Fat facts, fads and fallacies

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Abstract

Fatty acids play an enormous role in health maintenance. While the chemical classification and vocabulary of fats is well elucidated, albeit unwieldy, the benefits and harms of this dietary group has only recently begun to be uncovered with the help of impartial research. Trans fatty acids, introduced to replace supposedly harmful saturated fats, may be responsible for much of our society's cardiovascular disease. Efforts to remove this source of injury will take years, but public education is just around the corner. The essential fatty acids, particularly the omega-3 group, have a multitude of disease modifying benefits which should make dietary supplementation a high priority. Saturated fats have been largely misrepresented by Industry. In contrast, legitimate research over the last two decades has slowly outlined the real benefits of naturally occurring palm oils, once considered taboo. Patients will soon benefit from this new paradigm as physicians teach the *fats of life.*

Keywords: fatty acids, trans fatty acids, saturated fats

1. Introduction

He that takes medicine and neglects diet,
wastes the skill of the physician.–*Chinese Proverb*

Dietary fats continue to be a misunderstood key ingredient in health. Our society has been cajoled into rejecting fats, them embracing them with the surge of interest in low carbohydrate diets. The key to optimal health lies in understanding the biochemistry of fats, their necessity in human nutrition and their role in disease. Science is now uncovering these connections; unfortunately, the food industry has the ability to influence outcomes of studies financed with the goal of promoting a favorable spin on the type of fat consumed – a twist designed to boost sales while inadvertently harming consumers.

2. Coming to biochemical terms

Fatty acids are the common denominators we share with other life forms. The oleic acid found in almonds, for example, is the same molecule that is found in cell membranes of the animal kingdom. Fatty acids are the source of nutritional support for many metabolic pathways from hormones to cell wall structure and function, yet this vital source of our diet is still viewed by the public as harmful [1]. Worse yet is the blind intake of unhealthy fats that may take years to manifest as disease in any one individual. The key is not only the amount of fat intake but the type of fat ingested.

Eicosanoids are powerful cell regulators which are derived from essential polyunsaturated fatty acids (PUFAs). Alpha linoleic acid (LNA) and linoleic acid (LA) are essential fatty acids (EFAs) and the generators of the eicosanoids known as the series 1, 2 and 3 prostaglandins (Table 1) [2].

Free fatty acids rarely exist *free* in nature. In dietary consumption they are present as triglycerides (solid fats and liquid oils) or as phosphatides in the cell walls of whole foods. Fatty acids are digested by bile acids and are then absorbed across the intestinal lumen with cholesterol, bound to chylomicrons in the blood where endothelial capillary enzymes cleave off fatty acids for metabolic use. The need for FA supply in the fasting state is met with direct conversion of glucose or amino acids. Of course, FAs can also be mobilized from body fat stores. The liver houses FA as very low density lipoproteins (VLDL). In the fasting state, the liver delivers fatty acids to the periphery using VLDL much like the chylomicrons do for intestinal transit. The low density lipoprotein (LDL) remnants left from this transport system are bound to extrahepatic cell receptors.

While synthesis and transport of FAs seem straightforward, their nomenclature is not and accounts for a considerable amount of confusion even in the scientific literature. Common nomenclature of the FA groups is based upon word derivatives from Greek or Latin. Butyric acid was named for its source – butter. Oleic acid is derived from olive oil and linoleic acid sprouted from the Latin word, *linum*, meaning flax, where linoleic acid (an essential PUFA) is found in abundance. But common names can only give a limited amount of information about FAs.

As one might recall from organic and biochemistry, several naming systems are in current usage; chemical structure is best described by common chemical naming systems. For example, palmitic acid (found in palm oil) is chemically known as hexadecanoic acid (hexadeca = 16 and "oic" = acid); thus describing a 16 carbon chain fatty acid molecule. When prefixed by descriptors such as w6,9 (e.g., *cis*- w6,9-octadecadienoic acid for linoleic acid), this would indicate where in the organic chain the double bond is located – it starts at position 6 and 9, beginning at the *w* or omega, or methyl end (Figure 1). The letter *n* or the Greek letter omega (Ω) is sometimes used interchangeably with *w* to signify this double bond position.

Structural formulas are somewhat unwieldy and have given rise to the use of shorthand notations. In the example noted in Figure 1, the shorthand notation would be condensed to 18:2w6. The number of carbon atoms being 18; while 2 denotes the number of double bonds and w6 the position from the methyl end where the first double bond appears.

To make the chemistry of FAs more confusing, another naming system was developed using the Greek letter delta (Δ) which is superscripted with numerals specifying the double bond positions. This system counts from the other end of the

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molecule (the carboxyl end). The above example, linoleic acid would then become 18:2$\Delta^{9,12}$. Thus, the double bonds are found at the ninth and twelfth position from the carboxyl (COOH) end.

3. Characteristics of fatty acids (FAs)

FAs are divided into the saturated fatty acids (SaFAs) and unsaturated fatty acids (UFAs). SaFAs are present in all edible fats and oils and most abundant in hard fats. SaFAs contain no double bonds (no "unsaturation") and are from 4 to 28 carbons in length. Lengths above 10 carbons are solid at body temperature. The medium chain triglycerides (MCTs) contain 6-12 carbons and are unique in that the body does not store them as fat, yet they are readily utilized for energy – explaining why the well known dosing of two tablespoons of MCT oil has become a favorite pre-workout meal.

Long chain FAs use their solidity at body temperature to build cell membranes, keeping unsaturated FAs from becoming active when mixed together at the cell membrane level. Their solid state, however, also functions to cause long chain FAs to aggregate, making platelets stickier. Cholesterol is in this category. With a melting point of 149ºC, the reality of its deposition in cells, arteries and organs is well known. SaFAs can be directly consumed from diets high in beef, pork, and dairy, but can also be manufactured from diets high in excess sugar which is directly converted to SaFAs. It is interesting to note that the typical American diet is notoriously abundant in both of these supply lines.

UFAs, on the other hand, do contain at least one double bond between carbon atoms and retain the same end structures of a methyl and carboxyl acid group. It is the presence of this double bond (one or more) that makes all the difference in their function and health consequences. Note in Figure 1 that linoleic acid is bent at the double bond. This is the normal *cis* configuration found in nature. This kinking in the molecule allows for poor aggregation, lower melting temperatures and a negative charge at the double bond is generated by the pair of extra electrons. Hence these UFA chains tend to repel one another allowing for greater dispersion. This becomes critical in the cell membranes where fluidity is required for nutrient transport and other cell membrane dynamics.

Monounsaturated fats (MUFAs) contain one single double bond. Oleic acid (18:1w9) is probably the best known and most important of these. Found in olive and other nut oils, oleic acid (OA) adds suppleness to arterial walls and lubricates our skin. Although an important PUFA, oleic acid is non-essential since it can be manufacture *de novo* from SaFAs. OA in excess can compete with the essential PUFAs and interfere with prostaglandin function due to competition with the conversion enzyme Δ6 desaturase [1].

Polyunsaturated fats (PUFAs) contain more than one double bond. The most important in this class are linoleic acid (LA) (Figure 1) and α-linoleic acid (LNA), whose chemical notation is 18:3w3. Both LA and LNA (and their derivatives) comprise the essential fatty acids in human nutrition. One might consider LA the "seed and corn" (omega 6) PUFA and LNA as a "fish and flaxseed" (omega 3) PUFA. The important health benefits of this class will be discussed in detail later in this article.

4. *Trans* fat – the phantom menace

The benefits of olive oil's oleic acid were changed drastically when it was converted to the *trans* form, elaidic acid. Other beneficial *cis* configured PUFAs soon followed at the insistence of the food industry's need for longer shelf life, a replacement for SaFAs and stable profits. Hard margarine, for example, soon contained as much as 60 percent of the total fats as elaidic acid. Animal sources are also known to contain TFAs due to bacterial hydrogenation of PUFAs in the rumen [3]. This amount of TFA from animal sources is probably of no health consequence if intake is only moderate.

Unfortunately, there was a serious downside to TFAs as a dietary source. As shown in Figures 2 and 3, the *trans* isomer of the naturally occurring PUFAs transforms the normally bent shape of the long chain structure into a straightened molecule.

Replacing SaFAs in foods with partially hydrogenated TFAs was perhaps a well meaning but nonetheless nutritional disaster for industrialized society. Reports began to surface in the early and mid-90's demonstrating that modest intake of *trans* fat could adversely affect lipoproteins and thus cardiovascular risk factors.

TFAs behave metabolically like SaFAs due to their tendency to resemble the saturated moieties, but this resemblance may not be responsible for their deleterious effects. On the other hand TFAs are able to mimic UFAs by competitive binding to desaturase enzymes thereby adversely affecting the normal metabolic conversion of UFAs. The combined effect which has concerned researchers is an apparent elevation of LDL, lipoprotein A [Lp(a)] and reduction in HDL [1]. Sundram *et al* looked specifically at elaidic acid's effects on lipids and comparing them to SaFAs and the natural *cis* 18:1 UFAs. Adverse lipid effects were similar between SaFAs and *trans* isomers of oleic acid in the study groups [4]. In addition to an elevation of plasma triglycerides due to TFA intake, studies of impaired flow mediated vasodilatation further suggest TFAs increase the risk of CAD through an adverse effect on endothelial function.[3] It may be this effect, rather than their mimicry of SaFAs that is the bad actor in this ongoing dietary drama.

Given the well established relationship these blood lipid abnormalities and increased rates of coronary artery disease (CAD), research interest soon began to wax extensively with a sharp focus on the health effects of TFAs. In one case control series of TFAs in angiographic confirmed CAD and matched disease free controls, plasma TFAs were significantly elevated in the disease laden group. Epidemiological data also appeared to confirm a causal relationship [5,6]. In a three year case controlled study, Dr. Clifton and colleagues confirmed epidemiological suspicions when adipose fat biopsies were correlated with dietary TFA intake and first myocardial infarction [7]. Up to 15 percent of tissue fatty acids have been found at necropsy to contain the *trans* fat double bonds [1].

TFAs have an adverse effect on fetal development. A negative association was noted between birth weight and TFAs in plasma of normal and premature infants. Other evidence noted that TFAs, including conjugated linoleic acid levels, were inversely related to length of gestation. TFAs may also impair insulin resistance, possible due to the effects on the ion channels in cell membranes [8-10]. The FDA will require labeling

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of TFAs on food packages as of January 1, 2006. There will be equal weight given to TFAs from industrial and animal (ruminant) sources. Therefore, one can expect milk to have a TFA listing.

Will the change in food labeling for *trans* fat make a difference in terms of cardiovascular health? Since TFAs will not be banned, much will depend upon public awareness. Many products are already labeled as to *trans* fat content and often packages will boldly proclaim TFA absence. When Denmark reduced its intake of industrially produced TFAs from 6 grams in 1976 to less than 1 gram in 1996, there was a concomitant 50 percent decrease in deaths from ischemic heart disease [3].

5. The omega factor – the essential fatty acids and health

References to the snake oil salesman of the old west conjure visions of a charlatan trade. But snake oil originally came from China. For thousands of years it had been used for inflammatory conditions as a topical application. Chinese laborers brought it to the US for treating the pains derived from long hours spent on railroad construction. U.S. Patent medicine promoters belittled the claim of this product to the point that many consider it a stigma; however in 1989, replication of this snake potion–oil from the original Chinese water snake–found that it was composed of over 20 percent eicosapentaenoic acid (EPA), an omega-3 derivative, one of the richest sources of omega-3 fatty acids known. Absorbed through the skin, EPA, the parent of series 3 prostaglandins, inhibits the pro-inflammatory effects of series 2 prostaglandins (Table 1) [2]. And as we shall see, this is only the beginning.

6. Omega basics (w6 and w3)

LA, an omega-6 fatty acid, and the two principle omega-3s, LNA and EPA, cannot be synthesized in humans and are known as essential fatty acids (EFAs). Their intake is necessary on a daily basis in order to prevent deficiency states that can comprise a laundry list of behavioral and systemic signs and symptoms. Indeed, prolonged deficiency of LA is fatal [2]. The overall function of EFAs is only now being elucidated and it is well established that EFAs function to maintain growth, tissue repair, oxygen transport, hemoglobin production and cell membrane integrity (to name only a few).

Requirements for the omega-6 LA acid are about 1-2 percent of calories (3-6 grams) to prevent a deficiency state. The optimal dosing depends upon general nutritional state, physical activity and stress. High doses of LA have been given without adverse effects to animals (up to 28 percent of calories), but intake of *natural* vitamin E (d-alpha tocopheryl) is necessary for proper utilization – about 1 unit of natural vitamin E for very 1500 parts LA. For example, an 18 gram ingestion of LA would require about 30 International units of vitamin E.[2] Nonetheless, high doses (particularly in the vitamin E deficient American diet) may be a health concern. Dietary sources of LA (i.e., corn oil) are abundant and levels can be excessive in some adults. Such excess could contribute to an overproduction of the proinflammatory series 2 prostaglandins.

Gamma linolenic acid (18:3w6) is the precursor for dihomogammalinolenic acid (DGLA). Its importance lies in its function as the precursor for the inflammatory moderating series-1 prostaglandins (Table 1). Found in relatively high amounts in evening primrose, borage, black currant and hemp oils, GLA can also be produced *in vivo* by desaturase enzyme activity on LA. Not unexpectedly, borage seed oil at levels of 1.4 g/d showed evidence of clinical improvement in rheumatoid arthritis.[11] Care should be taken when supplementing GLA in cancer patients, since GLA not supplemented with omega-3 PUFAs enhances tumor formation and growth [12].

Arachidonic acid (AA or 18:4w6) deserves mention (Table 1,2). AA is unlikely to be deficient in modern diets due to its prevalence in corn oil and corn oil products used in feed for cattle and hogs. AA is the precursor for the proinflammatory cyclooxygenase and lipoxygenase enzymes which, in turn, produce the series-2 prostanoids and leukotrienes. Besides being proinflammatory, high AA intake also promotes cholelithiasis through promotion of mucin production by the gallbladder [13].

Optimal intake of the omega-3 LNA is between 2 and 9 grams per day and is readily available in plant sources, particularly flaxseed (Table 3). LNA's importance resides in its biochemical transformation. Through enzymatic elongation and desaturation it is converted in vivo to EPA and then to DHA via the Δ-6 desaturase enzyme (Table 2).

For maximizing health, the *omega-6:omega-3* ratio should hover around 4:1 (although this varies widely around the world). Since the beginning of the 19th Century, omega-3 PUFA consumption has decreased by one-sixth and omega-6 PUFA intake has doubled. Flax oil is the richest source of LNA and hemp oil has the closest to ideal ratio of *omega-6:omega-3* (3:1). Although hemp contains marijuana, consuming hemp oil is legal; but traces of tetrahydrocannabinol (THC) may surface during urine drug testing.

With the notable exception of the EPA containing purslane plant (*Claytonia perfoliata*) and an recently discovered algae source of DHA, these EFAs are scarce in the vegetable kingdom – but are highly concentrated in the skin and liver of some fish and reptiles (e.g., codfish and the Chinese water snake). EPA is the parent of the much needed series 3 prostaglandins and leukotrienes that moderate the proinflammatory activity of AA derived series 2 prostaglandins (Table I). EPA can be converted from dietary sources of LNA, but dietary intake of LNA is generally not sufficient. Conversion further requires the Δ-6 desaturase enzyme which can be sluggish if vitamin intake is deficient [1]. High levels of TFAs will also slow this conversion.

The omega-3 PUFA docosahexaenoic acid (DHA, or 22:6w3) is well known for its effect on the growth and development of the central nervous system. Attention deficit disorders and developmental diseases of the visual cortex are examples of DHA deficiencies during early growth and development. Levels of DHA (and its companion docosapentaenoic acid) in breast milk are directly correlated with the mother's intake of fish oil 14].

7. Omega 3 PUFAs in Disease Prevention

In the 1970's the first epidemiological studies [15] began to surface reporting the beneficial effects of the w-3 PUFAs on CHD. More recently, the U.S. Physicians Health Study [16]

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demonstrated that consumption of at least 1 fish meal per week reduced the risk of cardiac death by 52 percent. While w-3 PUFAs probably have several mechanisms which promote cardiac protection, their ability to stabilize the electrical activity of cardiac myocytes through inhibition of the sarcolemmal ion channels and prolonging the refractory period has received notable attention [17].

Antithrombotic properties found in the w-3 class of PUFAs are but one more addition to a wealth of benefits. Beyond reducing fibrinogen and increasing tissue plasminogen activator levels, EPA is known to inhibit synthesis of thromboxane A_2, the prostaglandin noted for vasoconstriction and platelet aggregation [18]. The vasodilatory effect of endothelial nitric oxide (NO) is enhanced by EPA and the reduction of free radicals by EPA further increases NO tissue levels [19]. EPA's reduction in platelet-derived growth factor (PDGF) may seem esoteric, but PDGF is a key chemotactic factor and mitogen for the smooth muscle cells and macrophages that play a role in atherosclerotic plaque formation (Table 4) [19].

Unlike the omega-6 PUFAs, dietary omega-3 oils do not lower HDL. Indeed, an increase in the most favorable anti-atherogenic subtype, HDL_2, is the norm [21]. Total reductions in cholesterol, triglyceride and apolipoprotein B production are also observed. Angiographic trials in patients randomized to omega-3 PUFAs have shown a modest improvement in CAD progression but analysis of re-stenosis rates after angioplasty failed to show an effect with omega-3 PUFA intake [22,23].

In the GISSI-Prevention Trial [24], 11,324 post-MI subjects in Italy–already on a relatively healthy Mediterranean diet–were followed for over 3 years and randomized to either vitamin E, DHA/EPA, both, or placebo. The results demonstrated a 20 percent reduction in cardiovascular events in the 850 mg/day EPA group. Such results are not limited to DHA and EPA; omega-3 PUFAs from both plant and animal sources have proven beneficial in cardiovascular disease prevention [25].

Reductions of 25 to 30 percent in triglycerides are noted with doses of 4 grams per day of w-3 PUFAs [26]. Intake of greater than 3 grams per day should be done under a physician's care since at this level, excess bleeding can be noted in some patients. Cardiovascular benefits are clearly seen at lower doses (1 g/day). Omega-3 PUFAs have only a mild hypotensive effect at the higher doses, limiting their usefulness as a primary therapy for hypertension. This hypotensive effect is most likely due to the endothelial effects of improved NO levels. More specific benefits to the heart include lowering of resting heart rate, decrease in left ventricular filling pressures and the previously mentioned antiarrhythmic effect.

Increasing attention is now being focused upon the role of w-3 PUFAs in inflammatory arthritis. Volker [27] demonstrated clinical improvement using w-3 PUFAs in patients with rheumatoid arthritis. Researchers at Cardiff University in Wales [28] brought attention to osteoarthritis in a trial showing that 86 percent of pre-operative patients with arthritis who took w-3 PUFAs in the form of cod liver oil on a daily basis, had absent or significantly reduced levels of the enzymes that cause cartilage damage compared to 26 percent of those given a placebo oil capsule. Specifically, in the w-3 group, there was a marked reduction in some of the enzymes responsible with joint pain - cartilage aggrecanases, the cytokines known as interleukin (IL)-1α and tumor necrosis factor (TNF)-α, and cyclooxygenases (COX-1 and COX-2). The implications of this biochemical pathway seem obvious, yet apparently underutilized in our pharmaceutical based health care system.

The anti-inflammatory properties of w-3 PUFAs extend beyond arthritic conditions. Belluzzi *et al* performed a one-year, double blind, placebo-controlled analysis of patients with Crohn's disease supplemented with 9 fish oil capsules per day. After one year 59 percent of the study group remained in remission, compared to 26 percent of controls ($p = 0.003$) [29].

Other benefits of w-3 supplementation have also been observed. Tepaske and colleagues found that a combination of w-3 PUFAs, L-arginine, and yeast RNA was effective as an oral immune enhancing nutritional supplement in high risk patients undergoing elective cardiac surgery improving both post-operative renal function and reducing the number of post-operative infections [30]. Recently, evidence of a benefit for age-related macular degeneration (AMD) surfaced after a 4.6 year prospective study showing that higher fish intake was associated with a lower risk of AMD [31]. Behind the eyes, DHA supplementation (1.5-1.8 grams/day) also revealed a lower rate of aggression in young adults using standard psychometric testing [32].

Since omega-3 PUFAs seem to be critical to the growth and maintenance of brain cells, especially cell membranes, the recent link between omega-3 deficiency and depression, even bipolar depression and schizophrenia is not surprising [33,34]. Further evidence of benefit in childhood asthma and migraine headaches place the clinical benefits of omega-3 supplementation into a category of necessity for the general population [35,36]. Overall, the evidence for omega-3 intake, at least for cardiovascular health, has impressed the FDA sufficiently that they have issued a qualified health claim for this oil in September 2004 [37]. A qualified health claim on a conventional food must be supported by credible scientific evidence. This allows labeling of food sources for a health claim for reduction of cardiovascular disease and requires a content of total EPA and DHA per serving. Indeed, it would appear that few other single dietary interventions have the potential therapeutic impact as proper omega-3 PUFA intake.

8. Other oils of interest

Trans fatty acids are not the only controversial fats. Reaction over the addition of olean (Olestra™) as a "fake fat" food additive has flooded the scientific community with a tsunami of debate. In the usual industry vs non-industry contest of facts, industry funded studies have claimed olean's safety while independent research shows a plethora of potential health problems [38. Indeed, in 2002, 19,700 reports of adverse reactions to olean were submitted to the FDA. Detractors fear that olean's ability to inhibit absorption of nutrients (e.g., fat soluble vitamins) may be preparing the consuming public for a avalanche of serious health consequences.

Functional foods, like the omega-3 PUFAs, also include the SaFA from coconuts (palm oil). In the 1950's when Keys and Taylor [39 announced that the heart disease epidemic was caused by hydrogenated vegetable oils, industry's solution was

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the introduction of partially hydrogenated oils (*trans* fatty acids); however, little change was needed since oils were already being hydrogenated at that time. During this same period other researchers reported the observation that ingestion of PUFAs lowered serum cholesterol. According to Enig [40] he original publications neglected to mention that the lowering was due to cholesterol moving into the peripheral tissues. The consequence of such widely publicized reports was to introduce "fear of fats" (at least certain fats) in the minds of millions of consumers.

The apprehension of cardiovascular consequences from coconut oil is based upon its chemical classification as a SaFA. But there are no final answers that satisfy all researchers on the health risks of SaFAs. Indeed, Keys' saturated fat theory took a major blow in 1992 when Framingham's Dr. William Castelli [41 declared "…in Framingham, Mass, the more saturated fat one ate, the more cholesterol one ate, the *more calories one ate,* the lower the person's serum cholesterol…the opposite of what the equations provided by Heisted etl (1965) and Keys et al (1957) would predict…"

In spite of Dr. Castelli's finding that the Framingham group that ate higher SaFA had lower cholesterol, the results only reported that the cohort with higher cholesterol had an increased risk of death from CAD. This begs the question: if SaFAs did not cause the elevated cholesterol (and resultant risk increase), what did? Questions such as this have led some researchers to claim that SaFAs have been misunderstood, or perhaps misrepresented, and only recently has science rediscovered their benefits [40]. Indeed, the low carbohydrate diet controversy has spurred intense debate over the benefits or harm of SaFAs. Advocates will correctly point out that "Industry" has manipulated studies for profit in the past, looking no further than the scientific *spin-doctoring* performed on lead, asbestos, urea foam insulation, and mercury in vaccines. The debate continues.

But concerning palm oil in particular, Blackburn et al reviewed the published literature on its effect on serum cholesterol and concluded that natural, unprocessed coconut oil is a neutral fat in terms of atherogenicity [42]. In fact, adverse reports of coconut oil on animals have been attributed to coconut oil's hydrogenation with resultant TFAs or feeding a coconut oil only diet that was devoid of all essential fatty acids. Approximately 50 percent of the fatty acids in coconut fat are lauric acid, a medium chain triglyceride, which has the additional beneficial function of being formed into monolaurin in the body. Monolaurin has anti-viral, antibacterial, and antiprotozoal activity in humans by its ability to destroy lipid coated viruses (e.g., HIV, herpes, cytomegalovirus, influenza, and *H. pylori*) [43]. Physicians and the public are then left to decide the benefits of SaFAs, particularly coconut oil.

Pure MCT oil is less controversial. Ingestion of these medium chain triglycerides (MCTs), besides being an athletic premeal, has been found to impede accumulation of body fat in rodents [44]. At least two studies have confirmed increased thermogenesis and decreased deposition of body fat in diets high in MCTs [45,46]. Coconut oil is readily available for some MCT content (lauric acid) in its pure form in health food stores. Pure MCT *oil* is a special order item and can be added to salads, popcorn or blended into fruit drinks. Research at McGill university may hasten access to MCT with a new commercial form of this oil [47]. Studies on this product revealed a lowering of cholesterol by at least 13 percent compared to olive oil at 4.5 percent. Using cross sectional magnetic resonance imaging, total body fat and fat volume were reduced in the study group. The phytosterols in the oil - the very same found in the common coconut - are touted as being the fat and cholesterol reducing moiety; and the commercial product is a coconut oil derivative. The benefit over standard palm oils appears to be the ability to cook with this blend at much higher temperatures than was previously possible. In the prescription section of this article, I will discuss another currently available alternative for healthier cooking.

Peanuts and peanut oil, an American staple, deserve special mention. Corinna *et al* in a peanut industry funded study, looked at the effects on serum triglycerides in 15 subjects ingesting 500 calories of peanuts in a 30 week double blind crossover analysis [48]. Results showed a mild favorable effect on triglyceride reduction, suggesting that peanuts were beneficial to cardiovascular risk profiles. But there are thorns in every rose garden as well as in some peanut patches. Concerns over peanut consumption are historically based on its inherent ability to produce allergic and anaphylactic reactions and the potential for contamination by aflatoxin - a natural toxin produced by certain strains of the mold *Aspergillus flavus* and *A. parasiticus* that grow on peanuts (as well as other nuts) stored in warm, humid environments. U.S. Government testing has kept aflatoxin levels at 20 parts per billion or less in standard commercial sources but concerns persist over raw peanuts sold in health food stores.

Toxins and allergy aside, peanut oil is a poor source of LNA compared to other nut sources (Table 3) [49]. Of equal concern is the fact that peanuts (along with canola and mustard) contain very long chain fatty acids (VLCFAs) which, along with other odd chain and branch chain PUFAs, are classified as "renegade fats" [50]. Such fats when incorporated into cell membranes can cause off key expression of membrane function resulting in cell death and apoptosis [51]. A healthier alternative might be almond butter, containing oleic and α-linoleic acid.

Physicians who wish more information on in-depth testing for lipid deficiencies can view a sample analysis (including extensive metabolic studies) at http://tinyurl.com/3s7pl. Metabolic and nutritional recommendations for correcting deficiencies are also obtainable from readily available scientific texts [1].

9. Conclusions – the prescription: oil

It is clear that our individual biochemistries function best when the proper fatty acids are supplied in the diet. The following is a list of recommendations that might prove beneficial based upon current evidence.

• Limiting TFAs in the diet is essential. Teaching patients to read labels and eschewing items containing "partially hydrogenated" oils of any type is paramount. For purists who wish to severely limit even naturally occurring TFAs in meat and dairy, skim milk and reduced intake of red meat products would suffice. Cooking with oils at high temperature will invariably lead to the formation of TFAs. One notable exception is cooking oil

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made from macadamia nuts. This oil is rich in healthy MUFAs (79 percent) and does not form TFAs when used at higher cooking temperatures [52].

• Fish consumption has been recommended as a source of omega-3 PUFAs; unfortunately, the larger the fish (the higher up the food chain), the greater the likelihood of mercury contamination. Fish to avoid for possible high mercury levels are shark, swordfish and tuna. Mercury free cod liver oil (CLO) is readily available in liquid and capsule formulations. One teaspoon of CLO will supply approximately one gram of omega-3 DHA/EPA (the equivalent of about 3 capsules). Concerns over excess vitamin A and D intake from cod liver oil ingestion would only be significant at the higher doses (2 to 3 teaspoons a day under physician supervision). However, CLO free of vitamin A and D is available with up to 1.8 grams of DHA/EPA per teaspoon. Flaxseed oil, containing 20 grams of LNA/100 grams of raw flaxseed, is also a superb source of w-3 LNA. Eggs containing up to 800 mg of omega-3 PUFAs are now available in retail stores as well.

• MCT oil sources (perhaps coconut and certainly the emerging "functional oils") deserve a second look for potential health benefits that have gone largely unnoticed for the past 5 decades. Consumers choosing "low carb lifestyles" will benefit from adding functional oils, as well as carefully balancing *omega-3/omega-6* ratios in their diets. It is likely that proper intake of these fats will provide improved energy and increased thermogenesis that will make natural, high fiber carbohydrates more appealing while at the same time moderating body fat.

Perhaps in the next decade the saturated fat dilemma will be solved; but at present there is little doubt over the danger of *trans* fat, regardless of the mechanism. Diet and health are so tightly intertwined that they are one. Consumption of the "natural balance" of dietary fats can be of substantial benefit for disease prevention. The advice of one ancient proverb sums it up the best: "When diet is wrong medicine is of no use. When diet is correct medicine is of no need."

Let the oil change begin…

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Figure 1. Chemical Structure of Linoleic Acid (Omega-6 Fatty Acid)

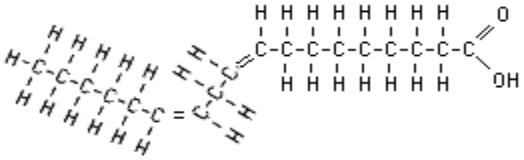


Figure 2. *Trans* fatty acid configuration

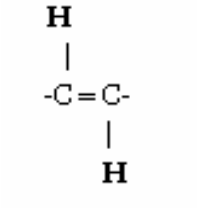
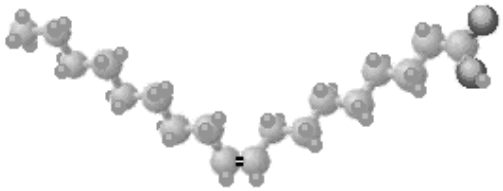
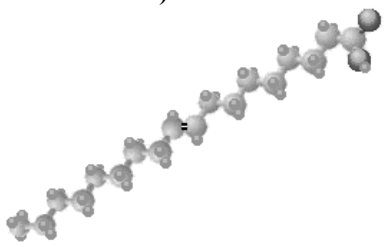


Figure 3. Oleic acid (*cis* and *trans* forms)



Cis-9-octadecenoic acid (Oleic acid)



Trans-9-octadecenoic acid (Elaidic acid)

Table 1. Prostaglandins derived from acid metabolism.[2]

Prostaglandin Series	Origin	Function (+) promotes; (-) inhibits; (+/-) mixed effects
1	DGLA (from LA elongation)	Inflammatory (-) Vasodilation (+) Thrombotic (-)
2	AA (from LA elongation and desaturation)	Inflammatory (+) Vasoconstriction (+) Thrombotic (+)
3	EPA (from LNA elongation or fish oil)	Inflammatory (+/-) Vasoconstriction (+/-) Thrombotic (-) weak effect

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Table 2. Pathways for w-3 and w-6 polyunsaturated fatty acid formation

w-6	*w-3*
Linoleic Acid	α-Linoleic Acid
(Seeds, Nuts, Vegetables)	(Legumes, Flaxseed, Vegetables)
↓	↓
Δ6 Desaturase	Δ6 Desaturase
↓	↓
γ-Linoleic Acid	Eicosapentaenoic Acid
(Found in Borage and Primrose Oils)	(Found in Fish Oil)
↓	↓
Arachidonic Acid	Docosahexaenoic Acid
(Found in Meat)	(Found in Fish Oil)
↓	↓
w-6 Eicosanoids	w-3 Eicosanoids
Thromboxane A_2	Prostacyclin
Leukotriene B_4	Thromboxane A_3 (reduced activity)
	Leukotriene B_5 (reduced activity)

Adapted from Harper[49]

Table 3. α-Linoleic Acid Sources (g LNA per 100 g raw source)[49]

Source	Content
Flaxseed	20.0
Butternuts	8.7
English walnuts	6.8
Soybeans (raw)	3.2
Leeks	0.7
Wheat germ	0.7
Purslane	0.4
Almonds	0.4
Pinto beans	0.3
Barley (bran)	0.3
Kale	0.2
Chickpeas	0.1
Avocados	0.1
Strawberries	0.1
Peanuts	0.003

Table 4. Proposed mechanisms of omega-3 PUFA cardiovascular beneft.

- Reduction of dysrhythmia potential
- Antithrombogenesis
- Triglyceride reduction
- Atherosclerotic plaque reduction
- Enhanced nitric oxide-induced endothelial relaxation
- Reduction in free radicals

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