Scientific Support for Chapter 14

Answering Questions and PEOs and Patient Conditions

"The molar ratio of the **sum of all antioxidants to PUFAs** [significantly PEOs; in particular, Parent omega-6] is on average **1:165**, ***thus one antioxidant molecule has to protect the large number of 165 PUFA molecules***. The [natural **predominant antioxidant** in low-density protein (**LDL)** is a-tocopherol [**vitamin E],** with an average of **6 molecules in each LDL** particle.

"The total number of fatty acids bound in the different lipid classes of an **LDL particle** with a molecular mass of 2.5 million is on average 2700, of which about 1/2 are polyunsaturated fatty acids (PUFAS), **mainly linoleic acid [Parent omega-6] (86%)**, with small amounts of arachidonic acids [10% of LA] and docosahexaenoic acid (DHA) [**a mere 2% of LA content**].

► **"…This shows that at least for our study group the *antioxidant status is not predictive* for the oxidation resistance of an individual LDL. The efficiency of vitamin E-dependent and the vitamin E independent oxidation resistance seem to be subject specific with *strong individual variation.***

"…[I]t is ***unlikely than LDL can become oxidized*** **in plasma to** the extent that it causes foam cell formation and the processes chemotactic and **cytotoxic properties**."

New reference:

► **"The most abundant fatty acid in human LDL is the polyunsaturated fatty acid, linoleate (18:2) [Parent omega-6].**

"**Oxidation of LDL** also leads to a striking ***depletion of polyunsaturated fatty acids***.

"On a molar basis, however, **LDL** contains ***several hundredfold more molecules of polyunsaturated fatty acids than these natural antioxidants***. [Note: Confirms Dr. Esterbaurer's journal article above.]

"Many different experimental protocols for generating oxidized LDL have been reported, and the degree of oxidation of the lipids and the amount of derivatization of apo B vary widely with different oxidation protocols. For example, ***only minimal physical and chemical changes related to oxidation are produced by a prolonged storage of LDL with oxygen*** or by incubation with low concentrations of copper ions. Low-density lipoprotein with **low degrees of oxidation**, frequently called minimally modified LDL, has **little** apo B fragmentation, **no** loss of the capacity to bind to the LDL receptor, and **no ability** to bind to the scavenger

receptor. Minimally modified LDL nevertheless has biologic properties that may be important in leading to the early development of atherosclerotic lesions.

"Laboratory studies have shown that it is difficult to oxidize LDL in the presence of either serum or plasma.

"Aside from participating in foam cell development, **oxidized LDL** has many other properties that could play a role in atherosclerosis. One of the biologic effects of oxidized LDL is its **cytotoxic effect on cultured endothelial cells [arterial intima]**…. **[O]xidized LDL**, presumably a lipid peroxide, **inhibits** the migration of endothelial cells." These authors point out that such an effect, if it persists in vivo, could **compromise the response of the endothelium to wound healing after injury**.

"**Among 12 populations with similar cholesterol levels** (clustered around "normal" levels-5.70 to 6.20 mmol per liter [220 to 240 mg per dl]), the ***blood pressure readings and the serum cholesterol levels were not predictive of ischemic heart disease mortality***. [NOTE: This is a major reason that DPA scans for arterial compliance — as in IOWA — is superior and much more useful than BP as a diagnostic tool. BP is outdated compared to PWV / DPA analysis.]

► **"Chin and associates presented convincing evidence that a lipid component in *oxidized LDL inactivates nitric oxide*."**

PEO Solution analysis: Nitric oxide opens the vessels and we see that defective, *adulterated* PEOs negatively impair NO output. **Solve the PEO deficiency and you will correct the nitric oxide issue at the same time**. Chapter 12 offered additional insight in this area via *endothelium-derived relaxing factor's requirement of fully functional PEOs in LDL-C.* We see Parent omega-6 is the major fatty acid in LDL. Its oxidation causes depletion of LA throughout (chain reaction), so once the oxidation problem starts, the worse it becomes. The *natural inability* of the small number of anti-oxidants compared to the huge amount of LDL's PEOs to protect all the PEO molecules is confirmed. It was never supposed to be an issue. Nature never thought we be consuming such high levels of adulterated PEOs. **Fully functional PEOs attached to LDL are naturally very resistant to damage**. Oxidized cholesterol is a direct cause of cardiovascular disease. That is why **LDL levels alone are meaningless in predicting cardiovascular disease—confirming that all the risk lies in the functionality of PEOs**.

Newsflash 2010: Calcium Supplements May Increase Risk Of Heart Attacks:[1]

> "An international team of researchers that reviewed data from several trials found that taking calcium **supplements was linked to a higher risk of** heart attack **and other cardiovascular events**; the authors called for new research to **re-assess** the **role of calcium supplements** in the treatment of osteoporosis.

1 July 30, **2010**, http://www.medicalnewstoday.com/articles/196310.php. Ref.: Bolland, Mark, J., et al., "Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis." *British Medical Journal*, 2010; DOI:10.1136/bmj.c3691.

"...[F]ound that people taking **calcium supplements had between 27 and 31 per cent higher [relative] risk of heart attack** than counterparts who took placebo.

"For the **11 studies** that yielded trial-level data, they found a **similar pattern**:

"Given that **calcium** supplements **only modestly improve bone density** and prevent fracture, their role in the **management of osteoporosis should be re-assessed**, *urged the authors*." [Note: The so-called "improvement" in density comes in completely the wrong manner—making the bone much less flexible and the patient much more likely for fracture.]

PEO Solution analysis: As you learned in Chapter 4, calcium merely deposits a mineral coating on the bone matrix—not improving the critical bone matrix in the least. Furthermore, a detrimental effect of this therapy is possible **acceleration of calcification of the plaque—the last stage of CVD disease**. PEOs are a much better solution to both prevent and treat osteoporosis without contraindications. *Textbook of Medical Physiology* makes clear that proper bone structure is in its matrix, not the deposited minerals. *Osteoporosis* is a bone matrix issue, not a mineral issue. Protein and PEOs comprise the critical bone matrix.

2011 Testosterone News...[2]

- "A decline in testosterone levels as men grow older is likely ***the result – not the cause*** – of deteriorating general health, say Australian scientists, whose new study finds that **age, in itself, has no effect on testosterone level in healthy older men**.
- "'We had originally expected age to have an effect on serum testosterone, so the **findings were a bit of a surprise**,' Handelsman said.
- **"Age had no effect on testosterone level**.
- "The message for patients and their doctors, Handelsman said, is 'older men with low testosterone levels **do not need testosterone therapy unless they have** ***diseases of their pituitary or testes***.'"

Sports Medicine Physicians: As you well understand, steroidal hormones have cholesterol as their substrate. With addition of fully functional PEOs, the cholesterol becomes *fully functional* **via its (esterified) PEOs.**

CASE STUDY "...By the way the **doctor was complimentary** and I think he is taking an extra interest because I take the **PEOs**. **The doctor did say that for a 68 year male my testosterone level was** ***nothing short of amazing***."

Allen W.

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2 www.sciencedaily.com/releases/2011/06/110607121129.htm. Ref.: The Endocrine Society (**2011**, June 7).

PEO Solution analysis: Everyone consuming commercial food will be overdosed on *estrogenic substances*. Men's sperm counts are significantly lower than in the past. PEOs give the building blocks to overcome this unnatural imbalance. Once again, we see that a ***cause is confused with its effect***. This study **analyzed blood testosterone levels in over 300 men nine separate times over a three-month timeframe**—there were no mistakes in measurement. Why would patient's testosterone levels become too low? Testosterone is derived with cholesterol as its substrate. When cholesterol's esterified Parent omega-6 is *adulterated* from food processing, the cholesterol structure is adulterated. Hence, testosterone's functionality is highly impaired. Ensure your patients have proper PEOs and steroidal-based hormonal issues—both male and female—will be minimized.

Note: The Endocrine Society's 94th annual meeting (June **2012**) presented reports of analyzing testosterone measurements in 1,500 patients. Two measurements were taken: baseline and five (5) years later. The average patient was 54 years old. The authors reported an **average DECLINE of less than 1% / year [This means a patient 95% of initial level 5 years later]**. I'd like to see the improved results with all patients taking PEOs.

Association of marine omega-3 fatty acid levels with telomeric aging in patients with coronary artery disease

TELOMERES—***Anti-Aging MAJOR Newsflash***: There is more to telomeres than we are told—they can lengthen…The article, "Association of marine omega-3 fatty acid levels with telomeric

aging in patients with coronary artery disease," confused many physicians:[3]

> "Little is known concerning the dynamic regulation of **telomere length over time**, although it has ***recently become apparent*** that **telomeres may lengthen as well as shorten**.
>
> …[T]herefore, **no definitive conclusions can be made about causality** [cause of telomere shortening]."

Telomeres and Aging." Telomere can lengthen

Here's the insight you and your patients need to know from the superb article published in **2008** by the American Physiological Society in the treatise, "Telomeres and Aging." **Telomere can lengthen**:[4]

> "**Telomerase** is a specialized reverse transcriptase **capable of extendin**g the 3′ end of chromosomes **by adding** TTAGGG repeats.
>
> "In the future attention undoubtedly will be centered on the **genome**, and with greater appreciation of its ***significance as a highly sensitive organ of the cell***, **monitoring** genomic activities and correcting common errors, **sensing the unusual and unexpected events, and responding to them [epigenetic environmental importance]**, often by restructuring the genome. We know about the components of genomes that could

3 Farzaneh-Far, Ramin, et al., *JAMA*, January 20, **2010**, Vol. 303, No. 3, pages 250-257.

4 Aubert, Geraldine and Lansdorp, Peter M., "Telomeres and Aging," *Physiol Rev* 88: 557–579, **2008**.

be made available for such restructuring. ***We know nothing, however, about how the cell senses danger and instigates responses to it that often are truly remarkable***.

"One of the most striking features of **telomeres** revealed by Q-FISH is the ***heterogeneity in the length of telomere repeats*** at individual chromosome ends. Some of this **diversity** is generated in somatic cells and not in the germline, and **specific chromosome ends** in clonally derived cells **can show an almost complete loss of telomere repeats**. Sporadic telomere losses complicate the relationship between telomere length and cell division history and potential. ***It is important to realize this uncertainty in the context of aging***.

"The **heterogeneity in telomere length** in chromosomes of normal cells has **complicated studies on the role of factors that regulate telomere length**.

▶ **"Studies reporting that (*oxidative*) *damage* of telomeric DNA could be *the major cause of telomere shortening in human cells* are less frequently cited, perhaps because these findings complicate notions about telomere loss acting as a simple "mitotic clock."**

"The **guanine-rich** nature of telomeric DNA makes it ***particularly vulnerable to oxidative damage***.

"Loss of telomeric DNA at the cellular level is well established and was shown to be related to replicative history and life span in somatic cells. However, at the ***level of tissues or of the entire organism, what is***

the impact of telomere shortening? Does aging cause telomere shortening, or does telomere shortening cause aging?

► "Accumulated data support the notion that the loss of telomere repeats in (stem) cells and lymphocytes contributes to human aging. **This notion is not widely accepted**, primarily because the gradual loss of telomere repeats with age in cells of various tissues is not easily measured and because the average telomere length ***shows a lot of variation between species and between individuals of the same age….***"

PEO Solution analysis: As predicted, oxidative damage is the significant cause of telomere damage that we can easily solve. ***Proteins can oxidize and PEOs mitigate against their damage***. There is enormous variability in telomere length and *a simple view that "longer is better" is simplistic and naïve*. Unfortunately, researchers often prefer "simplistic" answers even if they are wrong, misleading other researchers.

These **2008 / 2010** journal articles makes clear that **telomere lengthening is possible**:

> "Over the 2.5 year period, 45% of the study participants showed maintenance of man bulk TL, whereas 30% showed telomere shortening, and, **unexpectedly, 24% showed telomere lengthenin**g."[5]

5 Epel, E.S., et al., "The rate of leukocyte telomere shorten predicts mortality from cardiovascular disease in elderly men," *Aging*, **2008**, Dec 4; 1(1):81-88.

> "Telomere **shortening** is **counteracted** by the cellular **enzyme telomerase**. "[6]

The **2010** article, "Is telomere length a biomarker of aging? A review," has this to say:[7]

> "Although telomere length is implicated in cellular again, the **evidence suggesting telomere length is a biomarker of aging** in humans is **equivocal**. These studies would benefit from longitudinal measures of both telomere length and aging-related parameters."

PEO Solution analysis: Because these studies are *not consistent* any suggested *cause/effect relationship should be re-evaluated*.

Real-Life Results

"I have been following your protocol about 18 months and they have been very helpful for me. I have read *The Hidden Story of Cancer* cover to cover.

I developed rheumatoid arthritis in late 2010 / early 2011, and was **put on the standard prednisolone and methotrexate**. While they helped greatly with the pain in the initial crisis, they were not things I wanted to be on long term. I weaned myself off them (and blood pressure medication as well) and **now take noth-**

6 Ornish, Dean, et al., "Increased telomerase activity and comprehensive lifestyle changes: a pilot study," *Lancet Oncology* **2008**; 9:1048-57**.**

7 Mather, Karen Anne, et al., *The Journals of Gerontology: Series A, Biological Sciences and Medical Sciences*, Volume 66A, Issue 2, **2010,** pages 202-213.

ing except your recommendations. Recent comprehensive blood tests were excellent all round, including cholesterol, triglycerides and inflammation markers.

I want to thank you so much for your work. I have been speaking about your work to anyone interested."

Edward S. (e-mail: 2013)

"I was listed in the hospital as having l**ow grade leukemia**. I left the hospital and started following your recommended PEO guidelines taking double the normal amount for close to 3 months. I am happy to report I do not have nor never have had any problems with my blood count again. My doctor always draws my blood to check and **for the past 5 years I have perfect blood count and no anemia.**

I was led to your "**PEO Solution**" because they **healed my sister of severe hives** after the **doctors gave up on her because she would not take any more steroid shots**. I have told many people about PEOs. I will be on them for life!"

Thanks, Lynda O. (e-mail: 2014)

"Brian,
A patient (L. S.) prescribed PEOs for 2 weeks told me:

1. Her hot flashes from menopause have gone away and
2. 'It used to be the Sahara Desert with my fiancé, but no longer!'

To say the least, she is very happy."

Jeff Matheson, MD
Ontario, Canada (e-mail: 2014)