

## **Scientific Support for Chapter 8: The Danger of Processed/ Adulterated Fats**

**We were told but few listened.....**

“Lipid oxidation products in food and atherogenesis,” by Stan Kubow, Ph.D. in Nutrition Reviews; Feb 1993; 51, 5, pages 33-40, gave us this *incredible insight but few were made aware of it:*

“An exploration of the possible consequences may be particularly relevant at this time, both because of increasing evidence of a **pathogenic role of endogenous lipid oxidation in a number of chronic and acute disease states** and because recent advances in analytical techniques such as gas chromatograph-mass spectrometry (GC-MS), high-pressure liquid chromatography (HPLC), and nuclear magnetic resonance (NMR) have enhanced the ability to identify and quantify lipid oxidation products in biological systems.

“For example, the **toxicity of lipid oxidation** products formed in situ [in the body] may have **little relevance to the properties of lipid oxidation products taken in orally** [already adulterated from food processing].

“Linoleic acid [**Parent omega-6**] **hydroperoxides can cause irreversible damage** to porcine pulmonary artery endothelial cells and injection of this compound into the bloodstream causes marked damage to aortic endothelial cells....It is of interest that macro-

phages take up LDL very slowly and **do not change to foam cells unless the LDL has been modified or oxidized.**

“The feeding of unheated but highly oxidized fish oils at levels of 10--20% in the diet of rats has resulted in a wide spectrum of injurious effects, including diarrhea, loss of appetite, growth retardation, cardiomyopathy, hemolytic anemia, and the accumulation of peroxides in adipose tissue. **In particular, high concentrations of C20-C22 polyunsaturated fatty acids found in certain fish species and fish oils cause such oils to oxidize readily on exposure to air.**

“The possibility of oxidative stress induced by absorption of preformed secondary lipid oxidation products in foods that readily go rancid, *such as fish oils*, has also been suggested by an immediate and marked increase in urinary malondialdehyde observed in rats after ingestion of a cod liver oil diet.”

### **Is steaming or pan-frying a fish filet better?**

Prepare to be shocked. *The Journal of Agricultural and Food Chemistry* has this to say:<sup>1</sup>

“Salmon fillets were steamed, or pan-fried without oil, with olive oil, with corn oil, or with partially hydrogenated plant oil.

“In particular, *fish and fish oils are highly susceptible to oxidation* due to their high content of polyunsaturated

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1 Al-Saghir, Sabri, et al., “Effects of Different Cooking Procedures on Lipid Quality and Cholesterol Oxidation of Farmed Salmon Fish (*Salmo salar*),” *J. Agric. Food Chem.* **2004**, 52, 5290–5296.

fatty acids (30-40%), mainly eicosapentaenoic acid (EPA, 20:5n-3) (5-18%) and docosahexaenoic acid (DHA, 22:6n-3) (1-12%). These **unique fatty acids distinguish fish lipids from other plant and animal lipids.**

“The sum of **cholesterol oxidation products (COPs)** increased after the heating processes from 0.9 mg/g in the raw sample to **6.0, 4.0, 4.4, 3.3, and 9.9 mg/g** extracted fat in pan fried **without oil**, with olive oil, corn oil, partially hydrogenated plant oil, and **steamed**, respectively.

“As expected, the lipid oxidation of the residues was advanced. The highest increase was found for the residues of cooking without oil which *revealed the highly oxidative potential of the fish oil* that drained out of the sample during cooking...

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- ▶ **There was a “700% increase in COPs with fish seared without oil.” In contrast, meats cooked without oil increased only “120%-315%.”**
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“In particular, steaming increased the total amount [COPs] by more than 1000%, which should be considered in nutritional recommendations, although the initial COPs content of salmon is low compared to other foods such as meat.” [As you already understand, any type of heat compared to the frigid temperatures fish live in, quickly ruins EPA/DHA.]

**PEO Solution** analysis: Shockingly, that steaming caused the most damage. The **least damage** was from **pan-frying** in hydrogenated oil. However, do NOT use that oil. Use a saturated fat like organic coconut or palm oil for the best results. Hydrogenation is a very harmful process. Heating the fish with no oil is unhealthy because the oil draining from the fish is exposed to high temperature. That's why the authors state, "As expected, the **lipid oxidation of the residues (oils) was advanced**. The highest increase was found for the **residues** of cooking without oil, which **revealed the highly oxidative potential of the fish oil** that drained out of the sample during cooking."

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### **Warned but Few Listened**

The 2001 journal article, "Health effects of oxidized heated oils," warned us but too few listened:<sup>2</sup>

- "The purpose of this report is to alert the food service industry, particularly the fast-food industry, of an emerging health issue. Considerable evidence has accumulated over the past two decades that heated cooking oils, especially polyunsaturated oils, may pose several types of health risks to consumers of fried foods and even people working near deep fat fryers. Heat degrades polyunsaturated fatty acids to toxic compounds; *saturated and monounsaturated fatty acids are resistant to heat-induced degradation*.
- "The thermally-induced oxidation of glycerol-bound **polyunsaturated fatty acids** (PUFAs) in foods and

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2 Grootveld, Martin, et al., *Foodservice Research International*, Vol. 13, 2001, pages 41-55.

culinary oils during standard frying or cooking episodes is a process that involves the prior generation of isomeric **conjugated hydroperoxydiene** (CHPD) species. These CHPDs fragment to form alkoxy radicals that, in turn, undergo  $\beta$ -scission to generate a **wide range of aldehydic products**. In view of the extremely **toxic nature of the aldehydic end-products generated**, the employment of PUFA-containing culinary oils for **domestic or commercial frying/cooking** episodes poses **health hazards** that have recently attracted much public and clinical interest.

- “Indeed, these **cytotoxic agents** have been implicated in the development and progression of **atherosclerosis** and its associated pathological sequelae such as ischemic **heart disease and peripheral vascular disease**, and have also been shown to exert gastropathic, **pro-inflammatory**, and genotoxicological properties. These phenomena are undoubtedly attributable to the **extremely high reactivity of aldehydes with critical biomolecules** (e.g., thiols such as glutathione; DNA, forming covalently-modified base adducts; and the Apolipoprotein B component of low-density lipoprotein, altering its biological characteristics.)”

**Pregnant women, beware:**

- “The administration of nonheated oil did not give rise to any changes in the rate of fetal malformations (the administration of either preheated or nonheated culinary oil did not lead to any modifications in the rate of reabsorptions, crown-rump length, or the somite number). These results demonstrate that the

administration of aldehyde-containing thermally stressed culinary oil is highly teratogenic [causing malformation of an embryo] in the rat, and *an attractive hypothesis is that the intake of such oxidized oils during pregnancy may be partially responsible for the neural tube defects found in humans. Differences in the type of heated oil used in standard frying or cooking processes may also be responsible for the differing rates of neural tube defects found among different populations.*"

It was known decades ago (1993) that oxidized oils, from food processing, caused thrombosis. Although quantitative analysis has greatly improved over the past 20 years, many researchers understood its importance back then:<sup>3</sup>

**"This review will examine the possible relevance of whole oxidized oils in food products to the process of atherogenesis.** For excellent in-depth reviews of the many prevailing issues in the area of **lipid oxidation, its products, and chronic diseases,** the reader is referred to a number of recent papers [18 papers are referenced in this 1993 article].

**"Lipid hydroperoxides have been shown to accelerate the atherosclerotic process** in terms of initiation of **endothelial injury,** the **progression phase** in which there is **accumulation of plaque,** and the **final termination phase** of thrombosis.

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3 Kubow, Stan, "Lipid oxidation products in food and atherogenesis," *Nutrition Reviews*, Feb 1993;51,2, pages 33-40.

**“It is of interest that **macrophages take up LDL very slowly** and *do not change to foam cells unless the LDL has been modified or oxidized.***

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**PEO Solution** analysis: CVD-damage caused by food processing has been known about for decades yet nearly nothing was done. Even today, its deleterious impact is not fully understood. Macrophages do *not* become foam cells unless there is oxidized cholesterol. You have already seen that its source is adulterated of Parent omega-6 form food processing.

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### **Adulterated, Non-functional PEOs Must Be Replaced with Functional PEOs**

Dr. David Horrobin was the world’s leading authority on Parent omega-6 and its derivatives. Horrobin’s superb article detailing EFA metabolic pathways states:<sup>4</sup>

**“...Thus high intakes of non-EFAs may lead to an increased requirement for EFAs. The *trans* and positional non-EFA isomers of EFAs may be particularly important in this context.** Such isomers can be formed in the rumen of cattle and so are found in small amounts in dairy products. However, *far greater quantities* of these isomers enter the human food chain as a result of *processing of vegetable oils*. To be effective as EFAs, all the double bonds of its molecule must be

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4 Horrobin, D.F., “Nutritional and medical importance of gamma-linoleic acid,” *Prog. Lipid Res.*, Vol. 31, No. 2: 163-194, 1992.

in the *cis*-configuration [*unadulterated*, fully functional PEOs]. However, such *cis* double bonds are relatively unstable and may lead to a reduced food shelf life or have other properties, which *make them inappropriate for inclusion in processed foods*.

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- ▶ “As a result surprisingly large amounts of linoleic acid [**Parent omega-6**] are *modified* to improve stability and handling characteristics. **Total daily intake of these modified linoleic acids [Parent omega-6], which have no EFA activity, is high, of the order of 6-12 g/day in Western countries. These modified *trans* and other derivatives are not only devoid of EFA activity themselves, they also compete with normal EFAs and interfere with their actions and metabolism.**”
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**An adulterated PEO is NO longer a PEO!**

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**PEO Solution** analysis: Dr. Horrobin hits the nail on the head. Recall the damage caused by just 0.5 gram of *processed* PEOs. Adulteration by food processors causing non-functionality of Parent omega-6 (LA) is at the core of many of our health epidemics. **This is why regardless of other interventions; the patient’s consumption of adulterated Parent omega-6 MUST be solved first in order to increase the outcomes of other protocols.**

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## **Root Cause of Defective Cholesterol and Membrane Alternation: Oxidation of Parent Omega-6**

“Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity:”<sup>5</sup>

“The effect of dietary **oxidized oil** on the lipid **composition, fluidity** and **function** of rat liver microsomes was studied. Male growing rats were fed diets containing 10 g/100 g of a fresh (control) **or oxidized** (experimental) linoleic acid [**Parent omega-6**]-rich preparation for 4 wk.

“...This [increased membrane fluidity] was due to **profound differences in lipid composition of the liver** microsomes, namely, a lower cholesterol to phospholipid molar ratio and a greater arachidonic acid content in the phospholipids of the rats **fed the experimental [oxidized] diet**.

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► *“The study demonstrated that ingestion of oxidized lipids caused profound alterations in membrane composition, fluidity and function...”*

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“The main differences observed in the composition of the microsomal **phospholipid** fatty acyl residues between the two groups were a **23.2% lower linoleic acid [Parent omega-6] level** and a 14.2% greater arachidonic acid level in the rats fed the oxidized linoleic acid diet.

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5 Hochgraf, Edna, et al., “Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity,” *J. Nutr.*127: 681–686, 1997.

**PEO Solution** analysis: It's all confirmed. **Adulterated/processed Parent omega-6 (from food processing) causes profound physiologic problems for patients.** The adulterated Parent omega-6 takes the place of the fully functional Parent omega-6 in phospholipids and we have a physiologic disaster. We will detail its cholesterol-connection in a subsequent chapter.

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## **Cancer and Mitochondria Defects From Parent Omega-6 Deficiency**

In April, 2009, I presented a paper at the 17th Annual World Congress on Anti-Aging Medicine in Orlando, Florida (April 2009), which utilized new research from 2007–2009 to identify the prime cause of cancer. Predictably, these findings caused quite a sensation. Numerous physicians met with me afterwards to applaud the presentation and discuss direct patient application in their practices. This article, “Cancer and Mitochondria Defects: New 21st Century Research,” was published in the *Townsend Letter for Physicians* (August/Sept. 2009), pp. 87–90), and is linked here: [www.brianpeskin.com/BP.com/publications/CancerMito-Town8.09.pdf](http://www.brianpeskin.com/BP.com/publications/CancerMito-Town8.09.pdf).

## **Metabolism and Longevity**

Physicians, regardless of specialty, can benefit from patients' increasing demands for anti-aging solutions. “Anti-Aging” Medicine has become the largest growing segment of medicine. As you have already discovered, **PEO Solution** offers unprecedented patient solutions for this new market segment.

Scientist Dr. A.J. Hulbert details the connection between PEOs and cell membranes in his paper, “Metabolism and

Longevity: Is There a Role for Membrane Fatty Acids?"<sup>6</sup> He discusses the damage to the cell membranes caused by lipid peroxidation. This is the process whereby lipids in cell membranes are degraded by peroxidation – free radicals grab electrons from the lipids, producing reactive molecules that become involved in a damaging chain reaction that weakens the cell membrane. **Of particular concern is the mitochondrial membrane, which helps to determine longevity.** It is mostly polyunsaturated fatty acids that are affected.

- “Although unknown in Rubner’s time [Rubner was a scientist studying metabolic rate and correlated longevity], one aspect of body composition of mammals also varies with body size, namely the *fatty acid composition of membranes*. Fatty acids **vary dramatically** in their **susceptibility to peroxidation** and the products of lipid peroxidation are very powerful reactive molecules that damage other cellular molecules. *It is apparent that membrane composition is regulated for each species.* The exceptional longevity of Homo sapiens combined with the limited knowledge of the fatty acid composition of human tissues support the potential *importance of mitochondrial membranes in determination of longevity.*
- “The insight that the exceptionally long-living species, Homo sapiens, potentially provides for understanding the mechanisms determining animal longevity, is that

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6 Hulbert, A.J., “Metabolism and Longevity: Is There a Role for Membrane Fatty Acids?” *Integrative and Comparative Biology*, Vol. 50, No. 5: 808–817.

*the fatty acid composition of mitochondrial membranes may be much more important than the composition of other cellular membranes."*

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**PEO Solution** analysis: As Dr. Hulbert makes clear, membrane lipid composition is specific to each species. Diet typically has a minimal effect on its composition *EXCEPT when unnaturally overdosed* (as with flax oil alone, fish oil, or *adulterated* oils). This excess of non-functional oil has to get incorporated *improperly* into tissue—it all can't simply be "burned up" for energy. **Physicians specializing in anti-aging know about the importance of mitochondrial function.** Dr. Hulbert emphasizes the importance of structural integrity and that requires plenty of *fully functional, unadulterated* Parent omega-6. I have written about mitochondrial composition—in particular, its dependence on fully functional Parent omega-6.

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Continuing with this amazing article, Dr. Hulbert explains what makes cell membranes so susceptible to adulteration by peroxidation:

- "In naturally occurring polyunsaturates [including PEOs], the  $-C=C-$  units [these are the double-bonded carbon units] are all separated by a single-bonded  $-C-$  [carbon] atom. The hydrogen atoms attached to each of these intermediate  $-C-$  atoms are called *bis-allylic* hydrogens, and have the lowest C-H [weakest] bond-energies of the fatty acid chain. This [weak bond] makes them the *most susceptible to attack by Reactive Oxygen Species (ROS)* [chemically reactive molecules which contain oxygen] produced during aerobic metabolism."<sup>7</sup>

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7 Ibid.

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***Bis-allylic* (weak-bonded) hydrogens in the cellular membrane are the most susceptible to attack.**

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Although the following was covered in the chapter it is worth repeating. Dr. Hulbert's explanation immediately exposes the dangers of fish oil:

- “**Docosahexaenoic acid** (22:6), which has six double bonds and consequently *five bis-allylic hydrogens per chain*, is **320 times more susceptible to [anti-oxidant] attack** than the common **monounsaturated oleic acid** (18:1) which has “no” *bis-allylic* hydrogens in its chain.”

... and warns of additional DNA and protein DAMAGE:

- “Membrane lipid peroxidation should not be perceived solely as a ‘damage to membranes’ scenario but also as a significant *endogenous source of damage to other cellular macromolecules*, such as proteins and DNA (including mutations).”

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**PEO Solution analysis: Aside from PhDs in chemistry, the medical profession is not used to this biochemical term or the superior *Peroxide Index (PI)*.** The more polyunsaturated the oil is, the more *bis-allylic* chains are present. Fish oil's DHA (22:6) has six of them and therefore, is enormously more reactive than the monounsaturated oleic (olive oil), which contains none, and seven times more reactive than Parent omega-6. To combat this oxidation, you body is

forced to “use up” its reserve of anti-oxidants, leaving other areas vulnerable.

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Dr. Hulbert also explains the chain reaction that occurs with the attack on polyunsaturated fatty acids:

- “Thus, **contrary to a common misconception**, it is “not” just the presence of double-bonds in a mixture of fatty acids [(often quantified as the “double-bond index” (DBI) or “unsaturation index” (UI)] that will determine the susceptibility of this mixture to oxidative damage, **but rather the precise types of fatty acids present in the mixture and their relative abundance....** In this way [of causing lipid membrane peroxidation], **ROS attack on a membrane** lipid bilayer (that contains polyunsaturated fatty acids) **differs from ROS attack on other cellular molecules** such as proteins, carbohydrates and nucleic acids. Whereas ROS attack on these other types of molecules will damage the molecule and likely stop them from performing their function, **ROS attack on membrane polyunsaturates will damage the molecule** (by converting it to a lipid hydroperoxide) and will **also produce another reactive molecule** that will in turn continue the oxidative damage to other molecules.... The products of lipid peroxidation, such as lipid hydroperoxides, can also undergo fragmentation to produce a broad range of reactive intermediates (called propagators) that can modify proteins and DNA to produce “advanced lipoxidation endproducts” (ALEs)...”

- ▶ “Membrane lipid peroxidation should not be perceived solely as a “damage to membranes” scenario but also as a significant *endogenous source of damage to other cellular macromolecules*, such as proteins and DNA (including mutations).”
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**PEO Solution** analysis: As can be seen from Dr. Hulbert’s work, the damage to cell lipid membranes is horrific... to the membranes themselves and far beyond ... to proteins and critical DNA, too. With Dr. Hulbert’s magnificent work, we can now more fully appreciate how PEO adulteration—whatever its cause—is the *primary* issue behind virtually all patient health issues.

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### **Life Span Based on Metabolic Rate and Membrane Composition**

Dr. Hulbert has much more to teach us with his incredible treatise entitled “Life and Death: Metabolic Rate, Membrane Composition, and Life Span of Animals.”<sup>8</sup> This paper is required reading if you really want to understand aging, free radicals, and tissue membranes. Some highlights (not necessarily in publication order) are:

- “*Mitochondrial cardiolipin* molecules are possible targets of oxygen free radical attack, due to their high content of polyunsaturated fatty acids and because of their location in the inner mitochondrial membrane near

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8 Hulbert, A.J., et al., Life and Death: Metabolic Rate, Membrane Composition, and Life Span of Animals,” *Physiological Reviews*, Vol. 87, October 2007, pages 1175–1213.

the site of ROS [Reactive Oxygen Species] production. In this regard, it has been recently demonstrated that mitochondrial-mediated ROS generation affects the activity of complex I, as well as complexes III and IV, via peroxidation of cardiolipin following oxyradical attack to its fatty acid constituents. These findings might *explain the decline in respiratory chain complexes observed in mitochondria isolated from aged animals and in pathophysiological conditions* that are characterized by an increase in the basal rate of the ROS production.”

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**PEO Solution analysis: The importance of cardiolipin** is once again emphasized. Keep the supply of fully functional PEOs, in particular Parent omega-6, high and this potential problem is significantly reduced.

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The treatise continues:

- “It is suggested that rapid constitutive recycling of membrane phospholipids **rather than selective in situ repair** is responsible for eliminating peroxidized phospholipids, with triacylglycerols **providing a dynamic pool of undamaged PUFA** (PEOs) for phospholipid resynthesis.”
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**PEO Solution analysis:** The body can either *attempt* to repair damaged PEOs or it can **simply replace the damaged PEOs with fully functional PEOs—IF they are available.** *In situ* repair is nearly impossible. **We all require daily ingestion of fully functional PEOs.**

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Continuing ...

- “The susceptibility of membrane lipids to oxidative damage is related to two traits. **The first is that oxygen and many radical species are several times more soluble in lipid membrane bilayers than in the aqueous solution.** The second property is related to the fact that not all fatty acid chains are equally susceptible to damage. **It is this second property that is the key to the link between membrane composition and oxidative damage to membranes.** The carbon atoms that are most susceptible to radical attack are the single-bonded carbons between the double-bonded carbons of the acyl chains.

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► “Furthermore, the *noncharged structure of aldehydes* allows them to migrate with relative ease through hydrophobic membranes and hydrophilic cytosolic media, thereby *extending the migration distance far from the production site*. On the basis of these features alone, *these carbonyl compounds can be more destructive than free radicals and may have far-reaching damaging effects on target sites both within and outside membranes.*

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- “...When such C• radicals are generated in the hydrophobic interior of membranes, a likely fate is combination with oxygen dissolved in the membrane. The resulting peroxy radical is **highly reactive: it can attack membrane proteins and can also oxidize adjacent PUFA [PEO] chains.** Thus the *initial reaction is repeated* and a free radical *chain reaction is propagated*.... [T]hese

can thus *disrupt the membrane* structure, altering fluidity and other functional properties of membranes....”

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- ▶ “...Thus *lipid peroxidation should not be perceived solely in a “damage to lipids” scenario, but should also be considered as a significant endogenous source of damage to other cellular macromolecules*, such as proteins and DNA (including mutations).
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- “In this way, variation in *membrane fatty acid composition*, by influencing lipid peroxidation, can have *significant effects on oxidative damage to many and varied cellular macromolecules*. For example, peroxidized cardiolipin in the mitochondrial membrane can inactivate cytochrome oxidase by mechanisms both similar to hydrogen peroxide and also mechanisms unique to organic hydroperoxides...”
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**PEO Solution** analysis: We can’t do anything about the increased solubility of oxygen in cellular membrane ® ROS. This is a physiologic fact. We can see that the aldehydes cause grave damage to lipids, proteins, DNA, and other biological entities—even far removed from their source! **The second property is a different story. It is important to note that the (compositional) material of the membrane DOES make a difference; oxygen diffusion in the cell membrane (at physiologic conditions) is approximately twice as fast as water!**<sup>9</sup>

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<sup>9</sup> Subczynski, W.K., et al., “Is the mammalian cell plasma membrane a barrier to oxygen transport?,” *Journal of General Physiology*, Vol. 100,

## SOLUTION...

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**Make sure the membrane is not unnaturally altered. This is accomplished in two distinct ways: First, make sure the body has sufficient PEOs. Second, make sure there is no overdosing of either the omega-6 or omega-3 series fatty acids, e.g. fish oil or flax oil without appropriate systemic PEO and their associated series derivative compensation. Balance is required and most importantly, we need to understand what “balance” means.**

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### **Unsaturation Index (UI) and Peroxidation Index (PI)**

Continuing with Dr. Hulbert’s article, “Life and Death: Metabolic Rate, Membrane Composition, and Life Span of Animals,” ...

- “The peroxidation index [PI] of a membrane is not the same as its unsaturation index [UI] (sometimes also called its “double-bond index”), which is a measure of the density of double bonds in the membrane.”

The respective measurements follow. It is important to note that the PI is based on actually measuring the *real-life* increase in peroxidation of the series of double-bonded lipids. *PI index is much more instructive in measuring relative PEO and PEO-derivative peroxide potentials.*

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1992: 69–87.

**Peroxidation index (PI):**  $0.025 \times (\% \text{ monoenoics}) + 1 \times (\% \text{ dienoics}) + 2 \times (\% \text{ trienoics}) + 4 \times (\% \text{ tetraenoics}) + 6 \times (\% \text{ pentaenoics}) + 8 \times (\% \text{ hexaenoics})$ .

**Unsaturation index** =  $1 \times (\% \text{ monoenoics}) + 2 \times (\% \text{ dienoics}) + 3 \times (\% \text{ trienoics}) + 4 \times (\% \text{ tetraenoics}) + 5 \times (\% \text{ pentaenoics}) + 6 \times (\% \text{ hexaenoics})$ .

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<b>Omega Chain Length</b>	<b>PI</b>
Oleic (olive Oil)	2.5
Parent omega-6	100
Parent omega-3	200
DHA	800

Parent omega-3 is *twice* as reactive as Parent omega-6 and DHA has an astronomical *8-fold* increase in reactivity compared to Parent omega-6. As will be detailed later, the body has sufficient antioxidant capability to protect normal, physiologic levels of polyunsaturated fatty acids throughout the body. As the world-leading biochemist Professor Gerhard Spiteller, Chairholder of Biochemistry, Institute of Organic Chemistry at the University of Bayreuth, Germany, makes clear, these are NOT an issue—Nature is consistent. However, it does NOT

have capacity to combat artificial OVERDOSES based on wrong nutritional advice.

**Tragically, we are (unknowingly)  
our own worst enemy!**

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It is only when we artificially introduce supra-physiologic amounts of improper lipids that the excessive cellular destruction and ROS oxidation problem exists. ***Despite the best of intentions, we cause ourselves grave harm.***

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**PEO Solution** analysis: This information goes a long way toward independent proof and verification of my thesis that we are ***ingesting pre-oxidized*** PEOs—in particular, adulterated Parent omega-6—and it is too late for an “antioxidant” to work, regardless of quantity because the oxidation damage is already done! When esterified cholesterol is analyzed, it will be “case closed.”

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**An example of the difference between Unsaturation Index (UI) and Peroxidation Index (PI):**

**The peroxidation index of a membrane is not the same as its unsaturation index—“double-bond index”—a measure of the density of double bonds in the membrane. For example, a membrane bilayer consisting solely of MUFA [monounsaturates like in the majority of olive oil] will have an **unsaturation index of 100 and a peroxidation index of 2.5**, while a membrane bilayer consisting of 95% SFA (saturated fat) and 5% DHA (with**

6 double-bonds) will have an **unsaturation index of 30 and a peroxidation index of 40.**

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**The 5% DHA-containing membrane is 16 times (40 / 2.5) more susceptible to peroxidative damage—a most surprising result!**

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The potential damage to all cell membranes, including mitochondrial membranes is immense.

### **With Fish Oil, EPA Rises, Linoleic Acid Decreases—Real-Life Results**

The superb article by Martijn B. Katan, et al., “Kinetics of the Incorporation of Dietary Fatty Acids Into Serum Cholesteryl Esters, Erythrocyte Membranes, and Adipose Tissue: An 18-month Controlled Study,” (*Journal of Lipid Research*, Vol. 38, 1997, pages 2012–2022) has this to say:

- “Tissue levels of n-3 fatty acids [fish oil] reflect dietary intake, but quantitative data about rate of **incorporation and levels as a function of intake** are scarce. We fed **58 men** 0, 3, 6, or 9g/d[ay] of **fish oil for 12 months** and monitored fatty acids in serum cholesteryl esters, erythrocytes, and subcutaneous fat during and after supplementation. Steady-state levels **increased by 3.9 ± 0.3 mass % points (± SE) for each extra gram of EPA eaten per day.** Incorporation of docosahexaenoic acid (DHA) was erratic; plateau values were **1.1 ± 0.1 mass % higher for every gram per day ingested.**

- ▶ “The rise **in EPA** [a significant component of fish oil] was compensated for largely by **decreases** in linoleic acid [**Parent omega-6**].”
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**PEO Solution** analysis: This experiment shows how the overdose of fish oil increases cholesterol esters—the body’s most significant fatty acid transport system. There was a corresponding increase per day of the erythrocytes, too. Natural triglyceride form (not synthetic esters) was used. Trappist monks were the population sample. **We see the fish oil overdose displacing critical Parent omega-6**—destroying critical mitochondrial functionality, etc. Given fish oil’s incredible high rate of oxidation, even a “little too much incorporation” (3.9% extra with a corresponding 3.9% less Parent omega-6) **causes horrific damage!**

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### **Oxidized Lipids Alter Membrane Integrity**

Ingestion of *adulterated* Parent omega-6 causes grave physiologic issues as demonstrated in the journal article, “Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity:”<sup>10</sup>

- “...This [increased membrane fluidity] was due to profound differences in lipid composition of the liver...
- “The study *demonstrated that ingestion of oxidized lipids caused profound alterations in membrane composition, fluidity and function...*”

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10 Hochgraf, Edna, et al., “Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity,” *J. Nutr.*127: 681-686, 1997.

- ▶ “The main differences observed in the composition of the microsomal **phospholipid** fatty acyl residues between the two groups were a **23.2% lower linoleic acid [Parent omega-6] level** and a 14.2% greater arachidonic acid level in the rats fed the *oxidized linoleic acid* [Parent omega-6] diet.”
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**PEO Solution** analysis: It is all confirmed. The *adulterated* Parent omega-6 *replaces* the fully functional Parent omega-6 in phospholipids and we have a physiologic disaster.

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**Both adulterated Parent omega-6 (from food processing) and overdoses of fish oil (EPA/DHA) cause profound physiologic problems for patients.**

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### **You Need to Know This... Markers of Lipid Oxidation—Malondialdehyde is tops...**

Malondialdehyde is an *in vivo* marker of oxidative stress (lipid peroxidation/rancidity). Reactive oxygen species degrade polyunsaturated lipids, forming malondialdehyde (MDA). This results in advanced lipoxidation end-products (ALE), analogous to advanced glycation end-products (AGE), so harmful to diabetics.

There is much to know before relying on specific lipid oxidation markers. The excellent medical journal article, “Lipid Peroxidation and the Thiobarbituric [TBA] Acid Assay:

Standardization of the Assay When Using Saturated and Unsaturated Fatty Acids," makes clear:<sup>11</sup>

- "Indeed supplementation with *polyunsaturated* [in particular, EPA/DHA], as opposed to saturated fatty acids results in a **statistically significant increase in lipid peroxidation in the plasma and liver.**
- "**Oxidative damage to DNA** in bone marrow was recorded in aged, but not young rats when a **polyunsaturated** diet was employed.
- "A plateau was reached where no additional decrease in the TBA-reactive species (TBARS) was observed regardless of increases in fatty acid concentration. This limitation to the TBA assay suggests the need for a standardized assay....

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▶ "The TBA Assay is not specific for *malondialdehyde* (MDA), one of the many breakdown products of degraded fatty acids. [The test is separately required.]

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- "**Caution is advocated for the interpretation** of in vitro [outside the body] lipid peroxidation *using only the TBA assay* without standardization..."

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11 Rael, Leonard, T., et al., "Lipid Peroxidation and the Thiobarbituric Acid Assay: Standardization of the Assay When Using Saturated and Unsaturated Fatty Acids," *Journal of Biochemistry and Molecular Biology*, Vol. 37, No. 6, November 2004, pages 749–752.

**PEO Solution** analysis: Be wary of those telling you there is “no issue” with EPA/DHA oxidizing in the body. Peroxidation level based on TBA will stop (reach an upper limit) prematurely before recording full damage—an inherent limitation of the particular test. Red blood cell oxidation levels are not a sufficient indicator—the actual tissue is what counts. Furthermore, researchers often don’t understand the limitations of measurement. Short-term damage may not present but long-term damage will, i.e., DNA destruction.

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*Malondialdehyde* measurement should be performed independently for increased accuracy of the damage assessment. Organ damage does occur, as shown decisively in the monkey experiment (chapter 7). It is “case closed.” *Lipids*, one of the world’s top medical journals in the field, makes clear how fish oil raises MDA. The article, “Malondialdehyde excretion by subjects consuming cod liver oil vs a concentrate of n-3 fatty acids,” states:<sup>12</sup>

- “Urinary malondialdehyde (MDA), an indicator of lipid peroxidation in the diet and in the tissues, was *determined in human adults* consuming a supplement of n-3 fatty acids derived from a pharmaceutical grade of **cod liver oil** (CLO) without added antioxidants vs a concentrate of n-3 acids containing dodecyl gallate and vitamin E. **MDA excretion increased immediately in the subjects consuming CLO but remained unchanged in those ingesting the concentrate for 50 days. The increase in the subjects taking CLO** was attributable to MDA in the oil. The results indicate that consuming

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12 Piche, LA, et al., *Lipids*, Volume 23, Number 4, 1988, pages 370–271

unstabilized fish oils as a source of n-3 fatty acids may entail exposure to **potentially toxic products of lipid peroxidation.**

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**PEO Solution** analysis: No amount of lipid “antioxidant/stabilization” outside of the body (in the container) can protect the supra-physiologic amounts of EPA/DHA from rancidity inside the body (*in vivo*). This was conclusively shown in the peroxidized monkey livers.

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The following article gave me great pause – “Margarines fortified with  $\mu$ -linolenic acid, eicosapentaenoic acid [EPA], or docosahexaenoic acid [DHA] alter the fatty acid composition of the erythrocytes but do not affect the antioxidant status of healthy adults”<sup>13</sup> – first because the study was conducted back in **2004!** Why is this just being published now, eight years later in **2012?** The study of 48 women and 26 men doesn’t answer this. Their published results don’t even make sense. It is well-known EPA/DHA at room temperature spontaneously turn rancid. Therefore, the problem is much worse at body temperature close to 100° F. They gave huge doses of ALA (parent omega-3) (4.4 grams/day), EPA (2.2 grams/day), and DHA (2.3 grams/day). Not surprisingly, RBC incorporation of these overdoses was significant: ALA incorporation increased by 104%, EPA incorporation increased by 394%, and DHA incorporation by 91% from normal. To support these increases, natural Parent omega-6 and AA (an important anti-CVD omega-6 derivative) significantly decreased.

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13 Egert, Sarah, et al., *Journal of Nutrition*, 142: 1638-1644, 2012.

Furthermore, it appears from their description that the women in the study were all taking birth control pills.

Participants were given (Unilever) **margarine, loaded with adulterated Parent omega-6**. If it wasn't unprocessed/organic, then it was necessarily adulterated; there was no exclusion by the researchers.

Researchers measured red blood fatty acids levels and plasma levels. They did not measure tissue/organ peroxidation/lipofuscin levels. *The study ran just 6 weeks, much too short to establish long-term damage*, as the journal article above describing long-term DNA damage with fish oil makes clear.

The article leads with the familiar "we don't know how it works but it does" disclaimer:

"The exact mechanisms through which (n-3) PUFA influence CVD are not well established...." [Note: "magical" metabolic pathways are not admissible.]

**"The plasma concentrations of lipid peroxidation product MDA significantly increased with the EPA and DHA intervention** compared with the ALA [parent omega-3] intervention. [Note: This is to be expected based on known physiology.]

## “Malondialdehyde Excretion by Subjects Consuming Cod Liver Oil vs a Concentrate of n-3 Fatty Acids

The journal article, “Malondialdehyde Excretion by Subjects Consuming Cod Liver Oil vs a Concentrate of n-3 Fatty Acids,” found the following:<sup>14</sup>

- “In Experiment 1, ingestion of CLO [cod liver oil] was associated with an increase in **MDA excretion in all six subjects. The mean increase of 37.5%**, from  $24.5 \pm 3.5$  ug to  $34.7 \pm 2.5$  ug MDA (mean + SEM), was significant ( $P < 0.01$ ) using a two-tailed paired t-test. [Note: This is “highly” significant.]
- “In Experiment 2, CLO ingestion again was associated with an increase in **MDA excretion in all subjects. The mean increase of 54.3%**, from  $31.7 \mu\text{g}$  to  $49.1 \mu\text{g}$  MDA/sample was highly significant ( $P < 0.001$ ).

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**PEO Solution** analysis: Of course, there is verification of lipid oxidation, because there has to be evidence. The researchers TRIED to say when using “stabilized” fish oil in another group of 6 patients, the oxidation as measured by urinary MDA was minimized. However, using Stat-Smart® analysis, we immediately found that **the result was NOT statistically significant**—there was more than a 5% error rate! They put this “little detail” in a footnote, but we won’t be fooled.

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Holman confirmed in 1954 that fish oil oxidizes effortlessly:

- “Docosahexaenoic acid (22:6), which has six double bonds and consequently five bis-allylic hydrogens per

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14 Piche, LA, et al., *Lipids* 23, 370–371 (1988)

chain, is 320-times more susceptible to ROS attack than the common monounsaturated oleic acid (18:1) which has 'no' bis-allylic hydrogens in its chain."<sup>15</sup>

### **Oils are Damaged when Heated for Cooking**

When assessing oxidative damage to lipids, at least two different chemical methods should be utilized. The investigator must understand each test's uses and limitations. For example, hydroperoxides do not accumulate at frying temperatures; instead, they decompose spontaneously, but their damage is significant.

Adulteration of oils both contained in the food and used in its preparation causes damage to the food; in the case of frying, the food also absorbs oil – potentially creating additional harm to the consumer of the food.

Dr. Dobargenes' warning of the oil in fry vats exceeding the limit for polar compounds is warranted (2003):<sup>16</sup>

- "Industrial continuous fryers had polar compounds ranging from 4.2% - **27.3%** and batch fryers found in **fast food fryers and restaurants** had 3.1 - **61.4%**!
- "**Restaurant and fast-food outlets oil quality data are in many cases short of complying** with the recommendations outlined at a recent international conference held in Germany (2000) where the maximal

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15 Holman, RT, "Autoxidation of fats and related substances," *Progress in Chemistry of Fats and Other Lipids*, edited by Holman RT, Lundberg WO, Malkin T. London: Pergamon, 1954, vol., 2, p. 51-98.

16 Saguy, I. S. and Dana, D., "Integrated approach to deep fat frying: engineering, nutrition, health and consumer aspects," *Journal of Food Engineering*, Vol. 56, 2003, pages 143-152.

concentrations of total polar compounds and polymer content were 24% and 12%, respectively. Data on oil quality in batch fryers in some European countries stress the fact that this issue is far from acceptable.

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- ▶ A recent study conducted in Europe indicated that even when TFA concentration is less than 1% in non-hydrogenated oil, its level in frying oils **could reach up to 50%** due to partially hydrogenated oil. Only Denmark has regulation on maximum allowed TFA (15%) in unused frying oil.
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- “Repeat use of frying oils may increase TFA concentration due to the exchange of fatty acids between the fried food and the oil as well as the high temperature and prolonged frying process. **For instance, an increased concentration of trans isomers of C18:1 [as in olive oil] was found in various vegetable oils used for beef frying.** Repeated **frying in sunflower** oil also resulted in an increased concentration of the TFA isomer C18:2.
- “A positive correlation was found with **thiobarbituric acid** reactive substances (TBARS), formed *from fatty acids with three or more double bonds*. This fraction contains MDA, which was **found to be mutagenic in many other studies. Prolonged frying caused a substantial rise in MDA concentration.** These findings need further study, as MDA is not typically monitored during frying.

- ▶ **“MDA was found to cause skin cancer in rats and created cross-linking with amino groups of DNA solution.** The Ames test indicated that **MDA is a mutagen causing DNA alterations** and reacting mainly with guanine and cytidine by a depletion of this base pair. **MDA can damage proteins and phospholipids by covalent bonding and cross-linking.** *Rats fed a diet containing MDA suffered from retarded growth, irregular intestinal activities, enlarged liver and kidneys, anaemia and low serum and liver vitamin E.*
- 

- **“Since a considerable amount of oil is absorbed in the product during frying (10-40%), frying in oil that contains mutagens could lead to consumption of foods containing hazardous compounds.** Usually the concentration of mutagens in the oil is low, but **high oil uptake may have health implications.** It is also possible that higher concentration could be absorbed in the food due to preferential uptake. It is worth noting that the concentrations used in the aforementioned experiments on rats are twice as high as the average human consumption of MDA.
- **“Low mutagenesis was found in oils exposed to severe frying conditions--uncommon in typical industry or household operations,** and no mutagenic activity was found in oil used for repeated frying of potatoes, onion rings or fish fillets. Thus it can be concluded that under controlled conditions, the level of exposure to mutagenic

compounds formed during frying should not comprise a real health hazard. Nevertheless, this topic needs further study before a final conclusion can be made.”

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**PEO Solution** analysis: Don't be confident that French fries or other fried foods have low or no mutagenic activity as reported here. That comment makes little sense and is inconsistent with the established science and inconsistent with numerous other experiments (as the researchers were forced to admit.).

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It's confirmed that consuming oxidized cholesterol-containing foods leads to oxidized cholesterol in plasma:<sup>17</sup>

- “In studies in humans, we have shown that the **quantity of oxidized fatty acids in the diet also correlates with the levels of oxidized lipids in postprandial serum chylomicrons / chylomicron remnants (CM/RM)**. Oxidized fatty acids in the diet are absorbed by the small intestine, incorporated into chylomicrons and chylomicron remnants, and appear in the bloodstream where they **contribute to the total body pool of oxidized lipid**.
- “In our initial studies, we determined the effect of different  $\alpha$ -epoxy cholesterol quantities in the test meal on  $\alpha$ -epoxy cholesterol levels in postprandial serum. **We found that the serum levels of  $\alpha$ -epoxy cholesterol**

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17 Staprans, I., et al., “Oxidized cholesterol in the diet is a source of oxidized lipoproteins in human serum,” *Journal of Lipid Research*, Vol. 44, 2003, pages 705-715.

**strongly correlated.** [Note: It was nearly a perfect correlation of  $r=1$ .]

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- **“Thus, our data clearly show that oxidized cholesterol, when ingested, is incorporated into CM/RM fraction and is transferred within the plasma compartment from exogenous to endogenous lipoproteins, and this transfer accounts at least partially for the presence of oxidized cholesterol in LDL and HDL in the circulation. It has been previously demonstrated in rabbits and in rodents that dietary oxidized cholesterol is absorbed and enters the circulation via CM/RM particles. As expected, in the present study we found that the absorption of dietary oxidized cholesterol in humans is similar to that observed in other species.”**
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**PEO Solution** analysis: Just as with EFAs, absorption and integration of oxidized cholesterol is the same in humans as in other species.

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Broiling buffalo meat increased oxidative rancidity by approximately 3-fold.<sup>18</sup>

- **“Oxidative rancidity of lipids is a serious problem during storage of meat and meat products and *TBA (2-thiobarbituric acid) value is the most commonly used parameter to measure it.***

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18 Rao, V.R., et al., “Effect of Cooking and Storage on Lipid Oxidation and Development of Cholesterol Oxidation Products in Water Buffalo Meat,” *Meat Science*, Vol. 43, No. 2, 1996, pages 179–185.

- “There was a **significant increase of approximately 3-fold in TBA from broiling.**”

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**PEO Solution analysis:** Malonaldehyde is a highly reactive three carbon dialdehyde produced as a byproduct of polyunsaturated fatty acid peroxidation and arachidonic acid metabolism. TBA is used to measure it.

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### **Fish oil is problematic... Cooking is not a necessary condition...**

The **2000** article titled, “Supplementation of postmenopausal women with fish oil rich in eicosapentaenoic acid and docosahexaenoic acid is not associated with greater in vivo lipid peroxidation compared with oils rich in oleate and linoleate as assessed by plasma malondialdehyde and F2-isoprostanes,” has buried on page 7:<sup>19</sup>

- “Whether normalized to **plasma** volume or plasma PUFA concentration, *plasma TBARS were significantly higher after fish-oil supplementation* than after sunflower-oil or safflower-oil supplementation [Parent omega-6 oils].

- 
- ▶ “After **fish-oil supplementation**, plasma **TBARS were > 21% higher** than after sunflower-oil supplementation and **23% higher** than after safflower-oil supplementation. [Note: with non-organic sunflower and safflower oils, those oils are adulterated to begin with.]
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19 Higdon, Jane V., et al., *Am J Clin Nutr* 2000;72:714-22.

- “The fact that the **TBARS** concentrations in plasma were nearly *10 times higher* than the **MDA** concentrations is likely due to the **lack of specificity of the TBA assay for MDA** and to artifactual production of MDA during the acid heating step of the TBA assay.
- “Many of the **assays** available for the measurement of **lipid peroxidation in vivo** lose their utility when **specific PUFA concentrations** in plasma vary as a result of changes in dietary intake. Instead of measuring overall lipid peroxidation, different assays measure the oxidation or decomposition of specific PUFAs.
- “The utility of the **F2-isoprostane** assay for comparing in vivo lipid peroxidation at different intakes of specific unsaturated fatty acids is **limited** because it *does not provide direct information about the peroxidation of 20:5n-3 [EPA] and 22:6n-3 [DHA].*”

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**PEO Solution** analysis: The bottom line is that EPA/DHA is damaged. Consequently, patients should avoid this obvious risk.

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**Dr. Rowan:**

### **The Toxic Impact of Heat on Foods—Pottenger’s Cats...**

- Pottenger was studying the adrenal gland and cortisol using cats
- He was donated lots of unwanted cats.

## Scientific Support for Chapter 8

- Overflowing with cats, he resorted to different feeds.
- He noticed that certain groups were healthier than others, so he decided to do a study on nutrition in the cats

### **Pottenger's Study:**

- The study involved 900 cats
- 600 were studied for their entire life span.
- All cats were placed in identical physical enclosures.
- The variable was their diets

### **His Groups:**

1. Raw meat group: 2/3 raw meat, 1/3 raw milk, and cod liver oil
2. Cooked meat group: 2/3 cooked meat, 1/3 raw milk, and cod liver oil

.....

Raw meat group observed over **three** generations.

1. Maintenance of a regular, broad face with prominent malar (pertaining to the cheek or cheek bone) and orbital arches, adequate nasal cavities, broad dental arches, and regular dentition.
2. The configuration of the female skull is different from the male skull, and each sex maintains his/her distinct anatomical features.
3. The membranes are firm and of good, pink color with no evidence of infection or degenerative change. Tissue tone is excellent, and the fur is of good quality with very little shedding noted.

4. In the older cats, particularly the males, engaging in fighting, the incisors are often missing, but inflammation and disease of the gums is seldom seen.
5. The calcium and phosphorus content of their femurs remains consistent.
6. Their internal organs show full development and normal function.
7. Over their life spans, they prove resistant to infections, to fleas, and to various other parasites, and show no signs of allergies. In general, they are gregarious, friendly, and predictable in their behavior patterns..
8. These cats reproduce one homogeneous generation after another with the average weight of the kittens at birth being 119 grams.
9. Miscarriages are rare, and the litters average five kittens with the mother cat nursing her young without difficulty

Now the results in the cooked meat group over three generations:

1. This group reproduces a reproduce a **heterogeneous** strain of kittens, each kitten in a litter being different in size and skeletal pattern.
2. When comparing the changes in configuration found in their x-rays, there are almost as many variations in the facial and dental structures of the second and third generation cooked-meat fed animals as there are animals.
3. **Evidence of deficiencies is written so plainly** on their faces that with a little training, any observer can be almost certain that a given cat has been subjected to a deficient diet or that it comes from a line of cats that has suffered from deficient nutrition.

## Scientific Support for Chapter 8

4. The long bones of cooked-meat cats tend to increase in length and decrease in diameter with the hind legs commonly increasing in length over the forelegs. The trabeculation (the internal structural mesh of the bones) becomes coarser and shows evidence of less calcium.
5. In the third generation, some of the bones become as soft as rubber, and a true condition of osteogenesis imperfecta (the inherited condition in which bones are abnormally brittle and subject to fractures) is present.
6. Heart problems; nearsightedness and farsightedness;
7. Under activity of the thyroid or inflammation of the thyroid gland;
8. Infections: of the kidney, of the liver, of the testes, of the ovaries, and of the bladder
9. Arthritis and inflammation: of the joints; inflammation of the nervous system with paralysis and meningitis—all occur commonly in these animals. A decrease in visceral volume is evidenced by the diminishing size of their thoracic and abdominal cavities.
10. Microscopic sections of organs show poor histological development of organs.
11. Behavioral anomalies, females overly aggressive, males docile.
12. Sexual deviations in same sexed animals.
13. Reproductive organ pathology – ovarian, uterine and sperm production anomalies.
14. Abortion in females rampant running 25% in the first generation to as high as 70% in the second.

15. Pests and intestinal parasite abound.
16. Skin lesions, allergies abound.
17. Pneumonia, empyema (abscess within chest space) and diarrhea are common causes of death.
18. High perinatal mortality of both mothers and kittens.
19. Kittens of this group average 19 grams less than raw meat nurtured kittens.

**Pottenger went on to study if the cats could regenerate or recover their health and found:**

1. It requires approximately four generations for the deficient cats to regenerate to a state of normal health after healthy diet reinstated.
2. However, because of the lack of reproductive efficiency, very few deficient animals regain the normal health noted before deficiency was imposed on their line of cats.
3. Improvement in resistance to disease is noted in the second generation regenerating cat, but allergic manifestations persist into the third generation.
4. In the third generation, skeletal and soft tissue changes are still noticeable, but to a lesser degree; and by the fourth, most of the severe deficiency signs and symptoms disappear—but seldom completely.

**A startling discovery:**

1. A female cat is subjected to a deficient diet for a period of 12 to 18 months, her reproductive efficiency is so reduced that she is never again able to give birth to normal kittens.

## Scientific Support for Chapter 8

2. Even after three or four years of eating an optimum diet, her kittens still show signs of deficiency in skeletal and dental development. When her kittens are maintained on an optimum diet, a gradual reversal and regeneration takes place.

Pottenger Studied the effects of raw vs. pasteurized, vs. evaporated mild added to diet (2/3 of diet) with similar results.

His findings almost exactly mirror what we are seeing now in younger human populations. It also mirrors the horrific rise in diseases in our younger human generations, autism collapse in sperm counts, immune dysfunction, etc.

*Journal of the American Medical Association* published a startling discovery in 1981.<sup>20</sup> Rats made deficient in zinc gave birth to offspring with immune defects. Even when zinc was restored to their diet, it took **four** generations for them to regain normal immune function! I first learned about this about 9 years later and it shook me regarding nutrition!

This exactly mirrors modern experiments on epigenetics, using nutritional deficiencies and environmental xenobiotics. Epigenetics is the science of DNA or gene expression. Your DNA might be perfectly intact. The problem is, dietary deficiencies, toxins, or even stress might compromise its expression. See, your DNA is like your computer's hard drive. You control what the hard drive does through your keyboard commands. Imagine missing a key (nutritional deficiency) or a heavy brick (toxin) loading one or more keys. Your hard drive, perfectly intact otherwise, is not going to perform!

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20 *JAMA*. 1981;245(1):53-58.

We are finding that nutritional or toxic damage to animals induces alteration in DNA EXPRESSION, not the genetic sequence. This has profound implications for the offspring and it takes about 4 generations to recover.

There is a Biblical passage about this: ‘punishing the children for the sin of the fathers to the third and fourth generation of those who hate me’ (Exodus 20:5). Seems the Bible (I am NOT a Bible thumper) accurately predicted epigenetic effects and their lasting effects over 3,000 years ago!

**We are in the throes of:**

1. Autism epidemics
2. Immune defects epidemics
3. Cancer epidemics
4. Allergy epidemics
5. Suicide and violent behavior epidemics.
6. Chronic disease epidemics.
7. Infertility epidemics.

COULD OUR DIET (or toxins) BE A KEY FACTOR? ***Could heated foods be a major culprit?***

Let’s turn to modern science.

IA recent article<sup>21</sup> on thermolyzed (heated) foods took up the subject. The authors even outlined the reasons for the study:

1. Diets are usually evaluated for calories, macro and micronutrients.
2. **Rarely are the effects of heat considered.**
3. Thermolysis of foods, however, can result in the formation of new products such as advanced glycation end-products (AGE).
4. A heterogeneous group of compounds that are formed by a complex series of parallel and sequential reactions called the **Maillard reactions**. (Nonenzymatic browning resulting from a chemical reaction between an amino acid and a reducing sugar from heat).

Maillard reactions are chemical alterations to molecules in food. They impart the unique taste and smell of cooked foods. However, they create compounds not found in nature, such as acrylamides, now known to be carcinogens. You'll find these reactions and molecules in the browning of various meats like steak, toasted bread, biscuits, malted barley or spirits. Also, in fried onions or any fried food, dried or condensed milk, roasted coffee, and the burnished surface/crust of many heated foods.

**Maillard reactions are accelerated by the following factors:**

1. High temperature, intermediate moisture levels, and alkaline conditions all promote the Maillard reaction.

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21 "A Thermolyzed Diet Increases Oxidative Stress, Plasma alpha aldehydes, and colonic inflammation in the Rat," *Experimental Diabetes & Aging*, Available online 8 June 2007, and *Chem Bio Interact*, Vol. 169, No. 2), pages 100-109, 2007.

PEO Solution

Alteration	Causative Agent	New Compounds
Hydrolysis	Moisture	Free Fatty acids / Diacylglycerols
Oxidation	Air	<ul style="list-style-type: none"><li>• Oxidized monomeric Triacylglycerols</li><li>• Oxidized dimeric and oligomeric triacylglycerols</li></ul>
Thermal alteration	Heat	<ul style="list-style-type: none"><li>• Volatile compounds (aldehydes, ketones, alcohols, hydrocarbons, etc.)</li><li>• Cyclic monomeric triacylglycerols</li><li>• Isomeric monomeric triacylglycerols</li><li>• Nonpolar dimeric /oligomeric triacylglycerols</li></ul>

## Scientific Support for Chapter 8

2. In cooking, low moisture levels are necessary mainly because water boils into steam at 100 °C (212 °F), whereas the Maillard reaction happens noticeably around 154 °C (309 °F): significant browning of food does not occur until all surface water is vaporized.

Hence, a limiting factor is the temperature. Far less of these reactions occur with cooking food in water or steaming.

For the science buffs, a 2013 article<sup>22</sup> is posted at <http://cen.acs.org/articles/90/i40/Maillard-Reaction-Turns-100.html>.

Here were the parameters for the heated foods diet study:

- 344 rats
- Fed normal diet for one week, then split into two groups, one a control diet, and the second group got the same diet which was thermolyzed (heated).
- Thermolyzed diet was chow prepared with a relatively short exposure of 122<sup>o</sup> C (252<sup>o</sup>F) for only 30 minutes in an atmosphere devoid of oxygen. Hence, oxygen could not be the culprit in findings, only heat!

### **Here are the key findings:**

1. Experimental group became thiamine deficient measured by transketolase.
2. Experimental group had increased oxidative stress measured by reduced GSH.

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22 *Chemical and Engineering News*, Volume 90, Issue 40, pp. 58-60, 2013.

3. Experimental group had increased markers of Maillard reactions measured by the presence of  $\alpha$  – oxoaldehydes (glyoxals and methyl glyoxals).
4. The protein adducts of these carbonyls and protein oxidation levels were increased.
5. Experimental diet also increased oxidative stress biomarkers in livers and colons.
6. Experimental diet increased macrophage infiltration into colons (fourfold).

**Here are some of the “baaad” biochemical effects found:**

1. Experimental group became *thiamine deficient* measured by transketolase
2. Experimental group had *increased oxidative stress* measured by reduced GSH.
3. Experimental group *had increased markers of Maillard reactions* measured by the presence of  $\alpha$  – oxoaldehydes (glyoxals and methyl glyoxals).
4. The protein adducts of these carbonyls and protein oxidation levels were increased.
5. Experimental diet also ***increased oxidative stress biomarkers*** in livers and colons
6. Experimental diet increased macrophage infiltration into colons (fourfold).

The methylglyoxal A generated by the Maillard reactions is not a good thing for you. An international study including our own NIH <sup>23</sup> found that undesirable free radicals are generated during the glycation reaction of amino acids with this compound.

**So, now, what about fats?**

All fats in nature that you eat are in the form of triglycerides. That means three fatty acids are joined by an “ester” molecular linkage to a glycerol molecule “backbone”. You’ve read how very important natural fats, in their original unadulterated states are. I’ll bet you don’t know what happens when you heat (“cook”) fats. Now, I don’t expect you to understand all the biochemical terms unless you are technically inclined. But consider if you’d like all these ***oxidized fats, aldehydes and volatile compounds*** to enter your precious body.

Heat breaks the bonds of the natural triglycerides making toxic free fatty acids. ***Circulating NEFA [non esterified fatty acids or free fatty acids] concentration is an independent risk factor for sudden death in middle-aged men. Some form of primary prevention could be envisaged in subjects at high risk of sudden death.***<sup>24</sup>

Obviously the prevention here could include not **overly** heating fats. Heat in the presence of oxygen not only breaks the glycerol ester bonds (releasing the free fatty acids) but also further damages the fats to these other non-naturally occurring compounds that can be dangerous to you as this chapter details.

Researchers have also found that a diet rich in raw vegetables lowers your risk of breast cancer, and eating lots of fruit reduces your risk for colon cancer, according to a study published in the May 1998 issue of

23 *J. Biol. Chem.* 1995, 270:28228-28233.

24 *Circulation* 2001 Aug 14;104(7):756-61

the journal *Epidemiology*. Including fresh fruit as part of your daily diet has been associated with fewer deaths from heart attacks and related problems, by as much as 24% (of course, as you have learned, this is a “relative risk”), according to a study published in the September 1996 issue of the *British Medical Journal*.

A few relevant articles you may be interested in showing the impediment of heating to digestion are:

**“Effect of meat cooking on physicochemical state and in vitro digestibility of myofibrillar proteins.”** [[http://www.ncbi.nlm.nih.gov/pubmed/18237130.](http://www.ncbi.nlm.nih.gov/pubmed/18237130)]

“The Effect of Cooking on the digestibility of meat.” [<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1254788/pdf/biochemj01123-0140.pdf> ]

There ‘s more from Dr. Rowen .....

For the technically inclined: These reactions take place in the unsaturated fatty acyl groups attached to the glyceridic backbone and, therefore, the stable final products are triglyceride monomers, dimers and oligomers containing modified and non-modified acyl groups. The concurrent formation of positional and geometrical isomers through auto-oxidation gives rise to **thousands** of individual new compounds and explains the poor information on the original structures. Studies on the mechanisms for their formation as well as for their structural analysis have been carried out after formation of simpler derivatives, i.e. fatty acid methyl esters. Formation of other groups of compounds present in used frying oils without extra oxygen, i.e.,

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**Diels-Alder dimers, cyclic fatty acids and positional and geometrical fatty acids are explained by thermal reactions and not by radical interaction. They are minor compounds of nutritional interest and paradoxically are much better known than the major oxidation compounds because of their stability and low polarity.**

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Remember, oxidized and otherwise damaged fats are the fundamental cause of vascular disease. Folks, **we are simply dumping in a horrible mishmash of molecules not found in nature, not native to foods, not metabolizable by the body, which could give rise to most any toxic bodily reaction, especially damaging to cell membranes.** I haven't covered trans fatty acids. You've heard a lot about them, like in margarine. But what you don't know is that the high heat in processing and clarifying cooking oil converts a lot of the native 'cis' fatty acids into 'trans.'

Now consider this. Most oils used for cooking are valued for their "smoke" points. That's the temperature at which the oil will visibly burn. Most vegetable oils are in the 215-265° C range. That's an awfully high temperature. Water boils at 100° C (212° F). Olive oil is a bit lower at 190° C. Even if your oil doesn't smoke, the higher ranges will induce conversion of cis bonds to damaging trans.

The ability of oil to withstand high temperatures before smoking is an additional reason fried foods are the worst things you can eat. Aside from the damaged oils, the heat wipes out whatever nutritional value might have been there.

If you don't believe me, do this simple experiment. Take any unsaturated oil and smear it on the inside of a pan. Heat it for a period of time at moderate heat. The longer you do this, the less "oily" the oil remains and the tackier (sticky) it becomes. This is due to molecular damage to the oils and cross-linking almost making the oil into a plastic. This is what is happening in your body in slow motion.

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But now consider the impact of heat on cholesterol oxidative damage in your food! (WE know that cholesterol isn't the bad guy. **It's oxidized cholesterol that is**. Oxidized cholesterol is taken up in your vascular endothelial cells and seen by your immune system as "foreign". An immune attack is launched against these foreign molecules and your own tissues take a huge hit).

So what does heat do to cholesterol? In fish cooked in vegetable oil, the sum of cholesterol oxidation products (COPs) increased after the heating processes from **0.9 microg/g in the raw sample** to 6.0, 4.0, 4.4, 3.3, and 9.9 microg/g extracted fat in pan-fried without oil, with olive oil, corn oil, partially hydrogenated plant oil, and steamed, respectively. A highly significant correlation was found between the fatty acid pattern and the total amount of COPs ( $r^2 = 0.973$ ,  $p < 0.001$ ). **(300%-900% increase in COPs).**<sup>25</sup> **Up to nearly a 10-fold increase in oxidized products!**

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Uncooked fresh butter had virtually NO COPs. So the **fears about the saturated fats in butter is hogwash**. Furthermore, as Prof. Peskin made clear, there is NO SATURATED FAT in an arterial occlusion.

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However, if you heat butter to 170-180 C (300<sup>o</sup> F), you'll start seeing COPs. Raw bacon rind (NEVER EAT IT) has no COPs, but cooked at 25 *J Agric Food Chem* 2004 Aug 11;52(16):5290-6.

routine frying temperatures of 170° C for 10 minutes and they form. (I'd rather have some COPs than trichinosis).

Routine cooking definitely increases COPs. Using an electric skillet at just 135° C, the COPs of cooked beef, veal, and pork increased by 1.7, 2.3 and 2.5 fold respectively. When cooked in an oven at 220° C for 60, 80. And 90 minutes respectively, the COPs levels increased by 3.5, 5.4 and 4.2 fold respectively.<sup>26</sup> Freshly prepared meat products are a minor source of COPs overall, but semi-prepared frozen meat products fried once and stored can increase COPs considerably.<sup>27</sup>

Many people look at fish as a healthier food than mammal meat. But here is a report<sup>28</sup> that even fish can deliver you COPs if heated. The authors aimed to look at routine fish preparation, as you might do in your own kitchen. **Strangely enough, the highest amount of COPs was found in steamed fish over pan-fried fish.** The authors felt that was due to the “longer heat exposure.” The authors concluded that even salmon, touted for its heart sparing effects could provide COPs as a potential toxicological health risk.

Contrary to popular belief that the highly unsaturated fatty acids improve membrane fluidity, the opposite may be occurring. Yes, these fatty acids have a lower melting point, hence are more fluid—perhaps TOO FLUID—another reason that Nature doesn't incorporate much of them, but it is even worse... **Over time, they become peroxidized far more easily.** And, these lipid oxidation products strongly contribute to membrane rigidity. These products are likely dominant factors in **creating age-related membrane rigidity.**

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26 *Journal of Food Science*, Vol. 66, No. 9, **2001**.

27 *JAOCs*, Col 77, no. 6 (**2000**).

28 *J. Agric. Food Chem.* **2004**, 52, 5290–5296.

A funny named animal, the naked mole rat, has the **highest longevity of any rodent** – up to 30 years. And, it has the **lowest amount of DHA in its membranes** of the rodent species. This makes their membranes far more resistant to peroxidation, and more similar to the longer-lived mammals.

**Similar findings in insects...** Queen bees are fed mouth to mouth by the workers, which workers consume pollen for their own food. Pollen is rich in polyunsaturates. Queen bee food is not loaded with polyunsaturates, which might be a major difference in the longevity of the queen (years) to her brood (weeks).

This is now seen in human research. Children born to nonagenarians have red cell membranes that are more resistant to peroxidation damage! So, **we see that membrane composition of susceptible fatty acids is associated with longevity cross species lines, from bees, to rodents, birds, and even humans.**

### **Weston Price, DDS...**

One of the greatest heroes of nutrition and health was Weston Price. A dentist, he traveled the world to aboriginal cultures to study how they ate, and how what they ate impacted their health, from their teeth to the rest of their bodies. You can't separate the health of one part of your body from another. In today's world *specialists* will tell you that you have 2 kidneys, a liver, a heart, teeth, etc., and what is going on with these organs is separate and distinct from each other.

**It was easy for Price to see that if you had dental caries, and not enough bone growth in your jaw to accommodate all your teeth, that you'd have other nutritional problems as well.** His observations were most profound, yet quite simple and LOGICAL! Yet his name was

not mentioned once in my prestigious medical school. I didn't learn of him, nor Francis Pottenger for at least 10 years after graduating. When you read the following, graciously posted on the Price Pottenger Foundation website, I think you'll be dismayed that those who rule this country have also ignored these maxims, to your detriment. What you read here is the foundation of why the "health" of the American people has fallen into disarray and plunder by those who are violating these maxims at every turn to farm (pharm) you for profit.

<http://ppnf.org/about/about-price-and-pottenger/dr-pottenger/traditional-diets/>.

## **THE FOODS THAT WERE EATEN**

Dr. Price's research shows that healthy traditional diets:

- Contain foods naturally rich in body-building nutrients
- Include minerals and fat-soluble vitamins found in butter, sea foods, fish oils and fatty animal organs
- Incorporate raw, unaltered proteins from meats, sea foods, nuts, raw dairy and sprouted seeds
- Use sweeteners rarely and sparingly

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Diets based on these guidelines provide optimal nutrition for preventing disease and slowing physical degeneration.

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### **How the foods were cultivated:**

In a healthy traditional diet, *how* foods are grown is as important as *what* foods are eaten. In a healthy traditional diet, foods are grown:

In natural, mineral-rich soil

Using NO chemical fertilizers

Using NO chemical pesticides

Growing foods in this way prevents the introduction of harmful chemicals into our food chain and our bodies.

**How the foods are preserved and prepared.**

Food preservation and preparation methods are also important components in the healthy traditional diet. In such diets, foods are:

- Eaten in season
- Preserved using methods of—earth storage, drying, freezing, culturing and pickling—that maintain or enhance the nutritional content of the food
- Eaten “whole” and unrefined, maintaining fiber and nutrient content
- Consumed raw or very gently and lightly cooked

Preparing and preserving foods using traditional methods ensures that we benefit from their full nutritional value.

**Lifestyle**

**Choices:**

In his studies, Dr. Price noted that healthy diets are enhanced by healthy lifestyle choices. The healthy tribes studied by Dr. Price:

- Engaged in regular, vigorous exercise through work, play, dance, and sports
- Had access to pure air and enjoyed abundant sunlight

## Scientific Support for Chapter 8

- Observed periods of partial abstinence from food (fasting), or regulated periods of under-eating
- Ate special protective foods in preparation for conception, pregnancy and lactation
- Spaced pregnancies apart to protect the health of the mother and the children
- Breast-fed their babies
- Instructed their children in the importance of maintaining traditional dietary and lifestyle principles

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These maxims are among the most logical and reasonable rules to live by that have ever been revealed. They are as basic to health as the Golden Rule of “do unto others as you would have done unto you.” Is basic to our relations with others. Yet, they are totally ignored in a profit and greed driven world.

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I say to the ignorant and arrogant political and disease maintenance establishment, “WAKE UP!” Stop taking payouts from those poisoning the planet to stay in office while not only our families but also your families pay the ultimate price of untimely physical degeneration.

### More from Professor Peskin:

**2008 Newsflash:** Just like fruits fulfill our natural “sweet tooth,” fats fulfill our appetite. Fats – not protein or carbohydrates.<sup>29</sup> Take 6 egg whites and cook them with no butter. You’ll be starving just 15 minutes after eating them. Compare this to adding 2 yolks. You’ll be full and contented. A landmark experiment – it’s only **FATs – NOT carbohydrates or proteins** which send signals to the brain saying you aren’t hungry. Here’s what was published in **2008**, if anyone would care to research it:

- “Here, we report that *duodenal infusion of fat stimulates* oleoylethanolamide (OEA) mobilization in the proximal small intestine, whereas infusion of *protein or carbohydrate does not*.
- ...[T]his lipid messenger *participates in the induction of satiety*
- ...[T]he **rapid onset of the OEA response (<30 minutes)**...
- ...**[P]rolonging the time interval between meals.**
- OEA production utilizes dietary oleic acid as a substrate and is disrupted in mutant mice lacking the membrane fatty-acid transporter *CD36*. Targeted disruption of *CD36* or *PPAR-α* **abrogates [ends] the satiety response induced by fat.**

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29 Schwartz, GJ, et al., “The Lipid Messenger OEA Links Dietary Fat Intake to Satiety,” *Cell Metabolism*, Vol. 8, Issue 4, Oct 8, **2008**, pages 281-288.

- The results suggest that activation of small-intestinal OEA mobilization, enabled by *CD36*-mediated uptake of dietary oleic acid, **serves as a molecular sensor linking fat ingestion to satiety.**
- In conclusion, our studies identify **OEA as a key physiological signal that specifically links dietary fat ingestion to across-meal satiety.**"

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**PEO Solution** analysis: Once again, the truth gets published but no one is made aware of it. ***ONLY fats fulfill your appetite, NOT protein or carbohydrates.***

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### **$\omega$ -6 Polyunsaturated fatty acids extend life span through the activation of autophagy**

O'Rourke, Eyleen, J., et al.,  $\omega$ -6 Polyunsaturated fatty acids extend life span through the activation of autophagy," (*Genes & Development*). Published in advance February 7, 2013, <http://genesdev.cshlp.org/content/27/4/429.full>, 2013:

"Supplementing *C. elegans* culture media with these  $\omega$ -6 PUFAs [long chain metabolites termed derivatives] increases their **resistance to starvation and extends their life span** in conditions of food abundance. Supplementation of *C. elegans* or **human epithelial cells** with these  $\omega$ -6 PUFAs *activates autophagy*, a cell recycling mechanism that **promotes starvation survival and slows aging.**

"We found that supplementation with **AA and DGLA [both Parent omega-6 derivatives]**, *but not with EPA,*

was sufficient to activate autophagy in ad libitum-fed *C. elegans*.

“Our data show that supplementation with  $\omega$ -6 PUFAs activates autophagy **in human epithelial cells**.

“These results show not only that dietary supplementation with  $\omega$ -6 PUFAs **activates a conserved cellular response** normally triggered by fasting, but also that long-term administration of  $\omega$ -6 PUFAs can render the beneficial effects of **low-caloric intake** even in ad libitum feeding conditions....”

## Appendix X from 24-Hour Diet: The Power of Parent Omega-6

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**Newsflash: Parent Omega-6 Increases Weight Loss: Known in 1973!**

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**You have already read about Doctor Cavallino's** experiment with “carboholics” in Italy. I have long been aware of the remarkable power of the correct *unadulterated* parent omega-6 to -3 ratio in decreasing carbohydrate cravings. However, even I had never seen the following medical journal article. I sincerely thank Canadian David Macphail for sending it to me. All the way back in 1973, physician H. Kasper proved there is an effect of greater weight loss and better blood chemistry, too, when parent omega-6 is added to the diet – REGARDLESS of CALORIES.

Here's what the study states<sup>30</sup>:

**“Despite a higher total caloric intake**, the weight-reducing effect clearly equals that of a standard clinical reducing diet of 1,000 kcal [even though patients consumed significantly more food].

“...A **maximal weight loss** was achieved in cases 1, 2, and 15 when they were **taking fats high in linoleic acid [parent omega-6]**.

“It was **striking to observe** that the **weight gain did not correlate with the caloric intake**. Particularly if fat was given in the form of corn oil [high in parent omega-6], a distinct discrepancy between the caloric intake and the response of the body weight was detectable.

“This phenomenon was less conspicuous if fat was taken in the form of olive oil.

“If **fat was exchanged** isocalorically **for glucose [carbohydrates]**, the **weight loss ceased**.

“The **cholesterol and triglyceride concentrations** in the serum, which had been raised at the beginning of the experiment, invariably showed a **tendency towards normalization** under this dietary program.” (Emphasis added.)

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30 Kasper, H., et al., “Response of body weight to a low carbohydrate, high fat diet in normal and obese subjects,” *The American Journal of Clinical Nutrition*, 26: February 1973, pages 197-204.

## ► *Lean-for-Life* Commentary

1. This experiment proves that the “calorie theory” is incorrect and that there is much more to the picture than merely “calories in minus calories used equals weight gain.”
  2. The parent omega-6 oil has a natural weight-loss property, whereas olive oil does not.
  3. When carbs were switched “calorie-for-calorie,” for fat, weight loss STOPPED. Once again, we clearly see the “low-fat/hi-carbohydrate diet” failing!
  4. Triglyceride and cholesterols significantly improved with the low carbohydrate/parent omega-6. We have a home-run!
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Following are some critical points, from the exceptional treatise by DF Horrobin, MD, PhD—a true medical genius, published in *Progress in Lipid Research*:<sup>31</sup>

**“The *n*-6 EFAs have at least four roles:** (1) The modulation of **membrane structure**. (2) The formation of short-lived local regulating molecules such as prostaglandins (**PGs**) and leukotrienes (**LT**), together often **known as eicosanoids**. (3) The control of the **water impermeability of the skin** and possibly the permeability of other membranes such as the gastrointestinal tract and the blood-brain barrier. (4) **The regulation of cholesterol transport and cholesterol**

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31 Horrobin, D.F., “Nutritional and medical importance of gamma-linoleic acid,” *Prog. Lipid Res.*, Vol. 31, 1992, No. 2, pages 163-194.

**synthesis. The membrane effects of the EFAs are possibly the most important.**

“The fluidity and flexibility of all membranes within the body are influenced by their EFA content. As a crude indicator, the effect of an EFA on membrane fluidity is determined by its concentration in the membrane and by the number of double bonds in the molecule (the product of concentration x the number of double bonds is sometimes known as the unsaturation index). **However, there is much more to the story than that.**

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**“The *n*-3 EFAs, even though they have as many or more double bonds as the *n*-6 EFAs are unable to reverse the features of *n*-6 EFA deficiency.**

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“The precise configuration of the double bonds must therefore be important and attempts to explain the rationale for this are just beginning.

**“The lipid configuration of the membrane** is important in itself, but also matters because it **influences the structure and behaviour of the many proteins in the membrane such as ion channels, receptors and ATPases [including insulin receptivity]**. These proteins are literally *afloat in a lipid sea and their function is dependent on the behaviour of that sea*. Good examples of this are studies on the effects of lipids on the binding of ligands to their receptors. The unsaturation of the

lipid medium in which the receptors are found has been reported to change the affinity for ligands, such as *steroid hormones*, and *peptides*, such as opioids and angiotensin. In general, the more unsaturated the lipid, the lower the affinity of the receptor for its ligand.

“Lipid unsaturation also influences membrane ‘fluidity.’ This is important in the **vascular system** and also in **any other situation in which cells move**, for example, during inflammation and immune responses. Red cell membranes, which have **reduced EFA levels** are “stiffer” than usual, and as a result **increase blood viscosity and reduce tissue oxygenation**.

“The third role of the EFAs is in the maintenance of the water impermeability of the skin. *In the absence of n-6 EFAs the skin loses its ‘water-proofing.’*”

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**“It is apparent from this brief description that a lack of or abnormal metabolism of EFAs could adversely influence every cell and every organ system in the body. There is therefore nothing inherently surprising in the concept that EFAs may have a role to play in modulating many different disease processes. [Note: This precisely explains why cancer can occur in any tissue—the most oxygen deficient.]**”

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“The EFA requirement may be increased in the presence of high rates of cell division.

This situation may be physiological (as in infancy) or **pathological (as in the presence of cancer, inflammation or rapid cellular repair after injury)**.

### **MEN need 5Xs More PEOs...**

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**“Gender has a major, but inadequately understood, impact on EFA requirements. *Male animals require a higher EFA intake than females.* This may in part be because females metabolise LA more rapidly and in part because they retain EFAs in tissues more effectively in the presence of EFA deficiency.**

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**“The total phospholipid (TPL) fraction, in contrast, does not change rapidly in response to feeding or fasting. Moreover it is relatively rich in the EFAs right along the metabolic chain. It can therefore be used as a guide to both EFA intake and EFA metabolism.”**

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**PEO Solution analysis: In research, plasma total phospholipids are the best quantitative measure of EFA status—*much superior to red blood cell analysis.***

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Horrobin’s treatise continues...

### **Confirmation of Small Derivative formation:**

**“GLA [Parent omega-6 derivative] is formed by the rate-limiting step of delta-6-desaturation and**

metabolised by the non-rate-limiting step of elongation to dihomogamma-linolenic acid (DGLA). It is therefore not surprising that **GLA is found in most tissues in only small amounts**. It normally makes up less than **0.2% of the fatty acids in phospholipids**, less than **0.1% of those in triglycerides** and less than **2.0% of those in cholesterol esters**. Furthermore, *GLA—the body's most important derivative* is found in tissue in only very small quantities. Furthermore, Parent omega-6 can modulate cytokine releaser directly, rather from its long-chain metabolites.

“The administration of **GLA leads to increased plasma PGE1 levels in humans** and increased macrophage PGE1 levels in rats. There can therefore be no doubt that GLA enhances the rate of formation of this very desirable substance.

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**“PGE1 has a quite extraordinary range of desirable actions. It dilates blood vessels and lowers blood pressure; it inhibits platelet aggregation; it inhibits cholesterol biosynthesis; it is an anti-inflammatory agent; it has a biphasic regulating effect on immune responses; and it stimulates cyclic AMP formation, thus being capable of inhibiting phospholipase A2, an enzyme important in releasing AA during inflammation.**

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**These desirable effects obviously have considerable therapeutic potential.**

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“The formation of PGE1 may explain why, *contrary to simplistic expectations*, but in accordance with a **prediction based on understanding** of PGE1 actions, the rise in AA levels following GLA administration to cells, animals or humans is *consistently followed by a fall, rather than a rise, in the levels of conversion of AA to potentially harmful metabolites like Thromboxane A2 or PGE2*. There is a tendency to consider arachidonic acid “a bad thing” because it can give rise to metabolites like thromboxane A2, PGE2, and leukotriene B4. In fact there is **no evidence at all that arachidonic acid is harmful so long as it stays as AA**. AA is an **essential constituent of membranes**. Adequate levels of DGLA seem important in keeping AA in membranes where it is desirable, and preventing conversion of AA to its possibly undesirable metabolites:

The amazing treatise continues.... showing **Parent omega-6 and its metabolites takes center stage....**

**“The n-3 EFAs are of major biological significance but they are simply not as important as the n-6 EFAs.**

“This is shown by the following facts:

“(1) When animals and humans are put on **diets deficient only in n-6 EFAs**, it is easy to show that they develop

multiple biochemical and biological abnormalities. In contrast it has proved extremely difficult to demonstrate biological abnormalities in animals deprived only of *n-3* EFAs. There are abnormalities in the brain, the retina, the heart and platelets and the *n-3* EFAs are undoubtedly important in modulating the functions of these organs, but *these abnormalities are not easy to demonstrate*.

“(2) When animals are *deprived of both n-3 and n-6 EFAs*, all the readily observed **abnormalities are quickly corrected by *n-6* EFAs alone. *N-3* EFAs alone do not correct any of the abnormalities, and make some, such as the capillary fragility, worse.**

“In order to express their normal biological effects, *n-3* EFAs **must be given with *n-6* EFAs** whereas the *n-6* EFAs are biologically active when given without *n-3* EFAs. [Note the significant difference. The 21<sup>st</sup> century solution is BOTH Parent omega-6 and Parent omega-3 in the proper ratios and quantities.]

“(3) In human milk and in most tissues in the body, the ratio of *n-6* to *n-3* EFAs lies within the range 3:1 to 9:1 [breast milk is 10:1 – most tissues 4:1-7:1; although stored body fat is much higher.]. This is true even of animals such as the zebra whose EFA intake is almost entirely in the form of ALA [Parent omega-3] from grass. **Thus even when most dietary EFAs are in the *n-3* form, the *n-6* EFAs are preferentially retained.**”

**PEO Solution** analysis: We see the much greater importance of Parent omega-6 and its metabolites compared to Parent omega-3 and its metabolites. Dr. Horrobin terms omega-6 series GLA the body's most important derivative.

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The Power of PEOs was known in 1956. The extraordinary nutritional scientist of Reading and Oxford, H.M. Sinclair, wrote a superb article in *The Lancet* titled, "**Deficiency of essential fatty acids and atherosclerosis, etcetera.**"<sup>32</sup> He warned that people won't believe it, stating, "**MY inclusion of 'etcetera' in the title invites the scorn we so readily pour I vendors of patient cure-alls.**" Tragically, he was correct and his advice was not properly integrated into the medical community. Today, you can remedy that mistake.

Journal highlights are:

- "First, there was an enormous increase in permeability of the skin [**epithelial tissue in all carcinomas**] in EFA [PEO] deficiency and there is an increase in **capillary fragility**...
- "Wealthier peoples are becoming increasingly sensitive to the carcinogenic effect of X rays through deficiency of EFA, the above facts become explicable provided those young children who died of leukaemia were irradiated when their pregnant mothers were had diagnostic radiography; a chemical carcinogen could hardly be responsible for their deaths. "In lower animals, in which we have carefully studied the skin lesion of EFA [PEO]-

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32 " April 7, 1956, pages 381-383.

deficiency and found a dramatic increase in permeability of the epidermis, we believe there is a structural fault perhaps through failure of the phospholipids containing EFA [PEO] to polymerise and form the impermeable barrier in the stratum granulosum. Phospholipids are rich in unsaturated fatty acids [PEOs] (though not so rich as cholesteryl esters... "So, as in the case of esterified cholesterol, we have an **abnormal type of phospholipid** being formed which may not only cause a structural defect in the skin which is responsible for the great increase in permeability but may also be outstandingly important ....

- "... [T]he nervous system is rich in phospholipids containing polythenoid fatty acids, and these are found together with highly **unsaturated cholesteryl esters in myelin**; the presence of abnormal types of the compounds that are known to important to it would be unlikely to leave function undisturbed; disseminated sclerosis is a disease of highly civilized countries being almost unknown in India and China [as of 1956], and other diseases in which the ectodermal neuroglia is effected may be relevant; since serum **EFA fall in acute infections...early mild dementia** appears to be becoming commoner in males. Thirdly, the **mitochondria** membrane probably contains phospholipids, and derangement of this through **deficiency of EFA [PEO] might me responsible for the uncoupling of oxidative phosphorylation found in such deficiency.**

- “Effects of EFA [PEO]-Deficiency: First, deficiency would be *likely to be at least five times commoner in males than in females*. “Secondly, we might expect deposition of cholesterol since cholesterol esterified with unusually saturated or with unnatural fatty acids is probably disposed of less readily...”

The article continues:

- “...I believe [abnormal esters/phospholipids] to be caused by a **pure dietary deficiency of essential fatty acids [PEOs]**... There is even more brilliance in his article, but these excerpts make it clear—his genius is evident.

Dr. Hugh Sinclair **predicted** the following patient ailments/disease/physiologic disorders from EFA deficiency:

- a) **Cardiovascular disease.**
- b) **Cancer**; in particular, increased skin cancer. [Note: Skin cancers are at epidemic levels with no end in sight, and cardiovascular disease (in all forms) is our #1 killer. CVD too has no end in sight. All carcinomas are enclosed by epithelial tissue—a PEO deficiency of Parent omega-6—is **DIRECTLY TIED to ALL CARCINOMAS**. Furthermore, only Parent omega-6 is contained in the arterial intima.]
- c) Dr. Sinclair understood the *damage X rays* may cause. **PEOs are highly protective against cancer treatment X ray damage**—See my book, *The Hidden Story of Cancer*.
- d) PEOs assist the nervous systems. **PEOs help fight MS.**

- e) Even **dementia** is addressed. This has become another epidemic directly related to PEO deficiency. Fish oils are worthless and coconut oil is not effective enough.
- f) PEOs vital role in **combating infection**.
- g) PEOs are directly **incorporated into mitochondrion**. This is a *top anti-aging secret and a key to cancer prevention*.
- h) Dr. Sinclair's key concept that leads us to lower **LDL-cholesterol** – esterification of cholesterol to PEOs – will be discussed in detail later.

See also

I.O.W.A. Experiment: <http://brianpeskin.com/BP.com/experiments/IOWA-Experiment-Results.pdf>

Cancer and Mitochondria Defects: New 21st Century Research, Townsend Letter For Physicians: <http://brianpeskin.com/BP.com/publications/CancerMito-Town8.09.pdf>