

# Statins and Increased Cancer: The Hidden Story and a New Solution

by Brian Peskin, BSEE

As early as the late 1990s, almost half of all Americans and Europeans died of heart disease.<sup>1</sup> It has been predicted that, by 2010, virtually all Americans will die of either heart disease or cancer. Atherosclerotic coronary artery disease (CAD), a “clogging” of the arteries, became the number-one killer of Americans in 2006, with cancer a close second. Surprisingly, in spite of widespread use of cholesterol-lowering drugs, heart disease remains the top killer in America. Could the medical establishment, by their unfettered support of the use of statins to lower cholesterol, inadvertently be exacerbating the rising incidence of cancer and heart disease? This article presents a causal link between increased widespread statin use for battling heart disease (by lowering low-density lipoprotein [LDL] cholesterol) and the concurrent significant increase in cancer.

## The Statin-Cancer Connection

An explosive article published in the 2007 issue of *Journal of the American College of Cardiology*<sup>2</sup> revealed that statins, previously reported to have relatively few serious side effects, can *significantly increase the risk of cancer*. Specifically, the increased risk of cancer has been significantly correlated with the lowering of LDL cholesterol – an unforeseen negative outcome. With statin use, the increase in cancer

deaths counteracts the *supposed* lower cardiac mortality associated with lower cholesterol, resulting in a neutral effect or increased overall mortality. Translation: with statin use, *even if you don't die of a heart attack, you will likely die of cancer*.

## Statins' Effectiveness Called Into Question

Prepare to be shocked. Statins, which represent huge profits to the pharmaceutical industry, have been the preferred drug of most cardiologists. However, *statins are now being shown to NOT PREVENT or reduce heart disease*. The inability of statins to have a positive impact on heart disease was predicted in a *Journal of the American Medical Association (JAMA)* article over ten years ago, which concluded that low cholesterol, by itself, did not significantly prevent heart disease:

Our findings do not support the hypothesis that hypercholesterolemia [high LDL cholesterol levels] or low HDL-C [high-density lipoprotein cholesterol – a.k.a. “good” cholesterol] are important risk factors for all-cause mortality, coronary heart disease mortality, or hospitalization for myocardial infarction or unstable angina in this cohort of persons older than 70 years.<sup>3</sup>

These (and other) poor outcomes prompted the recent medical journal article entitled “LDL Cholesterol: “Bad Cholesterol or Bad Science,” published

in the *Journal of American Physicians and Surgeons*, which included these conclusions:<sup>4</sup>

- “No tightly controlled clinical trial has ever conclusively demonstrated that LDL cholesterol reductions can prevent cardiovascular disease or increase longevity.
- “The concept that LDL is bad cholesterol is a *simplicistic and scientifically untenable hypothesis*.”

As this article was going to press, the *Journal of American College of Cardiology* (2007;50[18]:1735-1741) published “Beyond Low-Density Lipoprotein Cholesterol – Defining the Role of Low-Density Lipoprotein Heterogeneity in Coronary Artery Disease,” stating more discouraging conclusions:

- “[D]espite more aggressive interventions by lowering LDL-C levels, the majority of CAD (coronary artery disease) events go undeterred [not prevented]...”
- “Measurement of apolipoprotein (apo)B has been shown in nearly all studies to outperform LDL-C and non-HDL-C as a predictor of CAD events and as an index of residual CAD risk.”

This recent finding and its implications will be the key to explaining the statin/cancer connection. ➤

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### ► Cholesterol-Lowering Drugs Were Known to Cause Cancer a Decade Ago

A dire warning about statin use was published by two physicians, Thomas B. Newman and Stephen B. Hulley,<sup>5</sup> at the University of California in San Francisco in 1996. This same warning was published in the cancer journals over a decade ago. One example appeared in *Cancer Research*:<sup>6</sup>

Several trials of cholesterol lowering with drugs to prevent cardiovascular disease events have demonstrated an increase in cancer incidents in the subjects treated with lipid-altering drugs. The trials were randomized, double-blinded, and lasted an average of five years.... A statistically significant excess of malignancy was seen in elderly subjects and women randomized to the drug groups."

None of these studies or their conclusions has ever been refuted, yet we continue to prescribe more and more cholesterol-lowering drugs. Are physicians missing something? Yes. Take the following, for example.

### Arterial Plaques – It's *Not* the Saturated Fat

For decades, saturated fat was blamed for the buildup of arterial plaque, the material that can significantly narrow the diameter of arteries. However, a landmark article published in the *Lancet* in 1994 shattered that myth.<sup>7</sup> The investigators analyzed plaque and found it contained more than ten different compounds, none of which consisted of saturated fat. There are also other independent analyses confirming the lack of saturated fat in any arterial plaque.<sup>8,9</sup>

### Arterial Plaque – Normally a Harmless Natural Repair Mechanism

As the vasculature ages, it is constantly repaired with new collagen. A number of other repair mechanisms are concurrently working, with cholesterol and Lp(a) lipoprotein

acting as "sticky patches" to seal cracks when injury or damage to an arterial wall occurs.

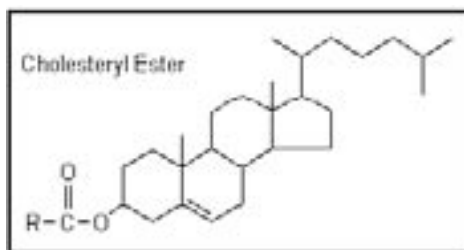
In healthy individuals, arterial plaques form as a result of these patching activities, but without serious consequences. However, in many individuals, the plaques do not disappear, but build up over time. To explain these perplexing observations, we need to explore cholesterol's makeup.

### Importance of Cholesterol – "Good" or "Bad" Terms Are Misleading

Cholesterol itself can't be "bad," because it is critical in the production of the hormones estrogen, progesterone, and testosterone,<sup>10</sup> keeping our skin water- and chemical-resistant, manufacturing bile salts for digestion of fats, forming our bones, and delivering precious Parent Essential Oils (PEOs) to all of our 100 trillion cells. Without plenty of cholesterol, we would all be dead.<sup>11</sup>

While free cholesterol does exist in the body, 80-90% is esterified, meaning it is chemically bound to a fatty acid, with a strong preference given to parent omega-6 (LA), as shown in Figure 1 (in which R represents the hydrocarbon portion of the fatty acid).

Figure 1: Cholesteryl Ester



### The Structure of Cholesterol Itself Never Changes

That's right; the structure of cholesterol itself never changes; the esterified component does. It is only the hydrocarbon [alkyl] portion of the ester group that changes. If you term something as "bad," presumably you want to get rid of it or at least get it as low as possible. This is what the pharmaceutical industry is saying.

However, if you got rid of all the LDL-C, you would be wiping out valuable fatty acids as well as a mechanism for removing oxidized fatty acids that should be removed from the body. It would be like stopping "garbage pick-up."

These cholesteryl esters are transported throughout the body in lipoprotein particles that are classified according to the ratio of protein to fat, or more simply, the density of the particle, in the following increasing order: chylomicrons, very low-density lipoprotein, intermediate density lipoprotein, low-density lipoprotein (LDL), and high-density lipoprotein (HDL).<sup>12</sup> LDL particles contain the highest percentage of cholesteryl esters (mainly parent omega-6, with a small portion of approximately three-percent parent omega-3).

### Importance of Esterified Cholesterol

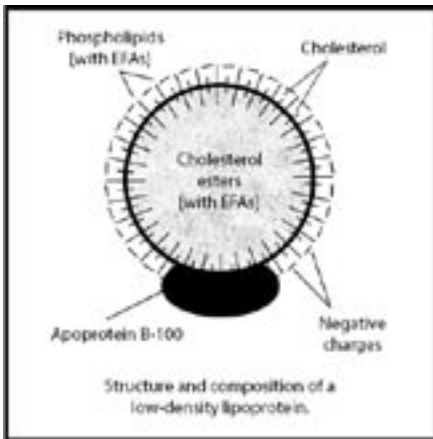
*Esterified* cholesterol comprises the majority of LDL (Figure 2). LDL is much more than just "cholesterol," although few people, including nutritionists and physicians, understand this. It is essential to understand the term cholesterol "esters" if you hope to understand the vital role of LDL in your body. Medical journals confirm this important fact: "LDL contains up to 80% lipid, including polyunsaturated fatty acids and cholesterol, mainly esters. *Linoleic acid, [is] one of the most abundant fatty acids in LDL...*"<sup>13</sup>

Furthermore, HM Sinclair, a top EFA researcher and famous English nutritional biochemist (bio available at: [http://www.britathsoc.org/bas\\_hugh\\_sinclair.html](http://www.britathsoc.org/bas_hugh_sinclair.html)), made clear in 1984 that about 20% of the free fatty acids of the phospholipids in both LDL and HDL are composed of parent omega-6, too.<sup>14</sup> America's top cardiology publication, the *Journal of American College of Cardiology* (2007;50[18]:1735-1741), published information stating that it is the *esterified* cholesterol that is the problem in heart disease, but didn't address the reasons why the problem occurs or offer ideas on how to solve it.

Esterification of LA with cholesterol was known as early as 1941<sup>15</sup> and is one of the keys to understanding the statin/cancer connection. However, due to widespread inaccurate terminology, we first need to discuss PEOs, Essential Fatty Acids (EFAs), and EFA derivatives.

### Figure 2: Structure and Composition of Low-Density Lipoprotein

(Textbook of Medical Physiology, p. 874, Courtesy W.B. Saunders)



### Parent Essential Oils: An Essential Difference

The term “Essential Fatty Acids” is so frequently misused that I was compelled to coin a new phrase, Parent Essential Oils (PEOs). “PEOs” refer to the only two true essential fatty acids: parent omega-6 (LA) and parent omega-3 (ALA). The term “parent” is used because these are the whole, unadulterated form of the only two essential fats your body demands, as they occur in nature. Once PEOs are consumed, your body changes only five to ten percent of them to “derivatives.”<sup>16-18</sup> That means 90-95% stay in the parent form in the cell and mitochondrial membranes.<sup>19,20</sup> There are a host of omega-6 and omega-3 derivative-based oils being marketed to physicians as EFAs that are, in fact, non-essential derivatives such as eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), and gamma-linolenic acid (GLA). Fish oils are made up almost exclusively of omega-3 derivatives. Scientifically and biochemically, calling these derivatives “EFAs” is wrong.

Derivatives are not EFAs because they are not essential – your body has the ability to make them as needed from the PEOs. Taking fish oil and other health-food-store “EFAs” often leads to pharmacological overdoses, which can be very harmful.

### Food Processing Adulterates Most Parent Omega-6

In the last several decades, processed foods – in particular, frozen foods and restaurant cooking oils – have increasingly incorporated trans fats (hydrogenated) and other unhealthy fats and oils resulting in less parent omega-6 (LA) for incorporation into cell membranes and conversion into arachidonic acid, which is a source of many prostanoids and leukotrienes used in inflammatory, immune, and signaling functions.<sup>21,22</sup>

Membrane fluidity increases when more PEOs (functional parent EFAs, in particular, parent omega-6) are available to incorporate in the membrane lipid bi-layer. *When natural PEOs are replaced by trans fats (hydrogenated), the fluidity changes, and that can cause significant reduction in critical cellular O<sub>2</sub> transfer.*

A category of synthetic fat that is increasingly used as a substitute for trans fats is inter-esterified fats termed IE fat. Consequently, IE has its own set of health problems such as abnormally raised resting blood glucose levels.<sup>24,25</sup>

It is important to understand that cooking oil manufacturers avoid omega-3 oils because they are much more unstable than the parent omega-6 series oils. Therefore, most omega-3 in the diet is unadulterated and of no concern in our analysis of adulterated PEOs. Many seeds, nuts, grains, eggs, etc. contain omega-3 and omega-6 unsaturated fatty acids, but typically the amount of omega-6 far outweighs the amount of omega-3; flax seeds are an exception.

Even when margarine and other hydrogenated products contain relatively few trans fats – as little as one to two percent – this translates to an enormous number of defective

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trans fat molecules. In absolute numbers, there are an enormous  $1 \times 10^{21}$  molecules in each tablespoon of oil. Therefore, the potential to cause great damage, either integrally in the cellular structure or in biochemical reactions, is highly significant since many of us consume much more than a single tablespoon of processed oil each day. Add to this number of defective oil molecules the huge number of defective fat molecules from other processed sources, and you should be terrified at what you, your family, and your patients have been consuming for decades.

### Avoiding Fat Isn't a CAD Solution – PEOs Are

As the *New England Journal of Medicine* makes clear, “Diets high in polyunsaturated fat (PEOs) have been more effective than low-fat, high-carbohydrate diets in lowering cholesterol as well as the incidence of heart disease.”<sup>26</sup> The key is making sure the PEOs are unadulterated.

### Otto Warburg, MD, PhD: “Lowered Cellular Oxygen Equals Cancer!”

Just as oxygen deprivation causes heart disease, sustained oxygen deprivation causes cancer, too. Over 70 years ago, the Nobel prize-winning physician and master chemist Otto Warburg, MD, PhD, demonstrated that a sustained reduction of 35% in the level of cellular oxygen causes cancer and does so each and every time the deficiency occurs for an extended period. Oxygen deprivation is cancer's prime cause, and the high ratio of fermentation to respiration is cancer's prime characteristic<sup>27,28</sup> (Townsend Letter; August 2007. Available at: [www.brianpeskin.com/townsend.html](http://www.brianpeskin.com/townsend.html)). Cancer's prime cause, cellular hypo-oxygenation (hypoxia), was directly proved by American research scientists in the 1950s.<sup>29,30</sup> Back then, they didn't know how to increase cellular oxygenation, whereas, today, we do, and this is the key in answering



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► why the “statin/increased cancer” connection occurs and how to prevent its tragic consequences:

1. Warburg proved depressed cellular respiration and phosphorylation are the cancer-causing effects of decreased cellular oxygen.<sup>31</sup>

2. Physico-chemical experiments (Campbell et al.<sup>32</sup>) show that parent omega-6 (LA) can bind twice as much oxygen and disassociates (releases its oxygen) at a much higher pressure (physiologically useful), much closer to hemoglobin, than non-essential oleic acid does.<sup>32</sup> Therefore, the 35% cancer-causing hypo-oxygenation (deprivation) threshold is breached

with insufficient or adulterated parent omega-6.

3. Oxygen disassociation curves for oleic acid compared with LA prove a 50% reduction in oxygen transfer is possible.

4. Decreased cellular oxygenation can therefore systemically occur in any membrane; any tissue in the body can become a potential cancer site.<sup>32</sup>

Campbell et al.'s seminal experiment<sup>32</sup> conclusively showed a 50% reduction in oxygenation when a PEO deficiency occurred. Now, imagine this effect coupled with already lowered parent omega-6 esterified cholesterol from statins. The chain of events is as follows:

Lowered Cholesterol = Fewer PEOs =  
Less Cellular O<sub>2</sub> = CANCER

The body has no cholesterol sensor because the absolute cholesterol number is irrelevant.

We have already explained in detail that the common link between LDL cholesterol and decreased oxygenation occurs because cholesterol is esterified with large amounts of parent omega-6 before it is combined with lipoprotein as LDL particles for transportation within the body.<sup>15,33</sup> Even though statins increase the uptake of LDL cholesterol from the blood, they decrease overall cellular LA because absolute levels of cholesterol are decreased. This affects oxygen transmission across the cell membranes, since the structure of the phospholipids that form a major portion of the cell membrane are a reflection of the composition of unsaturated fatty acids and bioavailability in the blood.<sup>23</sup> It is known that fatty acids component of cell membrane phospholipids reflect diet.<sup>28</sup>

### Clinical Results: PEOs Combat Cancer

Physicians utilizing the Peskin Protocol report significant improvements in patient outcomes across a broad spectrum of disease conditions. Following is a description of the results when one of America's top cancer consultants and researchers incorporates the Peskin Protocol:

Peskin's *The Hidden Story of Cancer* has provided a great breakthrough in the treatment of our cancer patients. The addition of 11,000 mg Peskin Protocol EFA capsules t.i.d., along with our protocol, has brought a dramatic difference; unbelievable and rapid improvement:

- **Patient 1:** 62-year-old male – four-pack-a-day smoker. Stage IV lung cancer (42 tumors). Advised six weeks to live, 13 months on protocol complete remission of the 42 tumors as verified by X-ray.
- **Patient 2:** 82-year-old male. Prostate cancer with PSA of 4280 and alkaline phosphatase of 2463. Patient on morphine. After eight days, morphine no longer required. Within two months, pt PSA at 0.4 and alkaline phosphatase at 63. Patient made full recovery.
- **Patient 3:** 62-year-old male. Very large tumor in esophagus, unable to eat. Three months later, tumor decreased 75-80%, and patient can now eat.
- **Patient 4:** Female. Stage IV pancreatic cancer. Told it was hopeless. Eight weeks later, tumor reduced 75%.
- **Patient 5:** 48-year-old male. Mandibular area cancer; stage IV, spread to base of tongue and jaw. Patient treated by G Tube as jaw was sutured shut. Complete remission in four months.

We believe Peskin Protocol EFAs are the “missing link” in cancer therapy. The cost of treating our patients has dropped from \$20,000 (US) per month to \$1,500 (US) per month by *completely eliminating hospitalization*. We saw no side-effects. Within two weeks, patients typically see a great physical and mental improvement.

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Stage 4 Cancer Researcher and Consultant  
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### Defective LDL Cholesterol Becomes a “Defective Delivery System”

With the consumption of organic, unprocessed, PEOs from natural sources such as walnuts, almonds Brazil nuts, sunflower seeds, or their (unprocessed) cooking oils rather than adulterated oils and trans fats, LDL cholesterol should be made up of significant amounts of properly functioning LA. However, since LDL cholesterol is the transport vehicle for PEO delivery into your cells, it does not care about the state of the essential fatty acids it is carrying. LDL cholesterol will transport adulterated essential fats already damaged by food processors into the cell. It is primarily the adulterated (defective) parent omega-6 that causes plaque, not saturated fat. So, while statins reduce LDL cholesterol by reducing the defective parent omega-6 from processed food it is carrying, and therefore reducing plaque, at the same time the statins are lowering the transport of vital oxygenating unadulterated PEOs into the cells. This is the reason why patient cholesterol

numbers steadily decrease, yet patient heart attacks continue to increase.

The popular belief, even among physicians, is that the evidence like the 2007 METEOR trial (Crouse III, J, et al. Effect of rosuvastatin on progression of carotid intima-media thickness in low-risk individuals with subclinical atherosclerosis: the METEOR trial. *JAMA*. 2007; 297: 1344-1353), for example, shows there is a decrease in heart attacks in patients taking statins. The facts are that although cholesterol was lowered and so halted progression of atherosclerosis, in the placebo group, no patient suffered a serious cardiovascular event, whereas in the treatment group (rosuvastatin), there were eight serious cardiovascular events including heart attack and angina, a bad outcome. In addition, this randomized controlled trial had a number of serious flaws that were pointed out in an editorial in *JAMA*, which accompanied the article (Lauer MS. *JAMA*. 2007;297:1376-8).

As this article was going to press, another negative, unexplainable, and baffling result of statins was published on Reuters, December 3, 2007 (available at: <http://www.drbriffa.com/blog/2007/03/30/hailed-meteor-trial-results-not-as-stellar-as-we-are-led-to-believe/> or <http://www.reuters.com/article/healthNews/idUSN2922862020071129>). It included the following:

- "...[B]affled by findings indicating lower cholesterol levels were not linked to reduced stroke deaths.
- "I think all we can say is that we don't really understand what's going on here....
- "Because most of the benefit of statins in preventing cardiovascular events can be ascribed to the LDL reduction, it is puzzling that LDL cholesterol is not associated with stroke risk."

For the first time, this baffling outcome is now both predictable and explained.

Any drug that *artificially* lowers cholesterol *also* lowers transport of cancer-fighting, oxygenating PEOs!

Lowered Cholesterol = Fewer PEOs =  
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The body has no cholesterol sensor because the absolute cholesterol number is irrelevant.

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**AA from parent omega-6 (LA) contributes to smooth working of vascular function and increased blood flow. AA provides eicosanoids for response to injury—acting as a healer—helping to heal vascular injury.**

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### Stop Blaming Cholesterol

LDL cholesterol continues to be improperly blamed for a myriad of health problems, while the real culprit is defective PEOs. LDL cholesterol has no alternative but to transport these killers throughout our body since, due to food processors' requirement for extended shelf-life in the oils they sell, we have insufficient properly functioning LA in our diets. The nutritionists never make this critical connection and incorrectly identify the "problem" as LDL cholesterol. To repeat: the reason for the ineffectiveness of statins to stop heart disease is they simply can't eliminate enough of the defective PEOs being transported in LDL *esterified* cholesterol. In addition, they simultaneously remove correctly functioning PEOs, because they reduce its cholesterol carrier – a doubly bad effect. You now understand why the absolute LDL number is irrelevant if the diet contains sufficient unadulterated PEOs. Statins don't discriminate between eliminating functional, unadulterated PEOs and nonfunctional, adulterated PEOs.

### Reducing LDL Cholesterol Increases Blood Clots and Facilitates Metastasis of Cancer

Defective parent omega-6 is also the root cause of thrombosis /blood clots forming in the arteries and then being unable to dissolve away naturally, as they do with external cuts. As referenced earlier, blood clots are a tremendous problem with cancer

cases, responsible for over 80% of the cancer mortality rate, because they facilitate cancer transport throughout the body when it would not have spread otherwise. This fact was known in 1958.<sup>34,35</sup>

Experiments from Florida Hospital Institute of Translational Research show that blood clots are often caused by biochemical factors contained in small cancerous tumors, like Tissue Factor (TF), that otherwise is found only in normal tissue – not in the blood – that normally causes *clotting only from vascular injury*. When cancer cells carrying TF enter the blood, small clots are formed on the cancer cell's surfaces. The blood platelets, which are small cells that stick to injured blood vessels to help prevent blood loss, then stick to the clot-covered cancer cell. This sticky "sandwich" of cancer cell, blood clot, and platelets is able to stick to the inside of the blood vessel wall. A clot provides a "safe haven" for the cancer cell, giving it the time it needs to squeeze between the cells that line the blood vessel and escape into the tissues, where it can multiply into a secondary tumor.

### Arachidonic Acid Is Important to Counteract Cancerous Clotting and CAD

Humans obtain arachidonic acid (AA) either ready-made in food or from the parent omega-6, if it is unadulterated. AA is not harmful: it is the precursor to prostacyclin – the most potent anti-aggregatory agent (natural "blood thinner") and inhibitor of platelet adhesion.<sup>36</sup> Lowering esterified LA through the lowering of LDL cholesterol automatically decreases the body's natural anti-



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➤ aggreatory AA.<sup>36</sup> In view of the above, this is a very bad effect as it will directly lead to increased risk of a blood clot and ultimately contracting cancer (and CAD).

### Atherogenesis, Adulterated PUFAs, and LDL Cholesterol: More Connections

The eminent researcher HM Sinclair published his finding that PEO deficiency causes an enormous permeability increase in skin along with increased capillary fragility.<sup>37</sup> We will use this information and connect it to the vascular system in an unexpected way.

### Intima is 100% Parent Omega-6

We need to know the innermost arterial layer, the intima, is epithelial tissue that is 100% parent omega-6 (LA); there is *no* omega-3 in skin.<sup>38,39</sup> The delicate intima requires unadulterated parent omega-6 and doesn't get enough because of surplus adulterated fats or because statins decrease LDL cholesterol, which transports the parent omega-6 and lowers the associated LA to hypo-oxygenating, cancer-causing levels.

The authors of a 1982 *British Medical Journal* (BMJ) article understood the Parent Essential Oil connection in 1982, but few of us heard the news reported in that article that LA and *most* polyunsaturated fatty acids, including AA and EPA,

were found to be lower (depleted) in heart attack victims. Their conclusion was that the fatty acid patterns of the phospholipids [PEOs] constitute an independent risk factor for heart disease.

This *BMJ* article "hits the nail on the head." Deficiency of PEOs is associated with increased heart attack risk. Don't think that the solution is to minimize parent omega-6 (along with parent omega-3), because of "oxidation" concerns. It is true that, in part, fats and oils oxidize for energy. Normal oxidation of fatty acids (for energy production) proceeds in the mitochondria via beta oxidation after activation by acyl-CoA synthetase.

Adulterated parent omega-6 deposits in cell membranes lead to *abnormal oxidation* – oxidation from adulterated oils at the site of vascular injury causing injurious inflammation. Abnormal oxidation involves formation of hydroperoxides from the double bonds of the PEOs. This harmful partial oxidation involves no energy (ATP) production.

All cells oxidize fuels for energy, and this is a normal process. However, food processing oxidizes PEOs, which prematurely *become nonfunctional foods*, causing vascular injury and destroying the body's inherent repair mechanism.

### Medical Journals Often Unknowingly Mislead

Medical journal publications and some of the pharmaceutical manufacturers continue with the

discredited theory that somehow your body's own cholesterol "causes" heart disease, so researchers continue to "discover" different types and sizes of cholesterol particles. Think about this conclusion: *The body has no cholesterol sensor because the absolute cholesterol number is irrelevant.*

### The Solution

1. Ensure that patient's diet contains generous amounts of unadulterated PEOs with a ratio of LA:ALA greater than 1:1 and less than 2.5:1 by eating unadulterated, unprocessed foods. To make simpler and easier with noncompliant patients, patients should consider supplements.<sup>41</sup>
2. Have patients minimize foods containing significant amounts of trans fats (hydrogenated), interesterified fats, and other adulterated hypo-oxygenating fats.

My research strongly supports the (prophylactic) use of an unprocessed organic supplement with a ratio of parent omega-6 to parent omega-3 between 1:1 and 2.5:1. With this ratio, suggested use is 725 mg. per 40 lb. of body weight (e.g., 3 grams for a 160-lb. person on a daily basis). I term this the "Peskin Protocol." Cancer patients require significantly more. (For an in-depth analysis of how this specific ratio is determined, see "The Scientific Calculation of the Optimum Omega-6/3 Ratio" at [www.CambridgeMedScience.org](http://www.CambridgeMedScience.org) ["Optimum PEO Ratio"] or [www.BrianPeskin.com](http://www.BrianPeskin.com) ["EFA Report"].) ♦



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