
Brian Peskin

Both the West and the Middle East share rising heart disease and cancer incidence rates. Brian Peskin, with David Sim, MD and Amid Habib, MD, review current research literature and suggest a causal link between increased widespread statin use for battling heart disease (by lowering LDL cholesterol) and the concurrent increase in cancer.

Statins and cancer – the hidden story

As early as the late 1990s, almost half of all Americans and Europeans died of heart disease.[1] By 2010, virtually all Americans are predicted to die either of 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. Now, in spite of widespread use of cholesterol-lowering drugs, heart disease remains the top killer in Western countries as well as in the Middle East.[2] In addition to the rising incidence rate of heart disease we also see a concurrent rise in cancer rates throughout the industrialised West and the Middle East. For example, we see a very troubling trend in cancer rates in the Gulf state of Oman. Oman, with a population of over two million citizens, established a countrywide cancer registry in 1994. Since undertaking this project they have recorded cancer rates comparable to Western countries. The cancer rate revealed during a five-year study (1993-1997), although less than in America and Europe is significant because it is rising.[3]

Egypt also has developed a significant cancer problem, prompting their National Cancer Institute (NCI), the largest comprehensive cancer center in Egypt, to state: "The present [cancer] hospital with its 550 beds is overloaded by patients referred from all over the country."[4] Egyptian women lead breast cancer contraction rates in the Arab world and, along with men, share the greatest percentage of lymphoma in that region of the world as well.

Lebanese men have the highest rates of both prostate and bladder cancer in the Arab world and Lebanese women have the greatest percentage of uterine cancer,[5] while breast cancer is the number one killer of women in the United Arab Emirates.[6]

Could the medical establishment inadvertently be exacerbating the rising incidence of both cancer and heart disease in both the West and the Middle East through widespread use of statins, their preferred cholesterol-lowering drug?

The Statin-Cancer Connection

An explosive article published in the 2007 issue of *Journal of the American College of Cardiology*[7] 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

It must be noted:

This article is not peer-reviewed.

Middle East Health publishes this article by Brian Peskin to highlight the question of a connection between statins and cancer, raised by the findings of Alsheikh-Ali A *et al*[7], which found a significant correlation between statin treatment and cancer mortality. This issue is contentious and clearly there needs to be further research. *Middle East Health* is not suggesting that doctors stop prescribing statins to their patients.

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 10 years ago when they concluded that low cholesterol, by itself, did not significantly prevent heart disease[8]: "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."

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*[9]: "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."

As this article was going to press, 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): "…despite 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.

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,[10] 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*[11]:

"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 randomised, double-blinded, and lasted an average of 5 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.

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.[12] The investigators analysed plaque and found it contained more than 10 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.[13,14]

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,[15] keeping our skin water- and chemical-resistant, manufacturing bile salts for digestion of fats, forming our bones, and delivering precious PEOs (Parent Essential Oils) to all the cells of our body. Without plenty of cholesterol, we would all be dead.[16]

> LDL cholesterol continues to be improperly blamed for a myriad of health problems while the real culprit is defective PEOs.

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, as shown in Figure 1 (in which R represents the hydrocarbon portion of the fatty acid).

The structure of cholesterol itself never changes

That's right; the esterified component changes. 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 oxidised fatty acids that should be removed from the body. It would be like stopping "garbage collection".

These cholesteryl esters are transported throughout the body in lipoprotein particles (Figure 2) 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).[17]

Importance of esterified cholesterol

Esterified cholesterol comprises the majority of LDL. 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 fact18: "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…"

Furthermore, HM Sinclair, a top EFA researcher and famous English nutritional biochemist; *(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.[19] America's top cardiology publication, the *Journal of American College of Cardiology* (2007;50(18):1735-1741), published that it is the esterified cholesterol that is the problem in heart disease, but didn't address the reason why or how to solve it.

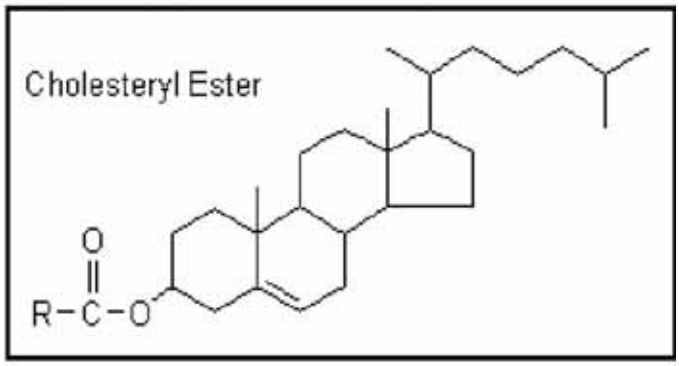

Figure 1

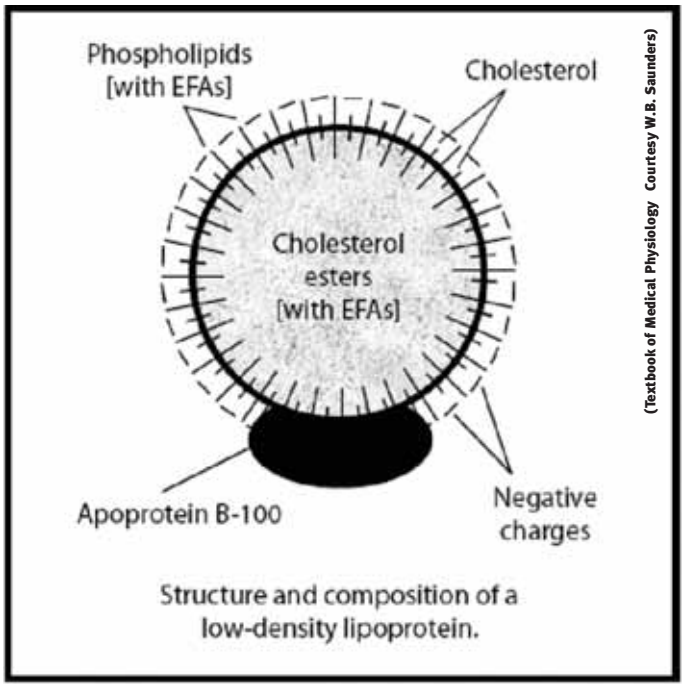

Figure 2.

Esterification of omega-6 with cholesterol was known as early as 1941,[20] and is one of the keys to understanding the statin-cancer connection. However, due to widespread inaccurate terminology, we first need to discuss PEOs, EFAs, and EFA derivatives.

Parent Essential Oils (PEOs): 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 5-10% of them to "derivatives."[21-23] That means 90-95% stay in the parent form in the cell and mitochondrial membranes.[24,25] 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 EPA (eicosapentaenoic acid), DHA (docosahexaenoic acid), and GLA (gamma-linolenic acid). 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 pmega-6

In the past 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 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.[26,27]

One of the important features of cell membranes is their fluidity, which results from local disordering of the bilayer induced by the cis double bonds of unadulterated unsaturated fatty acids.[28] Membrane fluidity increases when more PEOs (functional parent EFAs, in particular, parent omega-6) are available to incorporate in the membrane lipid bilayer. When natural PEOs are replaced by trans fats (hydrogenated), the fluidity changes, and that can cause significant reduction in critical cellular O2 transfer.

A category of synthetic fat that is increasingly used as a substitute for trans fats is interesterified fats termed IE fat. Consequently, IE has its own set of health problems such as abnormally raised resting blood glucose levels.[29,30]

Commentary

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 1-2% – this translates to an enormous number of defective trans fat molecules. In absolute numbers there are an enormous $1x10^{21}$ 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."[31] 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.[32,33] (In the August 2007 *Townsend Letter for Physicians* at *(www.brianpeskin.com/townsend.html)* Cancer's prime cause, cellular hypo-oxygenation (hypoxia), was directly proved by American research scientists in the 1950s.[34,35] Back then they didn't know how to increase cellular oxygenation; whereas today we do, and this is the key in answering why the "statin-cancer" connection occurs and how to prevent its tragic consequences.

Commentary

1. Warburg proved depressed cellular respiration and phosphorylation are the cancer-causing effects of decreased cellular oxygen.[36]
2. Physico-chemical experiments *(Campbell, et al.*[37]*)* 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 haemoglobin, than non-essential oleic acid does.[37] 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 omega-6, 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.[37]
5. Campbell et al.'s seminal experiment[37] 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: **Lowered Cholesterol = Fewer PEOs = Less Cellular O_2 = Cancer.**

In summary

We have 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.[20,38] 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.[28]

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 METEOR (2007) trial, 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 eight serious cardiovascular events including heart attack and angina, a bad outcome. *(www.drbriffa.com/blog/2007/03/30/hailed-meteor-trial-results-not-as-stellar-as-we-are-led-to-believe/)*. In addition, this randomised 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).

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. This is 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.[39,40] Experiments from Florida Hospital Institute of Translational Research shows that blood clots are often caused by biochemical factors contained in small cancerous tumours, like TF (Tissue Factor), that is otherwise 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 tumour.

AA 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

Statins not linked to reduced stroke deaths

As this article was going to press another negative, unexplainable and baffling result of statins was published by Reuters, 3 December 2007 *(www.reuters.com/article/healthNews/idUSN2922862020071129)* which highlighted these points:

"…Doctors baffled by findings indicating lower cholesterol levels were not linked to reduced stroke deaths.

Dr Sarah Lewington of the University of Oxford in Britain was quoted as saying: "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."

"I think all we can say is that we don't really understand what's going on here. And we need to know more about cholesterol and more about stroke subtypes to find out what's going on."

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!"

the precursor to prostacyclin – the most potent anti-aggretory agent (natural "blood thinner") and inhibitor of platelet adhesion.[41] Lowering esterified LA through the lowering of LDL cholesterol automatically decreases the body's natural anti-aggretory AA.[41] 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.[42] 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 which is 100% parent omega-6; there is no omega-3 in skin.[43,44] 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 the following journal article understood the Parent Essential Oil connection in 1982, but few of us heard the news reported in *British Medical Journal*[45] in 1982 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.

Commentary

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 minimise parent omega-6 (along with parent omega-3), because of "oxidation" concerns. It is true that, in part, fats and oils oxidise 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 oxidise fuels for energy and this is a normal process. However, food processing oxidises PEOs prematurely forming nonfunctional foods which cause vascular injury and destroy the body's inherent repair mechanism.

The solution

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.[46]

Have patients minimise 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 of between 1:1 and 2.5:1. With this ratio, suggested use is 725 mg per 18 kg of body weight (e.g. 3 grammes for a 72.5-kg. person on a daily basis). I term this the "Peskin Protocol." 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. ("Optimum PEO Ratio")* or *www.BrianPeskin.com* ("EFA Report").

The Author

Brian Peskin is the primary contributing medical researcher at the Cambridge International Institute for Medical Science *(www.CambridgeMedScience.org)*. He has exclusively devoted the past five years to the cause and solution of cancer. This article is based on information in *The Hidden Story of Cancer*, written by Brian Peskin with clinical researcher Amid Habib, MD, FAAP, FACE. This book is available from Pinnacle Press, P.O. Box 56507, Houston, Texas 77256, USA, or by phoning +1-713-979-0065 internationally.

Brian Peskin earned his Bachelor of Science degree in Electrical Engineering from Massachusetts Institute of Technology (MIT) in 1979. He founded the field of Life-Systems Engineering Science in 1995. Peskin was appointed adjunct professor at Texas Southern University in the Department of Pharmacy and Health Science from 1998-1999. Peskin combines theory with practicality in a very unique way. He is the first to acknowledge he is often the messenger of the information already published in leading medical textbooks and medical journals. His unique insights have on occasion led a few malcontents to label him a "quack".

- For more information visit *www.BrianPeskin.com* and *www.CambridgeMedScience.org.*

David Sim, MD is a leading cardiologist practicing in Boise, Idaho, USA.

Amid Habib, MD is a leading endocrinologist practicing in Orlando, Florida, USA.

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Arachidonic acid

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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