As early as the late 1990s, almost half of all Americans and Europeans died of heart disease. 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. 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.

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.”

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

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* 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
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: “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: “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 undetected [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, 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:

“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.32 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.33,34

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,11 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.36

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).

**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 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...”

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." 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.

Esterification of omega-6 with cholesterol was known as early as 1941, 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.” That means 90-95% stay in the parent form in the cell and mitochondrial membranes. 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 omega-6**

In the past several decades, processed foods - in particular, frozen foods and
restaurant cooking oils - have increasingly incorpo-
rated trans fats (hydro-
genated) 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 inflam-
matory, immune, and
signaling functions.26,27

One of the important
features of cell membranes is
their fluidity, which results
from local disordering of the
cis double bonds of unadulter-
ated 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 bi-
layer. When natural PEOs
are replaced by trans fats
(hydrogenated), the fluidity
changes, and that can cause
significant reduction in crit-
cial 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 under-
stand 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
omeg-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 prod-
ucts contain relatively few
trans fats – as little as 1-2% –
this translates to an enor-
mous number of defective
trans fat molecules. In
absolute numbers there are
an enormous 1x1021 mole-
cules 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 table-
spoon 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 solu-
tion – PEOs are
As the New England Journal
of Medicine makes clear:
"Diets high in polyunsatu-
rated fat (PEOs) have been
more effective than low-fat,
high-carbohydrate diets in
lowering cholesterol as well
as the incidence of heart
disease." The key is making
sure the PEOs are unadulter-
ated.

Otto Warburg, MD, PhD:
"Lowered cellular oxygen
equals cancer!"
Just as oxygen deprivation
causes heart disease,
sustained oxygen depriva-
tion causes cancer, too. Over
70 years ago, the Nobel
prize-winning physician
and master chemist Otto
Warburg, MD, PhD, demon-
strated 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/townse
nd.html) Cancer’s prime
cause, cellular hypo-
oxidation (hypoxia), was
directly proved by
American research scien-
tists in the 1950s.34,35 Back
then they didn’t know how
to increase cellular oxygena-
tion; 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 respi-
ration and phosphoryla-
tion are the cancer-
causing effects of
decreased cellular
oxygen.36
2. Physico-chemical ex-
periments (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 haemo-
globin, than non essen-
tial oleic acid does.37
Therefore, the 35% cancer-causing hypo-
oxidation (depriva-
tion) threshold is
breached with insuffi-
cient 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 there-
to fore systemically occur
in any membrane – any
tissue in the body can
become a potential
cancer site.37
5. Campbell et.al.’s seminal
experiment38 conclu-
sively showed a 50%
reduction in oxygena-
tion when a PEO defi-
ciency 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 O2
= 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.39,40 Even though
statins increase the uptake
of LDL cholesterol from the
blood, they decrease
overall cellular LA because
absolute levels of chole-
sterol 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 1958.29,40 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
the precursor to prosta- 
clyn - the most potent anti
aggregatory agent (natural
“blood thinner”) and
inhibitor of platelet adhe-
sion.41 Lowering esterified
LA through the lowering of
LDL cholesterol automati-
cally decreases the body’s
natural anti-aggregatory 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 ulti-
mately contracting cancer
(and CAD).

Atherogenesis, adulterated
PUFAs, and LDL choles-
terol: More connections
The eminent researcher HM
Sinclair published his
finding that PEO deficiency
causes an enormous perme-
ability increase in skin
along with increased capil-
lary fragility.43 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 inner-
most 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 42 in 1982
that LA and most polynu-
saturated 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 phospho-
lipids [PEOs] constitute an
independent risk factor for
heart disease.

Commentary
This BMJ article “hits the
nail on the head”. Deficiency of PEOs is associ-
ated 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 produc-
tion) proceeds in the mito-
chondria 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 forma-
tion of hydroperoxides from
the double bonds of the
PEOs. This harmful partial
oxidation involves no energy
(AMP) 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), interester-
ified fats, and other adul-
tered 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”).

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