



C.I.I.M.S.

CambridgeMedScience.org

CAMBRIDGE INTERNATIONAL INSTITUTE FOR MEDICAL SCIENCE

Stephen Cavallino, M.D. - Founder & Chairman (Italy) • Amid Habib, M.D. • David Sim, M.D. • Robert Nemer, D.O.

THE PHYSICIAN'S CONCISE GUIDE TO:

- 12 -

A New Look at Cholesterol, Clogged Arteries & EFAs

*Reprinted with permission by Pinnacle Press
from **The Hidden Story of Cancer.***

© 2007



Dedicated to advancing and publicizing breakthrough discoveries in the health sciences

There is simply no one better in the 21st century at developing practical health-related solutions based on the world's leading medical and nutritional science. **"Science - Not opinion" is Brian's trademark.** When Brian is through explaining a topic it is "case closed!" When he says it, you "can take the information to the bank!"

Unlike most of his peers' recommendations, Brian's health and nutritional recommendations have stood the test of time. **Brian has never had to reverse or significantly alter any of his medical reports—reports that have tackled everything from the dangers of soy, to the wrongly popularized need for fiber in the diet, to his warning about the potential harm of supplementing with copious amounts of omega-3.** In 1995 he published the report "Fiber Fiction" and finally, eleven years later, others in research are acknowledging the silliness of recommending fiber in the diet of a human being. Brian's latest crusade is to warn of the dangers of excess omega-3 (in particular, fish oil) and how it will lead to increased cases of skin cancer. The list goes on and on...

Brian received an appointment as an Adjunct Professor at Texas Southern University in the Department of Pharmacy and Health Sciences (1998-1999). **The former president of the University said of his discoveries: "...His nutritional discoveries and practical applications through *Life-Systems Engineering* are unprecedented."** Brian earned his Bachelor of Science degree in Electrical Engineering from Massachusetts Institute of Technology (MIT) in 1979. Brian founded the field of *Life-Systems Engineering Science* in 1995. This field is defined as *The New Science of Maximizing Desired Results by Working Cooperatively with the Natural Processes of Living Systems*. To many, Brian is THE MOST TRUSTED AUTHORITY ON HEALTH AND NUTRITION IN THE WORLD.

Brian continues to be a featured guest on hundreds of radio and television shows both nationally and internationally. His sheer number of accomplishments during the last decade of the 20th century and into the 21st century are unprecedented and uniquely designate him as the #1 authority in the world of what really works and why. Forget listening to the popular press or most popular so-called health magazines. Their editors simply don't understand the complicated science that they write about – they merely "parrot" what everyone else says without independent scientific verification. Their recommendations often have no basis in reality of how the body works, based on its physiology.

Brian has dedicated his life to provide the truth – which is almost always opposite to what everyone says. Here's why Brian is the #1 man in America to listen to when it comes to your health.

The Easy Solution: The Peskin Protocol PEOs™

Parent Essential Oils (PEOs™): The DIFFERENCE

I am often asked how my EFA-based recommendations differ from others. The answer is simple but very significant. The term “Essential Fatty Acids” is being misused so frequently that I was compelled to coin a new phrase, *Parent Essential Oils* (PEOs™).

This term “Parent Essential Oils” refers 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 a small percentage of them—about 5%—into other biochemicals called “derivatives,” while **leaving the remaining 95% in parent form**.

This is crucial to understand. There are a host of omega-6 and omega-3 oils being sold as EFAs that are *not* EFAs, but rather nonessential derivatives such as EPA, DHA, and GLA. Fish oils are made up almost exclusively of omega-3 *derivatives*. Scientifically and biochemically, calling derivatives such as EPA, DHA and GLA by the term “EFA” is wrong. **Derivatives are not EFAs because they are not essential**—your body has the ability to make them *as needed*. My research has shown that supplementing with the derivatives so commonly found in the marketplace and mislabeled as “EFAs” can easily be harmful to your health.

Why are the parent forms—PEOs™—so important? Many of the EFAs sold in the stores consist of manufactured EFA derivatives. To be clear, your body doesn’t need or want these derivatives, because it makes its own derivatives out of the **Parent Essential Oils** (PEOs™) you consume *as it needs them*. Taking fish oil and other health-food-store “EFAs” often overdoses you with derivatives, which can be very harmful.

Don’t make the common “EFA mistake” by unknowingly substituting derivatives for parents! **Since the term has become so confused by so many it is time to focus on the essence of what they are and why they are so vital to our health and well being.**

**From this point forward it is Parent Essential Oils (PEOs™)
that get center stage.**

A New Look at LDL Cholesterol, Clogged Arteries and PEOs™

Cholesterol-lowering drugs are among the pharmaceutical industry's top money makers. Billions and billions of dollars are spent each year in the hopes of "lowering the 'bad' cholesterol." You will soon discover how wrong this method is, why heart disease or cancer can't be helped sufficiently by these drugs or this approach, and which substance transported by LDL cholesterol is *really* the villain for which cholesterol mistakenly gets a bad rap.

I owe tremendous thanks to Dr. David Sim, a leading interventional cardiologist, for his significant assistance in developing this section. I also thank Dr. Stephen Cavallino, a leading emergency physician practicing in Italy, who was desperately looking for the reason why the majority of his emergency room heart patients **suffered heart attacks in spite of normal cholesterol levels**. The following section represents a collaborative effort. After reading this section you will discover more about the function of cholesterol than likely many physicians understand.

Drug company advertisements on television seem calculated to "scare us to death" when they contain statements like these:

"When diet and exercise don't lower cholesterol enough...."

"Cholesterol comes from the foods you eat and your grandparents"

"The 'bad' cholesterol"

The advertisement may even make this amazing statement: **"Not shown to prevent heart attack or heart disease...."**

Scary, isn't it? We are told that "bad" cholesterol (LDL) isn't low enough, your **likelihood of a heart attack isn't even lowered by the drug**, yet we **never question the reasoning** behind statements that cholesterol should **be made *artificially* lower than its natural levels**. It's called the power of advertising. Cholesterol advertisements are on television constantly. Soon after seeing them, we automatically think that lowering cholesterol is "the answer" to decreased heart disease when nothing could be further from the scientific truth.

The same method was used in the early 1980s to sell quartz heaters. The ads appeared to be based on science. They became a fad because these heaters promised to be "much more efficient" and energy-saving, too. Both facts are wrong. There is no difference in quartz vs. any other space-heaters being sold. Nonetheless, even some scientists got fooled into buying them. As physicist extraordinaire Dr. Lewis Epstein states, "Have you ever noticed that if you don't really understand something, but you know the 'right words,'

people who also do not understand will often think that you do?"¹This is why we can be misled by "official-sounding" sources. Don't forget Nicolas Tesla's warning on page 20 to always think clearly, not just deeply. *Life-Systems Engineering Science* always predicates any conclusion on this insight.

You need to know that *heart disease was nearly nonexistent in 1920 when the American diet was based on meat and potatoes*. In fact, the inventor of the EKG was told his invention wasn't needed because heart disease (myocardial infarction) didn't significantly exist. There were few cereal or milk advertisements back then, so milk and cereal weren't heavily consumed.

* * *

Compare this with the situation today. Pharmaceutical companies never let up on television and print advertising in an attempt to get us to lower our cholesterol. This section will answer why you are told to lower LDL cholesterol and will give you the medical facts about cholesterol that big pharmaceutical companies hide from physicians. When a pharmaceutical company's drug test fails, they may try to stop the study before the negative results are published. They may even attempt to discredit any negative information about the drug. The physician may never know of the negative effects of a drug that he so glowingly recommends; the drug rep only gave the positives to him, so both he and the public is misled.

Statin drugs are those used to control cholesterol levels in the body. A 2001 study found,

"Statins and polyunsaturated fatty acids have similar actions.... In view of the similarity of their actions and that statins influence essential fatty acid metabolism, it is suggested that EFAs and their metabolites may serve as secondary messengers of the action of statins...."²

These statements mean that **EFAs (PEOs™) naturally accomplish what statin drugs try to do by decreasing cholesterol levels**. While this by itself can help speed blood flow, this is not the most important thing to know about PEOs™ in relation to cholesterol and clogged arteries.

1 *Thinking Physics: Practical Lessons in Critical Thinking*, Lewis Carroll Epstein, Ph.D., Insight Press, San Francisco, CA, 1987. All of the heater's energy goes into heat because there is nowhere else for a heater's energy go. Fans and reflectors disperse the heat but they have been used for decades and are nothing new.

2 "Essential Fatty Acids as Possible Mediators of the Action of Statins," *Prostaglandins, Leukotrienes and Essential Fatty Acids*, Vol. 65, No. 1, July 2001.

Arterial Plaques—It’s Not the Saturated Fat—It’s the Adulterated Parent Omega-6 that Clogs Arteries and Impedes Blood Flow!

Contrary to what we have heard for decades, it is **not** the saturated fat you eat that clogs your arteries! How do we know this? A 1994 *Lancet* article reported investigating the components of arterial plaques. In an aortic artery clog, they found that **there are over ten different compounds in arterial plaque, but NO saturated fat.**³

There *was* some cholesterol in the clog. This is explained by the fact that cholesterol acts as a protective healer for arterial cuts and bruises. So what is the predominant component of a clog? You probably guessed it—the *adulterated* omega-6 polyunsaturated oils we have spoken about so extensively—those that start out containing good PEOs™ but are ruined during commercial food processing and sold at the supermarket in thousands of products.

Many analyses have been carried out regarding arterial clogs and published in the medical journals, but few physicians have seen them. The average person has little, if any, chance of ever seeing the truth. Two of these publications are listed below.⁴

Contrary to what we have heard for decades it is **not the cholesterol itself that is clogging your arteries.** Something to think about is the fact that a cat, a true carnivore, lives on a diet of 100% meat. They consume lots of cholesterol, saturated fat, and “red” meat. By “popular wisdom” cats should be suffering massive heart attacks on a regular basis, but they don’t. Contrary to popular belief, humans are much closer to a wolf with a 4-pint stomach that can eat once every few days than to a cow or sheep with an 8 ½ gallon stomach that has to eat constantly.⁵

As the medical textbook, *Molecular Biology of the Cell* on page 481 makes clear, **cholesterol is necessary** for the structural integrity of the lipid bi-layer (the structure **in each of our 100 trillion cell membranes**).

This is precisely why the *Journal of the American Medical Association*, No. 272, pgs 1335-1340, 1994, published an article stating that **cholesterol-lowering drugs do not work significantly to prevent heart disease.** In 1993, a report titled “Cholesterol Screening and Treatment” was released by the University of Leeds in England. Drugs for lowering high cholesterol levels were given to a study’s participants. *The patients whose cholesterol was artificially lowered with drugs developed heart disease just as frequently as the drug-free cholesterol group.* There were more health problems among the group taking the drugs! The authors stated that few people identified purely on the basis of cholesterol levels will

3 “Dietary polyunsaturated fatty acids and compositions of human aortic plaque,” Felton, CV, et al., *Lancet*; 344:1195-1196, 1994.

4 Waddington, E., et al., “Identification and quantification of unique fatty acid and oxidative products in human atherosclerotic plaque using high-performance lipid chromatography,” *Annals of Biochemistry*; 292:234-244, 2001; Kuhn, H., et al., “Structure elucidation of oxygenated lipids in human atherosclerotic lesions,” *Eicosanoids*; 5:17-22, 1992.

5 http://www.second-opinions.co.uk/carn_herb_comparison.html

benefit from drug treatment. The study *discourages general cholesterol screening*. Despite these findings, England's estimated number of prescriptions for cholesterol-lowering drugs is increasing by 20% per year.

This is the reason why cholesterol drugs can't do the job:

An explosive article published in the 2007 issue of *Journal of the American College of Cardiology*⁶ 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 (low density lipoprotein) 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*. Wouldn't it be more desirable to lead a full life while also avoiding both cancer and heart disease?

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 the *Journal of the American Medical Association (JAMA)* over ten years ago when they concluded that low cholesterol, by itself, did not significantly prevent heart disease⁷:

"Our findings do not support the hypothesis that hypercholesterolemia [high LDL cholesterol levels] or low HDL-C [high density lipoprotein cholesterol—aka "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." (Emphasis added.)

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*⁸:

- "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 *simplistic and scientifically untenable hypothesis*." (Emphasis added.)

6 Alsheikh-Ali A, et al., "Effect of the magnitude of lipid lowering on risk of elevated enzymes, rhabdomyolysis, and cancer," *J Am Coll Cardiol*. 2007;50:409-418.

7 Krumholz HM, et al., "Lack of association between cholesterol and coronary heart disease mortality and morbidity and all-cause mortality in persons older than 70 years," *JAMA*. 1994;272:1335-1340.

8 Colpo A., "LDL Cholesterol: 'Bad' cholesterol or bad science," *J Am Phys Surg*. 2005;10:83-89.

The *Journal of American College of Cardiology* published “Beyond Low-Density Lipoprotein Cholesterol: Defining the Role of Low-Density Lipoprotein Heterogeneity in Coronary Artery Disease,” reporting more discouraging findings (Mudd et al, 2007; 50:1735-1741):

- “[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.” (Emphasis added.)

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

The popular belief, even among physicians, is that the evidence like the 2007 METEOR trial (“Effect of Rosuvastatin on Progression of Carotid Intima-Media Thickness in Low-Risk Individuals With Subclinical Atherosclerosis: The METEOR Trial,” Crouse III, J, et al., *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 halted progression of atherosclerosis*, in the placebo group no patient suffered a serious cardiovascular event whereas in *the treatment group (rosuvastatin) there were 8 serious cardiovascular events including heart attack and angina, a bad outcome.*⁹ 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).

Another negative, unexplainable and baffling result of statins was published on Reuters, 3 December 2007¹⁰:

- “...[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.

“LDL [cholesterol] contains up to 80% lipid [fats and oils], including polyunsaturated fatty acids and cholesterol, mainly esters. **Linoleic acid (LA), one of the most abundant fatty acids in LDL...**”¹¹ (Emphasis added.)

It’s what the cholesterol is transporting, **the adulterated fats**, that is the problem.

An article in *Human Nutrition: Clinical Nutrition* explains that it is *parent omega-6* that makes up most of the fatty acids in LDL and HDL cholesterol:

9 <http://www.drbriffa.com/blog/2007/03/30/hailed-meteor-trial-results-not-as-stellar-as-we-are-led-to-believe/>.

10 <http://www.reuters.com/article/healthNews/idUSN2922862020071129>.

Atherosclerosis Reports; 6:477-484, 2004.

“Linoleic acid [parent omega-6] comprises about 55 per cent [the majority] of the fatty acids in cholesterol esters of LDL and HDL, AND about 20% of the free fatty acids in the phospholipids in each class...”

“...It must also be remembered that all tissues need EFA which must come from the diet and for most tissues *through the plasma* [blood] where they are almost entirely transported in lipoproteins, mainly in their cholesterol esters and phospholipids.”¹² (Emphasis added.)

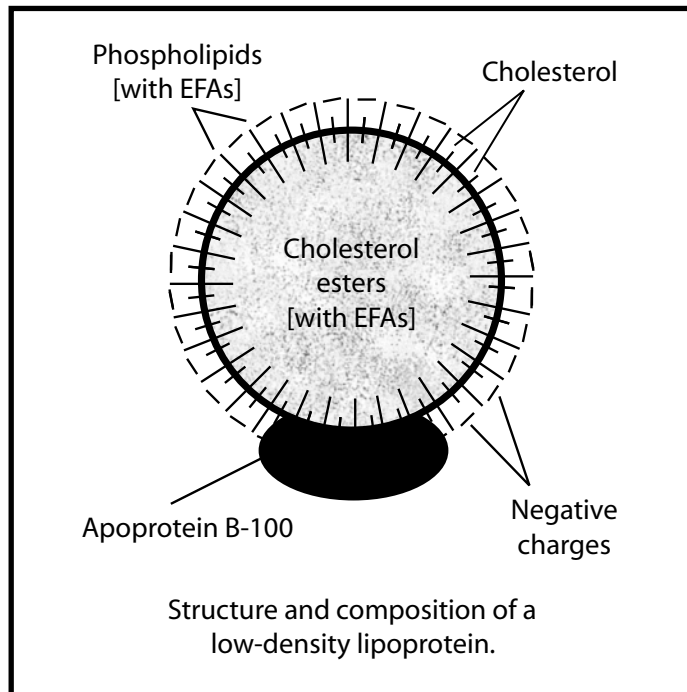
► *Life-Systems Engineering Science Commentary*

We clearly see that **parent omega-6, linoleic acid, comprises 75% (3/4th) – 55 percent PLUS 20 percent – of the plasma cholesterol-related structure.** Virtually every cell membrane in your body is composed of a phospholipid bilayer – with a FLUID CONSISTENCY comparable to light oil¹³ – and plenty of cholesterol-related compounds are in each cell membrane, too. Don’t let anyone ever tell you that natural fats are “bad.” One hundred trillion cells need lots of EFA-containing natural fats; in particular parent omega-6. If just a little of this parent omega-6 is defective, reducing its ability to absorb oxygen and perform other cellular functions, it acts as a direct cause of both heart disease and cancer.

The **oxidized** parent omega 6 **in the phospholipids** found in the lipoproteins—fats surrounded by protein in the bloodstream for easy transportation—AND defective parent omega 6 in the cholesterol esters are the main causes of heart disease—**not the cholesterol itself**. Any condition leading to decreased blood flow, with its consequent increased likelihood of forming clots, helps a localized cancer to spread (metastasis)! We need to avoid slow blood flow.

12 “Essential Fatty Acids in Perspective,” Sinclair, H.M., *Human Nutrition: Clinical Nutrition*, (1984) 38C, pages 245-260.

13 http://www.abbysenior.com/biology/transport_across_membranes.htm. “The phospholipid bilayer is the structural element that forms the physical boundary of the cell membrane. Materials which can dissolve in fat, like alcohol, can move across phospholipid bilayer with ease. Water **soluble** substances are unable to cross through the bilayer and must enter the cell through channel proteins. The cell membrane is made up a phospholipid bilayer [double layer] with proteins embedded in it. **The phospholipid bilayer has a fluid consistency, comparable to light oil [because it is oil-based].**” (Emphasis added.)



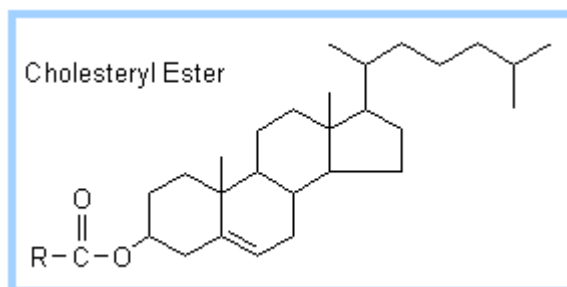
Textbook of Medical Physiology, pg 874

Esterified cholesterol comprises the **majority** of LDL. LDL is the acronym for **Low Density Lipoprotein**. LDL is more than “cholesterol” although many people, including nutritionists and physicians, don’t understand this. It is essential to understand the term cholesterol “esters” if you hope to understand the vital role of LDL in your body. *Harper’s Illustrated Biochemistry* (26th edition) on page 219 addresses this important issue in their description:

“Cholesterol is present in tissues and in plasma either as free-cholesterol or **in a storage form, combined** with a long-chain fatty acid [**containing PEOs™**] as a **cholesterol ester**. In plasma, both forms are transported in **lipoproteins**.” (Emphasis added.)

And from *Harper’s Illustrated Biochemistry*, pg 224, we discover that dietary cholesterol is tied to PEOs™, too:

“Of the **cholesterol absorbed, 80 - 90% is esterified [with PEOs™]** with long-chain fatty acids in the intestinal musoca.” (Emphasis added.)



Note: Some of the fatty acids in the body are esterified with cholesterol (see drawing above). **The structure of cholesterol itself never changes, merely the esterified moiety—the acyl side chain.** That’s a **big** difference that most physicians and nutritionist may not understand. This is a simple condensation reaction, removing the water, catalyzed by the enzyme ACAT (Acyl CoA: Cholesterol Acyl Transferase) between a fatty acid and cholesterol. R symbolizes the hydrocarbon portion of the fatty acid. For example, if oleic acid were esterified with cholesterol, then R would be $-C_7H_{14}CH=CH-C_8H_{17}$ with the double bond in cis configuration. (Thanks to Dr. Marissa Carter, Ph.D. in biochemistry, for the clarification.)

EFA Deficiency = Defective Cholesterol Structure

It was known in 1941 that EFA deficiency caused a defective cholesterol structure and in 1956 that **carbohydrates are also a culprit in causing defective cholesterol structure**, but the popular press rarely mentions these facts:

“Cholesterol is normally esterified with unsaturated fatty acid [PEOs™]¹⁴ and when—as in our experiments—these are extremely deficient in the body it is esterified **with much more saturated fatty acids** synthesized in the body **from carbohydrate.**”¹⁵ (Emphasis added.)

1965: An Important Experiment Furnishing WRONG RESULTS

An important experiment was performed in 1965, long before the pharmaceutical companies created what I term the “bad cholesterol annuity.” This experiment was performed at the Karolinska Institute in Sweden. (Note: a committee from this Institute appoints the laureates for the Nobel Prize in Physiology or Medicine.) In their experiment, the researchers fed patients different oils to determine fat absorption parameters; the outcome was amazing:

- “...[T]here is also a **preferential incorporation of oleic acid** [a monounsaturated, omega-9 such as that found in olive oil] into the cholesterol esters, relative to other fatty acids tested [including parent omega-6].
- “It is clear from these results [in humans] that the process of lymph cholesterol ester formation during fat absorption **showed far greater affinity for dietary oleic acid** than for the other fatty acids studied.

14 Kelsey, F.E., Longenecker, H.E., *J. Biol. Chem.*, 1941, Vol. 139, page 727

15 H.M. Sinclair, “Deficiency of Essential Fatty Acids and Atherosclerosis, Etcetera,” *Lancet*, April 7, 1956.

- “During fatty acid absorption lymph cholesterol ester formation showed **marked specificity for oleic acid** relative to other fatty acids tested [including parent omega-6].¹⁶ (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

These results were completely different from those reported in previously published articles. For example, it was known in **1941 that cholesterol prefers PEOs™** in its structure over omega-9. (*The Journal of Biological Chemistry*, 1941, Vol. 139, page 727.) Also, an opposite finding was published again in 1956 in one of the world’s top medical journals, *Lancet*, because Dr. Sinclair knew it that cholesterol prefers PEOs™ in its structure. (H.M. Sinclair, “Deficiency of Essential Fatty Acids and Atherosclerosis, Etcetera,” *Lancet*, April 7, 1956.) Experiments in 1976 and 1990 got much different results than the researchers of 1965. As Dr. Carter, Ph.D. states, there are a myriad of technical reasons why the experimenters in 1965 could have made this error.

1976: An Important Experiment: Defective Cholesterol = Lack of Oxygen

In 1976, the medical journal *Pediatrics* investigated abnormal fatty acid composition and impaired oxygen supply. They showed EFA deficiency leads to the exact conditions Dr. Warburg showed was always cancer-causing – lack of cellular oxygen.

Proof: Insufficient Parent Omega-6 is Cancer-Causing!

You will be amazed by a discovery made more than 30 years ago and it is the **direct proof of how defective/insufficient parent omega-6 (LA) DE-OXYGENATES by 50% – well in EXCESS of the 35% deficiency that Dr. Warburg proved was cancer-causing.**

Here’s what the investigators found:

- “...[W]e have proposed that the **cellular lipids** may be involved in the **facilitation and regulation** of the supply of oxygen to the cells...
- “We have already reported that, although the saturates, such as palmitates, have little or no affinity for oxygen, the unsaturates [including PEOs™] are capable of undergoing reversible **oxygenation in response to changes in oxygen pressure**. Because two unsaturated carbon-carbon bonds are required for the reaction, each linoleic [parent omega-6] molecule can bind with one molecule

¹⁶ “Intestinal Absorption and Esterification of C-Labeled Fatty Acids in Man,” *Journal of Clinical Investigation*, Blomstrand, Rolf, et al., Vol. 44, No. 11, 1965.

of oxygen with it, but **two oleic molecules must bind one oxygen** between them. (Note: Parent omega-6 is twice as effective in oxygen transfer.

- “Underwood’s group has shown that, in cystic fibrosis, the abnormality in fatty acid composition is not restricted to the erythrocytes and plasma. **Interference with the movement of oxygen could then occur at any cell membrane** so that there could be a **general reduction in the supply of cellular oxygen** throughout the body....
- “[S]uch a condition could **depress the rate of *cellular respiration*, *phosphorylation***, and all energy-dependent processes.
- “...[I]t seems possible that that many of their symptoms may result from **essential fatty acid (linoleic) deficiency**, leading to the **decrease in the availability of cellular oxygen for respiration.**”¹⁷ (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

1. Physical-chemical experiments show that linoleic acid (parent omega-6) **can bind twice as much oxygen** and disassociates (releases its oxygen) at a much higher pressure (physiologically useful), much closer to hemoglobin, **than oleic acid** does.
2. **Oxygen disassociation curves for oleic acid compared with linoleic acid**, parent omega-6, **shows a 50% reduction in oxygen transfer.**

I discovered this article belatedly in 2006, after the first edition of this book had been printed. Dr. Campbell and his team should be congratulated for conclusively proving, on a biochemical basis, that an EFA deficiency of functional parent omega-6 sets up the exact conditions Dr. Warburg showed were cancer-causing: lack of cellular respiration. Dr. Campbell, et al., performed both brilliant theoretical and experimental work! It is tragic they never met Dr. Warburg, since I believe together they might have solved the anticancer puzzle much sooner.

This experiment conclusively shows oxygenation decreased 50% when an EFA deficiency occurred. None of us can afford this consequence if we want to maintain our cancer-free health.

1990: “Effects of Lipids on Cancer Therapy”: Cell Membrane Structure Modification

The journal article in *Nutrition Reviews* confirmed the preferential use of parent omega-6 over omega-9 in cell membranes:

17 Campbell IM, Crozier DN, Caton RB: Abnormal fatty acid composition and impaired oxygen supply in cystic fibrosis patients. *Pediatrics* 57, 480-486, 1976.

- “The structure of membrane proteins and the carbohydrate chains of membrane glycoproteins is genetically determined and does not change in response to differences in the type of amino acids or sugars available to the cell. By contrast, **the structure of the phospholipids that make up the lipid bilayer of the membrane depends to a considerable extent on the type of fatty acid available in the extracellular fluid.**
- “The plasma membrane of the L1210 murine leukemia cells from the animals **fed sunflower seed oil have a totally polyunsaturated fatty acid content 18% greater than** that of animals **fed coconut oil** [a highly saturated fat]. This increase is primarily due to an increase in linoleic acid [parent omega-6].
- “Conversely, tumor cells from **animals fed the saturated fat-rich coconut oil diet have a greater proportion of monounsaturated fatty acids**, particularly oleic acid (18:1).”¹⁸ (Emphasis added.)

These animal experiments in 1990 showed that **in the presence of insufficient un-processed parent omega-6**, the cell structure will **incorporate oleic acid** (non-essential omega-9 **such as that found in olive oil**) instead. So we have discovered that both the cell itself and the cholesterol-structure of the cell require plenty of parent omega-6.

Therefore, the researchers in 1965 must have made a mistake.

2001: Consequences of ω -6 Oleate Desaturase Deficiency on Lipid Dynamics and Functional Properties of the Mitochondrial Membrane:

An analysis of defective mitochondria in the *fad2* mutant of *Arabidopsis thaliana* from an article in *The Journal of Biological Chemistry* confirms substitution of parent omega-6 with non-EFA oleic acid (omega-9):

- “Experiments were carried out with the *fad2* mutant of *Arabidopsis thaliana*, which belongs to a family of monogenetic mutants **deficient in fatty acid desaturase activities.**
- “**Oxidative phosphorylation parameters** such as oxidation rates and activation energy of electron transport **were analyzed.**
- “...A **drastic reduction in the amount** of PUFA, linoleic acid (18:2) [**parent omega-6**], and linolenic acid (18:3) [**parent omega-3**] [was] observed in *fad2* mitochondria.
- “**As a consequence, the amount of oleic acid (18:1)** [non-essential omega-9] was **considerably enhanced (~ 10 times)** since it represented about 75% of total fatty acids.

18 Burns, C.P. and Spector, A.A., “Effects of Lipids on Cancer Therapy, *Nutrition Reviews* 48, No.6, 233-240, 1990 pages 381-383.

- “Functional parameters such as **oxygen consumption rate under phosphorylating and nonphosphorylating conditions and proton permeability of the inner mitochondrial membrane were significantly reduced...**”¹⁹ (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

This experiment made use of genetically defective mitochondria, once again showing that non-essential omega-9 was substituted for the essential parent omega-6. As the last point above shows, the *prime* cancer-causing condition appeared: the oxygen consumption was significantly reduced.

Why the 1965 Experiment Went Wrong?

We can now return to the experiment furnishing wrong results starting on page 282. Their published results make no biochemical sense. That is, there would be no good biological reason to see such a result. In 1965 those medical researchers thought that a non-essential fat, oleic acid, was *supposed to be* preferentially incorporated into the cholesterol structure. In fact, this only occurs when there is not enough parent omega-6 or it is defective. They had no understanding that the cholesterol was a major transport mechanism of PEOs™ into the cell.

Could these scientists at one of the world’s most prestigious institutes for higher education in medicine have made a fundamental mistake; one that today’s cancer researchers continue to make: Unknowingly substituting a “purified” but non-functional omega-6 for functional parent omega-6?²⁰ (Dr. Carter offers insight on this point in the footnote below.) Regardless of the reasons their results were wrong, they were still published. Publishing of incorrect results by medical journals is another reason that medical researchers don’t know what to believe and often don’t believe what is published in their own medical journals.

19 Caiveau, O., et al., “Consequences of ω -6 Oleate Desaturase Deficiency on Lipid Dynamics and Functional Properties of the Mitochondrial Membranes of *Arabidopsis thaliana*,” *The Journal of Biological Chemistry*, Vol. 276, No. 8, February, 23, 2001, pages 5788-5794.

20 Conducting fat metabolism studies in humans is fraught with complications, even today. In principle, Blomstrand, et al.’s experimental design was a reasonable one for its time, though there are a number of potential sources of error, which we can’t quantify since the level of experimental detail in the paper is insufficient. (1) The experiments did not use healthy subjects (most had various stages of cancer); (2) We do not know their overall PUFA status before the experiment was started (this would have influenced how the fatty acids were ultimately used by the body); (3) The investigators used ¹⁴C radiolabeled fatty acids to follow the fate of several types of fatty acid, including linoleic acid, but although the radiolabeled fatty acids were pure, because not all the radioactivity was recovered from the experiment, (> 50% unaccounted for), we do not know if the results they obtained are completely correct; (4) **We do not know the source of the non-radiolabeled fatty acids they used, nor their purities (the linoleic acid, parent omega-6, could have been substantially impure).** (5) The experimental work-up of body fluids and analytical analysis employed did not preclude the possibility that errors were made; (6) Finally, only 4 subjects were used in the experiment, which is a very small number. For these reasons, the results of the experiment cannot be generalized to all humans.

Defective LDL Cholesterol Becomes a “Poison Delivery System”

As you have already discovered, huge numbers of molecules in the omega 6-based cooking oils are ruined by commercial food processing. The body then incorporates these adulterated oils **into** the LDL cholesterol. With the consumption and transport of **defective**, cancer-causing processed oils, LDL cholesterol acts like a “poison delivery system,” bringing deadly trans fats and other ruined oils into the cells. It is primarily the oxidized (altered) parent omega-6 that clogs the arteries, NOT saturated fat! Renowned interventional cardiologist, Dr. David Sim, makes a great analogy that anyone can understand:

“It’s like building a wall without having enough bricks. You use another material and ‘fill the hole,’ but it doesn’t work correctly. The same thing happens when cholesterol doesn’t have enough parent omega-6 to incorporate.”

In nature, with the consumption of organic, unprocessed PEOs™ rather than adulterated oils and trans fats, LDL cholesterol should be made up of significant amounts of properly functioning “parent” omega-6, linoleic acid (LA), and as a result it will not be harmful. Furthermore, it is **the natural transporter of parent omega-6 and parent omega-3 into the cells**. That’s why it is not necessary to lower LDL cholesterol, nor is the absolute LDL number as important, when the diet contains sufficient unadulterated PEOs™. Also note the body has no natural “cholesterol sensor” in the bloodstream. Unlike sodium, calcium, and glucose levels, your body does not need to maintain a strict cholesterol level. For example, glucose levels are maintained to an amazingly tight 0.1% (just 1 teaspoon of sugar per every thousand teaspoons of blood) in each of us! So Nature implemented biological sensor mechanisms only if required. **There is no need for a cholesterol sensor because the absolute number is irrelevant.**

This is THE REASON the medical profession has offered us no insight into why our cholesterol numbers keep plummeting, yet heart attacks continue to increase. **LDL cholesterol is improperly blamed** for a myriad of health problems when the real culprit is defective EFAs. LDL cholesterol has no alternative but to transport these killers throughout our body since we have inadequate amounts of properly functioning LA in our diets. The “experts” never make this critical connection and pinpoint the **real** “problem” with LDL. The cholesterol-lowering drugs simply can’t lower the defective omega-6 enough.

The daily television and print advertisements bombard us with doomsday comments about “bad cholesterol” coming from your genetics and the food you eat. LDL cholesterol isn’t bad in and of itself. If it was, we’d all be dead and the human species would have ceased eons ago. An appropriate analogy is the situation of a drunk driver causing an accident—the drunk driver is like bad EFAs, and the auto-mobile is like cholesterol. The cancer institutes and pharmaceutical companies would have you ban all automobiles (cholesterol) **INSTEAD** of addressing the problem by **eliminating** the drunk driver (**bad EFAs**).

Perhaps for the first time, utilizing the biochemical and physiological properties of EFAs, this explanation of cholesterol finally makes sense.

The Failure of Cholesterol-Lowering Drugs: The Drugs Can't Lower Enough the Defective Parent Omega-6

Hence the reason for the ineffectiveness of cholesterol-lowering drugs above – **they simply can't eliminate enough of the defective EFAs being transported to work well.** This is why the medical journal article titled "LDL Cholesterol: 'Bad' cholesterol or Bad Science," published in *Journal of American Physicians and Surgeons*, Vol 10, No. 3, Fall 2005, by Anthony Colpo, stated:

"Among elderly Belgians, higher levels of *oxidized* LDL were accompanied by a significantly increased risk of heart attack *regardless of* total LDL levels.

"...However, there was *no association between oxidized LDL concentrations and total LDL* levels [in Japanese patients undergoing surgery to remove plaque].

"No tightly controlled clinical trial has ever conclusively demonstrated that LDL cholesterol reductions can prevent cardiovascular disease or increase longevity." (Emphasis added.)

You can see why the absolute LDL number is not very important *if the diet contains sufficient unadulterated PEOs™*. (Also take note that the body has no natural "cholesterol sensor" in the bloodstream – but it *would* if its levels had to be maintained within exact limits.)²¹

LDL cholesterol transports PEOs™ **into** your cells. Any drug that **artificially** lowers cholesterol **ALSO lowers transport of cancer-fighting PEOs™!**

Triglycerides are transported by LDL, too. You need to know that the medical journal *Circulation*²² reported in 2000 that SUBSTANTIAL increased risk of heart disease results from increased triglycerides **independent of cholesterol levels**. Why would we expect this result? Because LDL transports cholesterol AND triglycerides.²³ Triglycerides can be formed from adulterated fats and oils, too. Fix the problem – too many bad fats and oils – instead of blaming the messenger LDL.

21 *Life-Systems Engineering Science* terms cholesterol a *dependent* variable. Recall from high school algebra that if you have three variables in an equation, you can select or change two of them, but the third variable is entirely determined by the other two. Cholesterol acts in exactly the same fashion. Cholesterol varies so that other more important factors can be rigidly maintained.

22 *Circulation* 2000;101:2777-2782.

23 (Voet) *Biochemistry*, page 317.

Do Cholesterol-Lowering Drugs Cause Cancer?

A dire warning was published in a 1995 study by two physicians, Thomas B. Newman and Stephen B. Hulley, at the University of California in San Francisco. They said widespread cholesterol testing for people under twenty years old should be abandoned. They were concerned that popular cholesterol-lowering drugs were being prescribed far too frequently – and often unnecessarily – for people who were at little risk of developing heart-related problems.

“Drugs to lower cholesterol may **cause** cancer ...”²⁴

Both the early drugs known as fibrates (glofibrate, gemfibrozil) and the newer drugs known as statins (Lipitor, Pravachol, Zocor), cause cancer in rodents at doses equivalent for mice to the doses used by man.

Cholesterol-lowering drugs are now prescribed at least ten times more often than just ten years ago, when Newman and Hulley first issued their warning. These physicians were concerned about the routine prescriptions for young people who had no serious risk factors. Yet young patients are now being given these drugs with the expectation they will be staying on them for twenty to thirty years, when **the long-term negative effects aren't known. Do you want to be one of the guinea pigs?** Here's what Drs. Newman and Hulley revealed:

- “...We tabulated rodent carcinogenicity [cancer-causing] data from the 1994 PDR [*Physician's Desk Reference*] for all drugs listed as hypolipidemics [cholesterol lowering]. For comparison, we selected a stratified random sample of hypertensive drugs. We also reviewed methods and interpretation of carcinogenicity studies in rodents and results of clinical trials in humans.
- “DATA SYNTHESIS – **All members of the two most popular classes of lipid-lowering drugs (the fibrates and the statins) cause cancer in rodents**, in some cases at levels of animal **exposure close to those prescribed in humans**.
- “In contrast, few of the hypertensive drugs have been found to be carcinogenic in rodents.
- “...[T]he **fibrates and statins should be avoided except in patients at high short-term risk** of coronary heart disease.” (Emphasis added.)

²⁴ “Drugs to Lower Cholesterol May Cause Cancer, Study Says,” David Perlman, *San Francisco Chronicle*, 1995; pre-pub. Ref., *JAMA*, vol. 275, pages 55-60, 1996.

Has this information been published in the cancer journals? **Yes, it has.** One example appeared in *Cancer Research* 64, 6831-6832, September 15, 2004, in the "Letter to the Editor" section, and was called, "Lipid-Altering Drugs: Decreasing Cardiovascular Disease at the Expense of Increasing Colon Cancer?" by Mark R. Goldstein, M.D. The article states:

- "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** (10, 11, 12, 13). **The trials were randomized, double-blinded, and lasted an average of 5 years.** The lipid-altering drugs were **statins or fibrates**, and the HDL cholesterol levels of the subjects randomized to the drug were raised by 5% or more for the duration of the trial period. A **statistically significant excess of malignancy** was seen in **elderly** subjects (12, 13) and **women** (11) randomized to the drug groups.
- "**Alarmingly, breast cancer** was diagnosed in **1** of 290 women in the **placebo** group and **12** of 286 women in the **pravastatin group** during a 5-year trial ($P = 0.002$; ref. 11). In another randomized study, involving elderly subjects with a mean age of 75 years at entry (13), the significant decrease in coronary death in subjects randomized to pravastatin equaled the **significant increase in cancer death** in the same subjects, leaving total mortality unchanged." (Emphasis added.).

Most people have likely heard *nothing* about the increased cancer risk incurred by taking cholesterol-lowering drugs. This reminds us of the Phen-Fen disaster. This combination drug was dispensed to virtually anyone who asked for it. It produced life-threatening disorders, and now millions of people may suffer its long-term health consequences.

► *Life-Systems* Engineering Science Commentary

These results must be taken seriously. Caution in conclusions from animal studies is a necessity. However, if drug manufacturers aren't monitoring or publicizing the drug's effect on cancer in humans, then we must take the responsibility ourselves.

As you will learn below, PEOs™ can be of significant help in reducing and preventing cardiovascular disorders, even as they protect the cells against cancer.

Lower Your Cholesterol With PEOs™

It is known that **polyunsaturated fats** (PEOs™) naturally **support healthy cholesterol levels** (*Textbook of Medical Physiology*, page 873). It was reported in *New England Journal of Medicine*, 337:1491-1499, that "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.**" (Emphasis added.) Have you been told these facts by your cardiologist? Probably not.

But, as you have already discovered, huge numbers of molecules of the omega-6-based cooking oils are ruined by commercial food processing. In the body these are incorporated

into the LDL cholesterol. With the consumption and transport of **defective**, cancer-causing processed oils, LDL cholesterol acts like a “**poison delivery system**,” bringing deadly transfats and other ruined oils into the cells. It is primarily the oxidized (adulterated) parent omega-6 that clogs the arteries, NOT the saturated fat; NOT the cholesterol.

This is THE REAL REASON that everyone keeps telling us to lower cholesterol at all costs – yet the medical profession has offered us no insight into the actual situation. So **LDL cholesterol is improperly blamed** for transporting defective PEOs™ when it has “no choice” other than to do so, because too few of us have enough properly functioning LA in our diets. The “experts” never make this critical connection and pinpoint the *real* “problem” with LDL.

LDL cholesterol acts like a “poison delivery system,” bringing deadly transfats and other ruined oils into the cells. **LDL cholesterol is improperly blamed**. An appropriate analogy would be the situation of a drunk driver causing an accident — the drunk driver is like the bad EFAs, and the automobile is like the cholesterol. The cancer institutes and pharmaceutical companies’ approach is to try to ban all automobiles (the cholesterol) INSTEAD of applying the correct solution: eliminating the problematic drunk driver (the bad EFAs).

The authors of the following medical journal article understood the connection in 1982, but few of us heard the news. “Fatty acid Composition of Serum Lipids Predicts Myocardial Infarction [Heart Attack],” *British Medical Journal*, Oct. 9, 1982, 285:993, reported that LA (**parent omega-6**) and **most** polyunsaturated fatty acids (PEOs™) including AA and EPA were **lower (depleted) in heart attack victims**. The fatty acid **patterns of the phospholipids** is an independent risk factor for heart disease.

► *Life-Systems Engineering Science Commentary*

This British medical journal article “hits the nail on the head.” Deficiency of EFAs is associated with increased heart attack risk.

So don’t let them scare you into believing that you should therefore minimize parent omega-6 (along with parent omega-3), because of “oxidation” concerns. This will lead you astray. It is true that fats and oils oxidize – that’s partly how they do their job. This is like saying never burn any wood for heat because it is “oxidizing.” Oxidation occurs in the process of producing the energy. In wintry climates you would freeze to death. The proper answer is to keep adding *more* wood to the fire, not less, so that the fire doesn’t go out!

The correct answer here is to take a daily supply of unprocessed, properly functioning PEOs™, not cut them out.²⁵

Furthermore, these consequences go beyond heart disease, because (1) ruined EFAs in arterial blockages cause decreased blood speed, and even worse, (2) it is clear that **because the analysis of aortic arterial plaque is so high in oxidized and ruined omega-6 polyunsaturated oils, consumption of defective polyunsaturated fats and oils is the most important reason your arteries become clogged.**

Additionally, they are also the root cause of blood clots forming in the arteries and not being able to dissolve away naturally, as they do on external cuts. As discussed earlier, blood clots are a tremendous problem with cancer cases, estimated to be responsible for *over 80% of the cancer mortality rate* because they facilitate cancer transport throughout the body when it would not have spread without blood clots.

The pharmaceutical manufacturers continue with the absurd theory (guess) that your body's own cholesterol "causes" heart disease, so they continue to "discover" different types and sizes of cholesterol particles. Then they furnish this "new" information to the physicians. This level of cholesterol detail obscures the main issue of slowing the blood and clogging arteries—just like the cancer community's constant focusing on secondary causes of cancer obscures Dr. Warburg's prime cause of cancer. I want to make it categorically clear that once the EFA issue is solved the cholesterol issue fades away.

One last bit of information. The fact that *transfats* clog arteries was known and published back in 1956 in *Lancet*—the world's most prestigious medical journal. The 16-page article warned about the massive heart disease epidemic that would come (and massive cancer rates along with it). Too few listened.

A Low-Fat Warning:

A low-fat diet automatically minimizes consumption of harmful *transfats* and other processed oils. That is the reason a low-fat diet may *temporarily* result in a reduced disease state—just like in lowered cholesterol, the amount of a cancer-causing, heart disease-causing poison is reduced. However, long-term you have also **deprived yourself of the required parent omega-6 and parent omega-3 consumption leading to an increased risk of heart attack and increased risk of cancer.** A *Life-Systems Engineering Science* analysis furnishes this insight

25 Further references: Waddington, E, et al., "Identification and quantification of unique fatty acid and oxidative products in human atherosclerotic plaque using high-performance lipid chromatography," *Annals of Biochemistry*; 292:234-244, 2001; Kuhn, H., et al., "Structure elucidation of oxygenated lipids in human atherosclerotic lesions," *Eicosanoids*; 5:17-22, 1992.