatures. Their stability offers some protection against the unstable PUFA.

Several of the degenerative conditions produced by the “essential fatty acids” can be reversed by use of saturated fats, varying in length from the short chains of coconut oil to the very long chains of waxes.

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When a person uses a drug, there is generally an awareness that the benefit has to be weighed against the side effects. But if something is treated as a “nutrient,” especially an “essential nutrient,” there is an implication that it won't produce undesirable side effects.

Over the last thirty years I have asked several prominent oil researchers what the evidence is that there is such a thing as an “essential fatty acid.” One professor cited a single publication about a solitary sick person who recovered from some sickness after being given some unsaturated fat. (If he had known of any better evidence, wouldn't he have mentioned it?) The others (if they answered at all) cited “Burr and Burr, 1929.” The surprising thing about that answer is that these people can consider any nutritional research from 1929 to be definitive. It's very much like quoting a 1929 opinion of a physicist regarding the procedure for making a hydrogen bomb. What was known about nutrition in 1929? Most of the B vitamins weren't even suspected, and it had been only two

or three years since “vitamin B” had been subdivided into two factors, the “antineuritic factor,” B_1, and the “growth factor,” B_2. Burr had no way of really understanding what deficiencies or toxicities were present in his experimental diet.

A few years after the first experiments, Burr put one of his “essential fatty acid deficient” rats under a bell jar to measure its metabolic rate, and found that the deficient animals were metabolizing 50% faster than rats that were given linoleic and linolenic acids as part of their diet. That was an important observation, but Burr didn't understand its implications. Later, many experiments showed that the polyunsaturated fats slowed metabolism by profoundly interfering with the function of the thyroid hormone and the cellular respiratory apparatus. Without the toxic fats, respiratory energy metabolism was very intense, and a diet that was nutritionally sufficient for a sluggish animal wouldn't necessarily be adequate for the vigorous animals.

Several publications between 1936 and 1944 made it very clear that Burr's basic animal diet was deficient in various nutrients, especially vitamin B_6. **The disease that appeared in Burr's animals could be cured by fat free B-vitamin preparations, or by purified vitamin B6 when it became available. A zinc deficiency produces similar symptoms,** and at the time Burr did his experiments, there was no information on the effects of fats on mineral absorption. If a diet is barely adequate in the essential minerals, increasing the metabolic rate, or decreasing intestinal absorption of minerals, will produce mineral deficiencies and metabolic problems.

Although “Burr's disease” clearly turned out to be a B-vitamin deficiency, probably combined with a mineral deficiency, it continues to be cited as the basis justifying the multibillion dollar industry that has grown up around the “essential” oils.

Two years before Burr's experiment, German researchers found that a fat-free diet prevented almost all spontaneous cancers in rats. Later work showed that the polyunsaturated fats both initiate and promote cancer. With that knowledge, the people who kept claiming that “linoleic, linolenic, and maybe arachidonic acid are the essential fatty acids,” should have devoted some effort to finding out how much of that “essential nutrient” was enough, so that people could minimize their consumption of the carcinogenic stuff.

Between the first and second world wars, cod liver oil was recommended as a vitamin supplement, at first as a source of vitamin A, and later as a source of vitamins A and D. But in the late 1940s, experimenters used it as the main fat in dogs' diet, and found that they all died from cancer, while the dogs on a standard diet had only a 5% cancer mortality. That sort of information, and the availability of synthetic vitamins, led to the decreased use of cod liver oil.

But around that time, the seed oil industry was in crisis because the use of those oils in paints and plastics was being displaced by new compounds made from petroleum. The industry needed new markets, and discovered ways to convince the public that seed oils were better than animal fats. They were called the “heart protective oils,” though human studies soon showed the same results that the animal studies had, namely, that they were toxic to the heart and increased the incidence of cancer.

The “lipid hypothesis” of heart disease argued that cholesterol in the blood caused atherosclerosis, and that the polyunsaturated oils lowered

the amount of cholesterol in the blood. Leaving behind the concept of nutritional essentiality, this allowed the industry (and their academic supporters, such as Frederick Stare at Harvard) to begin promoting the oils as having drug-like therapeutic properties. Larger amounts of polyunsaturated fat were supposed to be more protective by lowering the cholesterol, and were to be substituted for the saturated fats, which supposedly raised cholesterol and increased heart disease, producing atherosclerotic plaques in the blood vessels and increasing the formation of blood clots.

Since all ordinary foods contain significant amounts of the polyunsaturated fats, there was no reason to think that, even if they were essential nutrients, people were likely to become deficient in them. So the idea of treating the seed oils as drug-like substances, to be taken in large amounts, appealed to the food oil industry.

Prostaglandins, which are produced in the body by oxidizing the polyunsaturated fatty acids, provided an opportunity for the drug industry to get involved in a new market, and **the prostaglandins offered a new way of arguing for the nutritional essentiality of linoleic and related acids: A whole system of "hormones" is made from these molecules.** Since some of the prostaglandins suppress immunity, cause inflammation and promote cancer growth, some people have divided them into the "good prostaglandins" and the "bad prostaglandins."

PGI2, or prostacyclin, is considered to be a good prostaglandin, because it causes vasodilatation, and so drug companies have made their own synthetic equivalents: Epoprostenol, iloprost, taprostene, ciprostene, UT-15, beraprost, and cicaprost. Some of these are being investigated for possible use in killing cancer.

But many very useful drugs that already existed, including cortisol and aspirin, were found to achieve some of their most important effects by inhibiting the formation of the prostaglandins. It was the body's load of polyunsaturated fats which made it very susceptible to inflammation, stress, trauma, infection, radiation, hormone imbalance, and other fundamental problems, and drugs like aspirin and cortisone, which limit the activation of the stored "essential fatty acids," gain their remarkable range of beneficial effects partly by the restraint they impose on those stored toxins.

Increasingly, the liberation of arachidonic acid from tissues during stress is seen as a central factor in all forms of stress, either acute (as in burns or exercise) or chronic (as in diabetes or aging). And, as the fat stores become more toxic, it seems that they more readily liberate the free fatty acids. (For example, see Iritani, et al., 1984)

During this same period, a few experimenters were finding that animals which were fed a diet lacking the "essential" fatty acids had some remarkable properties**:** They consumed oxygen and calories at a very high rate, their mitochondria were unusually tough and stable, their tissues could be transplanted into other animals without provoking immunological rejection, and they were very hard to kill by trauma and a wide variety of toxins that easily provoke lethal shock in animals on the usual diet. As the Germans had seen in 1927, they had a low susceptibility to cancer, and new studies were showing that they weren't susceptible to various fibrotic conditions, including alcoholic liver cirrhosis.

In 1967 a major nutrition publication, *Present Knowledge in Nutrition,*

published Hartroft and Porta's observation that the “age pigment,” lipofuscin, was formed in proportion to the amount of polyunsaturated fat and oxidants in the diet. The new interest in organ transplantation led to the discovery that the polyunsaturated fats prolonged graft survival, by suppressing the immune system. Immunosuppression was considered to have a role in the carcinogenicity of the “essential” fatty acids.

Around the same time, there were studies that showed that unsaturated fats retarded brain development and produced obesity.

Substances very much like the prostaglandins, called isoprostanes and neuroprostanes, are formed spontaneously from highly unsaturated fatty acids, and are useful as indicators of the rate of lipid peroxidation in the body. Most of the products of lipid peroxidation are toxic, as a result of their reactions with proteins, DNA, and the mitochondria. The age-related glycation products that are usually blamed on sugar, are largely the result of peroxidation of the polyunsaturated fatty acids.

Through the 1970s, this sort of information about the harmful effects of the PUFA was being slowly assimilated by the culture, though many dietitians still spoke of “the essential fatty acids, vitamin F.” By 1980, it looked as though responsible researchers would see the promotion of cancer, heart disease, mitochondrial damage, hypothyroidism and immunosuppression caused by the polyunsaturated fats as their most important feature, and would see that there had never been a basis for believing that they were essential nutrients.

But then, without acknowledging that there had been a problem with the doctrine of essentiality, fat researchers just started changing the subject, shifting the public discourse to safer, more profitable topics. The fats that had been called essential, but that had so many toxic effects, were no longer emphasized, and the failed idea of “essentiality” was shifted to different categories of polyunsaturated fats.

The addition of the long chain highly unsaturated fats to baby food formulas was recently approved, on the basis of their supposed “essentiality for brain development.” One of the newer arguments for the essentiality of the PUFA is that “they are needed for making cell membranes.” But human cells can grow and divide in artificial culture solutions which contain none of the polyunsaturated fats, and no one has claimed that they are growing “without membranes.”

The long chain fats found in fish and some algae don't interfere with animal enzymes as strongly as the seed oils do, and so by comparison, they aren't so harmful. They are also so unstable that relatively little of them is stored in the tissues. (And when they are used as food additives, it's necessary to use antioxidants to keep them from becoming smelly and acutely toxic.)

When meat is grilled at a high temperature, the normally spaced double bonds in PUFA migrate towards each other, becoming more stable, so that linoleic acid is turned into “conjugated linoleic acid.” This analog of the “essential” linoleic acid competes against the linoleic acid in tissues, and protects against cancer, atherosclerosis, inflammation and other effects of the normal PUFA. Presumably, anything which interferes with the essential fatty acids is protective, when the organism contains dangerous amounts of PUFA. Even the trans-isomers of the unsaturated fatty acids (found in butterfat, and convertible into conjugated linoleic acid) can be protective against cancer.

In the 1980s the oil promoters were becoming more sophisticated, and

were publishing many experiments in which the fish oils were compared with corn oil, or safflower, or soy oil, and in many of those experiments, the animals' health was better when they didn't eat the very toxic seed oils, that contained the “essential fatty acids,” linoleic and linoleic acids.

Besides comparing the fish oils to the stronger toxins, another trick is to take advantage of the same immunosuppressive property that had seemed troublesome, and to emphasize their ability to temporarily alleviate some autoimmune or allergic diseases. X-rays were once used that way, to treat arthritis and ringworm, for example.

And, knowing that cancer cells have the ability to consume large amounts of fatty acids, they would test these fats in tissue culture dishes, and demonstrate that they were poisonous, cytotoxic, to the fast growing cancer cells. Although they caused cancer in animals, if they could be shown to kill cancer cells in a dish, they could be sold as anticancer drugs/nutrients, with the special mystique of being “essential fatty acids.” Strangely, their ability to kill cancer cells under some circumstances and to suppress some immunological reactions is being promoted in close association with the doctrine that these fats are nutritionally essential.

Arachidonic acid is made from linoleic acid, and so those two oils were considered as roughly equivalent in their ability to meet our nutritional needs, but a large part of current research is devoted to showing the details of how fish oils protect against arachidonic acid. The “balance” between the omega -3 and the omega -6 fatty acids is increasingly being presented as a defense against the toxic omega -6 fats. But the accumulation of unsaturated fats with aging makes any defense increasingly difficult, and the extreme instability of the highly unsaturated omega -3 fats creates additional problems.

PUFA and x-rays have many biological effects in common. They are immunosuppressive, but they produce their own inflammatory reactions, starting with increased permeability of capillaries, disturbed coagulation and proteolysis, and producing fibrosis and tumefaction or tissue atrophy. This isn't just a coincidence, since ionizing radiation attacks the highly unstable polyunsaturated molecules, simply accelerating processes that ordinarily happen more slowly as a result of stress and aging.

Prolonged stress eventually tends to be a self-sustaining process, impairing the efficient respiratory production of energy, converting muscle tissue to amino acids, suppressing the thyroid, and activating further mobilization of fatty acids. Fatty acids are mobilized from within the structure of cells by phospholipases, and from fat tissues by other lipases.

The highly unsaturated fatty acids, as well as the ordinary “essential fatty acids,” act directly to increase capillary permeability, even without conversion into prostaglandins, and they interfere in many ways with the clotting and clot removal systems. The effects of PUFA taken in a meal probably disturb the clotting system more than the same quantity of saturated fat, contrary to many of the older publications. The PUFA are widely believed to prevent clotting, but when cod liver oil is given to “EFA deficient” animals, it activates the formation of clots (Hornstra, et al., 1989). An opposite effect is seen when a long chain fatty acid synergizes with aspirin, to restrain clotting (Molina, et al., 2003).

Fibrosis is a generalized consequence of the abnormal capillary permeability produced by things that disrupt the clotting system.

Estrogen, with its known contribution to the formation of blood clots and edema and fibrosis and tumors, achieves part of its effect by maintaining a chronically high level of free fatty acids, preferentially liberating arachidonic acid, rather than saturated fatty acids.

Butter, beef fat, and lamb fat are the only mostly saturated fats produced on a large scale in the U.S., and the cheapness/profitability of the seed oils made it easy to displace them. But, in the face of the immense amount of propagandistic “health” claims that have been made against the saturated fats, it's instructive to look at some of their actual effects, especially on the clotting system, and the related fibrotic reactions.

The saturated fatty acids are very unreactive chemically. Coconut oil, despite containing about 1% of the unstable PUFA, can be left in a bucket at room temperature for a year or more without showing any evidence of deterioration, suggesting that the predominance of saturated fat acts as an antioxidant for the unsaturated molecules. In the body, the saturated fats seem to act the same way, preventing or even reversing many of the conditions caused by oxidation of fats.

The stress-induced liberation of arachidonic acid causes blood vessels to leak, and this allows fibrin to escape from the blood stream, into the basement membrane and beyond into the extracellular matrix, where it produces fibrosis. (Cancer, autoimmune diseases, and heart disease involve the same inflammatory, thrombotic, fibrotic processes as the nominal fibroses.) Scleroderma, liver cirrhosis, fibrosis of the lungs, heart, and other organs, and all the diseases in which fibrous tissue becomes dense and progressively contracts, involve similar processes, and the treatments which are successful are those that stop the inflammation produced by the oxidation of the polyunsaturated fatty acids.

Retroperitoneal fibrosis is now known to be produced by estrogen, and is treated by antiestrogenic and antiserotonergic drugs, but as early as 1940 Alejandro Lipschutz demonstrated that chronic exposure to very low doses of estrogen produced fibromas in essentially every part of the body. Earlier, Loeb had studied the action of large doses of estrogen, which produced fibrosis of the uterus, as if it had accelerated aging. Following Lipschutz' work, in which he demonstrated the “antifibromatogenic” actions of pregnenolone and progesterone, several Argentine researchers showed that progesterone prevented and cured abdominal adhesions and other fibrotic conditions, including retroperitoneal fibrosis.

Since estrogen produces both leakiness of the capillaries and excessive formation of fibrin, its effects will be seen first in the organs where it concentrates, but eventually anywhere capillaries leak fibrin. Estrogen activates the phospholipase which liberates arachidonic acid, and progesterone inhibits that phospholipase.

As the fat tissues become more burdened with arachidonic acid, they release it more easily in response to moderately lipolytic stress signals. This could explain the increased levels of free fatty acids and lipid peroxidation that occur with aging. In animals that are “deficient” in the polyunsaturated fatty acids, adrenalin doesn't have the lipolytic effect that it does in animals on the standard diet. With aging, there is not only a tendency to have chronically higher free fatty acids in the blood, but for those fatty acids to be more unsaturated. The phospholipids of mitochondria and microsomes become more unsaturated with aging (Laganiere and Yu, 1993, Lee, et al., 1999). In the human retina there is a similar accumulation of PUFA with aging (Nourooz-Zadeh and Pereira,

1999), which implies that the aged retina will be more easily damaged by light.

Several studies suggest that a high degree of unsaturation in the fats is fundamentally related to the aging process, since long lived species have a lower degree of unsaturation in their fats. Caloric restriction decreases the age-related accumulation of the fatty acids with 4 and 5 double bonds.

Although publicity has emphasized the anti-inflammatory effects of fish oil, experiments show that it is extremely effective in producing alcohol-related liver cirrhosis. Breakdown products of polyunsaturated fats (isoprostanes and 4-HNE) are found in the blood of people with alcoholic liver disease (Aleynik, et al., 1998). In the absence of polyunsaturated fats, alcohol doesn't produce cirrhosis. Saturated fats allow the fibrosis to regress**:**

“A diet enriched in saturated fatty acids effectively reverses alcohol-induced necrosis, inflammation, and fibrosis despite continued alcohol consumption. The therapeutic effects of saturated fatty acids may be explained, at least in part, by reduced endotoxemia and lipid peroxidation….” (Nanji, et al., 1995, 2001)

In these studies, the animals were switched from fish oil to either palm oil or medium chain triglycerides (a major fraction of coconut oil). In other studies, Knittel, et al. (1995), show that fibrinogen, in “a clotting-like process,” is involved in the development of liver fibrosis, and that this appears to provide a basis for the growth of additional extracellular matrix.

Brown, et al. (1989), discussed this developmental process (leaky capillaries, fibrosis) in relation to wound healing, lung disease, and tumor growth.

The relatively few studies of fish oil and linoleic acid that compare them with palmitic acid or coconut oil have produced some very important results. For example, pigs exposed to endotoxin developed severe lung problems (resembling “shock lung”) when they had been on a diet with either fish oil or Intralipid (which is mostly linoleic acid, used for intravenous feeding in hospitals), but not after palmitic acid (Wolfe, et al., 2002).

Eating low-fat seafood (sole, whitefish, turbot, scallops, oysters, lobster, shrimp, squid, etc.) once in a while can provide useful trace minerals, without much risk. However, fish from some parts of the ocean contain industrial contaminants in the fat, and large fish such as tuna, swordfish, Chilean sea bass and halibut contain toxic amounts of mercury in the muscles. Chilean sea bass (Patagonian toothfish) is very high in fat, too.

About ten years ago I met a young man with a degenerative brain disease, and was interested in the fact that he (working on a fishing boat) had been eating almost a pound of salmon per day for several years. There is now enough information regarding the neurotoxic effects of fish oil to justify avoidance of the fatty fish.

Some of the current advertising is promoting fish oil to prevent cancer, so it's important to remember that there are many studies showing that it increases cancer.

The developmental and physiological significance of the type of fatty acid in the diet has been established for a long time, but cultural stereotypes

and commercial interests are threatened by it, so it can't be discussed publicly.

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