Scientific Support for Chapter 2

More about False Positives

To calculate the number of false positives, divide the number of healthy people tested sick (9,999) by the total of all people who tested sick either correctly or incorrectly (9,999 + 99). [A group of 9,999 (healthy people tested sick) /10,098 (99 sick people tested sick + 9,999 healthy people tested sick) = 99% incorrect results.]

Because we tested a large number of people (1,000,000), even a small degree of inaccuracy—just 1% (typically considered very small)—produces a large error compared with the number of people who really have the disease.

When it comes to statistics, being correct is not always as simple or obvious as they may appear.

Proving the Point: The Folly of Raising HDL Levels

> "These results challenge several established views about plasma HDL cholesterol."
>
> "On the basis of the association between the *LIPG* Asn 396Ser allele and HDL cholesterol, the 5.5-mg/dL *increase in HDL* ***should have translated into a 13% decreased risk of MI***. "The people who are carriers of the HDL-boosting variant should have had a reduced risk of heart attack, ***but to our surprise, there was no association between the gene variant and heart-attack risk***."

"So we have these two lines of evidence, one from the single variant and another from a group of 14 variants, that lead to the same conclusion—that people who are genetically predisposed to having ***higher HDL-cholesterol levels are not protected from heart-attack risk, as would be expected***."

"…What is not known is whether that association [lower HDL=increased heart disease risk] ***is a causal relationship or an indirect [a mere association]...***"

Stat-Smart example from *New York Times*

Excerpts from ***New York Times*** article on Wednesday, October 17, 2012, page A18, by Anahad O'Connor, titled, "Cholesterol is falling in adults, study finds":

"Researchers examined a nationally representative sample of tens of thousands of Americans over the last two decades and recorded a decline of 10 points in average total cholesterol—to 196 mg/dL from 206 mg/dL….

"Two other trends in the last decade may have also been factors, the researchers said: declines in smoking and a drop in carbohydrate consumption.

"Dr. David J. Frid, a cardiologist at the Cleveland Clinic, said the findings were unexpected given the high rates of obesity and Type II diabetes. He pointed to a ***30 percent drop in deaths from heart disease nationwide, and said the cholesterol data might be related.***"

This last statement should give you great pause.

Two current examples of poor "studies"

In the analysis published online April 9, **2012** titled, "**Efficacy of Omega-3 Fatty Acid Supplements (Eicosapentaenoic Acid and Docosahexaenoic Acid) in the Secondary Prevention of Cardiovascular Disease:** A Meta-analysis of Randomized, Double-blind, Placebo-Controlled Trials**,"** Sang Mi Kwak, et al., *Archives of Internal Medicine,* published online April 9, **2012**. doi:10.1001/archinternmed.2012.262. ***They retrieved*** 1007 articles but only found a mere 14 trials ***with 20,000 patients that were qualified as adequate to include in the analysi***s.

In the analysis titled "Association Between Omega-3 Fatty Acid Supplementation and Risk of Major Cardiovascular Disease Events: A Systematic Review and Meta-analysis," Evangelos C. Rizos, et al., published in *Journal of American Medical Association* (JAMA), **2012**;308(10) 1024-1033, ***they reviewed*** **3635 citations but only accepted 20 studies** ***of 68,000 patients that were adequate for analysis***.

Scientific Support for Chapter 3

How to Determine the NNT

The number needed to treat (NNT) is equal to the reciprocal of the absolute risk. The reciprocal is the amount obtained when dividing the number one by another quantity. In an experiment, the reciprocal is obtained by dividing the number "1" by the difference in effectiveness. In the case of statins, the NNT is 100, obtained by dividing the number "1" by 1%, i.e. 1/.01 = 100.

What is a "p-value?"

Statistics is mathematical and therefore tends to be extremely detailed and difficult. Fortunately, the essential concepts describing clinical trials and experiments are relatively simple:

1. The first value looked at by physicians and others (and mistakenly too often assumed to be the only important value in a clinical trial) is the "p-value " or 1 – (p-value), meaning the probability (p) that a particular intervention's experimental result occurred by chance alone, i.e., **the drug doesn't really work but appears to work**.

If the p-value were set at a 95% confidence level, then it would mean that if 100 trials were done by a different experimenter, 95 of them would include the same variance between the means (averages) in each trial as those achieved by the original experimenter. You could expect the same type of results 95 out of 100 times—**5% of the time you'd accept results implying the intervention worked when it didn't work**. That's the price paid for a 95% confidence level. If you increased the confidence level to 99% (by increasing sample size and/or the required number

of successes in the intervention arm), the accuracy is greatly increased but the cost of the trial would likely go up too. (That is, unless the intervention worked extremely well, such as in the IOWA experiment, discussed later in this Scientific Support.)

When the study or experiment is repeated many times using the same general group of people, this same 5% "**successful result**" will occur **when the experiment is actually a failure**, but, again, it is entirely due to chance alone. *The item of interest (drug or nutraceutical) may not have worked at all*, but we may be led to think that it did work based on the false positives. **That's why the more studies performed = the greater likelihood of false results being accepted as true**.

Never forget, the experimental **failures** are much more telling than "successes." It is not merely judicial "preponderance of the evidence."

2. Typically, the p-value is set to 0.95 (at a 95% confidence level you get an inherent 5% possible error rate allowed) for the medical intervention to be considered "statistically significant." If p = 0.95 then the study would be termed a 95% confidence level study (although a bit more information is still required). A 0.99 (1% error rate) or 0.995, p-value (0.5% error rate) would be even better because there would be much less of a random chance effect behaving as though the drug worked when it really didn't, thereby fooling both the physician and patient. Never forget that with p=95%, even if the drug didn't work, there is still a 5% chance that you would get these pseudo-positive results 5% of the time, making it *appear like the drug did work*.

Never forget: This 5% error rate means 1 out of 20 times you can be FOOLED into thinking FAILURE is SUCCESS.

This means that IF 15,000 "studies" of fish oil showed success (which isn't true), 750 of the studies that actually failed would be wrongly deemed successes—*fooling both physicians along with their patients*. Numerous fish oil studies do fail and we are hearing more and more about them, but due to the huge number of continual studies, many supposed "successes" are true failures. Physicians need to understand this fact.

Once again, a 95% p-value means that if this experiment were carried out in the same patient population sample 100 separate times, every time showing the drug being tested didn't work, then this same result would be included at least 95% of the time; however, a *false-positive result* **would occur entirely randomly 5% of the time,** *although the drug was actually a complete FAILURE***—*5 of those failed trials would appear to be successes*.**

It's easy to mislead those who don't understand statistics—almost everyone. All a company has to do is to conduct many studies and then purposely select only those that randomly show a "positive" result. Don't mention the failures, and, presto, you have a "successful" drug! All you need is lots and lots of money.

The p-value is NOT a measure of the size or magnitude of the effect of the drug. That is a completely different issue and has to do with the means (difference of the averages between both groups). Many physicians and patients don't understand this critical fact and mistakenly think that a p-value alone is all that is needed. Wrong, wrong, wrong—it is only part of the picture.

It is true that the MINIMUM p-value should be at least 95%; however, even IF the study has a "significant" effect with the intervention, then one must ask this next critical question:

How Strong is the Effect? A Little, or a Lot?

You need to ask "*What is the magnitude of the positive effect*?" A positive effect can range from a very small negligible effect to a tremendous effect. It may work on everyone, but with very little positive effect.

What is considered a significant amount or a significant effect?

If more than 51% (the majority) of a group doesn't respond **in absolute numbers** (NOT relative measures discounting sample size) to the drug, then I am not impressed, and you shouldn't be, either. **If something works, it should work on nearly every patient**—the majority being, as a minimum, greater than 51% of all patients.

Typically today, if just 20% of the treated group—the degree considered "clinically effective"—obtains any positive effect (regardless of how little that effect), it is considered a huge success. **This really means 80% FAILURE**.

Is the Item Measured Significant, or a Worthless "Surrogate" (Association)?

Low NNT is a necessary, but not an entirely sufficient condition, to be able to claim victory. **Is there a DIRECT cause/effect relationship**? This is absolutely required because if it isn't, you are being misled. Stains decrease low-density cholesterol

(LDL-C) with an NNT =1: a superb job. However, this doesn't significantly translate into stopping and reversing CVD.

A Worthless Surrogate—NOT the Specific Desired Result—is Often Used

Even though statins lower LDL-cholesterol, CVD is not significantly reduced.

The tragic truth was only recently accepted in **2012**. This still hasn't stopped the pharmaceutical companies and physicians who rely on those drugs from saying that lowered LDL cholesterol is all that counts in preventing cardiovascular disease. This has been proven incorrect, and patients are paying for this mistake with their health.

Therefore, one **cannot** assume the "disease" is solved when a worthless "surrogate" (association) is used **instead** of measuring the result itself, such as *how many heart attacks occur with and without statins* (the answer is nearly the same amount). This means that statins are ineffective at stopping heart disease.

A recent example: The JUPITER (Justification for the Use of Statins in Primary Prevention) Failure Hailed as a Success

Of course, from the above, it goes without saying that there must first be a direct cause/effect relationship to the disease. If you treat 100 patients with a drug and all 100 improve, the drug's number needed to treat (NNT) is 1 (100 patients/100 successes). If you treat 100 patients and only 1 patient responds positively, the NNT would be 100 (100 patients treated/1 positive response). This is an awful result— a 99% failure rate.

The 2008 JUPITER study obfuscated the fact that numerous attempts had been made to prove the "cholesterol theory" (the lower the patient's LDL-C, the greater the prevention of CVD), by attempting to make the case that the real mode of action of statin drugs was C-reactive protein (CRP) reduction from the statin. However, there is one tragic flaw in this argument: CRP—the protein that shows up in elevated levels in response to inflammation—is not a reliable prognostic indicator of cardiovascular events; there are better markers. An article entitled *Largest-Ever Meta-Analysis Finds CRP Is Unlikely to Be Causal for CVD* reports that scientists of the Cambridge-based Emerging Risk Factors Collaboration (ERFC) found:

> "[A]lthough CRP concentration was linearly associated with CHD (coronary heart disease), stroke, and vascular mortality, as well as nonvascular mortality, statistical adjustment for conventional cardiovascular risk factors resulted in considerable weakening of associations."

An Example of Misleading Statistics

In the JUPITER Study, **the NNT was 240 for statins[1] in preventing any stroke.** ***This is a 99.58% failure rate.*** **The "relative risk" statistics were used instead and disguised as a hazard ratio—essentially a time-valued relative risk—of 0.52 (52%)); p-value was 0.002). The NNT in this study was not stated**.

This means that the JUPITER Study had an **undisclosed** NNT of 240 (**99.6% FAILURE**) for preventing any stroke— instead, a

---

1 Peskin, Brian Scott, "The Failure of Statins: A New Physiologic Solution to Cardiovascular Disease, Medical Therapeutics," *American Academy of Anti-Aging Medicine*, 2010, chapter 230, pages 259–273.

hazard ratio of 0.52 (appearing as a 52% success) was published, thus **making the trial appear immensely more successful than it actually was**.

What *appears* more impressive—a 0.4 success rate (**99.6% FAILURE** rate) or a 52% success rate (48% FAILURE rate)? Physicians are deceived along with their patients.

An Example of Modern-Day Low NNTs and High Effectiveness: IOWA Experiment

(*See* brianpeskin.com/BP.com/experiments IOWA-Experiment-Results.pdf (for entire screening information.)

There is a non-interventional way to screen subjects for arterial flexibility. It is called photoplethysmography with digital pulse analysis. This particular experiment was called the IOWA experiment—**I**nvestigating **O**ils **W**ith respect to **A**rterial health. The details will be described later but here were the results so you can see how both NNTs and p-values can be low, a high degree of significant effectiveness.

Long-term Use in Subjects with PEO Formulation Screened with Photoplethysmography

---

Significant differences (p-value=0.0015) with an experimental error of the mean (+ or -) 5 years. Subjects' cardiovascular biological age (average of) 8.8 years lower than their actual physical age.

Notice two points: People taking PEOs long-term had arterial flexibility 8.8 years lower—a younger cardiovascular "biological

age" than expected. Of the 34 subjects who were screened, 25 subjects improved. This is a very significant effectiveness measured either by absolute or relative measures.

On average, the "biological age" difference was significant, almost a decade!

The probability that this was a random chance occurrence was < 0.0015. You can take this result "to the bank."

Note: The typical clinical study uses a 5% cutoff. This is 30 times more confident!

The overall effectiveness was that 73% of the people taking the PEOs screened much younger than their biological age would suggest: an NNT = 1.4.

Short-term Improvement in Subjects with PEO Formulation Screened with Photoplethysmography

---

Significant differences (p-value=0.0099) with an experimental error of the mean (+ or -) 5 years. Subjects' cardiovascular biological age (average of) 7.2 years lower than their actual physical age.

On average, the "biological age" difference was significant—more than seven years "biologically younger" than expected.

The probability that this was a random chance occurrence was < 0.0099. You can take this result "to the bank." Note: This is 5 times more confident than a 5% cutoff!

The overall effectiveness in the short-term was that 44% of the people taking the PEOs screened much younger than their biological age would suggest: an NNT = 2.3. Of 16 subjects, 7 subjects rapidly improved. I like to see 80+% improvement effectiveness in screening for interventions but the timeframe was short to impact the cardiovascular system so significantly—less than a year.

PEOs Versus Fish Oil Subjects Who Discontinued Fish Oil Supplementation, Replacing it with PEO Formulation Screened with Photoplethysmography

---

Significant differences (p-value=0.0001) with an experimental error of the mean (+ or -) 5 years. Subjects' cardiovascular biological age (average of) 11.1 years lower than their actual physical age.

On average, the "biological age" difference was significant: more than 11 years "biologically younger" than expected.

The probability that this was a random chance occurrence was < 0.0001. You can take this result "to the bank." Note: This is 500 times more confident than a 5% cutoff!

> The overall effectiveness was that 87% of the people taking the PEOs screened much younger than their biological age would suggest: an NNT = 1.2. Of 15 subjects, 13 subjects improved. This translates to a 87% effectiveness—in just 3.5 months with PEO use (on average). Because this effectiveness is about double the screening for just PEOs alone—44%—the conclusion is that **SIMPLY STOPPING FISH OIL gave nearly everyone a 4-year improvement in increased arterial health**!

These general PEO results and PEOs versus fish oil results—in screening for arterial flexibility—are incredible and predictable, as you will soon discover in later chapters.

Always ask for the SAMPLE SIZE, since without it you cannot draw any meaningful conclusions.

Always ask for the ABSOLUTE RISK DIFFERENCE BETWEEN BOTH GROUPS (NNTs), since without it you cannot draw any meaningful conclusions.

Alpha-Linolenic Acid and Risk of Nonfatal Acute Myocardial Infarction

Alpha-Linolenic Acid and Risk of Nonfatal Acute Myocardial Infarction, Hannia Campos, H., Ana Baylin, A., and Walter C. Willett, W.C., "Alpha-Linolenic Acid and Risk of Nonfatal Acute Myocardial Infarction," Circulation, 2008; 118:339-345.

- "Greater alpha-linolenic acid [**parent omega-3**] associated with **lower risk of myocardial infarction.**
- "Similarly, **low intakes of alpha-linolenic acid** can be found in developing countries where **cardiovascular disease is on the rise**.
- "**Fish intake was similar in cases and controls**, and the *variation within each group was large*….
- **Fish** or eicosapentaenoic acid [**EPA**] and docosahexaenoic acid [**DHA**] intake at the levels found in this population **did not modify the observed association**."
- ***Conclusions*** – Consumption of vegetable oils rich in alpha-linolenic acid [**parent omega-3**] could confer **important cardiovascular protection**. The apparent protective effect of alpha-linolenic acid is **most evident among subjects with low intakes**.
- "In summary, consumption of vegetable oils rich in alpha-linolenic acid [***parent omega-3***] could confer ***important cardiovascular protection***."

[Important Note: This result is independent of the level of fish consumption. Given all of fish oils supposed miraculous claims, didn't these researchers wonder why? However, the researchers understand that the parent omega-3 did something the derivatives didn't do.]

Scientific Support for Chapter 4

"Protein Consumption and Bone Mineral Density in the Elderly: The Rancho Bernardo Study," Promislow, J, et al., *American Journal of Epidemiology,* ***2002****,* Vol. 156, No 7, pages 636-644:

- "This association was ***statistically significant in women [MORE protein = DECREASE in OP]***. For every *15-g/day increase in animal protein* intake, BMD [*bone mineral density*] *increased* by 0.016 g/cm2 at the hip *(p* = 0.005), 0.012 *g/cm2* at the femoral neck *(p* = 0.02), 0.015 *g/cm2* a the spine *(p* = 0.08), and *0.010 g/cm2* for the total body *(* = 0.04).

- "Conversely, a ***negative association between vegetable protein*** and BMD was observed in both sexes.

- "This study supports a ***protective role for dietary animal protein*** in the skeletal health of elderly women.

- "***Clinical trials*** in hip fracture patients have ***consistently*** observed that patients who receive ***protein*** supplements ***experience significantly improved recoveries and reduced bone loss***. Dietary protein has historically been investigated largely in regard to its effect on calcium balance.

- "However, ***protein itself is an important structural component*** of bone, accounting for approximately ***half of bone volume and one fourth of bone mass, including the skeletal matrix.***"

2006 Study Shows High Protein Diet *Did Not Increase Bone Loss*

"The effect of a low carbohydrate [**higher protein**] **diet** on bone turnover." *Osteoporosis International* (Carter, JD, et al., **2006**, May 23 [Epub ahead of print]).

If patients follow a "low-carbohydrate diet," they are consuming a much greater amount of protein, i.e., a higher-protein diet. Here's the conclusion you need to know:

- **strict low-carbohydrate [high protein] diet had *n effect* on bone loss** for adults **following an Atkins-type [high protein] diet for weight loss**, a three-month study by rheumatologists at the University of South Florida found."

- "Patients on the low carbohydrate diet **did lose weight** but the diet **did not** appear to **compromise bone integrity or lead to bone loss**."

- "'**I was <u>surprised</u>** by the results,' Dr. Carter said.

"**<u>The difference in bone turnover</u>** between the low carbohydrate dieters and the non-dieters **<u>was insignificant</u>** after three months."

2008, *British Medical Journal* reports calcium supplements increase cardiovascular events and brain lesions.

Bolland, MJ, et al., "Vascular events in healthy older women receiving calcium supplementation: randomised controlled trial," *British Medical Journal* **2008**; 336:262-266.

- "**Myocardial infarction was more commonly reported in the calcium group** than in the placebo group (45 events in 31 women vs 19 events in 14 women, P=0.01).

- "CONCLUSION: **Calcium supplementation** in healthy postmenopausal women is associated with **upward trends in cardiovascular event rates**. This potentially **detrimental effect** should be balanced against the likely benefits of calcium on bone. [Note: Calcium supplements are not required for anyone unless the patient suffers a pathophysiologic disorder.]

- "The finding of an **adverse trend in vascular events with calcium supplementation is not necessarily surprising**, since calcium supplements acutely ***elevate serum calcium levels possibly accelerating vascular calcification***, which is predictive of vascular event rates. **High calcium intakes** have also been **associated with brain lesions** on magnetic resonance imaging scans and with **vascular calcification** and mortality in patients who receive dialysis."

Protein Positively impacts blood chemistry — Verified in 2005 OMNIHEART STUDY[1]

If you are a cardiologist, you likely saw this on-line at the cardiologist's news journal, *theheart.org*. Once again,

---

1 http://www.theheart.org/printArticle.do?primaryKey=601763, ref: Appel, JL, et al., "Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids." Ref.: *Journal of the American Medical Society* 2005; 294:2455-2464.

we see what "sounds good" again failing to predict the *real-life* results of your patients:

- "**Turning conventional dietary wisdom on its head** results of the OMNIHEART study indicate that substituting **proteins** or **unsaturated fats** [PEOs] for carbohydrates within the context of a healthy diet can **reduce blood pressure and improve lipid profiles**.
- "…**Compared** with participants eating the **carbohydrate-rich diet**, those eating the **protein-rich diet** had greater **reductions in blood pressure**, **LDL**, and **triglycerides**…
- "... [Dr. Barbara] Howard also took issue with the study's focus on monounsaturated fats, saying she would have preferred a study emphasizing polyunsaturated fats [**PEOs**], which **are known** to have a **better effect on cardiovascular risk than monounsaturated fats**.

Report from the Association of Official Analytical Chemists International (AOAC) (2005):

- "**Digestibility of protein** in **traditional diets from developing countries** such as India, Guatemala, and Brazil is **considerably lower** compared to that of protein in typical North American diets.
- "The presence of **less digestible protein** fractions, high levels of **insoluble fiber**, and high concentrations of ***anti-nutritional factors*** in the diets of developing countries, which are ***based on less refined*** **cereals and grain**

legumes [soybeans, peanuts, beans, lentils, chickpeas, etc.] as major sources of protein, are **responsible for *poor digestibility* of protein**.

- "**Anti-nutritional factors may occur naturally**, such as glucosinolates in mustard and rapeseed [Canola] protein products, trypsin [required for digestion] inhibitors and hemagglutinins [causing heart attacks] in **legumes** tannins in legumes and **cereals**, **phytates** in cereals and oilseeds [husks only], and gossypol in cottonseed protein products.

- "The presence of **high levels of dietary trypsin inhibitors** from **soybeans, kidney beans, or other grain legumes** can cause **substantial reductions in protein and amino acid digestibilities** (up to 50%) in rats and pigs."

Ref.: Kimura, Yasumi, et al., "Meat, fish and fat intake in relation to subsite-specific risk of colorectal cancer: The Kukuoka Colorectal Cancer Study, *Cancer Science*, 2007, Vol.98; (4):590-597.

- "Recent studies published in the journal *Cancer Science* (from the Fukuoka Colorectal Cancer Study), a population-based case-control study, covering 782 cases and 793 controls) have ***disproved the myth*** that consumption of **red meat increases *colorectal cancer***:

- "Researchers have run a **large case-controlled study in Japan**, examining associations of meat, fish and fat intake with risk of colorectal cancer…"

- "[F]ound that intake of **beef**/pork, processed meat, total fat, **saturated fat** or n-6 PUFA [**parent omega-6**] showed **no clear association** with the overall or subsite specific risk of **colorectal cancer**.

- "Our findings **DO NOT SUPPORT** the hypothesis [guess] that **consumption of red meat increases colorectal cancer risk**…

2009—More confirmation that meat, in and of itself, is not colon cancer causing.

Key, TC, et al., Cancer incidence in vegetarians: results from the European Prospective Investigation into Cancer and Nutrition (EPIC-Oxford)," *American Journal of Clinical Nutrition* **2009**;89(suppl):1620S–6S.

- "This was a prospective study of *63,550 men and women* recruited throughout the United Kingdom in the 1990s. **Cancer incidence was followed through nationwide cancer registries**

- "Within the study, the incidence of **all cancers combined was lower** among **vegetarians** than among meat eaters, **but** the **incidence of colorectal cancer was higher in vegetarians than in meat eaters**. "Our observation that the incidence of **colorectal cancer** is **higher among vegetarians than among meat eaters** in the EPIC-Oxford study **is surprising**...."

Malhotra, SL, "*Epidemiology of Ischaemic Heart Disease in India with Special Reference to Causation," Br Heart J*. 1967 November; 29(6): 895–905.

- "Our data, however, **do not support this association of** high fat [**from significant meat consumption**] intake with the liability to develop ischaemic heart disease, because while in the **north** the consumption of fats, most of which are **animal fats, is *19 times more* than in the south (Indian Council of Medical Research, 1964**), the **disease is *7 times less* in the north than** in the south. Moreover, while the **milk fats eaten in the north** have a preponderance of **saturated fatty acids**, the seed oils used in **the south** are mainly composed of **unsaturated fatty acids** (Indian Council of Medical Research, 1963). [Note: Dr. Rowen donates his time and expertise to Indian patients during his yearly trek to India. He reports extensive adulteration of their cooking oils.]

- "This **inverse association is noteworthy**, especially because **others have also observed this association of high intake of animal fats and freedom from cardiovascular disease** (Shaffer *et al.*, 1964; Shaper, Jones, and Kyobe, 1961; Mann, Shaffer, and Rich, 1965). This evidence from other studies and our additional findings of the **inverse association** of a low intake of total as well as animal fats [**meat eaters**] and a high frequency of **ischaemic heart disease** in the south [vegetarians] are **contrary to the view** that it is the quantity of dietary fats and their degree of saturation [saturated fat] that bear responsibility for cases of this disease.

- "**Neither** smoking, nor socio-economic factors, ***nor physical activity*** of work, nor even stress and strain **have provided any tenable associations with the**

immunity from or a liability **to develop ischaemic heart disease**, in the data presented in this paper. **Nor is there any incontrovertible evidence that the total amount of fat in the diet bears responsibility for the production of this disease**.

- "*Physical Exercise versus Inactivity.* **An unexpected and extraordinary finding in our data is that mortality in the *sedentary* occupation of clerks is lower** than the physically active occupation of fitters. Furthermore, in the same type of physical jobs, 15 times higher in the south [vegetarian fitters]. [Note: Patients can't merely "exercise away" a nutritional deficiency or solve the consumption of adulterated PEOs by exercise.]

Carbohydrates, *Not Proteins*, Destroy Good Blood Chemistry

Chapter 5 will discuss the effects of carbohydrates in more detail. Dr. Gerald Reaven—the physician who coined "metabolic syndrome"—shows by experiment that **increasing carbohydrate consumption by only 20% yields their poor and disturbing results via altered blood chemistry** (Stanford University School of Medicine: *American Journal of Cardiology* **2000** 85:45-48, (Dr. Gerald Reaven)). To the contrary, protein doesn't have a deleterious effect on blood chemistry.

Even a High-Fat / *High-Protein Diet* with *Lots of Cholesterol is Insignificant*

Cholesterol comes from animal-based foods, not plant-based foods. Dr. Raven shows this again in his journal article

(Reaven, GM, et al., "Insulin resistance, dietary cholesterol, and cholesterol concentration in postmenopausal women," *Metabolism – Clinical and Experimental*; Vol. 50 (5), May **2001**, pages 594-597. A key finding was that consumption of high amounts of cholesterol-containing foods **did not result in a proportionate increase in** blood cholesterol. You may find amazing, as I did, the effectiveness of the body's automatic regulation of cholesterol:

"With even a 30% fat diet, ***increasing dietary cholesterol*** from 319 mg to 941 mg per day [close to ***a huge 300% increase***], the blood LDL-C was only a ***mere 6% increase*** [20 points]!"

Even insulin-resistant women did not experience a significant cholesterol increase!

From Dr. Rowen:

Let's look further. Vitamin C is also crucial. Mount Sinai researchers[2] have shown in an animal model that the vitamin actively protects against osteoporosis. Mice were given ovariectomies and were compared to mice given a sham operation. The ones with ovaries removed were further subdivided to receive vitamin C or no vitamin C. The researchers found that mice absent ovaries that received vitamin C had bone mineral density matching that of mice with intact ovaries. Essentially, vitamin C effectively replaced ovarian function in protecting bone! The nutrient does this by activating osteoblasts (bone-forming cells) to become fully active. Where do you get vitamin C? Certainly NOT from animal flesh or animal protein at all. You get this crucial nutrient from fruit and vegetables, and it is partially destroyed by heat! Hence, my preference for living foods!

Now let's look at a recently published study which, in my opinion, slam-dunks the issue. A simple nutrient, potassium citrate (together with vitamin D and calcium), was found to significantly increase bone mineral density in elderly osteoporosis-free men and women. Dr. Reto Krapf of Switerland, one of the authors, told Reuters Health, "By neutralizing the acid we generate by our diet, it is possible to slow or possibly reverse the age-related decline in bone density and bone mass." Their research comes on top of earlier work that osteopenia (mild osteoporosis) can be reversed with potassium citrate. The supplement completely neutralized their bodies' acid production, and they had lower calcium excretion at 6 and 12 months of the study. High-resolution tests confirmed more bone density and improved bone structure.

---

2 *Science Daily,* October 9, 2012

Having said this, I do agree with the good professor regarding the protein need for healthy bone. After all, the bone matrix is protein, and calcium is ushered in to harden this protein base. But, clearly from the above, it is not necessary that the protein you eat be all or even mostly animal protein. In fact, consumption of dairy, with all its calcium and protein, hasn't been shown epidemiologically to protect. Why? Cow milk is loaded with phosphorus. Your body will excrete calcium along with the load of phosphorus. This suggests that dairy will be largely pH- and osteoporosis-neutral on your body. "Mild" is considered pH neutral by clinicians. Finally, another problem with most dairy is that it has been pasteurized. The heat destroys certain nutrients, including enzymes necessary to free up calcium and other nutrients for absorption.

With regards to cancer, I agree with Prof. Peskin that it's largely processed meat that raises risk. Processed meat has foreign chemicals, and other man-made adulteration, that would not be good for any God-made creature. On the other hand, meat requires cooking to kill potentially horrific contaminating bacteria—at least for human consumption. I've yet to see a lion roast a zebra. I'll be discussing the toxic impact of heat on your food in Chapter ___. Additionally, meat made for human consumption may be also loaded with hormones, pesticides, and other chemicals dumped into the animals to increase corporate profit at the expense of your health. I have no issue with you eating organic grass- or range-fed animals or wild fish from unpolluted waters.

Now to me, the greatest mind in history regarding diet was Weston Price, DDS, who authored Nutrition and Physical Degeneration in 1939. He traveled to aboriginal cultures all over the world and found that their usual state of good health was not tarnished UNTIL they adopted what I call "the white man's food." That is the term I use to refer to the processed and adulterated foods of western man. Clearly,

eating meat was not a problem for aboriginal cultures. However, I do feel you can eat too much, even in an aboriginal group.

Samuel Hutton wrote Health Conditions and Disease Incidence Among the Eskimos of Labrador after observing Inuits from 1902-1913, when they were still eating their aboriginal high meat/seafood diet. Indeed, they were extremely robust and healthy when young. But, by middle age, their vital organs began to break down. They aged rapidly, and suffered severe osteoporosis. (Hmmmm.) They also had low resistance to infectious disease. Hutton confirmed the fact that cancer and other diseases of civilization were rare in the Inuits but had this to say about their life expectancy: "Old age sets in at fifty and its signs are strongly marked at sixty. In the years beyond sixty, the Eskimo is aged and feeble. Comparatively few live beyond sixty and only a very few reach seventy…." Missionaries had left careful records of these facts for over 100 years. These observations were written years before the current controversies over the "right" diet, so we are getting information at the source.

Now I can't tell you how much protein is too much. But, I firmly believe we only need about 30–40 grams of high-quality protein per day. **What is high-quality protein? It is a protein that has all the essential amino acids, and is DIGESTIBLE.** Plant proteins can lack certain essential amino acids, for sure, but, in contrast to animal protein, they are seemingly more digestible. Can you get enough protein from plants? Well, I do my rigorous hiking as a raw food VEGAN, and I do quite well! I believe that plant amino acids are more readily available than those in meat.

True, many plant proteins are deficient in one or more amino acids. But look at our great ape cousins. They forage and eat a large variety of vegetarian foods, and no dairy. They have plenty of amino acids to build muscles far stronger than our own. The variety of plant sources ensures

a good quantity of all essential amino acids. (I'll have more on our great primate cousins in a later chapter.)

Furthermore, contrary to popular belief, many plants other than the scorned soy have complete amino acid profiles. Spinach and other leafy greens, for example, are packed with protein. In fact, 30 grams of spinach contain 1 gram of highly digestible, full-spectrum protein, or close to 3%. While not on a par with animal flesh by weight, it clearly has what I need. I average about 1 pound per day of a mixture of green, leafy veggies, in addition to a wide variety of other plants. With one pound being 454 grams, I get about 15 grams of protein from these greens ALONE. Nuts, seeds, and fermented dairy add to my base. My blood-essential amino acids are all well within an acceptable range.

There is epidemiological evidence of the superiority of plant-protein-based diets. Campbell reported in The China Study that all-cause mortality rose in proportion to the amount of animal protein eaten in several rural China villages. While there are some holes in the methodology, the observation is hard to refute. Former President Bill Clinton had serious heart disease. In September 2004, he underwent quadruple bypass. In 2010, he adopted the suggested diet. He effectively lived as a vegan, eating vegetables, fruit, legumes, and a morning protein shake. The New York Times (August 18, 2011) reported that he quickly lost 24 pounds and returned to his college weight.

The longest-lived peoples in the world today include: the Abkasia of Southern Russia, the Vicalbamba Indians of the high Andes, the Hunza of north Pakistan, Okinawans, and the California-based Seventh-day Adventists. John Robbins, in Healthy at 100: The Scientifically Proven Secrets of the World's Healthiest and The Longest-Lived Peoples, describes the lifestyles and dietary patterns

of these long-lived cultures. He reports that the calorie breakdown of the food the first three groups ate daily was between 69–73% carbohydrates, 15–18% from fat and 10–13% from protein. With the exception of Okinawans, who eat fish, these are largely vegetarian societies. Overall daily calories ranged between 1,700–1,800, while the Abkhasia ate 90% plant foods and the Vilcabamba and Hunza ate 99% plant foods.

All three ate low amounts of salt, zero sugar or processed food, and had no incidence of obesity and other common diseases.

Robbins also discussed the Okinawan people. Also a long-lived group, they do eat a more animal-based diet (fish) but had a similar lifestyle as the other groups.

In America, the Seventh-day Adventists continue the pattern. They are culturally a rather homogenous lot for America, and are the longest-lived cultural group in our country. Those who choose a vegetarian lifestyle live, on average, about four to five years longer than their carnivorous counterparts, and use the medical system far less.

One medical report on Adventists tells it clearly: The lifetime risk of ischemic heart disease was reduced by 31% in those who consumed nuts frequently and by 37% in male vegetarians compared with nonvegetarians. Cancers of the colon and prostate were significantly more likely in nonvegetarian Adventists (relative risk—RR—of 1.88 and 1.54, respectively), and frequent beef consumers also had higher risk of bladder cancer. Intake of legumes was negatively associated with a risk of colon cancer in nonvegetarians, or a risk of pancreatic cancer. Higher consumption of all fruit or dried fruit was associated with lower risks of lung, prostate, and pancreatic cancers. Cross-sectional data suggest vegetarian Seventh-day Adventists have lower risks of diabetes mellitus, hypertension, and arthritis than nonvegetarians. Thus, among Seventh-day Adventists, vegetarians

are healthier than nonvegetarians. But this cannot be ascribed only to the absence of **meat**.[3]

I want to emphasis caution in interpreting the conclusion that the health of the vegetarians is due to absence of meat only. Those who are vegetarian might be taking many steps to preserve their bodies. However, these epidemiological findings remain consistent with my observations and many other reports, one of which I'll mention.

The Oxford Vegetarian Study[4] is a United Kingdom prospective study of 6000 vegetarians and 5000 nonvegetarian control subjects. They were recruited between 1980 and 1984 and followed for 12 years. All-cause mortality in the whole group was half that of the population of England and Wales. After adjusting for smoking, body mass index, and social class, death rates were lower in non-meat eaters than in meat eaters for each of the mortality endpoints studied, including vascular disease and malignancy. More animal products were positively associated with coronary disease. Non-meat eaters had only half the risk of meat eaters of requiring an emergency appendectomy, but, vegans were found at risk for iodine deficiency. The authors stated, "The health of vegetarians in this study is generally good and compares favorably with that of the non-vegetarian control subjects."

This study did state, however, that the size of the study precluded meaningful investigation of mortality from specific diet-related cancers, and recommended the EPIC study, which was in progress at the time. The EPIC study ultimately did show a higher colon cancer rate in vegetarians taken

---

3 Fraser, GE, "Associations between diet and cancer, ischemic heart disease, and all-cause mortality in non-Hispanic white California Seventh-day Adventists," *Am J Clin Nutr* 1999;70(suppl.):532S-538S.

4 Appleby, Paul, N, et al., "The Oxford Vegetarian Study: an overview," *Am J Clin Nutr* 1999;70(suppl):525S–31S.

from the general population, whereas the study of Adventist vegetarians showed a significantly lower rate, which would indicate that there were unknown variables at work. The EPIC study was reported by Prof. Peskin earlier in this chapter.

Finally, more intake of particular amino acids, including essential, might not be better. Consider the essential amino acid methionine. Some recent research[5] has found that restricting this essential amino acid in animals provides similar longevity enhancement as calorie restriction in rodents. Restriction of methionine reduces mitochondrial oxidative damage, reduces mitochondrial membrane unsaturation (important as per Prof. Peskin), and decreases five different markers of protein oxidation (markers of damage) in rat heart and liver mitochondria. This research group found that methionine supplementation increased oxidative damage in rat liver mitochondria. They expressed concern that the methionine in the average western diet is 2–3.3 fold higher than the average adult requirement. Furthermore, there is an abundance of literature now that methionine restriction might be a cornerstone of cancer treatment. Perhaps that's one reason why Max Gerson, MD was so successful years ago in CURING cancer with raw vegan diets and juicing. These food sources do have low methionine relative to methionine abundance in meat.

Clinically I definitely find that certain people do better eating some animal protein. Among these are those with blood group O. These people may have more direct links to our Paleolithic ancestors

---

5 Gomez, J., et. al., "Effect of methionine dietary supplementation on mitochondrial oxygen radical generation and oxidative DNA damage in rat liver and heart," *Journal of Bioenergetics and Biomembranes*, vol 41, issue 3, PP 309–321.

eating a hunter-gatherer diet. They also, in my experience, are highly intolerant of grains. People with blood type A seem to do quite well with little or no animal flesh. I haven't seen enough type Bs to make a definitive clinical opinion, but some seem to do better with at least a small amount of animal protein. You can find more on eating for blood type in the book by Peter D'Adamo, Eat Right for Your Type. I don't agree with everything he says, but I do find clinically that his position on blood type O is the most accurate of the four blood types.

The basis for anyone's diet should be Living Foods (uncooked), regardless of blood type or anything else. I encourage all my patients to eat 75% of their diet as nature made it (other than chopping, blending, etc.). ***With the other 25%, I don't care, just as long as it is not fast, fried, refined or processed.*** Those making this change respond so much better clinically that this change alone could run Pharma out of business. I don't encourage grains, especially those containing gluten, because grains must be processed or cooked.

Weight loss? Clinically it is not problematic for those following this plan. Metabolic syndrome (insulin resistance) and Type 2 diabetes (adult) in the USA is epidemic. Ninety percent of these problems in our country are related to diet-induced obesity. Gabriel Cousens, MD, in his book, There Is A Cure For Diabetes, demonstrates absolute cures of the problem following a raw food diet. I've found the same cures/improvements in my patients independently.

I'll have more for you in my chapter devoted to Living Foods, where I will use myself as the example.

While Prof. Peskin and I have a different take on diet here, there remains a common thread. We'll be getting to a surprising common denominator, which we both believe trumps our individual views on vegetarian vs. non-vegetarian. For anyone who eats, the PEO Solution is for you!

Addendum. Just after completing this chapter, a major article[6] was published basically confirming my decades warnings about red meat. It even took the good professor by surprise. It was NO surprise to me, but clearly slammed the meat advocates of the world. This is such an important finding and issue that I decided to provide you with the uncut version of information I presented to my *Second Opinion* readers. I do think that after reading and digesting this information, you'll come to my side on the red meat issue. This report confirms the analysis of the professor on cholesterol, but comes to a different conclusion regarding the safety of red meat for completely different reasons.

> *Folks, sometimes human intrigue leads us to some amazing discoveries, even in conventional medicine. Such has just happened with a study exploring, a bit more deeply, a possible connection between heart disease and red meat.*
>
> *You have repeatedly heard in the press that red meat might not be so good for you. Of course, the cattle industry's back bristles at every one of these challenges.*
>
> *Years ago, we were led to believe it's all the fat or cholesterol in red meat, especially if it's marbled. Then, we were led to believe that the culprit is processed meat. Indeed processed meat is far worse. It's famed for its nitrites and other toxic additives. But more recently, unprocessed red meat has been linked to greater heart disease.*
>
> *Now this goes against what those advocating the Paleolithic diet believe. That diet is touted as the answer to*

6 Koeth, et. al, Intestinal microbiota metabolism of l-carnitine, a nutrient in red meat, promotes atherosclerosis, *Nature Med*, April 7, 2013.

just about everything. Eating as our "caveman ancestors." A diet of animals they hunted, and plant materials they gathered. Sounds lovely, doesn't it? Just eat like our ancestors and you might not get heart disease? Well, you just might get heart disease after all. And new research strongly suggests that you can. Furthermore, this research may fully explain the "mystery" of why our Egyptian ancestors had heart disease (discovered on CT scans of mummies). The news media would have you believe that since these ancestors had coronary disease 4000 years ago, and they were not eating a "modern" diet, that humans are condemned to have heart disease simply by being human.

I reported this finding regarding mummies, and told you otherwise—that there had to be other factors, such as what they were eating and how they prepared their food, even if it were not processed like today's "food." I am simply delighted to bring you research from the prestigious Cleveland Clinic that provides the link between coronary disease in mummies to the heart disease of today. And best of all for me, this discovered link validates everything I have been bringing to you in these pages since I took over the helm at SO. Hold on to your hats, as I tie a fascinating puzzle together for your consideration and action.

Dr. Stanley Hazen is section head of preventive cardiology and rehabilitation at the Cleveland Clinic. There, scientists and clinicians wondered out loud about the cholesterol/fat theory of red meat as a cause of coronary disease. ***See, the cholesterol and saturated fat theory just didn't pan out. Even the mainstream is finally "getting it."*** *Hazen's group thought that there might be something else*

in red meat that was a key culprit, trumping its cholesterol and marbled fat.

The researchers kept samples of blood on more than 10,000 patients and followed them for the subsequent development of heart disease. Then, they started looking for the unknown. They found higher levels of a common and needed molecule for human physiology in their blood—carnitine. But it turned out that it wasn't just carnitine itself. We need carnitine for mitochondria to work. They found that higher carnitine was associated with another molecule, induced by ingestion of carnitine. Specifically, intestinal bacteria convert carnitine into a little-known molecule. The intestines absorb that molecule. Then, once in the liver, it is further converted into a molecule called TMAO (trimethylamine-N-oxide). ***Their research led them to conclude that TMAO carries a 10-fold risk for heart disease compared to cholesterol****.*

I'll tell you more about it in a moment.

The researchers combined some novel findings in mice and human subjects, including THEMSELVES. Hazen was actually a part of the human side of the study!

First though, the mice. The team found that chronic supplementation of carnitine, which is structurally similar to amino acids, reduced "reverse cholesterol transport." That is the process by which your body picks up and removes cholesterol from your arteries and delivers it back to your liver for elimination through the bile. You do NOT want that process hindered.

Now the underlying factor in these mice was their intestinal bacterial (flora) composition. The chronic carnitine supplementation altered their flora. It encouraged growth

of organisms that broke down carnitine (and choline) into TMAO. This did not happen if the intestinal flora of the mice was suppressed with powerful antibiotics. In other words, bacteria did the transformation.

The researchers then took this information and did a study on a small group of 6 humans, including Hazen. They simply fed them all a juicy sizzling sirloin steak. They wanted to know if eating the steak would raise TMAO levels. The answer was YES! TMAO levels simply soared! But there was a single notable exception. One of them didn't get a TMAO burst. That person had been a vegan for more than a year.

This peculiar finding was confirmed with additional research on 23 vegetarian/vegans and 51 meat eaters. The meat eaters had more TMAO in their blood. And, the meat eaters readily converted supplemental carnitine into TMAO. ***The vegetarians/vegans did not make the risky conversion! And, the researchers admitted that they were shocked at this finding!***

Many researchers were quick to compliment the report. Dr. Daniel J. Rader, a heart disease researcher at the University of Pennsylvania School of Medicine, said, "It's really a beautiful combination of mouse studies and human studies to tell a story I find quite plausible." Dr. Frank Sacks is a professor of cardiovascular disease prevention at the Harvard School of Public Health. He called the findings impressive, saying, "I don't have any reason to doubt it, but it is kind of amazing." Lora Hooper, an associate professor of immunology and microbiology at the University of Texas Southwestern Medical Center is a paleo diet follower. Her comment about the study: "YIKES!"

Of course researchers are already saying this will lead to new treatments for heart disease, perhaps including an antibiotic to wipe out the specific organism responsible. Regardless, TMAO may become a viable new blood test to assess your risk, and that test is in the works. I think the former (antibiotics) is ludicrous. What about the bacteria we rely on to make vitamin K and other key nutrients we need? On the other hand, a test for TMAO in your blood is a terrific idea.

Of course, critical questions remain. Would people reduce their heart attack risk if they lowered their blood TMAO levels? An association between TMAO levels in the blood and heart disease risk ***does not necessarily mean that one causes the other*** *[as detailed in chapters 2–3]. And which gut bacteria in particular are the culprits?*

There also are questions about the safety of supplements, like energy drinks and those used in bodybuilding. Such supplements often contain carnitine, a substance found mostly in red meat.

But the investigators' extensive experiments in both humans and animals, published April 8, 2013, in Nature Medicine, have persuaded scientists not connected with the study to seriously consider this new theory of why red meat eaten too often might be bad for people.

Folks, let me summarize these findings, and connect the dots for you in a manner you won't hear from these fine researchers. Hazen's group previously found TMAO to be a far greater predictor of heart disease than cholesterol. (Remember, I've repeatedly told you that the cholesterol theory just doesn't hold up. This may partially explain why.) Their laboratory studies determined that TMAO enables

cholesterol to get into artery walls and also prevents the body from excreting excess cholesterol. You might remember me repeatedly telling you that it's NOT cholesterol. It's what your body (or cooking or other handling) does to cholesterol.

In this case, bacteria in your gut convert a nutrient molecule into a bacterial by-product. That by-product goes to your liver, which converts the by-product molecule into TMAO, a now proven ***activator*** *of cholesterol pathogenesis. Vegetarians don't have this problem. Meat eaters do. And, their levels of TMAO "spike" upon presentation of carnitine either in meat or supplements. Vegetarians don't so spike. And, meat eaters given an antibiotic to temporarily wipe out their gut bacteria also did not spike. The elegant research proved that the culprit was intestinal bacteria feasting on the carnitine, and converting it to something you don't want in your body.*

Now, my comments. I also say "YIKES!"—but only on a positive note for all the heat I've taken for my dietary stands. Please remember the report I gave you on bacteria in your gut contributing to your weight. See, what you eat determines the kind of bacteria you harbor. Obese people are harboring bacteria that thrive on what they are eating. The problem for them is that these organisms further contribute to their obesity by enabling more calorie absorption from "foods" that would otherwise not be digestible. The bacteria certainly win that way. You don't!

Here we see that meat eaters are harboring an organism that converts readily available carnitine in meat to a nasty compound. Naturally, your gut will draw out and select those organisms if you are providing a fertile ground for them by

regularly eating carnitine-rich meat. (Fish, chicken and dairy have carnitine but in lesser amounts.)

Now this fascinating work connects many dots. It explains why I rarely, if ever, see heart disease in vegetarians who eat high-quality food. It provides a crucial missing link into the debunked cholesterol hypothesis. TMAO is 10 times the risk of cholesterol! And, it can explain why we found heart disease in Egyptian mummies. Generally it was the rich who could afford the expensive mummification process. And throughout history, the wealthy have eaten red meat. It then might not have mattered that the Egyptians had little in the way of modern processed foods. Perhaps they also fried food as well. They did have olive oil!

So what reasonable action should you take? There are two questions raised: 1) what about meat in your diet, and 2) what about carnitine supplements? Three ounces of red meat provide 95 mg of carnitine. Pork has about a third of that, and fish far less. (I think pork is quite bad for other reasons. Its flesh is similar to humans, making digestion and elimination of similar toxins we carry a real problem.)

Supplements may contain as much or more carnitine than three ounces of red meat. But, I am not running from carnitine as a supplement at this time. Why? Because I believe that your gut flora are more dependent on what you eat than what you supplement. I've used carnitine myself from time to time. Mitochondria need it. And considerable research does support its benefit.

I have long believed that eating meat is not ideal for humans, especially when I look at our closest cousins in nature—the great apes. We cook meat. Carnivorous animals

don't. Muscle protein we eat is also different than the organ meat that carnivores prefer. The former is far more difficult to digest and absorb. Consequently, undigested flesh protein works its way through your gut, potentially provoking a feeding frenzy by some "bad" bacteria. Since we know that slender people have a far different bacterial flora than obese people, I suggest that meat eaters also have a different flora (as compared to vegetarians). This might lead to their higher risk of vascular disease.

Finally, consider the great carnivores. The lion, a larger mammal than us, has a digestive tract of only about 12 feet. Ours is about 30 feet. Seems the lion "knows" he better poop out protein digestion products more quickly than we humans, who retain intestinal contents far longer with our long gut.

I'm not suggesting that you totally eliminate red meat, nor have I in the past. I'm vegetarian for spiritual reasons. If you want your steak, have it, but please de-emphasize red meat as a primary food. (I wonder how many heart attacks our government brought on with its bogus "food pyramid" with meat at the top when I was growing up.) Consider having red meat once weekly or, even better, as a treat once every two weeks. A by-product of less meat consumption will be a far lower carbon impact on the environment. You can't imagine the environmental devastation occurring as large amounts of the Amazon forests are leveled to grow cattle feed or to raise cattle for human food.

As mentioned, poultry, fish and dairy have far less carnitine. Fish might be the best choice as it easily flakes, and may be the most digestible. However, you then run into the problem with the lack of labeling of the farmed/toxic fish

and new "Frankenfish." And, fish can be highly polluted. So, just remember— moderation.

Please follow the Living Foods Diet 75% of the time. I don't care what you do with the remaining 25% so long as it is not fast, fried, refined or processed. But now, I certainly feel I have more weight behind me in encouraging you to make red meat a minor portion of that 20%. Please remember that you first heard this paradigm in ***Second Opinion.*** I do wonder how my predecessor at SO will react to this news. He's been a long-term advocate of a meat-based diet. Many people to this day believe that we are getting all the vegetation we need by eating grass-eating cows, since it is grass that sustains them. Well, if cows are providing us all the vegetation we need, seems that our intestinal bacteria just haven't learned that yet.

Scientific Support for Chapter 5

Voet, Donald; Voit, Judith G., *Biochemistry*, 2nd Edition (John Wiley & Sons, 1995), 790, from the chapter "Adipose Tissue":

> "Adipose tissue obtains most of its fatty acids from the liver or from the diet.... as described in Section 23-1. Fatty acids are activated by the formation of the corresponding fatty acyl-CoA and then esterfied [for storage] with ***glycerol-3-phosphate*** to form the ***stored triacylglycerols*** [body fat] (Section 23-4F). The ***glycerol-3-phosphate arises*** from the reduction of dihydroxyacetone phosphate, which ***must be*** glycolytically ***generated from glucose*** because ***adipocytes*** [body fat] ***lack a kinase*** that phosphorlates endogenous glycerol."

Veronique Douard and Ronaldo P. Ferraris, "Regulation of the fructose transporter GLUT5 in health and disease," *Am J Physiol Endocrinol Metab* 295: E227–E237, 2008

- "In this review, we describe the regulation of GLUT5 not only in the ***intestine and testis***, where it was first discovered, but also in the ***kidney, skeletal muscle, fat tissue***, and ***brain*** where increasing numbers of cell types have been found to have GLUT5.
- "Most of the increase in ***consumption*** is derived from ***refined or processed*** fructose. "

- "***Fructose is the sweetest*** of all natural sugars…

- "The myriad effects of fructose are possible ***only if fructose reaches physiologically significant concentrations*** i the plasma and extracellular fluids and if subsequently transported into cells of various organ systems, thereby potentially altering normal metabolism in those organs....

- "In healthy humans consuming high-fructose or -sucrose diets, ***serum fructose can reach 0.2–0.5 mM, but this concentration is still very low compared with normal blood glucose levels (5.5mM)*** [Note: 5.5mM is the 70–90 mg/dl) measurement all diabetics and their physicians know well.]

- "However, it is clear that the rate of ***glycolysis can be stimulated by fructose*** because its entrance into glycolysis skips the two main regulatory enzymes (glucokinase and PFK-1).

PEO Solution analysis: Naturally occurring fructose from unprocessed whole fruits is no issue whatsoever. However, physicians treating cancer patients need to be aware of the following 2 key matters:

If you continue to think that there is merit in using the glycemic index, there are other significant problems with using this measure. Professor of Nutrition Julie Miller Jones, Ph.D., at the College of St. Catherine in St. Paul, Minnesota (past holder of the 3M Endowed Chair in Science), has reviewed the current research and tells us of some important Glycemic Index drawbacks. The following excerpts

are from her publication "Contraindications and Challenges: A Look at the Glycemic Index":

"...**Surprisingly**, the **day-to-day variation** in the same subject [person] is often greater than [the] **variation between subjects [people].**

"The **food eaten at the previous meal can also affect the glycemic response** at the current meal...."

As early as 1944, Dr. Blake Donaldson at New York City hospital used radioisotope tagging to prove that carbohydrates were rapidly converted to body fat—**significantly more body fat was added to your frame from carbohydrates than from eating fat or protein.** Now, many years later, university professors of nutrition often aren't even aware of this. Therefore, their embarrassing "expert" recommendations are often harmful and should embarrass them.

Humans can't digest fiber.

Here is what the *Textbook of Medical Physiology,* 9th edition, page 834 states:

> "However, **no enzymes** capable of hydrolyzing [breaking down] cellulose [fiber] are secreted **in the human digestive tract. Consequently, fiber cannot be considered a food for the human being**."

Fiber/Colon Cancer—those people eating the most fiber get the *most* colon cancer!

The *New England Journal of Medicine* (Jan. 21, 1999, Vol. 340, No 3) reported that:

- Fiber did nothing to improve "colon efficiency."

The following year *Lancet* (October 14, **2000**; 356:1286-1287, 1300-1306), the world's premier medical journal, published the same finding again:

- Those people eating the **most fiber get the *most* colon cancer**! The fiber found worthless to protect against colon cancer was **the highly promoted *soluble* fiber**

Nutritionists and too many physicians didn't see this information. Don't let them or cereal manufacturers mislead you into thinking fiber is healthy.

Albion Research Laboratories Agrees *Fiber Leaches Minerals*

Albion Research Notes – A Compilation of Vital Research Updates On Human Nutrition, Albion Laboratories, Clearfield, UT (Vol. 6, No. 2, June 1997) stated:

> "**Natural sources of fiber, such as cereals and fruits, generally have a *depressing effect* on absorption of minerals** such as calcium, iron, zinc, and copper. Imagine taking **mineral supplements** and ***still going into a negative balance for the very minerals that are being supplemented***!"

Women Eating the MOST Fiber Get LEAST Calcium Retention

Once again, the fiber fallacy is presented in the *Journal of Clinical Nutrition*, 2000, 71:466-471:

> Women eating the most fiber and the lowest amount of fat had 20% lower calcium retention.

Body AUTOMATICALLY Converts Glycogen and Stored Fat to Sugar AS NEEDED

Basic Medical Biochemistry tells us on pages 28-29, 394, and 428:

> The **body can convert** glycogen (stored **carbohydrate** in the liver and muscles) into glucose **whenever needed** AND can **also convert** our **fat** reserves **to glucose** (blood sugar) **as needed** in a special process called *glucogenesis*.

Blood Sugar AUTOMATICALLY Balanced

Textbook of Medical Physiology on page 863 states:

> "The normal blood glucose concentration in a person who has **not eaten a meal within the past 3 to 4 hours** is about **90 mg/dl**." Note: This amounts to less than 1 teaspoon of sugar—the NORMAL amount of sugar we desire.

Don't be fooled by anyone telling you that "blood sugar balancing" for non-diabetic has to be maintained by eating carbohydrate-based foods, multiple times each day.

Insulin, a Response to Carbohydrates, Makes You Fat!

It's all in the *Textbook of Medical Physiology* on pages 974-975:

"…**[I]nsulin promotes deposition of fat** in these cells.

"**Insulin promotes glucose transport** through the cell membrane into fat cells [**making fat cells larger**]….

"Therefore, when **insulin is not available** [caused by the response to carbohydrates], even **storage of large amounts of fatty acids** transported from the liver in the lipoproteins is almost **blocked**.

"**All aspects of fat breakdown** and use for providing energy are **greatly enhanced in the absence of insulin** [generated from carbohydrates]."

Minimize the insulin production and you AUTOMATICALLY **m**inimize the fat production, too.

Carbohydrate Diet Clogs Your Arteries, Too.

As *Journal of American Medical Association*; **2000**; 283:221-228 makes clear:

Elevated **insulin** [generated **from eating carbohydrates**] causes blood clotting, which **blocks arteries**.

Carbohydrate Diet AWFUL for Diabetics

A carbohydrate diet is awful for a diabetic. The *American Journal of Clinical Nutrition*, October 1997; 66:4(S) states:

"In type II diabetics, the **carbohydrate diet** led to **impaired** glycemic [**blood sugar**] and insulin responses. As well as to hypertriglyceridema [**high triglycerides**

No Carbohydrate Required in Human Diet

Nutrition for Fitness and Sport by Melvin H. Williams, Brown and Benchmark Publishers, Chicago, 1995, answers this on page 87. From what the nutritional experts, the government, and physicians have told us for decades, we would expect the answer to be "lots of carbohydrates," but it isn't. In fact, the answer is shocking:

> "However, the National Research Council has **not established an RDA for carbohydrates**, probably because the **body can adapt to a carbohydrate-free diet** and **manufacture the glucose** it needs from parts of protein and fat."

Carbohydrates Eaten vs. Tissue Weight

Student Companion for Stryer's Biochemistry makes it clear on page 321:

> "**In the human diet, carbohydrates** constitute approximately **half** the total caloric intake [closer to 60% now], **yet only 1**% of **tissue weight** is carbohydrate."

What has eating a 50-60% carbohydrate diet done to us, given that only one percent of our bodies is composed of carbohydrate? World-wide rampant obesity and **diabetes** epidemics!

Carbohydrates INSIGNIFICANT for All Biochemical Functions

The carbohydrate present in nucleic acids, glycoproteins, glycolipids, and cofactors, although functionally essential, contributes relatively little to the weight of the body. An

insignificant amount of carbohydrate is required for these components. Only some carbohydrate is stored as glycogen, but the amount is relatively small compared to the storage of adipose tissue (fat) and protein as muscle mass. Don't be misled.

Glycemic Index Definition

The glycemic index (GI), developed in 1981, uses glucose as a standard of comparison with other carbohydrates as a measure of how quickly they enter the bloodstream. Glucose is given a value of 100. There are many problems with this method; in particular different food combinations raise havoc with the system. The GI system is misleading, too. The results are inconsistent. A much better system is the absolute glycemic LOAD—the amount of carbohydrate eaten.

Glycemic Index (GI)—A Worthless Measure

Flint, et al., *British Journal of Nutrition* **2004** Jun; 91(6):979-89, confirmed this upsetting finding and published in **2003**:

> "…**No association** was found between predicted and measured GI.
>
> "…There was **no association** between GI and II [Insulin **Index —the amount of insulin** generated].
>
> "…In conclusion, the present results show that the **GI** of **mixed meals** calculated by table values **does not predict the measured GI**…." (

Carbohydrate NOT NEEDED for Fat Burning

Stryer's Biochemistry (4th edition) pages 612 and 638 makes it quite clear with their quote:

> "Fat ***does not*** burn in the flame of carbohydrates."

Metabolism DECREASED, not increased, with Carbohydrates

Textbook of Medical Physiology page 908 makes clear:

> **Carbohydrates slow the metabolism compared to consuming natural fats and proteins**.

Never forget this important scientific fact. *The Textbook of Medical Physiology* makes it quite clear on page 866 that **no carbohydrates** (**or proteins**) are **required for energy production**, **just your own body fat**:

> "When the **fat that has been stored** in the adipose tissue is to be used elsewhere in the body, usually **to provide energy**, it must first be transported to other tissue. It is **transported mainly in the form of *free fatty acid***…. Despite the minute amount of free fatty acid in the blood, its rate of "turnover" is extremely rapid….
>
> "One can calculate that at this rate, almost **all the normal energy requirements of the body can be provided** by oxidation of the transported free fatty acid **without using any carbohydrates or proteins for energy**."

There you have it. NO carbohydrates are required for energy AND protein won't be "stolen from your muscles" for energy, either.

Never Forget the Number "One"—your entire bloodstream contains only 1 little teaspoon of sugar

Basic Medical Biochemistry – A Clinical Approach on pages 472-473 makes clear:

> Blood glucose levels are kept at approximately 80 milligrams per deciliter – about 1 teaspoon (actually 0.8 teaspoons) in the bloodstream (actually just 8/10^{ths} of a teaspoon) **throughout the day when not eating, AUTOMATICALLY**.

If your body allowed more than this in your system the toxic by-products would kill you, as they do uncontrolled diabetics. High blood sugar levels also encourage rampant yeast infection in women.

IMPORTANT NOTE: Blood sugar levels are controlled to 1 part in 1,000—a VERY TIGHT tolerance in everyone, or you are diabetic!

Carbohydrate Energy Not Used Immediately Makes You FAT

Textbook of Medical Physiology makes this clear on page 869:

> "Whenever a greater quantity of **carbohydrates enters the body than can be used immediately** for energy or stored in the form of glycogen (just an insignificant bit), the **excess is rapidly converted** into triglycerides and stored in this form in the **adipose tissue [body fat**]."

No Insulin (response to carbohydrates)
= No Fat Storage

As the *Textbook of Medical Physiology* on page 870 states:

"When **no insulin is available** [response to carbohydrates]…**fats are poorly**, if at all **synthesized** [**you don't get fat**]…"

Carbohydrates STOP Fat-burning COLD

Textbook of Medical Physiology references this fact numerous times on pages 974, 975, and 977. Anyone who is overweight is always consuming too many of those fattening, diabetes-causing carbohydrates! Here's another on page 871:

"Thus, an excess of **carbohydrates** in the diet **not only acts as a fat-sparer** [**you won't burn you own body fat**] but **also increases the fat in the fat stores [making you fatter]**. In fact, all **the excess carbohydrate not used** [immediately] for energy or stored in the small glycogen deposits of the body is **converted to fat and stored** as such."

Specific Sugars NOT Required—Your Body Makes Them

Excess carbohydrates (more than a mere 4 ounces a day) prevent the body from burning fat, and increase stored body fat because

as *Basic Medical Biochemistry – A Clinical Approach* on pages 24 and 394 and *Textbook of Medical Physiology*, pages 869, 871, 936, *state*:

> "Specific sugars [**carbohydrates**] **ARE NOT REQUIRED** in the diet." Note: This is because **your body makes them**.

Fat is stored ONLY When You Eat Carbohydrates

As *Basic Medical Biochemistry – A Clinical Approach*, pages 476, 510-12, makes clear, Adipose tissue (fat) is stored ONLY when carbohydrates are eaten. From *Principles of Medical Biochemistry*, page 372:

> "…[F]atty acids [from eating fat] cannot be converted into carbohydrates. **Carbohydrates, on the other hand, can be converted** into triglycerides [**excess body fat**]." and
>
> "…[E]xcess **energy from dietary carbohydrate** is **stored away** as triglyceride in adipose tissue [**body fat**]."

(Voet's) *Biochemistry,* second edition, published by John Wiley & Sons, 1995, gives more insight into carbohydrates and excess body fat (adipose tissue) on page 790, in the chapter titled "Adipose Tissue":

> "Adipose tissue obtains most of its fatty acids from the liver or from the diet as described in Section 23-1. Fatty acids are activated by the formation of the corresponding fatty acyl-CoA and then esterfied [for storage] with **glycerol-3-phosphate** to form the stored triacylglycerols

(Section 23-4F). The **glycerol-3-phosphate arises** from the reduction of dihydroxyacetone phosphate, which **must be** glycolytically **generated from glucose** because adipocytes [body fat] lack a kinase that phosphorlates endogenous glycerol."

This is complicated biochemistry explaining why excess fat can ***only be stored when a person eats carbohydrates*** and is one of the reasons why nutritionists don't have a clue about it and why most physicians get misled.

The quote continues:

"Adipocytes hydrolyze triacylglycerols to fatty acids and glycerol in response to the levels of glucagon, epinephrine, and insulin through a reaction catalyzed by hormone-sensitive lipase (Section 23-5). If **glycerol-3-phosphate is abundant [from carbohydrates]**, many of the fatty acids so formed are **reesterfied** to triaglycerols [**body fat**]. Indeed, the average turnover time for triacylglycerols in adipocytes is only a few days. If, however, **glycerol-3-phosphate is in short supply**, the **fatty acids** are released into the bloodstream [**used for energy**]. The rate of glucose uptake by adipocytes, which is regulated by **insulin** [response to carbohydrates] as well as **glucose availability** [from food], is therefore,

> also a **controlling factor** in triacylglycerol [**body fat**] formation and mobilization."

Do other medical textbooks confirm this fact? Yes. *Harpers Illustrated Biochemistry* (26th edition), page 214, states in the section titled "The Provision of Glycerol-3 Phosphate Regulates Esterification: Lipolysis is Controlled by Hormone-Sensitive Lipase (Figure 25-7)":

> "Triacylglycerol is synthesized from acyl-CoA and **glycerol 3-phosphate** (Figure 24-2). Because the enzyme glycerol kinase is not expressed in adipose tissue, ***glycerol cannot be utilized*** **for the provision of glycerol 3-phosphate**, which **MUST be supplied from [dietary] glucose [from carbohydrates]** via glycolysis [**breakdown of sugar]**."

High Carbohydrate Diet CAUSES Saturated Fat

Journal of Biological Chemistry and *Lancet* tell us that cholesterol is normally combined with a special type of fat called an EFA (Essential Fatty Acid]. On a high carbohydrate/low fat diet these EFAs are in short supply so saturated fats MADE FROM carbohydrates are tied to the cholesterol INSTEAD of what is suppose to be there—the healthy essential fats.

> "Cholesterol is **normally esterified with unsaturated fatty acid** [you will learn about these in the next chapter][7] and **when**—as in our experiments—these are extremely

---

7 Kelsey, F.E., Longenecker, H.E., *Journal of Biological Chemistry*, 1941, Vol. 139, page 727.

> **deficient** in the body it is **esterified [combined] with** much more **saturated fatty acids synthesized in the body from carbohydrate**."[8]

Eat too many carbs and lots of saturated fat is made from them. Few of us understand that carbohydrates make saturated fat.

Here's what *Basic Medical Biochemistry* on page 503-504 has to say:

> "When an excess of **dietary carbohydrate is consumed**, glucose is converted to acetyl CoA, which provides the 2-carbon units that condense in a series of reactions on the fatty acid synthase complex, producing palmitate [**THE BODY'S #1 saturated fat**]…."

Therefore, it is quite clear that carbohydrates produce the saturated fat that everyone complains about.

Carbohydrates are NOT Body's Preferred Energy Source

As *Basic Medical Biochemistry – A Clinical Approa*ch, pages 29, 272, 357 and 359, make clear, glucose [from **carbohydrates**] is **NOT the body's preferred energy** source; fatty acids are.

Carbohydrates Raise Both Insulin and Cholesterol Levels

Basic Medical Biochemistry, pages 475 and 566, make clear that:

> **Insulin** production, a response to consumption of carbohydrate, **raises cholesterol** levels.

---

8 H.M. Sinclair, "Deficiency of Essential Fatty Acids and Atherosclerosis, Etcetera," *Lancet*, April 7, 1956.

Glucose (Sugar from Carbohydrates) Causes Diabetes!

Diabetes **2001**; 50:1683-1690 makes this quite clear:

- "Our results underscore the **importance of tight glucose (sugar) control** in **limiting beta-cell** [insulin producing] **destruction**…"

The authors are stating here that carbohydrates are a cause of the destruction of your pancreas.

Carbohydrates are *Not* the "Feel Good Fix"; Moods are *Not Improved by Eating Carbohydrates*:

International Journal of Obesity and Metabolic Disorders, Oct. 21, 1997; (10):860-864, makes this clear in the article, "Psychological and metabolic responses of carbohydrate-craving obese patients to carbohydrate: fat, and protein rich meals." Their findings:

Moods are NOT improved by eating carbohydrates.

How Much Carbohydrate is Stored?

The Student Companion for Stryer's Biochemistry, page 624, gives us the answer:

> The normal 150-pound person stores about 250 grams—just **½ pound** of glycogen [hydrated storage form] and 25 grams—just **1/20th pound** of glucose. Compare this small amount to the significant amounts of stored body fat we have! These figures make it evident that humans can rely on stores of carbohydrates for only a short time.

Carbohydrates are NOT Your Body's Preferred Energy Source! Never forget this crucial fact.

If you think that you have already heard about all the evils of carbohydrates, think again. Here's another dreadful consequence of carbohydrates that, in addition to making you fat, will significantly impair your health.

Carbohydrates Contribute to Cellular EFA Deficiency and Insulin Resistance, Making Your Diabetes Even Worse!

For years I knew that somehow, in addition to overdosing on carbs, diabetics were deficient in essential parent omega-6 (EFAs) in the cell membranes. This would impair insulin effectiveness and cause insulin resistance (insulin doesn't work effectively). We have a worldwide diabetes epidemic and must do everything possible to stop its proliferation. I thank Patricia Kane, Ph.D. for directing my attention to this vital information.

You know that carbohydrates generate an insulin response, provoking fat storage, since insulin is a fat storage hormone. The more fat you have, the more of a certain chemical, called Lp-PLA(2), is generated. Studies show that Lp-PLA(2) REMOVES precious parent omega-6 from cell membranes! If the cell membrane is deficient in EFAs, insulin transport into the cell is compromised and **your risk of insulin resistance significantly increases**. Who needs this additional diabetes risk factor?

Minimize those fattening, diabetes-causing carbohydrates and you'll be on the path to radiant health!

Here is how the journals report it:

> "Phospholipase A2 (PLA(2)) hydrolyzes [removes] fatty acids from membrane phospholipids [the cell membranes comprised mainly of parent omega-6].[9]

The *Journal of Diabetes Complications* confirms that PLA(2) is **higher in overweight people** and diabetics:[10]

> "Lipoprotein-associated phospholipase A(2) production is **significantly increased in diabetics**.
>
> "Lp-PLA(2) was **significantly correlated** with **waist-hip ratio**.
>
> "Lp-PLA(2) was **significantly higher** in subjects with the metabolic syndrome [diabetic] than in those without it."

Furthermore, as the recent article titled, "Elevated Lp-PLA2 levels predict incident CHD independent of traditional risk factors," in *Journal of American College of Cardiology* (**2008**; 51: 913-919), makes clear, the Lp-PLA(2) enzyme is higher in diabetics than in non-diabetics. As would be expected from the decreased parent omega-6 in cells, heart disease would soon follow and it does:

---

9 "Essential fatty acids in the brain," Haag, M., *Can J Psychiatry*, **2003** Apr; 48(3): 195-203.

10 "The role of lipoprotein-associated phospholipase A(2) in the metabolic syndrome and diabetes," Noto, H., Chitkara, P., Raskin, P., *J Diabetes Complications*, **2006** Nov-Dec;20(6): 343-8.

"Elevated levels of lipoprotein-associated phospholipase A2 (Lp-PLA2), an enzyme involved in the proatherogenic [heart disease] process, are associated with coronary heart disease (CHD) events independent of traditional risk factors…

"Lp-PLA2 was a strong and independent predictor of fatal and nonfatal CHD events over and above other traditional risk factors.

"After adjustment for age and gender only, **elevated Lp-PLA2 levels were significantly associated with risk of incident CHD…**

"In **2003**, the Food and Drug Administration granted market clearance to an Lp-PLA2 test for coronary heart disease [yet few cardiologists use this marker]"

***** IMPORTANT NOTE *****

If you've been eating lots of carbs prior to starting your lower carb regimen, be sure to gradually reduce your carbohydrate intake. Don't suddenly change your diet. A sudden dietary change can shock your body, negatively affecting your digestion and could cause a sugar low that can last a week or so. To avoid this, start reducing your carb portions to about half for about a month. Then each subsequent week reduce them a little more. For increased fat burning reduce carb intake to *20-30* grams—for maintenance or with vigorous exercise, keep carbs at *60* grams (12 tsp) or less.

Breaking the Carbohydrate Craving With PEOs—Dr. Cavallino's results

Experiment in Italy for Overweight People with Carbohydrate Addiction Shows PEOs Eliminate Carbohydrate Cravings, Reduce Appetite, and Increase Energy and Alertness[11]

Dr. Steven Cavallino conducted an eight-week experiment with a group of his patients who were already following a higher protein/lower carbohydrate diet, as Chapter 4 details. The experiment compared certain physical manifestations among these patients, both prior to and during PEO supplementation, based on the guidelines I have recommended. Dr. Cavallino states:

> Four weeks prior to starting their PEO supplementation, all patients were told the value of a higher protein/lower carbohydrate diet based on information in this book. All patients were on this higher protein/lower

---

11 Since 2002, Steven Cavallino, M.D., has been the official physician and nutritionist for the famous Italian beauty pageant, **La Piu del Mondo**—"The Most Beautiful Women in the World." His experiment confirms PEOs reduce patient cravings for sweets and naturally reduce hunger. Dr. Cavallino has been the official physician for the **Italian National Flag Football League** for the past eight years. Currently, Dr. Cavallino is also using Prof. Peskin's recommendations in another one of his passions, **sports medicine**, stating, "**These proven real-life results now enable athletes to defeat the common lactic acid (muscle burning) and pain syndrome post-workout.** The exercise component of any weight-loss program becomes much more enjoyable with this new discovery."

carbohydrate diet for a minimum of four weeks *before* PEO supplementation. There were 10 patients: 8 women and 2 men.

- All patients were on a higher protein/lower carbohydrate diet **before** and **after PEO supplementation.**
- Patients were given a PEO formulation based on Professor Peskin's recommendations consisting of an organic/unprocessed blend with **more parent omega-6 than parent omega-3.**
- **All patients were initially self-described "carbohydrate addicts."**

All patients agreed to collaborate, knowing that many foods were not permitted for the entire eight weeks (four weeks prior to initiating the PEO supplementation and four weeks with PEO supplementation). **Patients agreed not to consume** fruit, pasta, pizza, rice, sweets, soda or soft drinks. All patients orally took 1,450 mg PEO formulation twice a day (2.9 gram total). **Patients were asked** to rate their responses to the regimen using four criteria, using from 1 to 4 asterisks.

All patients (100%) initially suffered from **intense carbohydrate cravings** and had little energy. Eighty percent (80%) of patients suffered from **constant hunger.** After PEO supplementation began, the following results were observed:

- The average patient **felt well and more at ease** with the higher protein/lower carbohydrate diet.
- **Overall appetite reduced in all 10 patients;** all noted a **GOOD to EXCELLENT** response, with **50% rating an EXCELLENT response.**
- **Carbohydrate cravings were reduced in all 10 patients—**a huge 100% success; 9 people rated this reduction EXCELLENT.
- **Energy and alertness increased** in all 10 patients: this was an EXCELLENT response—a huge 100% success.
- **Weight loss goal was reached** in all 10 patients.

Real-life results were achieved. I am positive about and thankful for Professor Peskin's assistance in showing scientifically that most carbohydrates are bad in relation to promoting obesity and diabetes, and that PEOs are essential for good health, with the objective to help us all to **lose weight without suffering.** I was able to obtain **excellent patient results adding the PEO supplementation program.**

Stephen Cavallino, M.D

October 22, **2005**

Chapter 6: Scientific Support

MRI SCAN & IMAGING CENTERS

mriscancenter.com

ROBERT L. KAGAN, M.D. • MEDICAL DIRECTOR

MRI, CT, PET & Ultrasounds

Accredited by the American College of Radiology

June 18, 2008

Jonathan Collin, MD
Letter to the Editor
Townsend Letter
911 Tyler Street
Pt. Townsend, WA 98368

Re: Parent Essential Oils (PEOs)
Brian Peskin, BSEE

Dear Dr. Collin:

In addition to my diagnostic radiology practice which includes CT, MRI, PET and Ultrasound examinations requested by healthcare providers for diagnostic purposes, I have a small private practice devoted to preventive medicine. These patients have yearly whole body scans utilizing a 64-slice multidetector CT scanner (MDCT) which includes coronary calcium scoring for detection of coronary artery disease (CAD). Also included is an extensive blood and urinalysis panel. The concept is that the whole body scan will detect anatomic abnormalities prior to their progression to a symptomatic phase and the laboratory testing (blood & urinalysis) will detect functional abnormalities in a preclinical stage. The most common pathology that I find is asymptomatic coronary artery disease (CAD) since the coronary calcium scoring detects hard plaque within the wall of the vessel. This build up of plaque within the wall of the vessel will occur many years prior to any symptomatology.

One of my patients, a 68-year old male, smoker, I have followed on yearly basis beginning in 2005. In addition to the calcium score, the test also provides the volume of plaque, which is the best number for follow up to evaluate of the progression of plaque burden. The score is based on the density of plaque but the volume is the amount of plaque. In spite of all routine conventional treatment which included blood pressure medication, a "statin" drug, high-dose niacin, co-enzyme Q-10, and a daily aspirin, his coronary plaque volume continued to progress, although an acceptable slow rate.

(continued)

3122 E. Commercial Boulevard • Fort Lauderdale, FL 33308 • (954) 772-8000 • FAX (954) 776-6356

Testimonial from Robert L. Kagan, page 2

Re: Parent Essential Oils (PEOs)
Brian Peskin, BSEE

Date:	Coronary Artery Total Plaque Burden (volume):
04-22-05	26
07-11-06	36
03-05-07	39
04-21-08	30

As you can see, for the first time from 2007 to 2008, the volume of plaque decreased from 39 to 30, which is a decrease of 22% when annualized on a yearly basis. ***I have never seen a decrease of coronary artery plaque volume by more than 5% in one year.*** My goal is usually just to stop the increase in plaque. Naturally, I was quite curious and called the patient to inquire about what else he was doing in addition to the traditional reduction in cardiac risk factors that I was aware of. He told me the only thing different about his regimen was the "oxygen pills" that he was taking for the past 8 months. Through my investigation, I finally traced the "oxygen pills" to the parent essential oils (PEO) advocated by Professor Brian Peskin. I was able to contact Professor Peskin who sent me a copy of his article recently published in your newsletter called "Vytorin Failure Explained – A New View of LDL". Needless to say, personally, I have stopped taking my "statin" drug (Lipitor) and I have now implemented Professor Peskin's "Parent Essential Oils" (PEOs) recommendation to my therapeutic regimen.

Thank you for publishing this important article. It should be required reading for any physician treating coronary artery disease (CAD) today.

Very truly yours,

Robert L. Kagan, MD, FCAP,
Medical Director, MRI Scan and Imaging Centers
RLK/lm

From: Robert Kagan [mailto:rkaganmd@bodyvision.pro]

Sent: Thursday, January 29, 2009 9:26 PM

I have important news regarding our study. Patient zero, who brought PEOs to my attention, came back for heart scan. **Last April he had a Cardiac calcium score that went down by 20% and the only thing it could be attributed to was the PEOs he started taking for cancer prevention**.

Well **his score now went up by slightly more than 100%** when calculated on an annual basis. My first question was when ***did you stop taking the PEOs?***

Sure enough, he stopped the end of August.

Remember, he did not receive the PEOs from me & I never counseled him on the study we were doing as I did for all the other patients I entered into the study.

He started back today and will come for a Calcium score every 3 months.

Regards,

Robert L. Kagan, MD, FCAP, Medical Director, MRI Scan and Imaging Centers

Fats fulfill our appetite

2008 Newsflash: Just like fruits fulfill our natural "sweet tooth," fats fulfill our appetite. Fats—not protein or carbohydrates.[12]

Take six egg whites and cook them with no butter. You'll be starving just 15 minutes after eating them. Compare this to adding two yolks. You'll be full and contented. As a landmark experiment shows—it's only **FATs—NOT carbohydrates or proteins**—which send signals to the brain saying you aren't hungry.

Here's what was published in **2008**:

- "Here, we report that ***duodenal infusion of fat stimulates*** oleoylethanolamide (OEA) mobilization in the proximal small intestine, whereas infusion of ***protein or carbohydrate does not.***
- "…[T]his lipid messenger ***participates in the induction of satiety.***
- "…[T]he **rapid onset of the OEA response (<30 minutes)**…
- "…**[P]rolonging the time interval between meals.**

12 Schwartz, GJ, et al., "The Lipid Messenger OEA Links Dietary Fat Intake to Satiety," *Cell Metabolism*, Vol. 8, Issue 4, Oct 8, **2008**, pages 281–288.

- "OEA production utilizes dietary oleic acid as a substrate and is disrupted in mutant mice lacking the membrane fatty-acid transporter CD36. Targeted disruption of CD36 or PPAR-α **abrogates [ends] the satiety response induced by fat.**
- "The results suggest that activation of small-intestinal OEA mobilization, enabled by CD36-mediated uptake of dietary oleic acid, **serves as a molecular sensor linking fat ingestion to satiety.**
- "In conclusion, our studies identify **OEA as a key physiological signal that specifically links dietary fat ingestion to across-meal satiety.**

PEO Solution analysis: Once again, the truth gets published but no one is made aware of it. *ONLY fats fulfill your appetite, NOT protein or carbohydrates*.

Omega-6 polyunsatuated fatty acids extend life span

O'Rourke, Eyleen, J., et al., "ω-6 Polyunsaturated fatty acids extend life span through the activation of autophagy," Genes & Development. Published in advance February 7, **2013**, http://genesdev.cshlp.org/content/27/4/429.full, **2013**:

> "Supplementing C. elegans culture media with these **ω-6 PUFAs** [long chain metabolites termed derivatives] increases their **resistance to starvation and extends**

their life span in conditions of food abundance. Supplementation of C. elegans or **human epithelial cells** with these ω-6 PUFAs activates autophagy, a cell recycling mechanism that **promotes starvation survival and slows aging**.

"We found that supplementation with **AA and DGLA [both Parent omega-6 derivatives],** ***but not with EPA,*** was sufficient to activate autophagy in ad libitum-fed C. elegans.

"Our data show that supplementation with ω-6 PUFAs activates autophagy **in human epithelial cells.**

"These results show not only that dietary supplementation with ω-6 PUFAs **activates a conserved cellular response** normally triggered by fasting, but also that long-term administration of **ω-6** PUFAs can render the beneficial effects of **low-caloric intake** even in ad libitum feeding conditions…."

Newsflash: Parent Omega-6 increases weight loss: Known in 1973!

You have already read about Doctor Cavallino's experiment with "carboholics" in Italy. I have long been aware of the remarkable power of the correct *un*adulterated parent omega-6 to -3 ratio in decreasing carbohydrate cravings. However, even I had never seen the following medical journal

article. I sincerely thank Canadian David Macphail for sending it to me. All the way back in 1973, physician H. Kasper proved there is an effect of greater weight loss and better blood chemistry, too, when parent omega-6 is added to the diet—REGARDLESS of CALORIES.

Here's what the study states[13]:

"**Despite a higher total caloric intake**, the weight-reducing effect clearly equals that of a standard clinical reducing diet of 1,000 kcal [even though patients consumed significantly more food].

"…A **maximal weight loss** was achieved in cases 1, 2, and 15 when they were **taking fats high in linoleic acid [parent omega-6]**.

"It was **striking to observe** that the **weight gain did *not* correlate with the caloric intake**. Particularly if fat was given in the form of corn oil [high in parent omega-6], a distinct discrepancy between the caloric intake and the response of the body weight was detectable.

"This phenomenon was less conspicuous if fat was taken in the form of olive oil.

"If **fat was exchanged** isocalorically **for** glucose [**carbohydrates**], the **weight loss ceased**.

---

13 Kasper, H., et al., "Response of body weight to a low carbohydrate, high fat diet in normal and obese subjects," *The American Journal of Clinical Nutrition*, 26: February 1973, pages 197-204.

> "The **cholesterol and triglyceride concentrations** in the serum, which had been raised at the beginning of the experiment, invariably showed a **tendency towards normalization** under this dietary program." (Emphasis added.)

◗ Lean-for-Life Commentary

This experiment proves that the "calorie theory" is incorrect and that there is much more to the picture than merely "calories in minus calories used equals weight gain."

The parent omega-6 oil has a natural weight-loss property, whereas olive oil does not.

When carbs were switched "calorie-for-calorie," for fat, weight loss STOPPED. Once again, we clearly see the "low-fat/hi-carbohydrate diet" failing!

Triglyceride and cholesterols significantly improved with the low carbohydrate/parent omega-6. We have a home-run!

IOWA Experiment

The IOWA experiment was presented to the American Academy of Anti-Aging Medicine (A4M) 18th Annual World Congress on Anti-Aging & Regenerative Medicine in Las Vegas, December 10, **2010**. **The presentation was titled "Fish Oil Fallacies: Physicians and Patients Beware," by Brian Peskin, BSEE. The IOWA screening experiment found in full at** http://www.brianpeskin.com/BP.com/experiments/IOWA-Experiment-Results.pdf.

Nutritional and medical importance of gamma-linoleic acid

Following are some critical points from the exceptional treatise by DF Horrobin, MD, PhD—a true medical genius, published in *Progress in Lipid Research*:[14]

> "**The *n-6* EFAs have at least four roles:** (1) The modulation of membrane structure. (2) The formation of short-lived local regulating molecules such as prostaglandins (**PGs**) and leukotrienes (LT), together often **known as eicosanoids**. (3) The control of the **water impermeability of the skin** and possibly the permeability of other membranes such as the gastro-intestinal tract and the blood-brain barrier. (4) **The regulation of cholesterol transport** and **cholesterol synthesis. The membrane effects of the EFAs are possibly the most important.**
>
> "The fluidity and flexibility of all membranes within the body are influenced by their EFA content. As a crude indicator, the effect of an EFA on membrane fluidity is determined by its concentration in the membrane and by the number of double bonds in the molecule (the product of concentration x the number of double bonds is sometimes known as the unsaturation index). **However, there is much more to the story than that.**

---

14 Horrobin, D.F., "Nutritional and medical importance of gamma-linoleic acid," *Prog. Lipid Res.*, Vol. 31, 1992, No. 2, pages 163–194.

► **"The n-3 EFAs, even though they have as many or more double bonds as the n-6 EFAs are unable to reverse the features of n-6 EFA deficiency.**

"The precise configuration of the double bonds must therefore be important and attempts to explain the rationale for this are just beginning.

"**The lipid configuration of the membrane** is important in itself, but also matters because it **influences the structure and behaviour of the many proteins in the membrane such as ion channels, receptors and ATPases [including insulin receptivity]**. These proteins are literally ***afloat in a lipid sea and their function is dependent on the behaviour of that sea.*** Good examples of this are studies on the effects of lipids on the binding of ligands to their receptors. The unsaturation of the lipid medium in which the receptors are found has been reported to change the affinity for ligands, such as *steroid hormones*, and *peptides*, such as opioids and angiotensin. In general, the more unsaturated the lipid, the lower the affinity of the receptor for its ligand.

"Lipid unsaturation also influences membrane 'fluidity.' This is important in the **vascular system** and also in **any other situation in which cells move**, for example, during inflammation and immune responses. Red cell membranes, which have **reduced EFA levels** are 'stiffer' than usual, and as a result **increase blood viscosity and reduce tissue oxygenation.**

"The third role of the EFAs is in the maintenance of the water impermeability of the skin. ***In the absence of n-6 EFAs the skin loses its 'water-proofing.'***

► "It is apparent from this brief description that a lack of or abnormal metabolism of EFAs **could adversely influence every cell and every organ system in the body.** There is therefore nothing inherently surprising in the concept that **EFAs may have a role to play in modulating many different disease processes.** [Note: ***This precisely explains why cancer can occur in any tissue—the most oxygen deficient.***]

"**The EFA requirement may be increased** in the presence of high rates of cell division.

"This situation may be physiological (as in infancy) or **pathological (as in the presence of cancer, inflammation or rapid cellular repair after injury)**."

► "**Gender has a major,** but inadequately understood, impact on **EFA requirements.** ***Male animals require a higher EFA intake than females.*** This may in part be because females metabolize LA more rapidly and in part because they retain EFAs in tissues more effectively in the presence of EFA deficiency. **[Note article below by H.M. Sinclair, "Deficiency of essential fatty acids and atherosclerosis, etcetera," stating that EPA deficiency is likely to be five times more common in males than females.]**

> **"The total phospholipid (TPL) fraction, in contrast, does not change rapidly in response to feeding or fasting.** Moreover it is relatively rich in the EFAs right along the metabolic chain. It can therefore be used as **a guide to both EFA intake and EFA metabolism**."

PEO Solution analysis: In research, plasma total phospholipids are the best quantitative measure of EFA status—*much superior to red blood cell analysis*.

Horrobin's treatise continues with a confirmation of Small Derivative formation:

> "GLA [Parent omega-6 derivative] is formed by the rate-limiting step of delta-6-desaturation and metabolised by the non-rate-limiting step of elongation to dihomogamma-linolenic acid (DGLA). It is therefore not surprising that **GLA is found in most tissues in only small amounts.** It normally makes up less than **0.2% of the fatty acids in phospholipids,** less than **0.1 % of those in triglycerides** and less than **2.0% of those in cholesterol esters**. Furthermore, ***GLA—the body's most important derivative*** is found in tissue in only very small quantities. Furthermore, Parent omega-6 can modulate cytokine releaser directly, rather from its long-chain metabolites.
>
> "The administration of **GLA leads to increased plasma PGE1 levels** in humans and increased macrophage PGEl levels in rats. There can therefore be no doubt

that GLA enhances the rate of formation of this very desirable substance.

► **"PGE1 has a quite extraordinary range of desirable actions.** It **dilates blood vessels** and **lowers blood pressure;** it **inhibits platelet aggregation;** it inhibits cholesterol biosynthesis; it is an **anti-inflammatory agent;** it has a biphasic **regulating effect on immune responses;** and it stimulates cyclic AMP formation, thus being capable of **inhibiting phospholipase A2, an enzyme important in releasing AA during inflammation.** These desirable effects obviously have **considerable therapeutic potential.**

"The formation of PGE1 may explain why, ***contrary to simplistic expectations,*** but in accordance with a **prediction based on understanding** of PGE1 actions, the rise in AA levels following GLA administration to cells, animals or humans is ***consistently followed by a fall, rather than a rise, in the levels of conversion of AA to potentially harmful metabolites like Thromboxane A2 or PGE2.*** There is a tendency to consider arachidonic acid 'a bad thing' because it can give rise to metabolites like thromboxane A2, PGE2, and leukotriene B4. In fact there is **no evidence at all that arachidonic acid is harmful so long as it stays as AA. AA is an essential constituent of membranes.** Adequate levels of DGLA seem important in keeping AA in membranes where it is desirable, and preventing conversion of AA to its possibly undesirable metabolites:

The amazing treatise continues.... showing **Parent omega-6 and its metabolites takes center stage**....

> "**The *n-3* EFAs are of major biological significance but they are simply not as important as the *n-6* EFAs.** This is shown by the following facts:
>
> "(1) When animals and humans are put on **diets deficient only in *n-6* EFAs**, it is easy to show that they develop multiple biochemical and biological abnorm∂alities. In contrast it has proved extremely difficult to demonstrate biological abnormalities in animals deprived only of *n-3* EFAs. There are abnormalities in the brain, the retina, the heart and platelets and the *n-3* EFAs are undoubtedly important in modulating the functions of these organs, but ***these abnormalities are not easy to demonstrate.***
>
> "(2) When animals are ***deprived of both n-3 and n-6 EFAs,*** all the readily observed **abnormalities are quickly corrected by *n-6* EFAs alone. *N-3* EFAs alone do not correct any of the abnormalities, and make some, such as the capillary fragility, worse.**
>
> "In order to express their normal biological effects, ***n-3* EFAs must be given with *n-6* EFAs** whereas the n-6 EFAs are biologically active when given without *n-3* EFAs. [Note the significant difference. The 21st century solution is BOTH Parent omega-6 and Parent omega-3 in the proper ratios and quantities.]

"(3) In human milk and in most tissues in the body, the ratio of *n-6* to *n-3* EFAs lies within the range 3:1 to 9:1. [Breast milk is 10:1—most tissues 4:1-7:1; although stored body fat is much higher.] This is true even of animals such as the zebra whose EFA intake is almost entirely in the form of ALA [Parent omega-3] from grass. **Thus even when most dietary EFAs are in the *n-3* form, the *n-6* EFAs are preferentially retained**."

PEO Solution analysis: We see the much greater importance of Parent omega-6 and its metabolites compared to Parent omega-3 and its metabolites. Dr. Horrobin terms omega-6 series GLA the body's most important derivative.

Deficiency of essential fatty acids and atherosclerosis

The power of PEOs was known in 1956. The extraordinary nutritional scientist of Reading and Oxford, H.M. Sinclair, wrote a superb article in *The Lancet* titled, "**Deficiency of essential fatty acids and atherosclerosis, etcetera."**[15] **He warned that people won't believe it,** in which he stated, **"MY inclusion of 'etcetera' in the title invites the scorn we so readily pour on vendors of patient cure-alls."** Tragically, he was correct and his advice was not properly integrated into the medical community. Today, you can remedy that mistake.

Journal highlights include:

---

15 April 7, 1956, pages 381–383.

- "First, there was an enormous increase in permeability of the skin **[epithelial tissue in all carcinomas]** in EFA [PEO] deficiency and there is an increase **in capillary fragility**...

- to the carcinogenic effect of X rays through deficiency of EFA; the above facts become explicable provided those young children who died of leukaemia were irradiated when their pregnant mothers had diagnostic radiography; a chemical carcinogen could hardly be responsible for their deaths.

- "In lower animals, in which we have carefully studied the skin lesion of EFA [PEO]-deficiency and found a dramatic increase in permeability of the epidermis, we believe there is a structural fault perhaps through failure of the phospholipids containing EFA [PEO] to polymerise and form the impermeable barrier in the stratum granulosum. Phospholipids are rich in unsaturated fatty acids [PEOs] (though not so rich as cholesteryl esters)...

- "So, as in the case of esterified cholesterol, we have an **abnormal type of phospholipid** being formed which may not only cause a structural defect in the skin which is responsible for the great increase in permeability but may also be outstandingly important...

- "... [T]he nervous system is rich in phospholipids containing polythenoid fatty acids, and these are found together with highly **unsaturated cholesteryl**

esters in myelin; the presence of abnormal types of the compounds that are known to important to it would be unlikely to leave function undisturbed; disseminated sclerosis is a disease of highly civilized countries being almost unknown in India and China [as of 1956], and other diseases in which the ectodermal neuroglia is effected may be relevant; since serum **EFA fall in acute infections**...early mild **dementia** appears to be becoming commoner in males. Thirdly, the **mitochondria** membrane probably contains phospholipids, and derangement of this through **deficiency of EFA [PEO] might me responsible for the uncoupling of oxidative phosphorylation found in such deficiency**.

- "Effects of EFA [PEO]-Deficiency: First, deficiency would be *likely to be at least five times commoner in males than in females*. "Secondly, we might expect deposition of cholesterol since cholesterol esterified with unusually saturated or with unnatural fatty acids is probably disposed of less readily..."

The article continues:

- "...I believe [abnormal esters/phospholipids] to be caused by a **pure dietary deficiency of essential fatty acids [PEOs]**...

There is even more brilliance in his article, but these excerpts make it clear—his genius is evident.

Dr. Sinclair **predicted** the following patient ailments/disease/physiologic disorders from EFA deficiency:

(a) **Cardiovascular disease**.

(b) **Cancer**: in particular, increased skin cancer. [Note: Skin cancers are at epidemic levels with no end in sight, and cardiovascular disease (in all forms) is our #1 killer. CVD, too, has no end in sight. All carcinomas are enclosed by epithelial tissue. A PEO deficiency of Parent omega-6 is DIRECTLY TIED to ALL CARCINOMAS. Furthermore, only Parent omega-6 is contained in the arterial intima.]

(c) Dr. Sinclair understood the damage that X rays may cause. **PEOs are highly protective against cancer treatment X ray damage.** *See* my book, *The Hidden Story of Cancer*.

(d) PEOs assist the nervous system. **PEOs help fight MS**.

(e) Even **dementia** is addressed. This has become another epidemic directly related to PEO deficiency. Fish oils are worthless and coconut oil is not effective enough.

(f) PEOs have a vital role in **combating infection**.

(g) PEOs are directly **incorporated into mitochondrion**. This is a ***top anti-aging secret and a key to cancer prevention***.

(h) Dr. Sinclair's key concept that PEOs lower **LDL-cholesterol**—esterification of cholesterol—will be discussed in detail later.

Fish Oil was known to spontaneously oxidize in 1990 as this *2002* journal article references:[16]

"…Oxidation of EPA leads to the **generation of a mixture** of aldehydes, peroxides, and other oxidation products…

"Highly polyunsaturated long-chained ***EPA is readily oxidized at room temperature*** **even in the absence of exogenous oxidizing reagents.**

► "More importantly, *in vivo*, **a large increase in tissue and plasma accumulation of fatty acid oxidation products is noted in subjects consuming fish oil** ***even after addition of antioxidant supplements*** to the diet, which suggests **extensive oxidation of omega-3 fatty acids such as EPA** ***in vivo***."

PEO Solution analysis: Once again, consumption of lots of fish and supplemental EPA/DHA is shown harmful, and **antioxidants can't help enough**. Safflower oil has a high content of Parent omega-6 (LA) with no EPA/DHA. When fish oil was added, the results were

---

16 Sethi, Sanjeev, "Oxidized omega-3 fatty acids in fish oil inhibit leukocyte-endothelial interactions through activation of PPARa," *Blood*, August 15, **2002**, Volume 100, No. 4., pages 1340-1356. [Note: These authors attempt to contrive a claim that this oxidation is "good."]

ruinous, as we expected: highly increased lipid peroxidation resulted and the animals died much earlier than the Parent omega-6 group (**56 weeks survival rate without** fish oil/**48-week with** fish oil). LDL-cholesterol and triglycerides were lower with fish oil, but you shall soon discover that these numbers, in and of themselves, are meaningless. All of the insight and explanation of the inconsistencies have to do with their structure—not their amount. It is all about their *un*adulterated structure—that is precisely why blood chemistry is NOT an accurate predictor of CVD. Premature patient death with "fine lipid chemistry" is not a good outcome! And this happens all too frequently, as cardiologists know all too well.

Scientific Support for Chapter 7

Inconvenient Truth #1: DHA and fish oil shown completely worthless in treatment for Alzheimer's.

Quinn, J, et al., "Docosahexaenoic Acid Supplementation and Cognitive Decline in Alzheimer Disease: A Randomized Trial, "*Journal of the American Medical Association*, November 3, **2010**, Vol. 304, No. 17, pages 1903–1911:

- "Conclusion: Supplementation with DHA [marine based oils] compared with placebo [no marine based oils] did *not slow the rate of cognitive and functional decline* in patients with mild to moderate Alzheimer disease. [Note: Since the condition was "moderate," patients were still quite capable of improvement.]

- "This study was designed to determine if supplementation with DHA would slow the rate of cognitive and functional decline in patients with *mild to moderate Alzheimer disease* Despite enrollment of the target population of individuals with low baseline DHA…

- "The *hypothesis* [guess] that DHA slows the progression of mild to moderate Alzheimer disease was not supported, so there is *no basis for recommending DHA supplementation for patients with Alzheimer disease*.

- "In summary, these results indicate that DHA supplementation is not useful for the population of individuals with mild to moderate Alzheimer disease."

<u>Inconvenient Truth #2</u>: Fish oil increases risk of colon cancer.

"Link Between Fish Oil And Increased Risk Of Colon Cancer In Mice," J. Fenton, et al., *Medical News Today (Colorectal Cancer)*, Article URL: www.medicalnewstoday.com/articles/203683.php#post, October 7, **2010**; and Woodworth, Hillary, L., et al., "Dietary Fish Oil Alters T Lymphocyte Cell Populations and Exacerbates Disease in a Mouse Model of Inflammatory Colitis," *Cancer Research*; 70(20); 7960–9; 0008-5472.CAN-10-1396; Published Online First August 26, **2010**; doi:10.1158/0008-5472.CAN-10-1396.

Following are the exact markers that were negatively impacted by Fenton's experiment that showed fish oil accelerating aggressive cancer. Again, it is significant to note that even the researcher was expecting completely opposite results; she wasn't even aware of the vast amount of negative fish oil studies until she experienced her own personal research failure and started researching the literature for other failures:

- "Contrary to expectations, DFO ***[dietary fish oil] induced*** **severe colitis and adenocarcinoma [epithelial tissue cancer of the colon] formation.** DFO consumption was associated with *decreased CD8+ cell frequency* ***and*** *diminished CD69 expression* on CD4+ and CD8+ T-cell populations. Mice consuming DFO **also exhibited** ***higher*** *FoxP3+ CD25+ CD4+ T regulatory cell frequency, FoxP3 expression,* and ***altered*** *L-selectin expression* during infection."

Additionally, the article stated:

- "'We found that mice developed deadly, late-stage colon cancer when given high doses of fish oil,' [Fenton] said.

- "More importantly, with the increased inflammation, i only took four weeks for the tumors to develop.
- "…not only the mice receiving the highest doses of DHA but those receiving *lower doses as well.*
- "'Our findings support a *growing body of literature implicating harmful effects of high doses of fish oil consumption in relation to certain diseases,'* Fenton said.
- "'We hypothesized [guessed] that feeding fish oil enriched with DHA to mice would decrease the cancer risk; **we actually found the opposite**.'
- " [Fenton] said people already receiving enough omega-3 fatty acids through their normal diet and foods have no need for added [fish oil] supplementation." (Emphasis added.)

Inconvenient Truth #3: Fish oil decreases proper immune system responses.

The International Society for the Study of Fatty Acids and Lipids (ISSFAL) 4th Congress, which met on June 4-9, 2000 in Tsukuba, Japan, and was reported in the article titled "Omega-3 Polyunsaturated Fatty Acids, Inflammation and Immunity," by Philip C. Calder, Institute of Human Nutrition, University of Southampton, Bassett Crescent End, Southampton, UK:

- "…[S]tudies indicate that at the levels used, fish oil [omega-3 derivatives] ***decrease*** **a wide range of immune cell responses** (natural killer cell, cytotoxic T lymphocyte activities, lymphocyte proliferation and production of IL-2 and IFN-y (1,2))…"

- **"...Recent studies have indicated that** relatively low levels **of the long chain omega-3 fatty acids (EPA or DHA)...are sufficient to** bring about some of the suppressive effects**..."**

- **"... This decrease (of inhibited lymphocyte proliferation and natural killer cell activity) causes** increased cellular bacteria **[infection] and** impaired [cancer] tumor cell killing**."**

Inconvenient Truth #4: Cod liver oil significantly increases risk of skin cancer.

Veirord, MB, et al., "Diet and Risk of Cutaneous Malignant Melanoma: A Prospective Study of 50,757 Norwegian Men and Women," *Int. J. Cancer:* 71,600-604 (1997)**:**

- "**A significant risk was found in women who used cod liver oil supplement**. [W]e found a **strong increased risk for the women using cod liver oil, a supplement rich in omega-3 fatty acids (EPA and DHA)**." [There was approximately **3xs more incidence of melanoma (the most dangerous type of skin cancer) in the cod liver oil users**.]

- ***increase is considered to be real*** and ***not due to chance***.

- "Mean time of follow-up was 12.4 years…. [Note: Sufficient time for an excellent analysis.]

- participants selected in an **unbiased manner**, the **high**

participation and response rate, the prospective design with *dietary data collected prior to onset of cancer* and a **complete follow-up** with regard to incidence of cancer, deaths, and emigration. The complete follow-up is secured by the procedure established by the **Cancer Registry**, ensuring that all physicians, hospital departments and histopathology laboratories in Norway are obligated to report malignant diseases to the Registry: as many as 98% of the **cases** were histologically [microscopic tissue analysis] **verified**." [Note: This guarantees superb tracking and confirmation of cancer cases.]

Four more studies confirming increased skin cancer:

Rogers, HW, et al., "Incidence Estimate of Nonmelanoma Skin Cancer in the United States, 2006," *Archives of Dermatology* Vol. 146 (No. 3), March **2010**, pages 283-287 reports:

- "The total number of procedures for **skin cancer in the Medicare fee-for-service population increased by 76.9**% from 1,158,298 in 1992 to 2,048517 in 2006.
- "Nonmelanoma skin cancer (NMSC) is the **most common malignancy** in the United States.
- "…[T]he incidence of **skin cancer in** the United States has **substantially increased** from 1992 through 2006 and **is now almost double the last published estimate** from 1994."

Linos, EL, et al., "Increasing burden of melanoma in the United States," *Journal of Investigative Dermatology*, **2009** July, 129(7): 1666-1674:

- "**Malignant melanoma** is one of the fastest growing **cancers worldwide**

- "Overall melanoma **incidence increased at 3.1%** (1992-2004) **per year**.

- "We observed that melanoma **incidence increased for both men and women** across all categories **of tumor thickness**, including **a significant 3.86%** ***annual increase*** **among thickest tumors** (>4mm)." [Important note: The researchers clearly stated the increase is not due to better reporting, but a true increase in severity.]

Journal of Investigative Dermatology, **2008** December; 128(12):2905-2908, "Recent trends in incidence of cutaneous melanoma among U.S. Caucasian young adults":

- "Among women, age adjusted annual incidence [of melanoma] per 100,000 increased from 5.5 in 1973 to 13.9 in 2004."

Actas Dermosifiliogr. **2010**;101(1) 39-46, "Changes in the incidence of skin cancer between 1978 and 2008," reports:

- "The incidence of skin cancer continues to increase and can now be considered a worldwide epidemic."

British Journal of Cancer (**2008**) 99, 1549-1554, "Cancer mortality in the United Kingdom: projections to the year 2025," reports:

- "Malignant melanoma projections are +48% [Note: Although absolute numbers are small, the percentage should decrease, not increase!]

Inconvenient Truth #7: Fish oil is WORTHLESS in preventing heart disease in Type I diabetic women.

"Women With Type 1 Diabetes Receive No Heart Benefit From Omega-3," *Medical News Today (Diabetes)*, Article URL: http://www.medicalnewstoday.com/articles/193107.php, June 28, 2010:

- "Consuming higher amounts of **omega-3 fatty acids [as found in fish oil] does not appear to lower heart disease risk for women with type 1 diabetes**, according to a University of Pittsburgh Graduate School of Public Health study presented at the *70th Scientific Sessions of the American Diabetes Association*.

- "Omega-3 fatty acids [omega-3 *derivatives*], primarily found in fish, [***supposedly***] promote heart health by preventing the buildup of cholesterol in the arteries. Little is known about the effect of consuming omega-3 in *people with type 1 diabetes, who are at much greater risk for heart disease*.

- "Although omega-3 [derivatives] is ***typically associated*** [not directly causal] with decreased risk for cardiovascular disease, this may not be the case for women who have type 1 diabetes…."

Inconvenient Truth #10: Glycemic (blood sugar) control WORSENS during fish oil administration:

Stacpoole, P, Alig, A., Ammon, L, and Crockett, E., "Dose-Response Effects of Dietary Marine Oil on Carbohydrate and Lipid Metabolism in Normal Subjects and Patients With

Hypertriglyceridemia," *Metabolism*, Vol. 38, No 10 (October), 1989, pages 946-956:

- "**The glycemic [blood sugar] control of [all of]** the four insulin dependent diabetic patients **worsened during the fish oil administration**.

- "…[T]he **insulin** dose of the subjects **had to be increased** throughout the six-month period of fish oil administration to maintain constant blood glucose and glycosylated hemoglobin concentrations (HbA1c—average blood sugar level).

- "Despite the stable bodyweight by patients on the basal diet, glycosylated hemoglobin [A1c] levels **after six months of fish oil administration increased 16% from 4.9% to 5.7%**. [Note: This is an awful effect for a diabetic.]

- "Another **important finding** of our investigation was that consumption of a ***fish oil*-enriched diet worsens glycemic tolerance**."

"Fish-oil supplementation reduces stimulation of plasma glucose fluxes during exercise in untrained males," *British Medical Journal of Nutrition* (2003), 90, 777–786.

- "It is concluded that fish oil reduced Rd [rate of glucose disappearance] glucose by **26% by reducing glucose metabolic clearance rate** …" [Note: This is an awful effect for a diabetic.]

- "[I]t was observed in healthy human subjects that a **3-week supplementation of the diet with fish oil** (6g/

day) **decreased by 40% the insulin response** [a horrific effect] to an oral glucose challenge without altering either endogenous glucose production or plasma utilisation.

- "[N]-3 long-chain fatty acids are incorporated into **membranes whose composition remains altered at least 18 weeks after interruption of fish-oil supplementation**…."

Inconvenient Truth #11: Consumption of "fatty fish" decreases insulin levels.

Karlström, BE, et al., "Fatty fish in the diet of patients with type 2 diabetes: comparison of the metabolic effects of foods rich in n-3 and n-6 fatty acids," *Am J Clin Nutr* **2011**;94:26–33.

- "The reduction in fasting blood glucose and in the glucose area under the curve during the day was significantly greater with the n-6 [from lean fish] than with the n-3 [fatty fish] diet [*Showing 21% less insulin production with fatty fish compared to lean, non-fatty fish*]."

Anthony P. Bimbo, "Raw material sources for the long-chain omega-3 market: Trends and sustainability. Part 2," April **2009**, www.aocs.org/Membership/FreeCover.cfn?itemnumber=1085, accessed 10.8.11.

Inconvenient Truth #12: Amount of supplemented DHA incorporated into the brain is insignificant.

Umhau, JC, et al., "Imaging incorporation of circulating docosahexaenoic acid [DHA] into the human brain using positron emission tomography," *Journal of Lipid Research*, Vol. 50, **2009**, pages 1259–1268:

- "The characteristics of brain DHA metabolism permit the use of an irreversible uptake model over the time course of a PET scan. This is because the other forms of plasma PUFA (i.e., esterified in lipoproteins) were shown not to contribute measurably to brain uptake and because circulating precursors of ARA (linoleic acid, 18:2n-6) and of DHA (alpha-linolenic acid, 18:3n-3) after entering the adult brain are largely lost by metabolism and are ***not elongated*** to ARA or ***DHA*** **[rather, staying in parent form].**

- "Docosahexaenoic acid (**DHA**; 22:6n-3) is a critical constituent of the brain, but its metabolism has **not been measured in the human brain** in vivo [in the body]. In monkeys, using positron emission tomography (PET), we first showed that intravenously injected [1-^{11}C] DHA mostly entered nonbrain organs, with **approximately 0.5% entering the brain**.

- "Then, using **PET** and intravenous [1-11C] DHA in 14 healthy adult humans, we **quantitatively imaged** regional rates of incorporation (K*) of DHA.

- "For the entire human brain, the net DHA incorporation rate Jin, the product of K*, and the unesterified plasma DHA concentration **equaled 3.8 ± 1.7 mg/day.**

- "This net rate is equivalent to the net **rate of DHA consumption by brain** and, considering the reported amount of DHA in brain, **indicates that the half-life of**

DHA in the human brain approximates 2.5 years. Thus, PET with [1-11C] DHA can be used to **quantify regional and global human brain DHA metabolism in relation to health and disease**."

"Alpha-Linolenic Acid Conversion Revisited," (www.fatsoflife.com) by Norman Salem, et al.

- "A recent article in the 2003 PUFA [Polyunsaturated Fatty Acid] Newsletter indicated that in adult men and women the 'average estimated conversion of … [EPA/DHA]… ***is likely to be an overestimate of the actual overall conversion rates for several reasons***.' We see even with this excessive estimate of the parent omega-3 derivative conversion that theoretically no more than 37% of them are converted to derivatives.

- "However, The article makes the case that, 'these amounts correspond to a conversion rate of one gram alpha-linolenic acid in the order of **< 0.02**% for total n-3 LC-PUFAs or **0.002% for conversion to DHA**'….[based on blood lipid conversion].

- "In conclusion, we believe the estimates and interpretations currently put forward as ***best estimates can be substantially improved***. The best estimates of alpha-linolenic acid **conversion to n-3 LC-PUFA [DHA/EPA] are much smaller than those claimed**. More rigorous determinations of n-3 fatty acid metabolism must serve as the foundation for more accurate nutritional conclusions and dietary recommendations."

Inconvenient Truth #13: EFA derivatives are made by the body "as needed."

"Flaxseed oil and fish-oil capsule consumption alters human red blood cell n–3 fatty acid composition: a multiple-dosing trial comparing 2 sources of n–3 fatty acid," *American Journal of Clinical Nutrition*, Vol. 88, No. 3, 801-809, September **2008**.[1]

- "Although an increased intake of dietary ALA might be e**xpected to upregulate ALA conversion**, *this has . . . **not been found**…*" [This means your body does not want more regardless how much can easily be made.]
- "Overall conversion rates of LA and ALA, calculated from peak.
- "[^{13}C] LCP concentrations adjusted for dietary influences on pool sizes of LA and ALA, were low and of similar magnitude overall for AA and EPA (**0.18% and 0.26%)**....
- "Few studies have attempted more than **relatively crude estimates of isotope transfer** from tracer into the various tracee pools, and it is recognized that AUC [area under the curve] ***values will overestimate true conversion rates*** and provide only approximate relative rates of transfer."[Note: Not using radioactive isotopes that directly appear in specific tissue so you can measure them is why so many health professionals have been

1 Hussein, Nahed, et al., *Journal of Lipid Research*, Volume 46, **2005**, pages 269–280.

misled; thinking the PEO-to-derivative conversion rates are much higher than they actually are.

Inconvenient Truth #14: The body only uses extremely small amounts of ALA to make DHA.

Pawlosky, RJ, et al., "Physiological compartmental analysis of alpha-linolenic acid metabolism in adult humans," *Lipids Res* **2001** 42: 1257–65.

- Research at the United States Department of Agriculture's USDA food composition laboratory concludes that: "Only about 0.2% of the plasma 18:3n-3 [ALA] was destined for synthesis of 20:5n-3 [EPA], approximately 63% of the plasma 20:5n-3 was accessible for production of 22:5n-3, and 37% of 22:5n-3 [0.23% of the 0.2% = 0.046% net ALA] was available for synthesis of 22:6n-3 [DHA]." Unlike what you are told by sellers of fish oil supplements, this is confirmation of the extremely small amounts your body uses to make DHA.

Inconvenient Truth #15: Amounts of EPA/DHA in fish oil are pharmacological plasma overdoses

There were other published warnings about the overestimate of parent-to derivative amounts. The article, "Comparison of bolus versus fractionated oral applications of [^{13}C]-linoleic acid in humans," *European Journal of Clinical Investigation*, Volume 29 Issue 7 (2001), Pages 603–609, had this to say regarding over-estimations of derivatives (EPA/DHA): "**Conclusions**: Using areas under the curve [the simple, standard method of analysis]

overestimates the conversion, because different residence times are ***not considered***."

Inconvenient Truth #16: Babies DO produce the omega-6 derivative, arachidonic acid (AA), and the omega-3 derivative, DHA.

Carnielli, V.P., et al., "The very low birth weight premature infant is capable of synthesizing arachidonic and docosahexaenoic acids from linoleic and linolenic acids," *Pediatric Research*, Vol. 40, No. 1, 1996, pages 169–174.

- "…[T]his clearly shows that **all infants were capable of actively synthesizing** the long chain polyunsaturated FA from their dietary precursors.

- "We report a **newly developed approach** which enabled us to measure *in vivo* [in the body] the biosynthesis of LCP with stable isotopes. The study shows that **small preterm infants are capable of converting both LA and LNA into LCP [long chain polyunsaturated fatty acid]**. We were also able to measure the ^{13}C enrichment of all major metabolites of the essential FA including C18:3n-6, which is the delta-6 desaturase product of LA and thought [guessed] to be the limiting step in EFA metabolism. "**The major finding of this study** is that the healthy preterm infant at approximately 1 month of age can desaturate and elongate LA and LNA into n-6 and n-3 LCP, respectively.

- "This observation suggests that the D6 desaturation *may not be a rate-limiting step* in our patients.

- "We chose to study preterm infants receiving a formula with a 10: 1 ratio of LA and LNA because this ratio is often found in human milk lipids. [Note: In this experiment, the infant was given adequate "parent" PEOs to ensure conversions.]

- "The duration of *our studies was far longer* than any other published work, and we show that at 168 hours the plasma phospholipid LCP were still highly enriched."

Inconvenient Truth #17: Fish oil increases platelet aggregation.

Knapp, H, et al., "In vivo indexes of platelet and vascular function during fish-oil administration in patients with atherosclerosis," *The New England Journal of Medicine*, Vol. 314, April 10, 1986, No. 15, pages 937–942: In patients with atherosclerosis, prostacyclin biosynthesis **fell** by a mean [average] of 42% during the fish-oil period.

- "...In patients with atherosclerosis, **prostacyclin** biosynthesis **fell** by a mean [average] of 42% during the fish-oil period."

Inconvenient Truth #18: Fish were found to be worthless in decreasing abnormal heart rhythm (called atrial fibrillation, or AF).

Jarrett D. Berry, MD, et. al., "Dietary Fish Intake and Incident Atrial Fibrillation," 15 March **2010**, *The American Journal of Cardiology,* V. 105, I. 6, 844–848.

Inconvenient Truth #19: Fish oil supplements increased sudden cardiac death in those with coronary heart disease.

Burr, et al., "Lack of benefit of dietary advice to men with angina: results of a controlled trial," *Eur J Clin Nutr* **2003**, 57:193–200.

Inconvenient Truth #20: Fish oil does not slow atherosclerosis.

Angerer, P., et al., "Effect of dietary supplementation with omega-3 fatty acids on progression of atherosclerosis [plaque buildup in interior of arteries] in carotid [heart to brain] arteries," *Cardiovascular Research*; 54:183–190, **2002**.

- Both **fish oil** groups and the control groups showed close to equal atherosclerotic progression (**getting more clogged**). Fish oil **did not stop thickening of the artery** On the contrary, the artery wall got thicker (bad) with fish oil ingestion!

- "In this group of selected patients with documented coronary artery disease, omega-3 PUFA [polyunsaturated fatty acids] **given for 2 years did not demonstrate an effect on slowing progression** of atherosclerosis in carotid arteries as measured by ultrasound." [Note: 1.65 grams per day of fish oil supplement were taken. This is a great enough dose to cause adverse immunity and bleeding effects.]

Sacks, Frank M., et al., "Controlled Trial of Fish Oil for Regression of Human Coronary Atherosclerosis," *Journal of the American College of Cardiology* Vol. 25, No. 7, June 1995: 1492–8.

- "Fish oil ***treatment for 2 years*** **DOES NOT** promote major **FAVORABLE CHANGES** in the diameter of atherosclerotic coronary arteries."

Inconvenient Truth #21: "Fatty fish does NOT prevent stroke; "lean fish" does prevent stroke.

Larsson, Susanna C, et al., "Fish consumption and risk of stroke in Swedish women," *Am J Clin Nutr* **2011**;93:487–93.

- "To our knowledge, this is the largest study (with respect to the number of stroke cases) to date to examine this association.

- ***lean fish was associated with a significant reduced risk*** of total stroke and cerebral infarction.

- "The consumption of salmon, whitefish, and char and herring and mackerel **[fatty /oil fish] was *not associated with [reduced] risk*** of total stroke or any stroke subtype.

- "Although an inverse association between fatty fish consumption and stroke is biologically plausible [although a silly guess as there is no significant metabolic pathway suggesting this], we observed no significant association between the consumption of salmon, whitefish, and char or herring and mackerel [all fatty/ oily fish] and risk of stroke in this study."

- "**Lean fish** consumption has been shown to ***reduce systolic and diastolic blood pressure*** in subjects with ischemic heart disease."

Inconvenient Truth #22: Fish oil does not decrease inflammation

Pot, GK, et al., "No effect of fish oil supplementation on serum inflammatory markers and their interrelationships: a randomized controlled trial in healthy, middle-aged individuals," *European Journal of Clinical Nutrition*, **2009** (62), 1353–1159.

- "In conclusion, ***this 12-week randomized, double-blind placebo-controlled*** intervention trial ***did not show*** that 1.5 g/day n-3 PUFA [fish oil] significantly affected the serum inflammatory response in healthy individuals, nor did patterns of inflammatory markers. Thus, a healthy ***middle-aged population may not benefit from fish oil as an anti-inflammatory agent***.

- "Overall, it seemed **that all serum inflammatory markers were increased rather than decreased after fish oil supplementation** than with placebo; however, these increases were not statistically significant.

Inconvenient Truth #24: Fish or its EPA/DHA does NOT help depression.

Lucas, Michel, et al., "Dietary intake of n-3 and n-6 fatty acids and the risk of clinical depression in women: a 10-y prospective follow-up study," *Am J Clin Nutr* **2011**;93:1337–43.

- "In conclusion, the results of this large longitudinal **study do not support a protective effect of long-chain n-3 fatty acids [EPA/DHA] or fish** intake on the risk of depression.

- "The use of 4 dietary assessments *over a period of 10 y[ears] was a unique strength of our study*
- "In this large prospective cohort of women, we found that **higher dietary intake of vegetable n-3, ALA [parent omega-3], was significantly associated with a lower risk of clinical depression**, especially among those who had the lowest intake of LA [which is often highly adulterated].
- "Compared with nonusers of fish-oil supplements, the **risk of clinical depression was unexpectedly high in the small fraction of women (n = 689) who reported fish-oil consumption** in 1990 only. [Note: Fish oil consumption was virtually nonexistent pre-1990. Fish oil's quick negative effect shocked the researchers.]
- "The risk of clinical depression increased with increasing LA intake. [Note: You will discover exactly why this is predictable in the next chapter along with how to prevent this negative effect from occurring.]
- "**We did not observe any association** between the risk of **clinical depression and fish consumption frequency or EPA+DHA intake from fish**."

Inconvenient Truth #26: Fish oil adversely affects chemotherapy.

Roodhart, Jeanine M.L., et al., "Mesenchymal Stem Cells Induce "Resistance to Chemotherapy through the Release of Platinum-Induced Fatty Acids," *Cancer Cel*l, **2011**; 20 (3): 370 DOI: 10.1016/j.

ccr.2011.08.010; www.medicalnewstoday.com/articles/234263.php.

- "Patients receiving virtually **all types of chemotherapy have been advised not to take fish oil supplements because they can make chemotherapy drugs ineffective** researchers from the University Medical Centre Utrecht, the Netherlands wrote in the journal *Cancer Cell*.
- "Cancer **patients commonly take fish oil supplements** in addition to their standard treatment.
- "Lead scientist, Professor Emile Voest, an oncologist, said: 'Whilst waiting for the results of further research, we **currently recommend that these products should not be used whilst people are undergoing chemotherapy**.'"

Scientific Support for Chapter 8: The Danger of Processed/ Adulterated Fats

Is steaming or pan-frying a fish filet better?

Prepare to be shocked. *The Journal of Agricultural and Food Chemistry* has this to say:[1]

> "Salmon fillets were steamed, or pan-fried without oil, with olive oil, with corn oil, or with partially hydrogenated plant oil.
>
> "In particular, ***fish and fish oils are highly susceptible to oxidation*** due to their high content of polyunsaturated fatty acids (30-40%), mainly eicosapentaenoic acid (**EPA**, 20:5n-3) (5-18%) and docosahexaenoic acid (**DHA**, 22:6n-3) (1-12%). These **unique fatty acids distinguish fish lipids from other plant and animal lipids**.
>
> "The sum of **cholesterol oxidation products** (**COPs**) increased after the heating processes from 0.9 *m*g/g in the raw sample to **6.0**, 4.0, 4.4, 3.3, and **9.9** *m*g/g extracted fat in pan fried **without oil**, with olive oil, corn oil, partially hydrogenated plant oil, and **steamed**, respectively.

---

1 Al-Saghir, Sabri, et al., "Effects of Different Cooking Procedures on Lipid Quality and Cholesterol Oxidation of Farmed Salmon Fish (*Salmo salar*)," *J. Agric. Food Chem.* **2004,** *52,* 5290–5296.

"As expected, the lipid oxidation of the residues was advanced. The highest increase was found for the residues of cooking without oil which ***revealed the highly oxidative potential of the fish oil*** that drained out of the sample during cooking…

► **There was a "700% increase in COPs with fish *seared without oil*." In contrast, meats cooked without oil increased only "120%–315%."**

"In particular, steaming increased the total amount [COPs] by more than 1000%, which should be considered in nutritional recommendations, although the initial COPs content of salmon is low compared to other foods such as meat." [As you already understand, any type of heat compared to the frigid temperatures fish live in, quickly ruins EPA/DHA.]

PEO Solution analysis: Shockingly, that steaming caused the most damage. The **least damage** was from **pan-frying** in hydrogenated oil. However, do NOT use that oil. Use a saturated fat like organic coconut or palm oil for the best results. Hydrogenation is a very harmful process. Heating the fish with no oil is unhealthy because the oil draining from the fish is exposed to high temperature. That's why the authors state, "As expected, the **lipid oxidation of the residues (oils) was advanced**. The highest increase was found for the **residues** of cooking without oil, which ***revealed the highly oxidative potential of the fish oil*** that drained out of the sample during cooking."

Warned but Few Listened

The **2001** journal article, "Health effects of oxidized heated oils," warned us but too few listened:[2]

- "The purpose of this report is to alert the food service industry, particularly the fast-food industry, of an emerging health issue. Considerable evidence has accumulated over the past two decades that heated cooking oils, especially polyunsaturated oils, may pose several types of health risks to consumers of fried foods and even people working near deep fat fryers. Heat degrades polyunsaturated fatty acids to toxic compounds; ***saturated and monounsaturated fatty acids are resistant to heat-induced degradation***.

- "The thermally-induced oxidation of glycerol-bound **polyunsaturated fatty acids** (PUFAs) in foods and culinary oils during standard frying or cooking episodes is a process that involves the prior generation of isomeric **conjugated hydroperoxydiene** (CHPD) species. These CHPDs fragment to form alkoxyl radicals that, in turn, undergo β-scission to generate a **wide range of aldehydic products**. In view of the extremely **toxic nature of the aldehydic end-products generated** the employment of PUFA-containing culinary oils for **domestic or commercial frying/cooking** episodes poses **health hazards** that have recently attracted much public and clinical interest.

---

2 Grootveld, Martin, et al., *Foodservice Research International*, Vol. 13, **2001**, pages 41–55.

- "Indeed, these **cytotoxic agents** have been implicated in the development and progression of **atherosclerosis** and its associated pathological sequelae such as ischemic **heart disease and peripheral vascular disease** and have also been shown to exert gastropathic, **pro-inflammatory**, and genotoxicological properties. These phenomena are undoubtedly attributable to the **extremely high reactivity of aldehydes with critical biomolecules** (e.g., thiols such as glutathione; DNA, forming covalently-modified base adducts; and the Apolipoprotein B component of low-density lipoprotein, altering its biological characteristics.)"

Pregnant women, beware:

- "The administration of nonheated oil did not give rise to any changes in the rate of fetal malformations (the administration of either preheated or nonheated culinary oil did not lead to any modifications in the rate of reabsorptions, crown-rump length, or the somite number). These results demonstrate that the administration of aldehyde-containing thermally stressed culinary oil is highly teratogenic [causing malformation of an embryo] in the rat, and ***an attractive hypothesis is that the intake of such oxidized oils during pregnancy may be partially responsible for the neural tube defects found in humans***. **Differences in the type of heated oil used in standard frying or cooking processes may also be responsible for the differing rates of neural tube defects found among different populations**."

It was known decades ago (1993) that oxidized oils, from food processing, caused thrombosis. Although quantitative analysis has greatly improved over the past 20 years, many researchers understood its importance back then:[3]

> "This review will examine the possible **relevance of whole oxidized oils in food products to the process of atherogenesis**. For excellent in-depth reviews of the many prevailing issues in the area of **lipid oxidation, its products, and chronic diseases**, the reader is referred to a number of recent papers [18 papers are referenced in this 1993 article].
>
> "**Lipid hydroperoxides have been shown to accelerate the atherosclerotic process** in terms of initiation of **endothelial injury**, the **progression phase** in which there is **accumulation of plaque,** and the **final termination phase** of thrombosis.
>
> "It is of interest that **macrophages take up LDL very slowly** and *do not change to foam cells unless* the **LDL has been modified or oxidized**.

PEO Solution analysis: CVD-damage caused by food processing has been known about for decades yet nearly nothing was done. Even today, its deleterious impact is not fully understood. Macrophages do *not* become foam cells unless there is oxidized cholesterol. You have already seen that its source is adulterated of Parent omega-6 form food processing.

---

3 Kubow, Stan, "Lipid oxidation products in food and atherogenesis," *Nutrition Reviews*, Feb 1993;51,2, pages 33-40.

Adulterated, Non-functional PEOs Must Be Replaced with Functional PEOs

Dr. David Horrobin was the world's leading authority on Parent omega-6 and its derivatives. Horrobin's superb article detailing EFA metabolic pathways states:[4]

> **"...Thus high intakes of non-EFAs may lead to an increased requirement for EFAs. The *trans* and positional non-EFA isomers of EFAs may be particularly important in this context.** Such isomers can be formed in the rumen of cattle and so are found in small amounts in dairy products. However, ***far greater quantities*** of these isomers enter the human food chain as a result of ***processing of vegetable oils.*** To be effective as EFAs, all the double bonds of its molecule must be in the cis-configuration [*un*adulterated, fully functional PEOs]. However, such *cis* double bonds are relatively unstable and may lead to a reduced food shelf life or have other properties, which *make them inappropriate for inclusion in processed foods*.

► **"***As a result surprisingly large amounts of linoleic acid* **[Parent omega-6]** *are* ***modified*** *to improve stability and handling characteristics***. Total daily intake of these modified linoleic acids [Parent omega-6], which have no EFA activity, is high, of the order of 6-12 g/day in Western countries. These modified *trans* and**

---

4 Horrobin, D.F., "Nutritional and medical importance of gamma-linoleic acid," *Prog. Lipid Res.*, Vol. 31, No. 2: 163–194, 1992.

> **other derivatives are not only devoid of EFA activity themselves, they also compete with normal EFAs and** ***interfere with their actions and metabolism.*****"**

An *adulterated* PEO is NO longer a PEO!

PEO Solution analysis: Dr. Horrobin hits the nail on the head. Recall the damage caused by just 0.5 gram of *processed* PEOs. Adulteration by food processors causing non-functionality of Parent omega-6 (LA) is at the core of many of our health epidemics. **This is why regardless of other interventions; the patient's consumption of *adulterated Parent omega-6 MUST be solved* first in order to increase the outcomes of other protocols.**

Root Cause of Defective Cholesterol and Membrane Alternation: Oxidation of Parent Omega-6

"Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity:"[5]

> "The effect of dietary **oxidized oil** on the lipid **composition**, **fluidity** and **function** of rat liver microsomes was studied. Male growing rats were fed diets containing 10 g/100 g of a fresh (control**) or oxidized** (experimental) linoleic acid [**Parent omega-6**]–rich preparation for 4 wk.

5 Hochgraf, Edna, et al., "Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity," *J. Nutr.*127: 681–686, 1997.

"…This [increased membrane fluidity] was due to **profound differences in lipid composition of the liver** microsomes, namely, a lower cholesterol to phospholipid molar ratio and a greater arachidonic acid content in the phospholipids of the rats **fed the experimental [oxidized] diet.**

► **"The study** ***demonstrated that ingestion of oxidized lipids caused profound alterations in membrane composition, fluidity and function….***

"The main differences observed in the composition of the microsomal **phospholipid** fatty acyl residues between the two groups were a **23.2% lower linoleic acid [Parent omega-6] level** and a 14.2% greater arachidonic acid level in the rats fed the oxidized linoleic acid diet.

PEO Solution analysis: It's all confirmed**. Adulterated/processed Parent omega-6 (from food processing) causes profound physiologic problems for patients**. The adulterated Parent omega-6 takes the place of the fully functional Parent omega-6 in phospholipids and we have a physiologic disaster. We will detail its cholesterol-connection in a subsequent chapter.

Cancer and Mitochondria Defects From Parent Omega-6 Deficiency

In April, **2009**, I presented a paper at the 17th Annual World Congress on Anti-Aging Medicine in Orlando, Florida (April **2009**), which utilized new research from **2007–2009** to identify the prime

cause of cancer. Predictably, these findings caused quite a sensation. Numerous physicians met with me afterwards to applaud the presentation and discuss direct patient application in their practices. This article, "Cancer and Mitochondria Defects: New 21st Century Research," was published in the *Townsend Letter for Physicians* (August/Sept. **2009**), pp. 87–90), and is linked here: www.brianpeskin.com/BP.com/publications/CancerMito-Town8.09.pdf**.**

Metabolism and Longevity

Physicians, regardless of specialty, can benefit from patients' increasing demands for anti-aging solutions. "Anti-Aging" Medicine has become the largest growing segment of medicine. As you have already discovered, **PEO Solution** offers unprecedented patient solutions for this new market segment.

Scientist Dr. A.J. Hulbert details the connection between PEOs and cell membranes in his paper, "Metabolism and Longevity: Is There a Role for Membrane Fatty Acids?"[6] He discusses the damage to the cell membranes caused by lipid peroxidation. This is the process whereby lipids in cell membranes are degraded by peroxidation—free radicals grab electrons from the lipids, producing reactive molecules that become involved in a damaging chain reaction that weakens the cell membrane. **Of particular concern is the mitochondrial membrane, which helps to determine longevity**. It is mostly polyunsaturated fatty acids that are affected.

---

6 Hulbert, A.J., "Metabolism and Longevity: Is There a Role for Membrane Fatty Acids?" *Integrative and Comparative Biology*, Vol. 50, No. 5: 808–817.

- "Although unknown in Rubner's time [Rubner was a scientist studying metabolic rate and correlated longevity], one aspect of body composition of mammals also varies with body size, namely the ***fatty acid composition of membranes***. Fatty acids **vary dramatically** in their **susceptibility to peroxidation** and the products of lipid peroxidation are very powerful reactive molecules that damage other cellular molecules. ***It is apparent that membrane composition is regulated for each species***. The exceptional longevity of Homo sapiens combined with the limited knowledge of the fatty acid composition of human tissues support the potential ***importance of mitochondrial membranes in determination of longevity***.
- "The insight that the exceptionally long-living species, Homo sapiens, potentially provides for understanding the mechanisms determining animal longevity, is that the ***fatty acid composition of mitochondrial membranes may be much more important than the composition of other cellular membranes***."

PEO Solution analysis**:** As Dr. Hulbert makes clear, membrane lipid composition is specific to each species. Diet typically has a minimal effect on its composition *EXCEPT when unnaturally overdosed* (as with flax oil alone, fish oil, or *adulterated* oils). This excess of non-functional oil has to get incorporated *improperly* into tissue—it all can't simply be "burned up" for energy. **Physicians specializing in anti-aging know about the importance of mitochondrial function**.

Dr. Hulbert emphasizes the importance of structural integrity and that requires plenty of *fully functional*, *unadulterated* Parent omega-6. I have written about mitochondrial composition—in particular, its dependence on fully functional Parent omega-6.

Continuing with this amazing article, Dr. Hulbert explains what makes cell membranes so susceptible to adulteration by peroxidation:

- "In naturally occurring polyunsaturates [including PEOs], the –C=C- units [these are the double-bonded carbon units] are all separated by a single-bonded –C- [carbon] atom. The hydrogen atoms attached to each of these intermediate –C– atoms are called ***bis-allylic*** hydrogens, and have the lowest C–H [weakest] bond-energies of the fatty acid chain. This [weak bond] makes them the ***most susceptible to attack by Reactive Oxygen Species (ROS)*** [chemically reactive molecules which contain oxygen] produced during aerobic metabolism."[7]

Bis-allylic (weak-bonded) hydrogens in the cellular membrane are the most susceptible to attack.

Although the following was covered in the chapter it is worth repeating. Dr. Hulbert's explanation immediately exposes the dangers of fish oil:

---

7 Ibid.

- "**Docosahexaenoic acid** (22:6), which has six double bonds and consequently *five bis-allylic hydrogens per chain*, is **320 times more susceptible to [anti-oxidant] attack** than the common **monounsaturated oleic acid** (18:1) which has ''no'' *bis-allylic* hydrogens in its chain."

… and warns of additional DNA and protein DAMAGE:

- "Membrane lipid peroxidation should not be perceived solely as a 'damage to membranes' scenario but also as a significant ***endogenous source of damage to other cellular macromolecules***, such as proteins and DNA (including mutations)."

PEO Solution analysis: **Aside from PhDs in chemistry, the medical profession is not used to this biochemical term or the superior *Peroxide Index* (PI).** The more polyunsaturated the oil is, the more *bis-allylic* chains are present. Fish oil's DHA (22:6) has six of them and therefore, is enormously more reactive than the monounsaturated oleic (olive oil), which contains none, and seven times more reactive than Parent omega-6. To combat this oxidation, you body is forced to "use up" its reserve of anti-oxidants, leaving other areas vulnerable.

Dr. Hulbert also explains the chain reaction that occurs with the attack on polyunsaturated fatty acids:

- "Thus, **contrary to a common misconception**, it is ''not'' just the presence of double-bonds in a mixture of fatty acids [(often quantified as the ''double-bond index'' (DBI) or ''unsaturation index'' (UI)] that will determine the susceptibility of this mixture to oxidative damage,

but rather the precise types of fatty acids present in the mixture and their relative abundance…. In this way [of causing lipid membrane peroxidation], **ROS attack on a membrane** lipid bilayer (that contains polyunsaturated fatty acids) **differs from ROS attack on other cellular molecules** such as proteins, carbohydrates and nucleic acids. Whereas ROS attack on these other types of molecules will damage the molecule and likely stop them from performing their function, **ROS attack on membrane polyunsaturates will damage the molecule** (by converting it to a lipid hydroperoxide) and will **also produce another reactive molecule** that will in turn continue the oxidative damage to other molecules…. The products of lipid peroxidation, such as lipid hydroperoxides, can also undergo fragmentation to produce a broad range of reactive intermediates (called propagators) that can modify proteins and DNA to produce ''advanced lipoxidation endproducts'' (ALEs)…"

► "Membrane lipid peroxidation should not be perceived solely as a ''damage to membranes'' scenario but also as a significant ***endogenous source of damage to other cellular macromolecules***, such as proteins and DNA (including mutations)."

PEO Solution analysis: As can be seen from Dr. Hulbert's work, the damage to cell lipid membranes is horrific… to the membranes themselves and far beyond … to proteins and critical DNA, too. With Dr.

Hulbert's magnificent work, we can now more fully appreciate how PEO adulteration—whatever its cause—is the *primary* issue behind virtually all patient health issues.

Life Span Based on Metabolic Rate and Membrane Composition

Dr. Hulbert has much more to teach us with his incredible treatise entitled "Life and Death: Metabolic Rate, Membrane Composition, and Life Span of Animals."[8] This paper is required reading if you really want to understand aging, free radicals, and tissue membranes. Some highlights (not necessarily in publication order) are:

- "***Mitochondrial cardiolipin*** molecules are possible targets of oxygen free radical attack, due to their high content of polyunsaturated fatty acids and because of their location in the inner mitochondrial membrane near the site of ROS [Reactive Oxygen Species] production. In this regard, it has been recently demonstrated that mitochondrial-mediated ROS generation affects the activity of complex I, as well as complexes III and IV, via peroxidation of cardiolipin following oxyradical attack to its fatty acid constituents. These findings might *explain the decline in respiratory chain* complexes observed in mitochondria isolated from *aged animals and in pathophysiological conditions* that are characterized by an increase in the basal rate of the ROS production."

8 Hulbert, A.J., et al., Life and Death: Metabolic Rate, Membrane Composition, and Life Span of Animals," *Physiological Reviews*, Vol. 87, October 2007, pages 1175–1213.

PEO Solution analysis: **The importance of cardiolipin** is once again emphasized. Keep the supply of fully functional PEOs, in particular Parent omega-6, high and this potential problem is significantly reduced.

The treatise continues:

- "It is suggested that rapid constitutive recycling of membrane phospholipids **rather than selective in situ repair** is responsible for eliminating peroxidized phospholipids, with triacylglycerols **providing a dynamic pool of undamaged PUFA** (PEOs) for phospholipid resynthesis."

PEO Solution analysis: The body can either *attempt* to repair damaged PEOs or it can **simply replace the damaged PEOs with fully functional PEOs—IF they are available.** *In situ* repair is nearly impossible. **We all require daily ingestion of fully functional PEOs.**

Continuing …

- "The susceptibility of membrane lipids to oxidative damage is related to two traits. **The first is that oxygen and many radical species are several times more soluble in lipid membrane bilayers than in the aqueous solution.** The second property is related to the fact that not all fatty acid chains are equally susceptible to damage. **It is this second property that is the key to the link between membrane composition and oxidative damage to membranes.** The carbon atoms that are most susceptible to radical attack are the single-bonded carbons between the double-bonded carbons of the acyl chains.

▶ "Furthermore, the ***noncharged structure of aldehydes*** allows them to migrate with relative ease through hydrophobic membranes and hydrophilic cytosolic media, thereby *extending the migration distance far from the production site*. On the basis of these features alone, ***these carbonyl compounds can be more destructive than free radicals and may have far-reaching damaging effects on target sites both within and outside membranes***.

• "…When such C• radicals are generated in the hydrophobic interior of membranes, a likely fate is combination with oxygen dissolved in the membrane. The resulting peroxyl **radical is highly reactive: it can attack membrane proteins and can also oxidize adjacent PUFA [PEO] chains**. Thus the *initial reaction is repeated* and a free radical *chain reaction is propagated*…. [T]hese can thus *disrupt the membrane* structure, altering fluidity and other functional properties of membranes…."

▶ "…Thus ***lipid peroxidation should not be perceived solely in a "damage to lipids" scenario, but should also be considered as a significant endogenous source of damage to other cellular macromolecules***, such as proteins and DNA (including mutations).

• "In this way, variation in ***membrane fatty acid composition***, by influencing lipid peroxidation, can have ***significant effects on oxidative damage to many and varied cellular macromolecules***. For example,

peroxidized cardiolipin in the mitochondrial membrane can inactivate cytochrome oxidase by mechanisms both similar to hydrogen peroxide and also mechanisms unique to organic hydroperoxides…"

PEO Solution analysis: We can't do anything about the increased solubility of oxygen in cellular membrane ® ROS. This is a physiologic fact. We can see that the aldehydes cause grave damage to lipids, proteins, DNA, and other biological entities—even far removed from their source! **The second property is a different story. It is important to note that the (compositional) material of the membrane DOES make a difference; oxygen diffusion in the cell membrane (at physiologic conditions) is approximately twice as fast as water!**[9]

SOLUTION…

Make sure the membrane is not unnaturally altered. This is accomplished in two distinct ways: First, make sure the body has sufficient PEOs. Second, make sure there is no overdosing of either the omega-6 or omega-3 series fatty acids, e.g. fish oil or flax oil without appropriate systemic PEO and their associated series derivative compensation. Balance is required and most importantly, we need to understand what "balance" means.

---

9 Subczynski, W.K., et al., "Is the mammalian cell plasma membrane a barrier to oxygen transport?," *Journal of General Physiology*, Vol. 100, 1992: 69–87.

Unsaturation Index (UI) and Peroxidation Index (PI)

Continuing with Dr. Hulbert's article, "Life and Death: Metabolic Rate, Membrane Composition, and Life Span of Animals," …

- "The peroxidation index [PI] of a membrane is not the same as its unsaturation index [UI] (sometimes also called its "double-bond index"), which is a measure of the density of double bonds in the membrane."

The respective measurements follow. It is important to note that the PI is based on actually measuring the *real-life* increase in peroxidation of the series of double-bonded lipids. *PI index is much more instructive in measuring relative PEO and PEO-derivative peroxide potentials.*

Peroxidation index (PI): 0.025 x (% monoenoics) + 1 x (% dienoics) + 2 x (% trienoics) 4 x (% tetraenoics) + 6 x (% pentaenoics) + 8 x (% hexaenoics).

Unsaturation index = 1 x (% monoenoics) + 2 x (% dienoics) + 3 x (% trienoics) + 4 x (% tetraenoics) + 5 x (% pentaenoics) + 6 x (% hexaenoics).

Omega Chain Length	**PI**
Oleic (olive Oil)	2.5

Parent omega-6	100
Parent omega-3	200
DHA	800

Parent omega-3 is *twice* as reactive as Parent omega-6 and DHA has an astronomical *8-fold* increase in reactivity compared to Parent omega-6. As will be detailed later, the body has sufficient antioxidant capability to protect normal, physiologic levels of polyunsaturated fatty acids throughout the body. As the world-leading biochemist Professor Gerhard Spiteller, Chairholder of Biochemistry, Institute of Organic Chemistry at the University of Bayreuth, Germany, makes clear, these are NOT an issue—Nature is consistent. However, it does NOT have capacity to combat artificial OVERDOSES based on wrong nutritional advice.

Tragically, we are (unknowingly)
our own worst enemy!

It is only when we artificially introduce supra-physiologic amounts of improper lipids that the excessive cellular destruction and ROS oxidation problem exists. ***Despite the best of intentions, we cause ourselves grave harm.***

PEO Solution analysis: This information goes a long way toward independent proof and verification of my thesis that we are ***ingesting pre-oxidized*** PEOs—in particular, adulterated Parent omega-6—and it is too late for an "antioxidant" to work, regardless of quantity be-

cause the oxidation damage is already done! When esterified cholesterol is analyzed, it will be "case closed."

An example of the difference between Unsaturation Index (UI) and Peroxidation Index (PI):

The peroxidation index of a membrane is not the same as its unsaturation index—"double-bond index"—a measure of the density of double bonds in the membrane. For example, a membrane bilayer consisting solely of MUFA [monounsaturates like in the majority of olive oil] will have an **unsaturation index of 100 and a peroxidation index of 2.5**, while a membrane bilayer consisting of 95% SFA (saturated fat) and 5% DHA (with 6 double-bonds) will have an **unsaturation index of 30 and a peroxidation index of 40**.

The 5% DHA-containing membrane is 16 times (40 / 2.5) more susceptible to peroxidative damage—a most surprising result!

The potential damage to all cell membranes, including mitochondrial membranes is immense.

With Fish Oil, EPA Rises, Linoleic Acid Decreases—Real-Life Results

The superb article by Martijn B. Katan, et al., "Kinetics of the Incorporation of Dietary Fatty Acids Into Serum Cholesteryl Esters, Erythrocyte Membranes, and Adipose Tissue: An

18-month Controlled Study," (*Journal of Lipid Research*, Vol. 38, 1997, pages 2012–2022) has this to say:

- "Tissue levels of n-3 fatty acids [fish oil] reflect dietary intake, but quantitative data about rate of **incorporation and levels as a function of intake** are scarce. We fed **58 men** 0, 3, 6, or 9g/d[ay] of **fish oil for 12 months** and monitored fatty acids in serum cholesteryl esters, erythrocytes, and subcutaneous fat during and after supplementation. Steady-state levels **increased by 3.9 ± 0.3 mass % points (± SE) for each** ***extra gram of EPA eaten per day***. Incorporation of docosahexaenoic acid (**DHA**) was erratic; plateau values were ***1.1 ± 0.1 mass % higher for every gram per day ingested***.

▶ "The rise **in EPA** [a significant component of fish oil] was compensated for largely by **decreases** in linoleic acid [**Parent omega-6**]."

PEO Solution analysis: This experiment shows how the overdose of fish oil increases cholesterol esters—the body's most significant fatty acid transport system. There was a corresponding increase per day of the erythrocytes, too. Natural triglyceride form (not synthetic esters) was used. Trappist monks were the population sample. ***We see the fish oil overdose displacing critical Parent omega-6***—destroying critical mitochondrial functionality, etc. Given fish oil's incredible high rate of oxidation, even a "little too much incorporation" (3.9% extra with a corresponding 3.9% less Parent omega-6) **causes horrific damage!**

Oxidized Lipids Alter Membrane Integrity

Ingestion of *adulterated* Parent omega-6 causes grave physiologic issues as demonstrated in the journal article, "Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity:"[10]

- "…This [increased membrane fluidity] was due to profound differences in lipid composition of the liver…
- "The study ***demonstrated that ingestion of oxidized lipids caused profound alterations in membrane composition, fluidity and function….***

▶ "The main differences observed in the composition of the microsomal **phospholipid** fatty acyl residues between the two groups were a ***23.2% lower linoleic acid [Parent omega-6] level*** and a 14.2% greater arachidonic acid level in the rats fed the *oxidized linoleic* acid [Parent omega-6] diet."

PEO Solution analysis: It is all confirmed. The *adulterated* Parent omega-6 *replaces* the fully functional Parent omega-6 in phospholipids and we have a physiologic disaster.

---

10 Hochgraf, Edna, et al., "Dietary Oxidized Linoleic Acid Modifies Lipid Composition of Rat Liver Microsomes and Increases Their Fluidity," *J. Nutr.*127: 681–686, 1997.

Both adulterated Parent omega-6 (from food processing) and overdoses of fish oil (EPA/DHA) cause profound physiologic problems for patients.

You Need to Know This... Markers of Lipid Oxidation—Malondialdehyde is tops...

Malondialdehyde is an *in vivo* marker of oxidative stress (lipid peroxidation/rancidity). Reactive oxygen species degrade polyunsaturated lipids, forming malondialdehyde (MDA). This results in advanced lipoxidation end-products (ALE), analogous to advanced glycation end-products (AGE), so harmful to diabetics.

There is much to know before relying on specific lipid oxidation markers. The excellent medical journal article, "Lipid Peroxidation and the Thiobarbituric [TBA] Acid Assay: Standardization of the Assay When Using Saturated and Unsaturated Fatty Acids," makes clear:[11]

- "Indeed supplementation with ***polyunsaturated*** [in particular, EPA/DHA], as opposed to saturated fatty acids results in **a statistically significant increase in lipid peroxidation in the plasma and liver**.

11 Rael, Leonard, T., et al., "Lipid Peroxidation and the Thiobarbituric Acid Assay: Standardization of the Assay When Using Saturated and Unsaturated Fatty Acids," *Journal of Biochemistry and Molecular Biology*, Vol. 37, No. 6, November **2004**, pages 749–752.

- "**Oxidative damage to DNA** in bone marrow was recorded in aged, but not young rats when a **polyunsaturated** diet was employed.

- "A plateau was reached where no additional decrease in the TBA-reactive species (TBARS) was observed regardless of increases in fatty acid concentration. This limitation to the TBA assay suggests the need for a standardized assay....

▶ "The TBA Assay is not specific for ***malondialdehyde*** (MDA), one of the many breakdown products of degraded fatty acids. [The test is separately required.]

- "**Caution is advocated for the interpretation** of in vitro [outside the body] lipid peroxidation ***using only the TBA assay*** without standardization..."

PEO Solution analysis: Be wary of those telling you there is "no issue" with EPA/DHA oxidizing in the body. Peroxidation level based on TBA will stop (reach an upper limit) prematurely before recording full damage—an inherent limitation of the particular test. Red blood cell oxidation levels are not a sufficient indicator—the actual tissue is what counts. Furthermore, researchers often don't understand the limitations of measurement. Short-term damage may not present but long-term damage will, i.e., DNA destruction.

Malondialdehyde measurement should be performed independently for increased accuracy of the damage assessment. Organ damage does occur, as shown decisively in the monkey

experiment (chapter 7). It is "case closed." *Lipids*, one of the world's top medical journals in the field, makes clear how fish oil raises MDA. The article, "Malondialdehyde excretion by subjects consuming cod liver oil vs a concentrate of n-3 fatty acids," states:[12]

- "Urinary malondialdehyde (MDA), an indicator of lipid peroxidation in the diet and in the tissues, was ***determined in human adults*** consuming a supplement of n-3 fatty acids derived from a pharmaceutical grade of **cod liver oil** (CLO) without added antioxidants vs a concentrate of n-3 acids containing dodecyl gallate and vitamin E. **MDA excretion increased immediately in the subjects consuming CLO but remained unchanged in those ingesting the concentrate for 50 days. The increase in the subjects taking CLO** was attributable to MDA in the oil. The results indicate that consuming unstabilized fish oils as a source of n-3 fatty acids may entail exposure to **potentially toxic products of lipid peroxidation**.

PEO Solution analysis: No amount of lipid "antioxidant/stabilization" outside of the body (in the container) can protect the supra-physiologic amounts of EPA/DHA from rancidity inside the body (*in vivo*). This was conclusively shown in the peroxidized monkey livers.

The following article gave me great pause —"Margarines fortified with μ-linolenic acid, eicosapentaenoic acid [EPA], or

12 Piche, LA, et al., *Lipids*, Volume 23, Number 4, 1988, pages 370–271

docosahexaenoic acid [DHA] alter the fatty acid composition of the erythrocytes but do not affect the antioxidant status of healthy adults"[13] —first because the study was conducted back in **2004**! Why is this just being published now, eight years later in **2012**? The study of 48 women and 26 men doesn't answer this. Their published results don't even make sense. It is well-known EPA/DHA at room temperature spontaneously turn rancid. Therefore, the problem is much worse at body temperature close to 100° F. They gave huge doses of ALA (parent omega-3) (4.4 grams/day), EPA (2.2 grams/day), and DHA (2.3 grams/day). Not surprisingly, RBC incorporation of these overdoses was significant: ALA incorporation increased by 104%, EPA incorporation increased by 394%, and DHA incorporation by 91% from normal. To support these increases, natural Parent omega-6 and AA (an important anti-CVD omega-6 derivative) significantly decreased.

Furthermore, it appears from their description that the women in the study were all taking birth control pills.

Participants were given (Unilever) **margarine, loaded with *adulterated* Parent omega-6**. If it wasn't unprocessed/organic, then it was necessarily adulterated; there was no exclusion by the researchers.

Researchers measured red blood fatty acids levels and plasma levels. They did not measure tissue/organ peroxidation/lipofuscin levels. ***The study ran just 6 weeks, much too short to establish long-term damage***, as the journal article above describing long-term DNA damage with fish oil makes clear.

---

13 Egert, Sarah, et al., *Journal of Nutrition*, 142: 1638-1644, 2012.

The article leads with the familiar "we don't know how it works but it does" disclaimer:

> "The exact mechanisms through which (n-3) PUFA influence CVD are not well established...." [Note: "magical" metabolic pathways are not admissible.]
>
> "**The plasma concentrations of lipid peroxidation product MDA significantly increased with the EPA and DHA intervention** compared with the ALA [parent omega-3] intervention. [Note: This is to be expected based on known physiology.]

"Malondialdehyde Excretion by Subjects Consuming Cod Liver Oil vs a Concentrate of n-3 Fatty Acids

The journal article, "Malondialdehyde Excretion by Subjects Consuming Cod Liver Oil vs a Concentrate of n-3 Fatty Acids," found the following:[14]

- "In Experiment 1, ingestion of CLO [cod liver oil] was associated with an increase in **MDA excretion in all six subjects. The mean increase of 37.5%**, from 24.5 ± 3.5 ug to 34.7 ± 2.5 ug MDA (mean + SEM), was significant ($P < 0.01$) using a two-tailed paired t-test. [Note: This is "highly" significant.]
- "In Experiment 2, CLO ingestion again was associated with an increase in **MDA excretion in all subjects**. **The mean increase of 54.3%**, from 31.7 **µg** to 49.1 **µg** MDA/ sample was highly significant ($P < 0.001$).

---

14 Piche, LA, et al., *Lipids 23,* 370–371 (1988)

PEO Solution analysis: Of course, there is verification of lipid oxidation, because there has to be evidence. The researchers TRIED to say when using "stabilized" fish oil in another group of 6 patients, the oxidation as measured by urinary MDA was minimized. However, using Stat-Smart® analysis, we immediately found that ***the result was NOT statistically significant*** —there was more than a 5% error rate! They put this "little detail" in a footnote, but we won't be fooled.

Holman confirmed in 1954 that fish oil oxidizes effortlessly:

- "Docosahexaenoic acid (22:6), which has six double bonds and consequently five bis-allylic hydrogens per chain, is 320-times more susceptible to ROS attack than the common monounsaturated oleic acid (18:1) which has 'no' bis-allylic hydrogens in its chain."[15]

Oils are Damaged when Heated for Cooking

When assessing oxidative damage to lipids, at least two different chemical methods should be utilized. The investigator must understand each test's uses and limitations. For example, hydroperoxides do not accumulate at frying temperatures; instead, they decompose spontaneously, but their damage is significant.

Adulteration of oils both contained in the food and used in its preparation causes damage to the food; in the case of frying,

15 Holman, RT, "Autoxidation of fats and related substances," *Progress in Chemistry of Fats and Other Lipids,* edited by Holman RT, Lundberg WO, Malkin T. London: Pergamon, 1954, vol., 2, p. 51–98.

the food also absorbs oil—potentially creating additional harm to the consumer of the food.

Dr. Dobargenes' warning of the oil in fry vats exceeding the limit for polar compounds is warranted (**2003**):[16]

- "Industrial continuous fryers had polar compounds ranging from 4.2% **- 27.3%** and batch fryers found in **fast food fryers and restaurants** had 3.1 **- 61.4%!**
- "**Restaurant and fast-food outlets oil quality data are in many cases short of complying** with the recommendations outlined at a recent international conference held in Germany (**2000**) where the maximal concentrations of total polar compounds and polymer content were 24% and 12%, respectively. Data on oil quality in batch fryers in some European countries stress the fact that this issue is far from acceptable.

▶ A recent study conducted in Europe indicated that even when **TFA** concentration is less than 1% in non-hydrogenated oil, its level in frying oils **could reach up to 50%** due to partially hydrogenated oil. Only Denmark has regulation on maximum allowed TFA (15%) in unused frying oil.

---

16 Saguy, I. S. and Dana, D., "Integrated approach to deep fat frying: engineering, nutrition, health and consumer aspects," *Journal of Food Engineering*, Vol. 56, **2003**, pages 143–152.

- "Repeat use of frying oils may increase TFA concentration due to the exchange of fatty acids between the fried food and the oil as well as the high temperature and prolonged frying process. **For instance, an increased concentration of trans isomers of C18:1 [as in olive oil] was found in various vegetable oils used for beef frying.** Repeated **frying in sunflower** oil also resulted in an increased concentration of the TFA isomer C18:2.

- "A positive correlation was found with **thiobarbituric acid** reactive substances (**TBARS**), formed ***from fatty acids with three or more double bonds***. This fraction **contains MDA**, which was **found to be mutagenic in many other studies. Prolonged frying caused a substantial rise in MDA concentration**. These findings need further study, as MDA is not typically monitored during frying.

► "**MDA was found to cause skin cancer in rats and created cross-linking with amino groups of DNA solution**. The Ames test indicated that **MDA is a mutagen causing DNA alterations** and reacting mainly with guanine and cytidine by a depletion of this base pair. **MDA can damage proteins and phospholipids by covalent bonding and cross-linking**. ***Rats fed a diet containing MDA suffered from retarded growth, irregular intestinal activities, enlarged liver and kidneys, anaemia and low serum and liver vitamin E.***

- "Since a **considerable amount of oil is absorbed in the product during frying (10–40%), frying in oil that contains mutagens could lead to consumption of foods containing hazardous compounds**. Usually the concentration of mutagens in the oil is low, but **high oil uptake may have health implications**. It is also possible that higher concentration could be absorbed in the food due to preferential uptake. It is worth noting that the concentrations used in the aforementioned experiments on rats are twice as high as the average human consumption of MDA.

- "Low mutagenesis was found in oils exposed to severe frying conditions––*uncommon in typical industry or household operations*, and no mutagenic activity was found in oil used for repeated frying of potatoes, onion rings or fish fillets. Thus it can be concluded that under controlled conditions, the level of exposure to mutagenic compounds formed during frying should not comprise a real health hazard. Nevertheless, this topic needs further study before a final conclusion can be made."

PEO Solution analysis: Don't be confident that French fries or other fried foods have low or no mutagenic activity as reported here. That comment makes little sense and is inconsistent with the established science and inconsistent with numerous other experiments (as the researchers were forced to admit.).

It's confirmed that consuming oxidized cholesterol-containing foods leads to oxidized cholesterol in plasma:[17]

- "In studies in humans, we have shown that the **quantity of oxidized fatty acids in the diet also correlates with the levels of oxidized lipids in postprandial serum chylomicrons / chylomicron remnants** (CM/RM). Oxidized fatty acids in the diet are absorbed by the small intestine, incorporated into chylomicrons and chylomicron remnants, and appear in the bloodstream where they **contribute to the total body pool of oxidized lipid**

- "In our initial studies, we determined the effect of different α-epoxy cholesterol quantities in the test meal on α-epoxy cholesterol levels in postprandial serum. **We found that the serum levels of a-epoxy cholesterol strongly correlated**. [Note: It was nearly a perfect correlation of r=1.]

- "Thus, our **data clearly show that oxidized cholesterol, when ingested, is incorporated into CM/RM fraction and is transferred within the plasma compartment from exogenous to endogenous lipoproteins**, and this transfer accounts at least partially for the presence of oxidized cholesterol in LDL and HDL in the circulation. It has been previously demonstrated in rabbits and in

17 Staprans, I., et al., "Oxidized cholesterol in the diet is a source of oxidized lipoproteins in human serum," *Journal of Lipid Research*, Vol. 44, **2003**, pages 705–715.

rodents that dietary oxidized cholesterol is absorbed and enters the circulation via CM/RM particles. As expected, in the present study **we found that the absorption of dietary oxidized cholesterol in humans is similar to that observed in other species**."

PEO Solution analysis: Just as with EFAs, absorption and integration of oxidized cholesterol is the same in humans as in other species.

Broiling buffalo meat increased oxidative rancidity by approximately 3-fold:[18]

- "Oxidative rancidity of lipids is a serious problem during storage of meat and meat products and ***TBA (2-thiobarbituric acid) value is the most commonly used parameter to measure it***.
- "There was a **significant increase of approximately 3-fold in TBA from broiling**."

PEO Solution analysis: Malonaldehyde is a highly reactive three carbon dialdehyde produced as a byproduct of polyunsaturated fatty acid peroxidation and arachidonic acid metabolism. TBA is used to measure it.

---

18 Rao, V.R., et al., "Effect of Cooking and Storage on Lipid Oxidation and Development of Cholesterol Oxidation Products in Water Buffalo Meat," *Meat Science*, Vol. 43, No. 2, 1996, pages 179–185.

Fish oil is problematic… Cooking is not a necessary condition…

The **2000** article titled, "Supplementation of postmenopausal women with fish oil rich in eicosapentaenoic acid and docosahexaenoic acid is not associated with greater in vivo lipid peroxidation compared with oils rich in oleate and linoleate as assessed by plasma malondialdehyde and F2-isoprostanes," has buried on page 7:[19]

- "Whether normalized to **plasma** volume or plasma PUFA concentration, ***plasma TBARS were significantly higher after fish-oil supplementation*** than after sunflower-oil or safflower-oil supplementation [Parent omega-6 oils].

► "After **fish-oil supplementation**, plasma **TBARS were > 21% higher** than after sunflower-oil supplementation and **23% higher** than after safflower-oil supplementation. [Note: with non-organic sunflower and safflower oils, those oils are adulterated to begin with.]

- "The fact that the **TBARS** concentrations in plasma were nearly ***10 times higher*** than the **MDA** concentrations is likely due to the **lack of specificity of the TBA assay for MDA** and to artifactual production of MDA during the acid heating step of the TBA assay.

- "Many of the **assays** available for the measurement of **lipid peroxidation in vivo lose their utility when**

---

19 Higdon, Jane V., et al., *Am J Clin Nutr* 2000;72:714–22.

specific PUFA concentrations in plasma vary as a result of changes in dietary intake. Instead of measuring overall lipid peroxidation, different assays measure the oxidation or decomposition of specific PUFAs.

- "The utility of the **F2-isoprostane** assay for comparing in vivo lipid peroxidation at different intakes of specific unsaturated fatty acids is **limited** because it ***does not provide direct information about the peroxidation of 20:5n-3 [EPA] and 22:6n-3 [DHA***]."

PEO Solution analysis: The bottom line is that EPA/DHA is damaged. Consequently, patients should avoid this obvious risk.

Dr. Rowan:

The Toxic Impact of Heat on Foods—Pottenger's Cats…

- Pottenger was studying the adrenal gland and cortisol using cats
- He was donated lots of unwanted cats.
- Overflowing with cats, he resorted to different feeds.
- He noticed that certain groups were healthier than others, so he decided to do a study on nutrition in the cats

Pottenger's Study:

- The study involved 900 cats

- 600 were studied for their entire life span.
- All cats were placed in identical physical enclosures.
- The variable was their diets

His Groups:

1. Raw meat group: 2/3 raw meat, 1/3 raw milk, and cod liver oil

2. Cooked meat group: 2/3 cooked meat, 1/3 raw milk, and cod liver oil

``````````````````````````````````

Raw meat group observed over **three** generations.

1. Maintenance of a regular, broad face with prominent malar (pertaining to the cheek or cheek bone) and orbital arches, adequate nasal cavities, broad dental arches, and regular dentition.

2. The configuration of the female skull is different from the male skull, and each sex maintains his/her distinct anatomical features.

3. The membranes are firm and of good, pink color with no evidence of infection or degenerative change. Tissue tone is excellent, and the fur is of good quality with very little shedding noted.

4. In the older cats, particularly the males, engaging in fighting, the incisors are often missing, but inflammation and disease of the gums is seldom seen.

5. The calcium and phosphorus content of their femurs remains consistent.

6. Their internal organs show full development and normal function.
``````````````````````````````````

7. Over their life spans, they prove resistant to infections, to fleas, and to various other parasites, and show no signs of allergies. In general, they are gregarious, friendly, and predictable in their behavior patterns..

8. These cats reproduce one homogeneous generation after another with the average weight of the kittens at birth being 119 grams.

9. Miscarriages are rare, and the litters average five kittens with the mother cat nursing her young without difficulty

Now the results in the cooked meat group over three generations:

1. This group reproduces a reproduce a ***heterogeneous*** strain of kittens, each kitten in a litter being different in size and skeletal pattern.

2. When comparing the changes in configuration found in their x-rays, there are almost as many variations in the facial and dental structures of the second and third generation cooked-meat fed animals as there are animals.

3. **Evidence of deficiencies is written so plainly** on their faces that with a little training, any observer can be almost certain that a given cat has been subjected to a deficient diet or that it comes from a line of cats that has suffered from deficient nutrition.

4. The long bones of cooked-meat cats tend to increase in length and decrease in diameter with the hind legs commonly increasing in length over the forelegs. The trabeculation (the internal structural mesh of the bones) becomes coarser and shows evidence of less calcium.

5. In the third generation, some of the bones become as soft as rubber, and a true condition of osteogenesis imperfecta (the inherited

condition in which bones are abnormally brittle and subject to fractures) is present.

6. Heart problems; nearsightedness and farsightedness;

7. Under activity of the thyroid or inflammation of the thyroid gland;

8. Infections: of the kidney, of the liver, of the testes, of the ovaries, and of the bladder

9. Arthritis and inflammation: of the joints; inflammation of the nervous system with paralysis and meningitis—all occur commonly in these animals. A decrease in visceral volume is evidenced by the diminishing size of their thoracic and abdominal cavities.

10. Microscopic sections of organs show poor histological development of organs.

11. Behavioral anomalies, females overly aggressive, males docile.

12. Sexual deviations in same sexed animals.

13. Reproductive organ pathology – ovarian, uterine and sperm production anomalies.

14. Abortion in females rampant running 25% in the first generation to as high as 70% in the second.

15. Pests and intestinal parasite abound.

16. Skin lesions, allergies abound.

17. Pneumonia, empyema (abscess within chest space) and diarrhea are common causes of death.

18. High perinatal mortality of both mothers and kittens.

19. Kittens of this group average 19 grams less than raw meat nurtured kittens.

Pottenger went on to study if the cats could regenerate or recover their health and found:

1 . It requires approximately four generations for the deficient cats to regenerate to a state of normal health after healthy diet reinstituted.

2. However, because of the lack of reproductive efficiency, very few deficient animals regain the normal health noted before deficiency was imposed on their line of cats.

3. Improvement in resistance to disease is noted in the second generation regenerating cat, but allergic manifestations persist into the third generation.

4. In the third generation, skeletal and soft tissue changes are still noticeable, but to a lesser degree; and by the fourth, most of the severe deficiency signs and symptoms disappear—but seldom completely.

A startling discovery:

1. A female cat is subjected to a deficient diet for a period of 12 to 18 months, her reproductive efficiency is so reduced that she is never again able to give birth to normal kittens.

2. Even after three or four years of eating an optimum diet, her kittens still show signs of deficiency in skeletal and dental development. When her kittens are maintained on an optimum diet, a gradual reversal and regeneration takes place.

Pottenger Studied the effects of raw vs. pasteurized, vs. evaporated mild added to diet (2/3 of diet) with similar results.

His findings almost exactly mirror what we are seeing now in younger human populations. It also mirrors the horrific rise in diseases in our younger human generations, autism collapse in sperm counts, immune dysfunction, etc.

Journal of the American Medical Association published a startling discovery in 1981.[20] Rats made deficient in zinc gave birth to offspring with immune defects. Even when zinc was restored to their diet, it took **four** generations for them to regain normal immune function! I first learned about this about 9 years later and it shook me regarding nutrition!

This exactly mirrors modern experiments on epigenetics, using nutritional deficiencies and environmental xenobiotics. Epigenetics is the science of DNA or gene expression. Your DNA might be perfectly intact. The problem is, dietary deficiencies, toxins, or even stress might compromise its expression. See, your DNA is like your computer's hard drive. You control what the hard drive does through your keyboard commands. Imagine missing a key (nutritional deficiency) or a heavy brick (toxin) loading one or more keys. Your hard drive, perfectly intact otherwise, is not going to perform!

We are finding that nutritional or toxic damage to animals induces alteration in DNA EXPRESSION, not the genetic sequence. This has profound implications for the offspring and it takes about 4 generations to recover.

---

20 *JAMA*. 1981;245(1):53-58.

There is a Biblical passage about this: 'punishing the children for the sin of the fathers to the third and fourth generation of those who hate me' (Exodus 20:5). Seems the Bible (I am NOT a Bible thumper) accurately predicted epigenetic effects and their lasting effects over 3,000 years ago!

We are in the throes of:

1. Autism epidemics

2. Immune defects epidemics

3. Cancer epidemics

4. Allergy epidemics

5. Suicide and violent behavior epidemics.

6. Chronic disease epidemics.

7. Infertility epidemics.

COULD OUR DIET (or toxins) BE A KEY FACTOR? ***Could heated foods be a major culprit?***

Let's turn to modern science.

IA recent article[21] on thermolyzed (heated) foods took up the subject. The authors even outlined the reasons for the study:

1. Diets are usually evaluated for calories, macro and micronutrients.

---

21 "A Thermolyzed Diet Increases Oxidative Stress, Plasma alpha aldehydes, and colonic inflammation in the Rat," *Experimental Diabetes & Aging, Available online 8 June 2007, and Chem Bio Interact*, Vol. 169, No. 2), pages 100-109, 2007.

2. **Rarely are the effects of heat considered**.

3. Thermolysis of foods, however, can result in the formation of new products such as advanced glycation end-products (AGE).

4.A heterogeneous group of compounds that are formed by a complex series of parallel and sequential reactions called the ***Maillard reactions.*** (Nonenzymatic browning resulting from a chemical reaction between an amino acid and a reducing sugar from heat).

Maillard reactions are chemical alterations to molecules in food. They impart the unique taste and smell of cooked foods. However, they create compounds not found in nature, such as acrylamides, now known to be carcinogens. You'll find these reactions and molecules in the browning of various meats like steak, toasted bread, biscuits, malted barley or spirits. Also, in fried onions or any fried food, dried or condensed milk, roasted coffee, and the burnished surface/crust of many heated foods.

Maillard reactions are accelerated by the following factors:

1. High temperature, intermediate moisture levels, and alkaline conditions all promote the Maillard reaction.

2. In cooking, low moisture levels are necessary mainly because water boils into steam at 100 °C (212 °F), whereas the Maillard reaction happens noticeably around 154 °C (309 °F): significant browning of food does not occur until all surface water is vaporized.

Hence, a limiting factor is the temperature. Far less of these reactions occur with cooking food in water or steaming.

For the science buffs, a 2013 article[22] is posted at http://cen.acs.org/articles/90/i40/Maillard-Reaction-Turns-100.html.

Here were the parameters for the heated foods diet study:

- 344 rats
- Fed normal diet for one week, then split into two groups, one a control diet, and the second group got the same diet which was thermolyzed (heated).
- Thermolyzed diet was chow prepared with a relatively short exposure of 122º C (252ºF) for only 30 minutes in an atmosphere devoid of oxygen. Hence, oxygen could not be the culprit in findings, only heat!

Here are the key findings:

1. Experimental group became thiamine deficient measured by transketolase.

2. Experimental group had increased oxidative stress measured by reduced GSH.

3. Experimental group had increased markers of Maillard reactions measured by the presence of α – oxoaldehydes (glyoxals and methyl glyoxals).

4. The protein adducts of these carbonyls and protein oxidation levels were increased.

---

22 *Chemical and Engineering News*, Volume 90, Issue 40, pp. 58-60, 2013.

5. Experimental diet also increased oxidative stress biomarkers in livers and colons.

6. Experimental diet increased macrophage infiltration into colons (fourfold).

Here are some of the "baaaad" biochemical effects found:

1. Experimental group became *thiamine deficient* measured by transketolase

2. Experimental group had *increased oxidative stress* measured by reduced GSH.

3. Experimental group *had increased markers of Maillard reactions* measured by the presence of α – oxoaldehydes (glyoxals and methyl glyoxals).

4. The protein adducts of these carbonyls and protein oxidation levels were increased.

5. Experimental diet also ***increased oxidative stress biom***arkers in livers and colons

6. Experimental diet increased macrophage infiltration into colons (fourfold).

Alteration	Causative Agent	New Compounds
Hydrolysis	Moisture	Free Fatty acids / Diacylglycerols
Oxidation	Air	• Oxidized monomeric Triacylglycerols • Oxidized dimeric and oligomeric triacylglycerols
Thermal alteration	Heat	• Volatile compounds (aldehydes, ketones, alcohols, hydrocarbons, etc.) • Cyclic monomeric triacylglycerols • Isomeric monomeric triacylglycerols • Nonpolar dimeric /oligomeric triacylglycerols

The methylglyoxal A generated by the Maillard reactions is not a good thing for you. An international study including our own NIH [23] found that undesirable free radicals are generated during the glycation reaction of amino acids with this compound.

So, now, what about fats?

All fats in nature that you eat are in the form of triglycerides. That means three fatty acids are joined by an "ester" molecular linkage to a glycerol molecule "backbone". You've read how very important natural fats, in their original unadulterated states are. I'll bet you don't know what happens when you heat ("cook") fats. Now, I don't expect you to understand all the biochemical terms unless you are technically inclined. But consider if you'd like all these ***oxidized fats, aldehydes and volatile compounds*** to enter your precious body.

Heat breaks the bonds of the natural triglycerides making toxic free fatty acids. ***Circulating NEFA*** **[non esterified fatty acids or free fatty acids]** ***concentration is an independent risk factor for sudden death in middle-aged men. Some form of primary prevention could be envisaged in subjects at high risk of sudden death.***[24]

Obviously the prevention here could include not **overly** heating fats. Heat in the presence of oxygen not only breaks the glycerol ester bonds (releasing the free fatty acids) but also further damages the fats to these other non-naturally occurring compounds that can be dangerous to you as this chapter details.

Researchers have also found that a diet rich in raw vegetables lowers your risk of breast cancer, and eating lots of fruit reduces your risk for

---

23 *J. Biol. Chem.* 1995, 270:28228-28233.

24 *Circulation* 2001 Aug 14;104(7):756-61

colon cancer, according to a study published in the May 1998 issue of the journal *Epidemiology*. Including fresh fruit as part of your daily diet has been associated with fewer deaths from heart attacks and related problems, by as much as 24% (of course, as you have learned, this is a "relative risk"), according to a study published in the September 1996 issue of the *British Medical Journal*.

A few relevant articles you may be interested in showing the impediment of heating to digestion are:

> "**Effect of meat cooking on physicochemical state and in vitro digestibility of myofibrillar proteins**." [http://www.ncbi.nlm.nih.gov/pubmed/18237130.]
>
> "The Effect of Cooking on the digestibility of meat." [http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1254788/pdf/biochemj01123-0140.pdf]

There 's more from Dr. Rowen …..

For the technically inclined: These reactions take place in the unsaturated fatty acyl groups attached to the glyceridic backbone and, therefore, the stable final products are triglyceride monomers, dimers and oligomers containing modified and non-modified acyl groups. The concurrent formation of positional and geometrical isomers through auto-oxidation gives rise to ***thousands*** of individual new compounds and explains the poor information on the original structures. Studies on the mechanisms for their formation as well as for their structural analysis have been carried out after formation of simpler derivatives, i.e. fatty acid methyl esters. Formation of other groups of compounds present in used frying oils without extra oxygen, i.e.,

Diels-Alder dimers, cyclic fatty acids and positional and geometrical fatty acids are *explained by thermal reactions and not by radical interaction.* They are minor compounds of nutritional interest and paradoxically are much better known than the major oxidation compounds because of their stability and low polarity.

Remember, oxidized and otherwise damaged fats are the fundamental cause of vascular disease. Folks, **we are simply dumping in a horrible mishmash of molecules not found in nature, not native to foods, not metabolizable by the body, which could give rise to most any toxic bodily reaction, especially damaging to cell membranes**. I haven't covered trans fatty acids. You've heard a lot about them, like in margarine. But what you don't know is that the high heat in processing and clarifying cooking oil converts a lot of the native 'cis' fatty acids into 'trans.'

Now consider this. Most oils used for cooking are valued for their "smoke" points. That's the temperature at which the oil will visibly burn. Most vegetable oils are in the 215-265º C range. That's an awfully high temperature. Water boils at 100º C (212º F). Olive oil is a bit lower at 190º C. Even if your oil doesn't smoke, the higher ranges will induce conversion of cis bonds to damaging trans.

The ability of oil to withstand high temperatures before smoking is an additional reason fried foods are the worst things you can eat. Aside from the damaged oils, the heat wipes out whatever nutritional value might have been there.

If you don't believe me, do this simple experiment. Take any unsaturated oil and smear it on the inside of a pan. Heat it for a period of time at moderate heat. The longer you do this, the less "oily" the oil remains and the tackier (sticky) it becomes. This is due to molecular damage to the oils and cross-linking almost making the oil into a plastic. This is what is happening in your body in slow motion.

But now consider the impact of heat on cholesterol oxidative damage in your food! (WE know that cholesterol isn't the bad guy. **It's *oxidized* cholesterol that is**. Oxidized cholesterol is taken up in your vascular endothelial cells and seen by your immune system as "foreign". An immune attack is launched against these foreign molecules and your own tissues take a huge hit).

So what does heat do to cholesterol? In fish cooked in vegetable oil, the sum of cholesterol oxidation products (COPs) increased after the heating processes from ***0.9 microg/g in the raw sample*** to 6.0, 4.0, 4.4, 3.3, and 9.9 microg/g extracted fat in pan-fried without oil, with olive oil, corn oil, partially hydrogenated plant oil, and steamed, respectively. A highly significant correlation was found between the fatty acid pattern and the total amount of COPs ($r^2 = 0.973$, $p < 0.001$). ***(300%-900% increase in COPs).***[25] **Up to nearly a 10-fold increase in oxidized products!**

Uncooked fresh butter had virtually NO COPs. So the **fears about the saturated fats in butter is hogwash**. Furthermore, as Prof. Peskin made clear, there is NO SATURATED FAT in an arterial occlusion.

25 *J Agric Food Chem* 2004 Aug 11;52(16):5290-6.

However, if you heat butter to 170-180 C (300^0 F), you'll start seeing COPs. Raw bacon rind (NEVER EAT IT) has no COPs, but cooked at routine frying temperatures of 170º C for 10 minutes and they form. (I'd rather have some COPs than trichinosis).

Routine cooking definitely increases COPs. Using an electric skillet at just 135º C, the COPs of cooked beef, veal, and pork increased by 1.7, 2.3 and 2.5 fold respectively. When cooked in an oven at 220º C for 60, 80. And 90 minutes respectively, the COPs levels increased by 3.5, 5.4 and 4.2 fold respectively.[26] Freshly prepared meat products are a minor source of COPs overall, but semi-prepared frozen meat products fried once and stored can increase COPs considerably.[27]

Many people look at fish as a healthier food than mammal meat. But here is a report[28] that even fish can deliver you COPs if heated. The authors aimed to look at routine fish preparation, as you might do in your own kitchen. **Strangely enough, the highest amount of COPs was found in steamed fish over pan-fried fish**. The authors felt that was due to the "longer heat exposure." The authors concluded that even salmon, touted for its heart sparing effects could provide COPs as a potential toxicological health risk.

Contrary to popular belief that the highly unsaturated fatty acids improve membrane fluidity, the opposite may be occurring. Yes, these fatty acids have a lower melting point, hence are more fluid—perhaps TOO FLUID—another reason that Nature doesn't incorporate much of

---

26 *Journal of Food Science*, Vol. 66, No. 9, **2001**.

27 *JAOCS*, Col 77, no. 6 (**2000**).

28 *J. Agric. Food Chem*. **2004**, 52, 5290–5296.

them, but it is even worse… **Over time, they become peroxidized far more easily**. And, these lipid oxidation products strongly contribute to membrane rigidity. These products are likely dominant factors in **creating age-related membrane rigidity**.

A funny named animal, the naked mole rat, has the **highest longevity of any rodent** – up to 30 years. And, it has the **lowest amount of DHA in its membranes** of the rodent species. This makes their membranes far more resistant to peroxidation, and more similar to the longer-lived mammals.

Similar findings in insects… Queen bees are fed mouth to mouth by the workers, which workers consume pollen for their own food. Pollen is rich in polyunsaturates. Queen bee food is not loaded with polyunsaturates, which might be a major difference in the longevity of the queen (years) to her brood (weeks).

This is now seen in human research. Children born to nonagenarians have red cell membranes that are more resistant to peroxidation damage! So, **we see that membrane composition of susceptible fatty acids is associated with longevity cross species lines, from bees, to rodents, birds, and even humans**.

Weston Price, DDS…

One of the greatest heroes of nutrition and health was Weston Price. A dentist, he traveled the world to aboriginal cultures to study how they ate, and how what they ate impacted their health, from their teeth to the rest of their bodies. You can't separate the health of one part of your body from another. In today's world *specialists* will tell you that you have 2 kidneys, a liver, a heart, teeth, etc,. and what is going on with these organs is seaparate and distinct from each other.

It was easy for Price to see that if you had dental caries, and not enough bone growth in your jaw to accommodate all your teeth, that you'd have other nutritional problems as well. His observations were most profound, yet quite simple and LOGICAL! Yet his name was not mentioned once in my prestigious medical school. I didn't learn of him, nor Francis Pottenger for at least 10 years after graduating. When you read the following, graciously posted on the Price Pottenger Foundation website, I think you'll be dismayed that those who rule this country have also ignored these maxims, to your detriment. What you read here is the foundation of why the "health" of the American people has fallen into disarray and plunder by those who are violating these maxims at every turn to farm (pharm) you for profit.

http://ppnf.org/about/about-price-and-pottenger/dr-pottenger/traditional-diets/.

THE FOODS THAT WERE EATEN

Dr. Price's research shows that healthy traditional diets:

- Contain foods naturally rich in body-building nutrients
- Include minerals and fat-soluble vitamins found in butter, sea foods, fish oils and fatty animal organs
- Incorporate raw, unaltered proteins from meats, sea foods, nuts, raw dairy and sprouted seeds
- Use sweeteners rarely and sparingly

Diets based on these guidelines provide optimal nutrition for preventing disease and slowing physical degeneration.

How the foods were cultivated: In a healthy traditional diet, *how* foods are grown is as important as *what* foods are eaten. In a healthy traditional diet, foods are grown:

In natural, mineral-rich soil

Using NO chemical fertilizers

Using NO chemical pesticides

Growing foods in this way prevents the introduction of harmful chemicals into our food chain and our bodies.

How the foods are preserved and prepared. Food preservation and preparation methods are also important components in the healthy traditional diet. In such diets, foods are:

- Eaten in season
- Preserved using methods of—earth storage, drying, freezing, culturing and pickling—that maintain or enhance the nutritional content of the food
- Eaten "whole" and unrefined, maintaining fiber and nutrient content
- Consumed raw or very gently and lightly cooked

Preparing and preserving foods using traditional methods ensures that we benefit from their full nutritional value.

Lifestyle Choices: In his studies, Dr. Price noted that healthy diets are enhanced by healthy lifestyle choices. The healthy tribes studied by Dr. Price:

- Engaged in regular, vigorous exercise through work, play, dance, and sports
- Had access to pure air and enjoyed abundant sunlight
- Observed periods of partial abstinence from food (fasting), or regulated periods of under-eating
- Ate special protective foods in preparation for conception, pregnancy and lactation
- Spaced pregnancies apart to protect the health of the mother and the children
- Breast-fed their babies
- Instructed their children in the importance of maintaining traditional dietary and lifestyle principles

---

These maxims are among the most logical and reasonable rules to live by that have ever been revealed. They are as basic to health as the Golden Rule of "do unto others as you would have done unto you." Is basic to our relations with others. Yet, they are totally ignored in a profit and greed driven world.

I say to the ignorant and arrogant political and disease maintenance establishment, "WAKE UP!" Stop taking payouts from those poisoning the planet to stay in office while not only our families but also your families pay the ultimate price of untimely physical degeneration.

More from Professor Peskin:

2008 Newsflash: Just like fruits fulfill our natural "sweet tooth," fats fulfill our appetite. Fats—not protein or carbohydrates.[29] Take 6 egg whites and cook them with no butter. You'll be starving just 15 minutes after eating them. Compare this to adding 2 yolks. You'll be full and contented. A landmark experiment — it's only **FATs — NOT carbohydrates or proteins** which send signals to the brain saying you aren't hungry. Here's what was published in **2008**, if anyone would care to research it:

- "Here, we report that ***duodenal infusion of fat stimulates*** oleoylethanolamide (OEA) mobilization in the proximal small intestine, whereas infusion of ***protein or carbohydrate does not***
- …[T]his lipid messenger ***participates in the induction of satiety***
- …[T]he **rapid onset of the OEA response (<30 minutes)**…
- …**[P]rolonging the time interval between meals**.
- OEA production utilizes dietary oleic acid as a substrate and is disrupted in mutant mice lacking the membrane fatty-acid transporter *CD36*. Targeted disruption of *CD36* or *PPAR*-α **abrogates [ends] the satiety response induced by fat**

---

29 Schwartz, GJ, et al., "The Lipid Messenger OEA Links Dietary Fat Intake to Satiety," *Cell Metabolism*, Vol. 8, Issue 4, Oct 8, **2008**, pages 281-288.

- The results suggest that activation of small-intestinal OEA mobilization, enabled by *CD36*-mediated uptake of dietary oleic acid, **serves as a molecular sensor linking fat ingestion to satiety**.
- In conclusion, our studies identify **OEA as a key physiological signal** that specifically **links dietary fat ingestion to across-meal satiety**.

PEO Solution analysis: Once again, the truth gets published but no one is made aware of it. ***ONLY fats fulfill your appetite, NOT protein or carbohydrates***.

ω-6 Polyunsaturated fatty acids extend life span through the activation of autophagy

O'Rourke, Eyleen, J., et al., **ω**-6 Polyunsaturated fatty acids extend life span through the activation of autophagy," (Genes & Development). Published in advance February 7, **2013**, http://genesdev.cshlp.org/content/27/4/429.full, **2013**:

> "Supplementing C. elegans culture media with these **ω-6 PUFAs** [long chain metabolites termed derivatives] increases their **resistance to starvation and extends their life span** in conditions of food abundance. Supplementation of C. elegans or **human epithelial cells** with these ω-6 PUFAs *activates autophagy*, a cell recycling mechanism that **promotes starvation survival** and **slows aging**.
>
> "We found that supplementation with **AA and DGLA [both Parent omega-6 derivatives]**, *but not with EPA*,

was sufficient to activate autophagy in ad libitum-fed C. elegans.

"Our data show that supplementation with ω-6 PUFAs activates autophagy **in human epithelial cells**.

"These results show not only that dietary supplementation with ω-6 PUFAs **activates a conserved cellular response** normally triggered by fasting, but also that long-term administration of ω**-6** PUFAs can render the beneficial effects of **low-caloric intake** even in ad libitum feeding conditions…."

Appendix X from 24-Hour Diet: The Power of Parent Omega-6

Newsflash: Parent Omega-6 Increases Weight Loss: Known in 1973!

You have already read about Doctor Cavallino's experiment with "carboholics" in Italy. I have long been aware of the remarkable power of the correct *un*adulterated parent omega-6 to -3 ratio in decreasing carbohydrate cravings. However, even I had never seen the following medical journal article. I sincerely thank Canadian David Macphail for sending it to me. All the way back in 1973, physician H. Kasper proved there is an effect of greater weight loss and better blood chemistry, too, when parent omega-6 is added to the diet — REGARDLESS of CALORIES.

Here's what the study states[30]:

"**Despite a higher total caloric intake**, the weight-reducing effect clearly equals that of a standard clinical reducing diet of 1,000 kcal [even though patients consumed significantly more food].

"…A **maximal weight loss** was achieved in cases 1, 2, and 15 when they were **taking fats high in linoleic acid [parent omega-6]**.

"It was **striking to observe** that the **weight gain did *not* correlate with the caloric intake**. Particularly if fat was given in the form of corn oil [high in parent omega-6], a distinct discrepancy between the caloric intake and the response of the body weight was detectable.

"This phenomenon was less conspicuous if fat was taken in the form of olive oil.

"If **fat was exchanged** isocalorically **for** glucose [**carbohydrates**], the **weight loss ceased**.

"The **cholesterol and triglyceride concentrations** in the serum, which had been raised at the beginning of the experiment, invariably showed a **tendency towards normalization** under this dietary program." (Emphasis added.)

---

30 Kasper, H., et al., "Response of body weight to a low carbohydrate, high fat diet in normal and obese subjects," *The American Journal of Clinical Nutrition*, 26: February 1973, pages 197-204.

◗ *Lean-for-Life* Commentary

1. This experiment proves that the "calorie theory" is incorrect and that there is much more to the picture than merely "calories in minus calories used equals weight gain."
2. The parent omega-6 oil has a natural weight-loss property, whereas olive oil does not.
3. When carbs were switched "calorie-for-calorie," for fat, weight loss STOPPED. Once again, we clearly see the "low-fat/hi-carbohydrate diet" failing!
4. Triglyceride and cholesterols significantly improved with the low carbohydrate/parent omega-6. We have a home-run!

Following are some critical points, from the exceptional treatise by DF Horrobin, MD, PhD—a true medical genius, published in *Progress in Lipid Research*:[31]

> "**The *n-6* EFAs have at least four roles:** (1) The modulation of **membrane structure**. (2) The formation of short-lived local regulating molecules such as prostaglandins (**PGs**) and leukotrienes (**LT**), together often **known as eicosanoids**. (3) The control of the **water impermeability of the skin** and possibly the permeability of other membranes such as the gastro-intestinal tract and the blood-brain barrier. (4) **The**

31 Horrobin, D.F., "Nutritional and medical importance of gamma-linoleic acid," *Prog. Lipid Res.*, Vol. 31, 1992, No. 2, pages 163-194.

regulation of cholesterol transport and **cholesterol synthesis**. **The membrane effects of the EFAs are possibly the most important**.

"The fluidity and flexibility of all membranes within the body are influenced by their EFA content. As a crude indicator, the effect of an EFA on membrane fluidity is determined by its concentration in the membrane and by the number of double bonds in the molecule (the product of concentration x the number of double bonds is sometimes known as the unsaturation index). **However, there is much more to the story than that**.

"The *n-3* EFAs, even though they have as many or more double bonds as the *n-6* EFAs are unable to reverse the features of *n-6* EFA deficiency.

"The precise configuration of the double bonds must therefore be important and attempts to explain the rationale for this are just beginning.

"**The lipid configuration of the membrane** is important in itself, but also matters because it **influences the structure and behaviour of the many proteins in the membrane such as ion channels, receptors and ATPases [including insulin receptivity]**. These proteins are literally ***afloat in a lipid sea and their function is dependent on the behaviour of that sea***. Good examples of this are studies on the effects of lipids on the binding

of ligands to their receptors. The unsaturation of the lipid medium in which the receptors are found has been reported to change the affinity for ligands, such as *steroid hormones*, and *peptides*, such as opioids and angiotensin. In general, the more unsaturated the lipid, the lower the affinity of the receptor for its ligand.

"Lipid unsaturation also influences membrane 'fluidity.' This is important in the **vascular system** and also in **any other situation in which cells move**, for example, during inflammation and immune responses. Red cell membranes, which have **reduced EFA levels** are "stiffer" than usual, and as a result **increase blood viscosity and reduce tissue oxygenation**.

"The third role of the EFAs is in the maintenance of the water impermeability of the skin. ***In the absence of n-6 EFAs the skin loses its 'water-proofing.'***

"It is apparent from this brief description that a lack of or abnormal metabolism of EFAs could adversely influence every cell and every organ system in the body. There is therefore nothing inherently surprising in the concept that EFAs may have a role to play in modulating many different disease processes. [Note: *This precisely explains why cancer can occur in any tissue—the most oxygen deficient.*]

"**The EFA requirement may be increased** in the presence of high rates of cell division.

This situation may be physiological (as in infancy) or **pathological (as in the presence of cancer, inflammation or rapid cellular repair after injury)**.

MEN need 5Xs More PEOs...

"Gender has a major, but inadequately understood, impact on EFA requirements. *Male animals require a higher EFA intake than females.* This may in part be because females metabolise LA more rapidly and in part because they retain EFAs in tissues more effectively in the presence of EFA deficiency.

"**The total phospholipid (TPL) fraction, in contrast, does not change rapidly in response to feeding or fasting**. Moreover it is relatively rich in the EFAs right along the metabolic chain. It can therefore be used as **a guide to both EFA intake and EFA metabolism**."

PEO Solution analysis: **In research,** plasma **total phospholipids are the best quantitative measure of EFA status**—*much superior to red blood cell analysis*.

Horrobin's treatise continues...

Confirmation of Small Derivative formation:

"GLA [Parent omega-6 derivative] is formed by the rate-limiting step of delta-6-desaturation and metabolised by the non-rate-limiting step of elongation to dihomogamma-linolenic acid (DGLA). It is therefore not surprising that **GLA is found in most tissues in only small amounts**. It normally makes up less than **0.2% of the fatty acids in phospholipids**, less than **0.1 % of those in triglycerides** and less than **2.0% of those in cholesterol esters**. Furthermore, ***GLA—the body's most important derivative*** is found in tissue in only very small quantities. Furthermore, Parent omega-6 can modulate cytokine releaser directly, rather from its long-chain metabolites.

"The administration of **GLA leads to increased plasma PGE1 levels in humans** and increased macrophage PGEl levels in rats. There can therefore be no doubt that GLA enhances the rate of formation of this very desirable substance.

"PGE1 has a quite extraordinary range of desirable actions. It dilates blood vessels and lowers blood pressure; it inhibits platelet aggregation; it inhibits cholesterol biosynthesis; it is an anti-inflammatory agent; it has a biphasic regulating effect on immune responses; and it stimulates cyclic AMP formation, thus being capable of inhibiting phospholipase A2, an enzyme important in releasing AA during inflammation.

These desirable effects obviously have considerable therapeutic potential.

"The formation of PGE1 may explain why, ***contrary to simplistic expectations***, but in accordance with a **prediction based on understanding** of PGE1 actions, the rise in AA levels following GLA administration to cells, animals or humans is ***consistently followed by a fall, rather than a rise, in the levels of conversion of AA to potentially harmful metabolites like Thromboxane A2 or PGE2***. There is a tendency to consider arachidonic acid "a bad thing" because it can give rise to metabolites like thromboxane A2, PGE2, and leukotriene B4. In fact there is **no evidence at all that arachidonic acid is harmful so long as it stays as AA**. AA is an **essential constituent** of **membranes**. Adequate levels of DGLA seem important in keeping AA in membranes where it is desirable, and preventing conversion of AA to its possibly undesirable metabolites:

The amazing treatise continues.... showing **Parent omega-6 and its metabolites takes center stage....**

"The *n-3* EFAs are of major biological significance but they are simply not as important as the *n-6* EFAs.

"This is shown by the following facts:

"(1) When animals and humans are put on **diets deficient only in *n-6* EFAs**, it is easy to show that they develop

multiple biochemical and biological abnormalities. In contrast it has proved extremely difficult to demonstrate biological abnormalities in animals deprived only of *n-3* EFAs. There are abnormalities in the brain, the retina, the heart and platelets and the *n* -3 EFAs are undoubtedly important in modulating the functions of these organs, but ***these abnormalities are not easy to demonstrate***.

"(2) When animals are ***deprived of both n-3 and n-6 EFAs***, all the readily observed **abnormalities are quickly corrected by *n-6* EFAs alone**. ***N-3* EFAs alone do not correct any of the abnormalities, and make some, such as the capillary fragility, worse**.

"In order to express their normal biological effects, ***n-3* EFAs must be given with *n-6* EFAs** whereas the ***n-6* EFAs** are biologically active when given without *n-3* EFAs. [Note the significant difference. The 21st century solution is BOTH Parent omega-6 and Parent omega-3 in the proper ratios and quantities.]

"(3) In human milk and in most tissues in the body, the ratio of *n-6* to *n-3* EFAs lies within the range 3:1 to 9:1 [breast milk is 10:1—most tissues 4:1-7:1; although stored body fat is much higher.]. This is true even of animals such as the zebra whose EFA intake is almost entirely in the form of ALA [Parent omega-3] from grass. **Thus even when most dietary EFAs are in the *n-3* form, the *n-6* EFAs are preferentially retained**."

PEO Solution analysis: We see the much greater importance of Parent omega-6 and its metabolites compared to Parent omega-3 and its metabolites. Dr. Horrobin terms omega-6 series GLA the body's most important derivative.

The Power of PEOs was known in 1956. The extraordinary nutritional scientist of Reading and Oxford, H.M. Sinclair, wrote a superb article in *The Lancet* titled, "**Deficiency of essential fatty acids and atherosclerosis, etcetera.**[32] **He warned that people won't believe it,** stating , **"MY inclusion of 'etcetera' in the title invites the scorn we so readily pour I vendors of patient cure-alls**." Tragically, he was correct and his advice was not properly integrated into the medical community. Today, you can remedy that mistake.

Journal highlights are:

- "First, there was an enormous increase in permeability of the skin [**epithelial tissue in all carcinomas**] in EFA [PEO] deficiency and there is an increase **in capillary fragility**...
- to the carcinogenic effect of X rays through deficiency of EFA, the above facts become explicable provided those young children who died of leukaemia were irradiated when their pregnant mothers were had diagnostic radiography; a chemical carcinogen could hardly be responsible for their deaths. "In lower animals, in which

32 " April 7, 1956, pages 381-383.

we have carefully studied the skin lesion of EFA [PEO]-deficiency and found a dramatic increase in permeability of the epidermis, we believe there is a structural fault perhaps through failure of the phospholipids containing EFA [PEO] to polymerise and form the impermeable barrier in the stratum granulosum. Phospholipids are rich in unsaturated fatty acids [PEOs] (though not so rich as cholesteryl esters... "So, as in the case of esterified cholesterol, we have an **abnormal type of phospholipid** being formed which may not only cause a structural defect in the skin which is responsible for the great increase in permeability but may also be outstandingly important ....

- "... [T]he nervous system is rich in phospholipids containing polythenoid fatty acids, and these are found together with highly **unsaturated cholesteryl esters in myelin**; the presence of abnormal types of the compounds that are known to important to it would be unlikely to leave function undisturbed; disseminated sclerosis is a disease of highly civilized countries being almost unknown in India and China [as of 1956], and other diseases in which the ectodermal neuroglia is effected may be relevant; since serum **EFA fall in acute infections**...early mild **dementia** appears to be becoming commoner in males. Thirdly, the **mitochondria** membrane probably contains phospholipids, and derangement of this through **deficiency of EFA [PEO] might me responsible for the uncoupling of oxidative phosphorylation found in such deficiency**

- "Effects of EFA [PEO]-Deficiency: First, deficiency would be *likely to be at least five times commoner in males than in females*. "Secondly, we might expect deposition of cholesterol since cholesterol esterified with unusually saturated or with unnatural fatty acids is probably disposed of less readily..."

The article continues:

- "...I believe [abnormal esters/phospholipids] to be caused by a **pure dietary deficiency of essential fatty acids [PEOs]**... There is even more brilliance in his article, but these excerpts make it clear—his genius is evident.

Dr. Hugh Sinclair **predicted** the following patient ailments/disease/physiologic disorders from EFA deficiency:

a) **Cardiovascular disease.**

b) **Cancer**; in particular, increased skin cancer. [Note: Skin cancers are at epidemic levels with no end in sight, and cardiovascular disease (in all forms) is our #1 killer. CVD too has no end in sight. All carcinomas are enclosed by epithelial tissue—a PEO deficiency of Parent omega-6—is DIRECTLY TIED to ALL CARCINOMAS. Furthermore, only Parent omega-6 is contained in the arterial intima.]

c) Dr. Sinclair understood the *damage X rays* may cause. **PEOs are highly protective against cancer treatment X ray damage**—*See* my book, *The Hidden Story of Cancer*.

d) PEOs assist the nervous systems. **PEOs help fight MS**.

e) Even **dementia** is addressed. This has become another

epidemic directly related to PEO deficiency. Fish oils are worthless and coconut oil is not effective enough.

f) PEOs vital role in **combating infection**.

g) PEOs are directly **incorporated into mitochondrion**. This is a ***top anti-aging secret and a key to cancer prevention***.

h) Dr. Sinclair's key concept that leads us to lower **LDL-cholesterol**—esterification of cholesterol to PEOs—will be discussed in detail later.

Scientific Support for Chapter 10

Our Secret for Natural Beauty

Dermatologists take note: The first experiments with EFAs concerned their wonderful effects to the skin:[33]

- "The effect of linoleic acid [**Parent omega-6**] on skin inflammation was perhaps the first "**medical" effect of the EFAs to be noted**. A technician working in the Burr's laboratory where the EFAs were discovered noted that his hand dermatitis improved when linoleic acid [Parent omega-6] intake was increased.

- "At the same time Hansen, a pediatrician friend of the Burrs, noted that the **dermatitis in EFA-deficient animals resembled atopic eczema in his patients**. Atopic eczema is the type of eczema which runs in families and usually manifests itself in the first year of life. Using crude techniques Hansen went on to show that the levels of unsaturated fatty acids in the blood [**in particular, Parent omega-6**] were ***low*** in patients with atopic **eczema**."

---

33 Horrobin, D.F., "Nutritional and medical importance of gamma-linoleic acid," *Prog. Lipid Res.,* Vol. 31, No. 2, pages 163-194, 1992.

"Detrimental Effect of an w-3 Fatty–Acid Enriched Diet on Wound Healing:"[34]

"The hypothesis that a diet enriched with *w-3* fatty acids could be detrimental to wound healing was tested in male rats fed complete diets **differing only in their fat** composition (17% menhaden oil [fish oil] + 3% corn oil *vs.* 20% com oil by weight) for 21 days before wounding and for 10 or 30 days after wounding.

"At 30 days, however, ***wounds*** **harvested from rats fed the menhaden [fish] oil diet were *significantly weaker* than those from corn oil-fed [parent omega-6] animals**. This difference in tensile strength was not explained by differential collagen accumulation, inasmuch as the collagen content of the sponges at 30 days was the same in both groups, Dietary consumption of a diet rich in ω-3 fatty acids [**fish oil**] may **conspire against the quality of wounds** by altering the fibroplastic or maturational phases of the healing response.

"Current results show that **substituting w-3 fatty acid [fish oil] for w-6 fatty acids in the diet is deleterious to the mechanical properties of wounds** at 30 days."

---

34 Albina, JE, et al., *Journal of Parenteral and Enteral Nutrition,* Vol. 17, No. 6, 1993, pages 519-521.

Dietary CLA and DHA modify skin properties in mice

The **2003** journal article, "Dietary CLA and DHA modify skin properties in mice," has this to say:[35]

> "No significant differences in lipid and collagen contents were detected among treatments, although the **FA composition in the skin was altered depending upon the FA composition of the supplemented oils**. Electron microscopy revealed that the subcutaneous tissue layers in the **CLA and DHA groups were significantly thinner** than **that in the high** linoleic acid [**Parent omega-6**] group, whereas no differences in the thickness of dermis layers were observed among the three groups. **These results suggest that skin properties in mice are readily modified by dietary FA sources within 4 wk of dietary oil supplementation**. [Note: Mice and humans have similar EFA-based metabolic pathways so the results are applicable to humans.]
>
> "…This suggest that LA [parent omega-6] is one of the ***important factors in maintaining healthy skin***."

Linos, Elini, et al., "Increasing burden of melanoma in the United States," *J Invest Dermatol*, 2009 July; 129(7): 1666–1674

> "Malignant melanoma is one of the fastest growing cancers worldwide."
>
> "We observed that **melanoma incidence increased for both men and women** across all categories of **tumor**

35 Oikawa, Daichi, et al., *Lipids*, 38, 609-614 (June **2003**).

> **thickness, including a significant 3.86% annual increase among the thickest tumors** (>4 mm)…. We do not believe that improvements in the reporting of thickness information over time could fully explain our observations of increasing incidence trends across all thickness categories, as demonstrated in our sensitivity analyses described above." [Note: There is a true, across-the-board increase in melanomas, regardless of specificity.]

Unfortunately, other countries follow America's wrong recommendations. The NutraIngredients.com article further states that Asia will soon consume more fish oil than the US in its article titled, "China to over take Western Europe in EPA & DHA oil consumption:"

> China is poised to overtake Western Europe in the consumption of EPA- and DHA-rich oils, according to research conducted by market analysts Frost & Sullivan and the Global Organization for EPA and DHA (GOED).
>
> In the US market, omega-3 dietary supplement sales are estimated at $1002m in **2009** up from $40m 16 years ago [**1993**]. [A **25-fold** increase.]
>
> **The biggest category for omega-3 oil consumption remains dietary supplements,** which account for 51,148t, according to the GOED/Frost and Sullivan report. But other categories are growing in importance.
>
> **Animal feed** accounts for 20,400t, followed by **food and beverage** (9561t), **infant** nutrition (3103t), **pharmaceuticals** (1391t) and clinical nutrition (70t).

Scientific Support for Chapter 11, Athlete Advantage

Effect of Hyperoxia on Maximal Oxygen Uptake, Blood Acid-base Balance, and Limitations to Exercise Tolerance

Astorino, TA and Robergs, RA, "Effect of Hyperoxia on Maximal Oxygen Uptake, Blood Acid-base Balance, and Limitations to Exercise Tolerance," *The Journal of Exercise Physiology*, Vol. 6, No. 2, May **2003**, pages 9-18.

The importance of increased *cellular* oxygen for increased performance is detailed next, "Effect of ***hyperoxia*** [**extra oxygen**] on VO2 max [maximal oxygen uptake], blood acid-base balance, and limitations to exercise tolerance:"

> "***Hyperoxia***, or an **increase** in inspired **oxygen concentration**, has been used by scientists to examine exercise metabolism and physical work capacity. It is apparent that ***hyperoxia increases VO2 max*** *and exercise* ***tolerance due to an increase in*** O_2 ***supply*** **to contracting muscle**. Furthermore, **hyperoxia increases PaO2** [Pulmonary Arterial Oxygen Tension], which may promote an enhanced diffusion of O2 in skeletal muscle. Compared to normoxia [normal O_2 levels], hyperoxia may reduce PCr [substance in muscles that facilitates energy for muscle contraction] degradation during the metabolic transient, **attenuating the magnitude of cellular disturbance** characteristic of near-maximal to **maximal exercise**.

"…During the next 30 years, research with improved experimental design and methodology supported early findings showing that **hyperoxia [super-oxygenation] improved work tolerance** ***independent of exercise mode***.

"An initial explanation for this **enhanced performance in hyperoxia is a greater VO2max mediated by enhanced oxygen delivery**. Consequently, it is evident that hyperoxia enhances **VO2max due to an increase in *oxygen delivery to active muscle.*** *This reduced perturbation of cellular homeostasis* would promote lesser acidosis in hyperoxia, leading to a better maintenance of contractile function and thus improved exercise tolerance.

"… Overall, these data suggest that in hyperoxia, a greater gradient for diffusion of **O2 from the capillary to the muscle mitochondria enhances VO2max**."

2011 *Journal of Strength and Conditioning Research* performed at the Exercise Physiology and Metabolism Laboratory, Department of Kinesiology and Health Education, The University of Texas at Austin, Austin, Texas `makes clear:

- "A **Low Carbohydrate-Protein Supplement Improves Endurance** Performance in Female Athletes."
- "It is likely that the greater performance seen with CHO [CHO is an abbreviation for carbohydrate, which is composed of carbon+hydrogen+oxygen] + PRO [protein] was a result of the CHO–PRO combination and the **use of a *mixture* of CHO sources [like fruit]**

- "In this study, **plasma glucose levels were significantly lower during exercise** [more fuel was being used by the muscles] as compared to CHO [levels]. However, plasma insulin levels were similar between trials; therefore, the lower plasma glucose levels cannot be attributed to an increase in insulin availability. **The combination of CHO and PRO could have increased glucose clearance from the blood at a greater rate than CHO alone**, resulting in lower blood glucose levels and increased exogenous CHO availability to the working muscle.

- "As in the study by Currell and Jeukendrup, **the glucose + fructose [like fruit] mixture improved performance by 8% in comparison to glucose only** Previous investigations in **our laboratory have additionally found improved efficacy when using a mixture of CHOs, in combination with a moderate PRO concentration**. Therefore, it appears this is a likely mechanism contributing to the improved TTE [transthoracic echocardiogram, which is a cardiac ultrasound examination that tests cardiac performance] we observed.

- "In summary, the addition of a moderate PRO concentration to a low concentration CHO mixture **improved endurance performance in comparison to a traditional 6% CHO sports drink** in trained female athletes. ***This improvement occurred despite CHO + PRO containing 50% less CHO and approximately 30% fewer calories than the traditional 6% CHO supplement.*** It is likely the greater performance seen with CHO + PRO

was a result of the combination of PRO and the use of a **mixture of CHO sources**."

Athletes need to know that published, "Fish-oil supplementation reduces stimulation of plasma glucose fluxes during exercise in untrained males":

- "It is concluded that **fish oil reduced Rd [rate of glucose disappearance] glucose by 26% by reducing glucose metabolic clearance rate** …"

- "[I]t was observed in healthy human subjects that a 3-week supplementation of the diet with **fish oil (6g/day) decreased by 40% the insulin response** to an oral glucose challenge without altering either endogenous glucose production or plasma glucose utilization.

- "[N]-3 long-chain fatty acids are incorporated into **membranes whose composition remains altered at least 18 weeks after interruption of fish-oil supplementation**…

- "The main observation of the present study is that a supplementation of the usual diet with **6 grams fish oil/day during a period of 3 weeks reduced stimulation of both HGP [hepatic glucose production] (-21%) and Rd glucose (-26%)** during exercise."

See "The Essential Role of Physiologic EFAs (PEOs) at http://www.brianpeskin.com/BP.com/reports/Sports-Medicine.pdf.

ScienceDaily
Your source for the latest research news

Mice Run Faster On High-grade Oil

ScienceDaily (July 1, 2009) — Between the 1932 and 2008 Olympic Games, world record times of the men's 100m sprint improved by 0.6 seconds due to improved training techniques and technological advances. Imagine if this improvement could be achieved by a simple change in diet. Scientists at the Research Institute of Wildlife Ecology in Austria have managed to achieve an equivalent feat in mice fed on a diet high in polyunsaturated fatty acids.

Mice fed on a diet high in polyunsaturated fatty acids can sprint faster. (Credit: iStockphoto/Sarah Salmela)

Polyunsaturated fatty acids are important dietary components which mammals cannot synthesize de novo. The research, to be presented on the 29th of June 2009 at the Society for Experimental Biology Annual Meeting, has shown that mice fed for two weeks on a diet high in sunflower oil, which contains n-6 polyunsaturated fatty acids, ran on average 0.19m/s faster than mice fed a diet rich in linseed oil, which is high in n-3 fatty acids.

This means that, over a 2 second sprint, a mouse fed on a high n-6 fatty acid diet would have a 0.4m advantage. This represents a 6.3% improvement which equals that achieved in the 100m world records over more than 75 years. For a mouse, or other small mammal, this would be significant in evolutionary terms when escaping from a predator or catching prey. "The results of the current study on mice suggest that moderate differences in dietary n-6/n-3 polyunsaturated fatty acid intake can have a biologically meaningful effect on maximum running speed", says Dr Christopher Turbill who will be presenting the research.

A previous study by the group, which looked at a range of mammal species, found that those with a relatively high n-6 fatty acid content in their skeletal muscles had a greater maximum running speed. Combined, these two studies suggest that diets enriched in these fatty acids "could also affect the maximum (or burst) running speed of other vertebrates, including humans," says Dr Turbill. "The application of this research to the performance of elite athletes (specifically those in sports that involve short distance sprints, including cycling) is uncertain, but in my opinion certainly deserves some further attention" he says.

Adapted from materials provided by Society for Experimental Biology, via EurekAlert!, a service of AAAS.

3

Increased Oxygen Without the Hyperbaric Chamber

The medical profession treats a very long list of conditions and diseases through the use of a hyperbaric oxygen chamber.[1] This is an enclosure, in which the patient lies or sits, that delivers pure oxygen under pressure to the body. The cells become saturated with ten to fifteen times more oxygen than they would at normal atmospheric pressure. In addition to speeding healing of many types of wounds and infections, this treatment has produced remarkable improvements in patients paralyzed with stroke, various neurological and orthopedic conditions including comas, patients with cerebral palsy, multiple sclerosis, macular degeneration in the eyes, and many other conditions.[2]

The **PEO Solution** program provides the next best oxygenator to the hyperbaric chamber in a practical way—nutritional supplementation with the correct PEOs. PEO supplementation allows increased *cellular* oxygenation on a daily basis. Oxygen has to penetrate into the tissue—not just bathe the surface—and both the hyperbaric chamber and PEOs—as specified in **PEO Solution**—accomplish this goal.

---

1 I wish to thank Robert E. Levine, M.D., of Los Angeles, California for his insight leading me to think about the existing medical uses of hyperbaric oxygen, and how EFAs represent the evolution of its benefits without its bulk.

2 Thanks to Alan Spiegel, M.D., National Hyperbaric Oxygen Therapy Center, Palm Harbor, Florida, for information about the use and applications of hyperbaric oxygen therapy and provided on his Center's website.

Hardworking Muscles Use Glycolysis for "Emergency" Short-Term Energy

ADVISORY: This section is somewhat technical but important to understand.

...

Normally, at least 95% of the time, muscles use oxygen for cellular respiration. However, under extreme physical endurance stress such as weight lifting, not enough oxygen can get to the muscle fast enough to keep up with increased energy requirements. Therefore, as a *temporary,* but *normal* phenomenon, the muscles switch over to "lactic acid fermentation" to get the energy they require. First, glucose is converted to pyruvate. Then, the pyruvate molecules are used for energy, producing lactic acid as a byproduct. (It is really just excess hydrogen ions but the pain still occurs—thanks to *The Townsend Letter for Physicians* and ND's Jade and Keoni Teta.) This acidic environment builds up in the muscles, causing what is commonly called a *lactic acid "burn."*

...

Excerpts from Nobel Prize-winner Otto Warburg's monumental *The Metabolism of Tumors in the Body*:[3]

> **"[That] normal tissues** under normal living conditions [resting] put no lactic acid **in the blood was confirmed.**
>
> "On the contrary, they [normal tissues] remove it from the blood.

---

3 Otto Warburg, et al., "The Metabolism of Tumors in the Body," *The Journal of General Physiology*, Vol. 8, 1928, pages 524-525.

> **"Normal cells form** lactic acid in general only **when their** oxygen is cut off or their respiration checked**.**
>
> **"...[D]uring forced bodily work [like weightlifting], the lactic acid of the blood increased.** In this case the diffusion of oxygen into the muscle cells is not sufficient to cover the oxygen requirement of the muscle**."**

The accumulated lactic acid has to be transported via the bloodstream to the liver and kidneys, where it converts to glucose to be used again.[4]

Muscles don't obtain significant energy (ATP) from fermentation. Respiration with oxygen is about 20 times more energy producing than the energy that can be produced from glycolysis utilizing the same amount of glucose. So why do our bodies use this inefficient system at all? Simple—it is quick! The energy is available much faster through fermentation. The muscle requires energy quickly, and Nature designed the ideal process to handle the situation.[5] The excellent science-based book, *Nutrition for Fitness and Sport*,[6] by Professor Melvin H. Williams, states, "… [T]he lactic acid system is used in sport events in which energy production is near maximal for 1 to 2 minutes, such as a 400- or 800-meter run."

---

4 *Harper's Illustrated Biochemistry, 26th edition*, **2003**, page 159.

5 http://biology.clc.uc.edu/courses/bio104/cellresp.htm and http://web. indstate.edu/thcme/mwking/glycolysis.html.

6 *Nutrition For Fitness and Sport*, Melvin H. Williams, Brown and Benchmark Publishers, Chicago, 1995, page 61.

Principles of Medical Biochemistry states:[7]

> "Skeletal muscle has to increase its ATP (energy) production more than twentyfold during bouts of vigorous contraction, for example, a 100-m sprint.
>
> "In this situation the supply of oxygen from the blood becomes a limiting factor, and the tissue depends to a large extent on the anaerobic glycolysis of glucose, and more importantly, of stored glycogen.
>
> "The lactate concentration in the blood rises 5- to tenfold in this situation."

We now know how to remedy a cellular oxygen deficiency so that the lactic acid produced in the muscles during strenuous workouts is more quickly used as fuel with oxygen. ***This reduction in acid "burn" proves the tissue's increased oxygenating capability***. We can now demonstrate that although the muscles still use fermentation short-term, their oxygenating capability has been raised to such an extent that the acid burn is minimized or eliminated.

Let's proceed with an in-depth discussion about the oxygenation/decreased lactic acid buildup discovery. *The Hidden Story of Cancer* details this regarding increased anti-cancer protection.

---

7 *Principles of Medical Biochemistry,* Gerhard Meisenberg and William H. Simmons, Mosby, Inc., New York, 1998, page 303.

Proof of Oxygenation with PEOs—Lactic Acid Burn is Stopped Cold: A "Do-It-Yourself" Test

If you have ever worked out with weights, then you have likely already experienced the so-called "lactic acid burn." It is a burning sensation that comes from acid buildup in your muscles, produced when they **ferment glucose for energy**—much in the same way that a cancer cell does. "Lactic acid burn" becomes a *problem of the past* when PEO supplements are properly used.

Here is a definitive test: First, take about 1,500 mg of a PEO-based oil supplement as recommended. Wait 20 minutes. Then you can simply take a heavy dumbbell and perform "biceps curls" until your arm is completely fatigued. If the muscle fails—you can't hold the dumbbell any longer *and* **there is NO BURN—***then* **you know that your tissues are fully oxygenated. If you get the "burn," keep following the PEO Solution and try again later.**

From the previous discussion, we can deduce what must be happening to prevent acid "burn," and therefore give practical proof that cellular oxygenation has increased. Because of the fact there is little to no lactic acid burn, there can be no *excessive* acid buildup. This can only have been accomplished in one of three possible ways:

1. **Oxygen** from the blood is **increased** so that fermentation by the muscle is no longer required.
2. **Oxygen** transfer in the muscle is **increased** so that fermentation by the muscle is no longer required.

3. **Oxygen** is **increased intracellularly in the muscle**, allowing the lactic acid to immediately be used in respiration—there is no excessive lactic acid buildup.

Biochemistry of Exercise & Training supplies the answer to the question of which one of the above three possibilities is occurring The following is quite technical. First, we need to understand the different types of muscle fiber:[8]

1. "**Type 1 fibres** have numerous **mitochondria** [factories for energy production using oxygen], mostly located close to the periphery of the fibre, near to the blood capillaries which provide a **rich supply of oxygen and nutrients**. These fibres possess a high capacity for **oxidative metabolism**, they resist fatigue and are specialized for the performance of repeated strong actions over prolonged periods. Note: These fibers are very red in color due to the presence of *myoglobin*, an intercellular respiratory pigment capable of binding oxygen.
2. "In comparison, **Type IIb fibres** …have about a threefold greater maximum power output than the Type I fibres, …**but greater glycogen [glucose]** and phosphocreatine **stores**…. A high activity of glycogenolytic and glycolytic enzymes endows Type IIb fibres with a high capacity for rapid (but relatively short-lived) ATP production when energy has to be released at rates in excess of that available from oxidative phosphoryla-

8 *Biochemistry of Exercise & Training,* Ron Maughan, Michael Gleeson, and Paul L. Greenhaff, Oxford University Press, New York, 1997, pages 12, 18-19, 43, 75-76, 141, and 197-207.

tion. In other words, they **possess a high anaerobic capacity**. It is perhaps worth noting here that **anaerobic respiration** (glycogenolysis and glycolysis) occurs **without the use of oxygen, but not necessarily in the absence of oxygen (anoxia), nor for that matter low oxygen availability (hypoxia)**… **Type IIa fibres** are red cells [although much paler than Type I] whose metabolic and physiological characteristics lie between the extreme properties of the two other fibre types."

PEO Solution analysis: Type I fibers require cellular oxygen-based respiration and require this oxygen-based mechanism for long-term endurance. However, the Type II fibers are more anaerobic (not utilizing oxygen directly) and will utilize lactic acid. What is of immense interest is the fact that **paradoxically, even though the anaerobic energy mechanism doesn't directly use oxygen, it still requires some oxygen for the glycolysis of both available glucose and utilization of stored glycogen to be maximized.**

You will soon discover, as you read the information in items #4 and #7 below, the completely unexpected fact that ***PEOs*** **maximize the *anaerobic* system so that endurance increases, too.**

3. "The **rate of lactate formation** is dependent primarily on the intensity of the exercise, but depends more on the relative exercise intensity (%VO2 max) [**maximum oxygen consumption**] than the absolute intensity… [M] uscle glycogen store can be used for anaerobic energy … lasting from 20 s [seconds] to 5 minutes… To **achieve high oxygen consumption, an effective system for the**

transfer of oxygen from the atmosphere to the site of utilization in the mitochondria of the exercising muscles is essential…

4. "Although the conversion of glucose to lactate is an **anaerobic process, it occurs EVEN WHEN OXYGEN IS FREELY AVAILABLE** to the muscle, and **lactate release does not necessarily imply that oxygen supply is inadequate**."

PEO Solution analysis: Based on these surprising and unexpected facts in items #3 and #4, **we would now expect a higher lactate output—not less lactate—as a result of the increased oxygenation efficiency that PEOs provide in an exercise such as biceps curls**. Anaerobic glycolysis is primarily used for muscular events lasting from 20 seconds to 5 minutes **AND more lactate (meaning more energy) is produced with more oxygen uptake and greater tissue oxygen transfer** (%VO2 max)—which we have seen that PEOs assist with. PEOs also allow maximal oxygen transfer into the muscles' mitochondria.

---

5. "After the initial one or two minutes of exercise, however, a steady state of oxygen delivery is achieved. The high oxidative capacity of the active muscle fibres ensures that **some of the lactate produced in the initial stages of exercise is taken up by these fibres and reconverted to pyruvate** which is then decarboxylated to acetyl-CoA and enters the TCA [tricarboxylic acid] cycle.[9]

---

9 This is also known as the *citric acid cycle* or *Krebs' Cycle*.

6. "…Lactate production will thus occur, even when there is no restriction on the oxygen availability to the muscle cells: the **accumulation of lactate in the blood is, therefore, largely a reflection of the activation of muscle fibres** in which the **glycolytic capacity** ***exceeds the capacity*** **for the oxidative metabolism** of pyruvate."

PEO Solution analysis: Type I muscle fibers have mitochondria (oxygen-based energy factories)] that can use the lactate waste product from the Type IIb fibers. This precisely explains the lack of acid burn even though there could still be a buildup of lactic acid—it simply gets used quickly.

Again, there will be lactic acid REGARDLESS of oxygenation level; it is a normal, yet unexpected result, as so often occurs in scientific study; it is simply the way your body works. Any good scientist **has to have the theory fit the *real-life* facts—not vice versa.**

Nutrition for Fitness and Sport (page 69) has this to say regarding training and lactic acid buildup and increased oxygen utilization:

---

7. "…Training **increased** both your **VO2 max** [maximum oxygen consumption] and your steady state threshold, which is the **ability to work at a greater percentage of your VO2 max without producing *excessive*** **lactic acid**—a causative factor in fatigue."

With PEOs, we get this *increased oxygen utilization without the additional expected training*—endurance increases and

recuperation time decreases, as you discovered from Dr. Cavallino's exceptional experimental results. Does it matter which single oxygenator or combination of the three possible factors causes the increased oxygenation? No, because each one of the three individual factors increases oxygenation!

With proper PEO supplementation, tissue's oxygenation level will increase significantly over time so that oxygen to the muscle is maximized; **hence, no "burn.** "Professional athletes understand this.

"The Lactic Acid Test," by Sam Walker, published in *The Wall Street Journal*; July 22, **2005**, page W1, gives us more *real-life* information:[10]

> "…[H]e [Lance Armstrong] produces **one-third less lactic acid than do other top cyclists** and **delivers oxygen** to his legs **at a rate higher** than all but maybe 100 of his fellow earthlings.

---

10 You may be wondering why Lance Armstrong got testicular cancer with a high level of oxygenation. This is what you need to know: He was treated for his cancer in 1996, meaning that the cancer had been developing for many years prior to that. Here's the reason: cycling causes abnormal irritation and stress in the testicular area. If Lance was deficient in EFAs back then, he'd have more bouts of exhaustion from oxygen transfer deficiency and be at greater risk of developing cancer. From an article in Paris, July 2000 (http://sportsmedicine.about.com/od/ cyclingworkouts/a/080100.htm) comes the following telling quote. "He [Lance] won the world championships in 1993, but he also **was forced to drop out of three of his first four Tours de France**, the most prestigious but correspondingly [most] difficult cycling race on earth, **because of exhaustion** or injury**." There is clearly a difference in Lance then and now regarding oxygen utilization!**

> **"...**Researchers at the U.S. Olympic Training Center have developed a series of four tests for things like **'maximum oxygen uptake'** and **'power output at lactate threshold'** that can determine whether someone has the natural ability to be a top endurance athlete."

Professional athletes supplementing with PEOs 20 minutes before a game have never before seen or felt anything like this effect.

Likewise, bodybuilders of any age are amazed, too. This is striking *real-life* **confirmation that EFAs, in the ratios this plan recommends, [d] oxygenate the tissues the way Nature intended. The exact means to prevent cancer—full oxygenation, as described so precisely by Dr. Warburg—is effectively accomplished, too.**

Scientific Support for Chapter 12

***2103 Newsflash* for OB/Gyns—Autism**: While autism has reached epidemic proportions, the **Power of the Parents** will give expectant mothers piece of mind by protecting their unborn child:[1] "Eating Healthy Fats [**Parent omega-6**] During Pregnancy Linked To Decreased Autism Rates; Omega-3 Fatty Acid [**Small amounts** of marine oil] Beneficial Until Threshold [Overdose],"

- "The study found that **women who consumed linoleic acid [Parent omega-6]—** a type of omega-6 acid found in vegetable oils, nuts, and seeds **—** were 34 percent ***less likely* to birth a child with autism**…

- "In analyses of extreme deciles of fat intake compared with the middle 80% of the distributions**, *ω -6 fatty acid and linoleic acid continued to show significant associations with ASD***

- In addition, ***risk was significantly elevated for those with the lowest 10% of intake of a -linolenic acid*** [**Parent omega-3**] (odds ratio = 2.23, 95% CI: 1.30, 3.84) and the lowest 5% of intake of linoleic acid (odds ratio = 2.20, 95% CI: 1.09, 4.46).

- "Significant associations with total ω-3 fatty acids, **which we had hypothesized to be of primary relevance** given their role in brain development, anti-inflammatory

---

1 http://www.medicaldaily.com. Ref.: Lyall K, et al., "Maternal Dietary Fat Intake In Association With Autism Spectrum Disorders.," *American Journal of Epidemiology*. June 28, 2013.

processes, and immune function, **were seen only when very low intakes were assessed**.

- **We did not see any association with eicosapentaenoic acid [omega-3 derivative] or docosahexaenoic acid [omega-3 derivative]**, which are 2 ω-3 fatty acids essential to fetal brain development, or arachidonic acid, which is also important in brain development.

- "Our results provide preliminary evidence that increased maternal intake of ω-6 [Parent omega-6] fatty acids could reduce risk [34% reduction] of off-spring ASD and that [only] very low intakes of ω-3 fatty acids and linoleic acid could increase risk."

▶ "For specific fatty acids, the only significant associations were seen with linoleic [Parent omega-6] and α-linolenic [Parent omega-3] acids, essential fatty acids that are required from dietary sources. Intakes of these fats, however, are highly correlated, and in analyses in which each of these fats was adjusted for the other, ***only linoleic acid [Parent omega-6] remained significant***.

Noda, Satoru, "Hypoxia upregulates adhesion ability to peritoneum through a transforming growth factor-β-dependent mechanism in diffuse-type gastric cancer cells," *European Journal of Cancer*, Volume 46, Issue 5, Pages 995-1005, March **2010**.

"**Hypoxic** environment exists in **most cancers** ... [Note: Hypoxia is present in *all cancers* to a greater or lesser extent].

> "In the present study, we investigated the effect **of hypoxia on adhesion ability of cancer cells** and found that hypoxia upregulates the adhesion….
>
> "The upregulation of α2-, α3- and α5-*integrin* by TGF-β under **hypoxic conditions** may be one of the mechanisms responsible for **the high metastatic potential of hypoxic DGC cells."**

The following article detailing the **importance of the cellular environment** was based on information **sponsored by the National Cancer Institute**, and other university cancer centers.

Medical News Today, October 25, **2012 —** Breast Cancer, "Genetic Changes Plus "Tumorous ***Environment***" Enable Breast Cancer Cells To Spread" has this to say:

- "A new study from Johns Hopkins researchers suggests that the lethal spread of breast cancer is as ***dependent on a*** *tumor's* ***protein-rich environment*** as on genetic changes inside tumor cells.
- "'The most dangerous aspect of breast cancer is its ability to **spread to distant sites, and most tumors are initially unable to do that**'
- "***If*** cancer cells are driven to disperse ***solely because of the genetic changes*** they carry, the researchers expected to see the tumor fragments behave similarly in both the healthy and tumorous environments. ***What they saw instead***, says Ewald, was a ***distinct difference*** As expected, 88 percent of tumor fragments sent cells crawling into the tumorous meshwork environment, the

first step in metastasis known as dissemination. But only 15 percent of tumor fragments sent cells crawling into the normal environment.

- "According to Ewald, ***these results indicate that the environment around a tumor plays a more direct role in cancer spread than previously thought***

- "**This tells us that tumors continue to listen to their environments**…"

Radiation

Radiation is often prescribed for patients. It has now been experimentally verified that radiation causes a significant increase in what is termed "cancer stem cells."[2] These findings confirm Dr. Warburg's warnings that radiation, after killing less virulent cancer cells, would make remaining cancer cells much more virulent (*The Hidden Story of Cancer* / Pinnacle-Press). Here's the article's warning along with quotes from the original article (Lagadec, C, et al., "Radiation-Induced Reprogramming of Breast Cancer Cells, *Stem Cells* **2012**;30:833-844):

- "...[R]adiation **treatment transforms cancer** cells **into treatment-resistant** breast **cancer stem cells**, even as it kills half of all tumor cells.

2 CancerScope: Oncology Issues in Focus by Carrie Printz, *Cancer* July 1, **2012**, page 3225.

- "...They also found that these **iBCSCs** (induced breast cancer stem cells) had ***more than a 30-fold increased ability to form tumors*** than the non-irradiated breast cancer cells.

Importantly, CSCs in breast cancer and glioma have been found to be reƌlatively ***resistant to radiation and chemotherapy*** compared with their non-tumorigenic progeny.

2013 *newsflash*: Women treated with radiation for breast cancer have increased risk of CVD.[3]

As published in the journal article, "**Risk of Ischemic Heart Disease in Women after Radiotherapy for Breast Cancer:**"

> **"Exposure of the heart to ionizing radiation during radiotherapy for breast cancer** increases the subsequent rate of ischemic heart disease. ***The increase is proportional to the mean dose to the heart, begins within a few years after exposure, and continues for at least 20 years***. Women with preexisting cardiac risk factors have greater absolute increases in risk from radiotherapy than other women.
>
> "In conclusion, we found that incidental exposure of the heart to radiotherapy for breast ***cancer increased the***

---

3 Darby, S., et al., *The New England Journal of Medicine* 2013; 368:987–998; Lagadec, C, et al., "Radiation-Induced Reprogramming of Breast Cancer Cells, *Stem Cells* **2012**;30:833–844.

rate of major coronary events by 7.4% per gray, with no apparent threshold. The percentage increase per unit increase in the mean dose of radiation to the heart was similar for women with and women without preexisting cardiac risk factors, which indicates that the absolute increases in risk for a given dose to the heart were larger for women with preexisting cardiac risk factors. Therefore, clinicians may wish to consider cardiac dose and cardiac risk factors as well as tumor control when making decisions about the use of radiotherapy for breast cancer."

Cancer

Oncology / Cardiology Newsflash: **Cancer patients at much greater risk for heart disease.** The Medscape article, "**Cancer Survivors at "Substantial" Risk of Cardiovascular Disease**" in **2009** makes clear:[4]

"**The largest cohort of childhood and adolescent cancer survivors ever** studied has indicated there is a **substantial risk of subsequent cardiac problems**, including congestive heart failure, myocardial infarction (MI), pericardial disease, or valvular abnormalities.

"***Cardiologists and oncologists will need to work together more and more*** due to the ***growing cardiotoxic risk associated with combination therapy for various malignancies***, say Dr Adriana Albini (Istituto di Ricerca e Cura a Carattere Scientifico MultiMedica,

4 http://www.medscape.com/viewarticle/713673?src=rss_

> Milan, Italy) and colleagues in the paper. "Today's oncologists must be fully aware of cardiovascular risks to avoid or prevent adverse cardiovascular effects, and ***cardiologists must now be ready to assist oncologists by performing evaluations relevant to the choice of therapy***," they write." The article summarizes the potential cardiovascular toxicities of a range of chemotherapeutics and chemopreventive agents and emphasize the importance of evaluating cardiovascular risk when patients enter into trials."

The cancer journal article's title is quite clear. "***Cardiotoxicity of anticancer drugs: the need for cardio-oncology and Cardio-oncological prevention***," (Albini A, et al., *J Natl Cancer Inst*. **2010** January 6; 102(1): 14–25). Its summary states:

> "Abstract: Due to the aging of the populations of developed countries and a common occurrence of risk factors, it is **increasingly probable that a patient may have both cancer and cardiovascular disease**. In **addition, cytotoxic agents and targeted therapies used to treat cancer**, including classic chemotherapeutic agents, monoclonal antibodies that target tyrosine kinase receptors, small molecule tyrosine kinase inhibitors, and even antiangiogenic drugs and chemoprevention agents such as cyclooxygenase-2 inhibitors, **all affect the cardiovascular system**. One of the reasons is that many agents reach targets in the microenvironment and do not affect only the tumor. **Combination therapy often amplifies cardiotoxicity, and radiotherapy [as detailed at the beginning of this chapter] can also**

cause heart problems, particularly when combined with chemotherapy.

"*In the past, cardiotoxic risk was less evident, but it is increasingly an issue, particularly with combination therapy and adjuvant therapy*.

"**Today's** ***oncologists must be fully aware of cardiovascular risks*** **to avoid or prevent adverse cardiovascular effects, and cardiologists must now be ready to assist oncologists by performing evaluations relevant to the choice of therapy. There is a need for cooperation between these two areas and for the development of a novel discipline, which could be termed cardio-oncology or onco-cardiology**…."

PEO Solution analysis: PEOs protect the cardiovascular system in patients undergoing any type of cancer therapy, positively assisting cancer patient mortality and increasing positive outcomes from therapy. PEOs are the ideal adjunct to any oncology protocol.

Cardiovascular disease

Newsflash: Cardiologists / Cardiovascular Disease Prevention: Parent omega-3 and Parent omega-6—not omega-3 series derivatives—**takes center stage** in the following 21st century analysis of elevated fibrinogen—the main component in coagulation processes. Although a critical substance to help cause clotting if you cut yourself to seal the wound, excess levels can cause buildup inside the vascular system. Furthermore, **fish**

oil is **not preferred—*LOW LEVELS* of EPA/DHA are *found most beneficial***. The **2010** journal article, "Elevated plasma fibrinogen caused by inadequate µ–linolenic acid [**Parent omega-3**] intake can be reduced by replacing fat with canola rapeseed [containing Parents] oil:"[5]

- "**Fibrinogen** is the main protein in coagulation processes and elevated levels, found in **prothrobotic and proinflammatory states**, and are associated with a **higher risk of CHD**, **strokes**, **diabetes**, and **Alzheimer disease and dementia**

- "…[M]ost of the **highest fibrinogen** values ***appeared at the lowest* range** of µ–linolenic acid [**Parent omega-3** and often combined with **low** LA (**Parent omega-6** levels.

- "…[D]emonstrates a new property for µ–linolenic acid [**Parent omega-3**] which is **physiologically of greater importance than** its own metabolism [**derivatives**.]

- "…[S]imilarly, a ***very low*, rather than a high EPA and DHA intake** combined with µ–linolenic acid [**Parent omega-3**] in most beneficial as regards the risk of CHD events.

- "µ–linolenic acid [**Parent omega-3**] should therefore be **the first 'omega-3' to be used in correcting the n-6/n-3PUFA imbalances in the body**."

---

5 Seppänen-Laasco, et al., *Prostaglandins, Leukotrienes and Essential Fatty Acids* 83 (**2010)**, 45-54.

Increased intake of alpha-linolenic acid (Parent omega-3) was associatedThe "Power of the Parents" was known in 2008…

Once again, marine oil was found worthless as this 21st century ***Circulation*** article attests:[6]

> "**Greater** alpha-linolenic acid [**Parent omega-3**] assessed either in adipose or by questionnaire was associated with **lower risk of myocardial infarction** [heart attack].
>
> "Similarly, **low intakes of alpha-linolenic acid** can be found in developing countries where **cardiovascular disease is on the rise**.
>
> "**Fish intake was similar in cases and controls**, and the variation within each group was large…. **Fish** or eicosapentaenoic acid [**EPA**] and docosahexaenoic acid [**DHA**] intake at the levels found in this population **did not modify the observed association**. [I want to make this very clear: **The amount of fish consumed didn't matter**. Given all of fish oils supposed miraculous claims, didn't these researchers wonder why? However, the researchers understand that the Parent omega-3 did something the derivatives didn't do.]
>
> "***Conclusions*** — In summary, **consumption of** vegetable oils rich in alpha-linolenic acid [**Parent omega-3**]could confer **important cardiovascular protection**."

---

6 Hannia Campos, PhD; Ana Baylin, MD, Dsc; Walter C. Willett, MD, DrPh, *Circulation*, **2008**;118:339-345.

This medical journal title says it all: "The beneficial effect of **α-linolenic acid** [**Parent oemga-3**] in coronary artery disease **is not questionable**."[7]

> "The **first prospective study** showing a beneficial effect of **ALA** (**Parent omega-3**) **on CAD** was conducted in **6250** middle-aged men of the usual care group of the Multiple Risk Factor Intervention Trial (1992).

"More recently, 2 large prospective studies in 76 283 nurses (1999) and 43 757 health professionals (1996) showed that **ALA** [Parent omega-3] was the only fatty acid that **protected against cardiac death and against nonfatal myocardial infarction, independently of other dietary or nondietary factors**." [Marine oils did nothing remarkable.]

2011 *Newsflash*: A Lower (glycemic) carbohydrate, higher protein diet is best to both prevent and slow tumor growth:[8]

"Abstract: Since **cancer cells depend on glucose more than normal cells**, we compared the effects of low carbohydrate (CHO) diets to a Western diet on the growth rate of tumors in mice. Taken together, our **findings offer a compelling preclinical illustration** of the ability of a **low CHO [carbohydrate] diet** in

---

7 Renaud, Serge, *Am J Clin Nutr* **2002**;76:903–6.

8 Ho, Victor, W., et al., "A Low Carbohydrate, High Protein Diet Slows Tumor Growth and Prevents Cancer Initiation," *Cancer Research*; 71(13), July 1, **2011**, pages 4484-4493.

not only **restricting weight gain** but also **cancer development and progression**.

> "Importantly, because glycolysis is far less efficient at generating ATP, most **cancer cells** require **higher levels of glucose** than normal cells to proliferate and survive, and this is why the glucose analog, fluorodeoxyglucose, is capable of detecting the majority of human tumors via positron emission tomography. [Note: Full details are in the book, *The Hidden Story of Cancer*, www.pinnacle-press.com.]
>
> "Consistent with this and our hypothesis that glucose supply is related to tumor growth, we found a ***positive correlation between plasma insulin levels and tumor size***. [Note: This supposed "hypothesis" is obvious. It is well known that tumors possess significantly more—on the order of 10-fold—insulin receptors than normal tissue. Respiration in the mitochondria is significantly impaired.]
>
> "Furthermore, **70%** (7 of 10) of mice on 5058 [**high carbohydrate diet**] developed **tumors** during their lifespan, with only 1 reaching normal life expectancy, whereas **less than 30%** (3 of 11) of the mice on the **15% CHO** diet developed **tumors**, with more than half reaching or exceeding normal life expectancy. Of note, in the 5 mice on the 15% CHO diet that exceeded normal life spans, only 1 had kidneys that showed above-

normal levels of protein in the urine. **These long-term mouse studies suggest that this 15% high *amylose* [easily digestible in humans] CHO, 58% protein, 26% fat diet is both safe and efficacious**."

PEO Solution analysis: This result is predictable. Because of the insulin response, *glycemic* carbohydrates—especially grains—make you fat unless immediately "burned" for energy. Cancer cells utilize glucose from carbohydrates as its prime fuel source. As expected, lactate levels were lower, too, confirming reduced tumor metabolism and growth. These facts are nothing new. The researchers mention that "high fat" is tumor promoting, but in research *adulterated cancer-causing* fats are nearly always used. **This experiment used 23% dietary fat.** Even with that amount, much of it adulterated, the results were still significant compared to the high carbohydrate diet.

Imagine this experiment's potential improvement with *fully functional, unadulterated* **PEOs. In fact, a seminal experiment was already performed with PEO pretreatment and the mice grafted with 2,000,000 cancer cells. See the extraordinary results at http://www.brianpeskin.com/BP.com/experiments.html and in the Scientific Support section.**

The above experimenter's results were consistent with the Korean study of cancer/blood glucose correlations. A superb **2005,** 10-year study of **over 1,000,000 people** confirms:[9]

- "…[T]he stratum [section] with the **highest fasting serum glucose** (>= 140 mg/dL (>=7.8mmol/L) **had higher death rates from all cancers combined ...** "
- "By cancer site, the association was strongest for pancreatic cancer… ***Significant associations*** were also found for cancers of the esophagus, liver, and colon/rectum, pancreas, and bile duct in men and of the liver and cervix in women, and there were ***significant trends*** **with glucose level for all cancers [referenced above]…"**
- "Of the 26,473 total cancer deaths in men and women 848 **[3.2%]** were estimated as attributable to having a **fasting glucose level of less than 90 mg/dL**."

The high blood glucose/increased cancer correlation was again confirmed and published in *PLoS Medicine* in December **2009**, which stated:[10]

- "**Glucose was significantly positively associated** with risk of overall incident and fatal **cancer**.

---

9 "Fasting Serum Glucose Level and Cancer Risk in Korean Men and Women," Sun Ha Jee, et al., *Journal of the American Medical Association* **2005**; 293:194-202.

10 Stocks, T., et al., "Blood Glucose and Risk of Incident and Fatal Cancer in the Metabolic Syndrome and Cancer Project (Me-Can): Analysis of Six Prospective Cohorts," PLoS Medicine | www.plosmedicine.org, December **2009** | Volume 6 | Issue 12 | e1000201.

- "In this large prospective cohort study, **elevated blood glucose was significantly** associated with an **increased risk of incident and fatal cancer at all sites combined** and of several specific cancers…."

- "**Results** from our study and those from the ***largest study reported to date***, on men and women in Korea, were **largely congruent** and together these studies provide **strong evidence that high blood glucose is a risk factor for cancer**…."

Here's another cancer journal confirmation you need to know: "Impact of Diabetes Mellitus on Outcomes in Patients With Colon Cancer," Jeffrey A. Meyerhardt, et al., *Journal of Clinical Oncology*, Vol 21, Issue 3 (February), 2003:433-440 states "Patients with diabetes mellitus and high-risk stage II and stage III colon cancer experienced a significantly higher rate of overall mortality and cancer recurrence…" and *National Cancer Institute (NCI Cancer Bulletin: Eliminating the suffering and death due to cancer)*, August 9, 2005, Volume 2/ Number 32 states in an article titled "New-Onset Diabetes is Possible Marker for Early Pancreatic Cancer," "This translates to a 3-year risk of pancreatic cancer of nearly 8 times higher than that of a person of similar age and sex in the general population, reported the scientists, lead by Dr. Suresh T. Chari."[11] Of course, Nobel Prize-winner Dr. Warburg, MD, PhD, in his brilliant paper, published in 1927, "The metabolism of tumors in the body," (*J Gen Physiol.* 1927 March 7; 8(6): 519–530) detailed how tumors voraciously want glucose.

---

11 Ref: *Gastroenterology* **2005,** Aug;129(2):504-11.

***Newsflash*... It is not open to discussion. If your patient has cancer, its severity will worsen with increased blood glucose levels.**

Newsflash 2010 Oncologist and Cardiologists need to know in patients treated with VEGF inhibitors...[12]

- "The VEGF [vascular endothelial growth factor] inhibitors are **potentially life-saving in the treatment of cancer**, the authors note, but their mechanism of action **can lead to cardiovascular adverse effects** which in some cases have led to treatment cessation and **even life-threatening consequences**
- "**Hypertension has been reported as an adverse event of all of these drugs**, and in some cases the BP elevation has been "dramatic," the authors write."

PEO Solution analysis: As the IOWA screening experiment clearly showed, PEOs are "the answer" to improved cardiovascular integrity. Increased arterial compliance mitigates potential adverse side effects.

12 Medscape published on May 14, **2010** an article by Zosia Chustecka, "New Recommendations for Monitoring BP in Cancer Patients on VEGF inhibitors" (http://www.medscape.com/viewarticle/721803_print). Ref.: *J Nat Cancer Inst*. **2010**;102;596-604.

Cardiovascular Disease

Next, we see the precise reason so many cardiovascular disease researchers get it completely WRONG:[13]

> "…[T]**he inconsistent results obtained in some studies with EPA and DHA could be attributed to** ***inadequate provision or utilization of n-6 fatty*** acids….
>
> "Under normal physiological conditions, **a balance is maintained between pro- and anti-inflammatory molecules**.
>
> "The **patchy manner in which atherosclerosis** occurs suggests that arterial walls undergo **regional disturbances** of metabolism that include the ***uncoupling of respiration and oxidative phosphorylation*** which may be characteristic of blood vessels being predisposed to the development of atherosclerosis.
>
> "…[A]therslerosis is a low-grade ***systemic inflammatory*** condition. One of the earliest signs of atherosclerosis is the development of ***abnormal mitochondria*** dysfunction triggers the disease. [Note: You'll recall mitochondrial *cardiolipin* requires fully functional Parent omega-6.]

---

13 Das, U.N., "A defect in the activity of D6 and D5 desaturases may be a factor in the initiation and progression of atherosclerosis," *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 76 (**2007**) 251- 268.

► **"Thus, on the whole, . . . EPA and DHA do not seem to have a very significant effect regarding blood lipids.**

"***Uncoupled respiration precedes atherosclerosis*** at lesion-prone sites **but not** at the sites **that are resistant to atherosclerosis**.

► **"Disease-free aortae have abundant concentration of the essential fatty acid-linoleate [Parent omega-6], whereas fatty streaks (an early stage of atherosclerosis) are deficient in EFAs [PEOs]."**

PEO Solution analysis: These researchers understand chronic inflammation. PGE_1. Is the body's most powerful anti-inflammatory—a metabolite of Parent omega-6. Sub-optimal amounts of *functional* Parent Omega-6 mislead researchers into (wrongly) thinking supra-pharmacologic amounts of omega-3 derivatives (marine oils) overcome this defect. WRONG, WRONG, WRONG… As was clearly stated, EPA/DHA aren't very significant regarding blood lipids. Later in this chapter you will discover the trials clearly detailing the importance of Parent omega-3, also. **Dr. Das suggests Warburg's *prime* cancer-cause, too—the uncoupling of cellular respiration**. Patients don't bleed to death from a wound because of this required life-saving *inflammatory response*. **Parent omega-6 and its derivative metabolites are the correct and natural anti-inflammatory answer.** Furthermore, fully functional Parent omega-6 stops atherosclerosis in its tracks. **PEO Solution** solves BOTH the *prime causes* of cancer and CVD.

Reversing Thrombosis (Blood Clots):

Cause of Thrombosis (Blood Clots): The (Esterified) LDL-C Connection

"Cholesterol esters are the predominant lipid fraction in all plaque types…

> Intimal [innermost arterial lining] macrophages contain substantial amounts of cholesterol esters, which are rich in PUFAs [PEOs]."[14]

PEO Solution analysis: The intima is all Parent omega-6—with virtually no Parent omega-3 or its derivatives. Cholesterol *esters* rich in PEOs are key.

More Confirmation It's the Unadulterated Parent Omega-6…

German physician Clause Weiss, MD, et al. states:[15]

> "In summary, infusion therapy with **PGE1** in patients with peripheral arterial occlusive disease (PAOD) ***reduces thrombin formation*** and results in a **decrease of fibrin degradation**. PGE1 may thus reduce fibrin

---

14 Felton CV, Crook D, et al., "**Relation of plaque lipid composition and morphology to the stability of human aortic plaque**," *Arteriosclerosis, Thrombosis, and Vascular Biology*, 1997;17:1337-1345.

15 "Hemostasis and fibrinolysis in patients with intermittent claudication: effects of prostaglandin E1," *Prostaglandins, Leukotrienes and Essential Fatty Acids*, Nov. **2000**; 63(5):271-277.

(thrombosis) deposition **involved in the pathogenesis of atherosclerosis**."

PEO Solution analysis: Because prostaglandin PGE1 is derived from Parent omega-6, ***unadulterated*** PEOs with a predominant LA (Parent Omega-6) component are the answer to Dr. Weiss' finding.

Newsflash: The April **2010** on-line journal for cardiologists, theheart.org -heartwire, had this amazing statement from Baylor College of Medicine's (Houston, Texas) Dr. Vijay Nambi:[16]

- "The **majority of heart attacks** that happen in the United States happen in people who are [supposedly] ***low or intermediate risk."***

PEO Solution analysis: The common measure for CVD risk must not be effective if most patients with heart attacks present with "low" or intermediate" risk. If the majority of CVD patients have so-called "low" or "intermediate" risk. Therefore, **the "standard risk factors" for CVD are WRONG**. This reminds me when my colleague in Italy, Stephen Cavallino, MD—Emergency Physician, told me that the **majority of heart attack patients had normal or low (LDL-C) cholesterol levels**—in the hospital every patient was measure on admittance. You have already discovered in Chapter 6 the true cause of CVD—*adulterated* Parent omega-6 from food ubiquitous processing. It is clear, that the greater the atherosclerosis, the less functional the

---

16 www.theheart.org, Lipid/Metabolic: "Carotid IMT and plaque presence improve prediction of coronary disease risk," April 9, 2010. Ref.: *Journal of the American College of Cardiology*, April 13, **2010**.

patient's Parent omega-6 is. Recall, intima, the innermost arterial lining contains no Parent omega-3 or omega-3 derivatives. GLA, an omega-6 derivative provides the basis for prostaglandin PGE_1—the body's most powerful anti-inflammatory.

PEO experiment in mice with cancer: http://www.brianpeskin.com/BP.com/experiments.html

---

Scientific Support for Chapter 13:

What Every Diabetic and "Pre-Diabetic" Needs to Know!

Kolata, Gina, "Research Questions Benefit of Low-Salt Diet, Drawing Criticism From C.D.C.," May 4, **2011**, page A17:[1]

- "A new study found that **low-salt diets** ***increase the risk*** of death from heart attacks and strokes and ***do not prevent high blood pressure***...
- "The investigators found that the **less salt** people ate, the **more likely they were to die** of heart disease...
- "'If the goal is to prevent hypertension' with lower sodium consumption, said the lead author, Dr. Jan A. Staessen, a professor of medicine at the University of Leuven, in Belgium**, 'this study shows it does not work.'**
- "...But, Dr. Alderman said, the new study is not the only one to find **adverse effects of low-sodium diets**. His own study, with people who had high blood pressure, found that **those who ate the** ***least salt*** **were** ***most likely to die***
- "...***Lowering salt consumption***, Dr. Alderman said, has consequences beyond blood pressure. It also, for example, ***increases insulin resistance***, which can increase the risk of heart disease."

---

1 Kolata, Gina, "Research Questions Benefit of Low-Salt Diet, Drawing Criticism From C.D.C.," May 4, **2011**, page A17.

The New York Times, **Gary Taubes, "Salt, We Misjudged You," June 3, 2012, pages 7-8":**[2]

- "Salt consumption **is said to** raise blood pressure, cause hypertension and increase the risk of premature death. This is why the **Department of Agriculture's** dietary guidelines still **consider salt Public Enemy No. 1**, coming before fats, sugars and alcohol. It's why the director of the **Centers for Disease Control and Prevention** has **suggested that reducing salt consumption is as critical** to long-term health **as quitting cigarettes**

- I was told then by Drummond Rennie, an editor for *The Journal of the American Medical Association*, that the ***authorities pushing the eat-less-salt message*** had "made a commitment to salt education that goes ***way beyond the scientific facts***.

- "A 1972 paper in *The New England Journal of Medicine* reported that the **less salt** people ate, the higher their levels of a substance secreted by the kidneys, called *renin*, which set off a physiological cascade of events that seemed to end with an **increased risk** of heart disease. In this scenario: ***eat less salt*, secrete more renin, get heart disease, *die prematurely***

- "When several agencies, including the Department of Agriculture and the Food and Drug Administration,

2 "Salt, We Misjudged You," June 3, **2012**, pages 7-8.

held a hearing last November to discuss how to go about getting Americans to eat less salt (as opposed to whether or not we should eat less salt), ***these proponents argued that the latest reports suggesting damage from lower-salt diets should simply be ignored***." [Note: Once again, we see opinion trumping valid medical science.]

Garg, R., et al., *Metabolism: clinical and experimental***, 60 (2011), 965-968, "Low-salt diet increases insulin resistance in healthy subjects":**[3]

- "Low dietary salt is recommended as one of the public health measures to decrease risk of cardiovascular disease. ***However, low salt intake stimulates aldosterone production*** ...We recently demonstrated an association between **aldosterone** and ***insulin resistance*** in healthy patients.
- "**Our study shows that low salt intake is associated with higher IR (insulin Resistance**)."

***The New York Times*, Kolata, Gina, October 19, 2012, page A17,** "Diabetes Study Ends Early With a Surprising Result":[4]

- "A large federal study of whether diet and weight loss can prevent heart attacks and strokes in overweight and obese people with Type 2 diabetes has **ended two years**

3 Garg, R., et al., *Metabolism: clinical and experimental*, 60 (**2011**), 965-968.

4 *The New York Times*, Kolata, Gina, October 19, **2012**, page A17.

ahead of schedule because the intensive program did not help.

- "...Like many, she had **assumed diet and exercise would help** ... "The study **randomly assigned 5,145** overweight or obese people with Type 2 diabetes to either a rigorous diet and exercise regimen or to sessions in which they got general health information. The diet involved 1,200 to 1,500 calories a day for those weighing less than 250 pounds and 1,500 to 1,800 calories a day for those weighing more. ***The exercise program was at least 175 minutes [nearly 3 hours] a week of moderate exercise***

- "But **11 years after** the study began, researchers concluded it was **futile to continue —** the two groups had **nearly identical rates of heart attacks, strokes and cardiovascular deaths**."

Diabetes induced Eczema and dry skin take a hike...:[5]

- "**Dermatitis** is consistently the first sign of EFA [PEO] deficiency in both animals and humans. **Itch** is the symptom that seems to respond most to GLA [an omega-6 derivative]. **There is also a significant reduction in the need for potentially harmful steroids**.

- "It was first reported in the **1950**s that **diabetic animals** ***required much more*** **linoleic acid [Parent omega-6]**

5 Horrobin, David, F., "Fatty acid metabolism in health and disease: the role of Δ6-desaturase," *American Journal of Clinical Nutrition*, *American Journal of Clinical Nutrition*, 1993:57(suppl):732S-737S.

than did normal animals. Linoleic acid [Parent omega-6] concentrations are almost always normal or slightly above normal in **diabetic patients**, whereas the concentrations of linoleic acid **metabolites are consistently below normal**.

► "Perhaps the main problem in the management of diabetes is the development of **long-term damage to the retina, the kidneys, the cardiovascular system, and the peripheral nerves**. Although there are many hypotheses, none have found universal acceptance and treatment is generally unsatisfactory. Good control of blood glucose may be beneficial, **but many well-controlled diabetics develop severe complications** whereas some poorly controlled diabetics do not. [Note: good control alone is insufficient as clearly demonstrated in recent studies like ACCORD presented at the end of this section.]

- "It is possible that the **increased requirement for EFAs is an important factor in the development of diabetic complications**. Neurophysiologically detectable ***damage to nerve function occurs in more than 90% of diabetics*** **The neuropathy leads to many further complications** including skin ulceration, limb amputation, impotence, and bladder."

Dr. Horrobin continues in another medical journal article:

- "There are ***multiple abnormalities of EFA-eicosanoid metabolism in diabetes*** because 5-desaturation is also impaired and there is a block in the conversion of DGLA

to PGE1. Because of this impaired 6-desaturation, **diabetics require higher amounts of EFAs than non-diabetics**. There have been several successful attempts to manage diabetic complications by the provision of very high levels of linoleic acid [Parent omega-6] intake.

▶ "These have shown convincingly that the development of ***cataract***, of ***retinopathy*** and of **cardiovascular damage** can all be ***slowed or stopped*** by the administration of large **daily doses of LA [Parent omega-6]**.

• "Of all the complications of diabetes, the **nerve damage** (**neuropathy**) seemed the one most likely to respond to GLA [**Parent omega-6 derivative**] treatment. This is because EFAs are required for neuronal structure, because second messengers derived from EF As such as PGE1 or diacylglycerol are required for normal neuronal function, and because nerve **microcirculation is also important in nerve function**. **Stiff red cells, such as might result from reduced amounts of EFAs in the membranes**, would impair nerve circulation and lead to a **reduced supply of oxygen** and nutrients.

▶ "**No one reviewing this literature** can have any doubts that GLA [Parent omega-6 derivative] is a **potent treatment** for experimental **inflammatory and auto immune disorders**…."

Controlling blood glucose alone (even with tight control of BP and cholesterol) wasn't sufficient, as this quote from *The New England of Medicine* publishing the seminal ACCORD study states:[6]

> "**Medical experts were stunned**. '**It's confusing and disturbing that this happened**,' said Dr. James Dove, president of the American College of Cardiology. 'For 50 years, we've talked about getting blood sugar very low. Everything in the literature would suggest this is the right thing to do,' he added. Among the study participants who were randomly assigned to get their blood sugar levels to nearly normal, ***there were 54 more deaths than in the group whose levels were less rigidly controlled***. The patients were in the study for an average of *four years* when investigators *called a halt* to the intensive blood sugar lowering and put all of them on the less intense regimen."

Dr. Horrobin has more to say:[7]

> "*Diabetes.* The decline of 6-desaturation in animal models of diabetes has been repeatedly and thoroughly documented both *in vitro* and *in vivo*. ***In human diabetics, the fatty acid compositions of plasma, serum, red cells and platelets are also consistent with impaired formation of GLA*** **[Parent omega-6 derivative]**. The

---

6 *The New England Journal of Medicine*, 2008; 358:2545-2559.

7 Horrobin, D.F., "Nutritional and medical importance of gamma-linoleic acid," *Prog. Lipid Res.*, Vol. 31, No. 2, pages 163-194, 1992.

low levels of unsaturated fat in blood in diabetes were actually noted as long ago as 1928! Administration of insulin to human diabetics changes the plasma fatty acid composition in a manner consistent with stimulation of 6-desaturation."

Cellular insulin Sensitivity Increases:[8]

"EPO (Evening Primrose oil) [**Parent omega-6 and GLA**] feeding for 4 months **significantly reduced erythrocyte membrane microviscosity** in the diabetic patients. EPO feeding also **dramatically increased the prostaglandin E_1** (PGE_1) on diabetic erythrocyte membranes to normal levels.

"We have also shown that **prostaglandin E_1** (PGE_1) receptor in human erythrocyte **membrane positively modifies the insulin effect on membrane microviscosity by lowering the physiological concentration of insulin needed** to produce a given effect.

► "…[E]rythrocyte membranes prepared from diabetic patients **show only 42% of the PGE_1 binding activity found in controls."**

---

8 Dutta-Roy, Asim, "Effect of Evening Primrose Oil Feeding on Erythrocyte Membrane Properties in Diabetes Mellitus," *Omega-6 Essential Fatty Acids: Pathophysiology and Roles in Clinical Medicine*, 1990, pages 505-511 (out of print).

"The Gracey HYPO-thesis" *for the CAUSE and CURE of Diabetes...*

After studying the field for over a decade, I have come to the conclusion that "Diabetes is caused by ***eating high glycemic foods too*** often which reduces **brain/nerve insulin production**. Glucose entry is increased into [and storage in] any 'peripheral tissues' having insulin receptors. Overeating of carbohydrates (*glycemic* insulin-generating foods) is a main cause of the worldwide-obesity epidemic. **Prof. Peskin's PEO discovery helps patients decrease their carbohydrate craving and cravings for sweets**, along with **improving cell membrane functionality so LESS INSULIN is REQUIRED.** It was previously thought that the brain was insulin-insensitive. This was wrong, very wrong. Neural cells require it.

Relative **hypoglycemia is** caused by eating too often—especially meals relatively high in *glycemic* [raising blood glucose] carbohydrates. ***Neuroglycopenia*** is a shortage of glucose (glycopenia) in the brain, usually ***due to hypoglycemia*** (a "relative" glucose low)**.** When anyone eats, especially carbohydrates/glucose, the bloodstream becomes flooded with it. As a supra-physiologic response, the pancreas releases insulin in response to increasing concentrations of glucose. Insulin forces glucose into [and storage in] many 'peripheral tissues.'

As a result, *blood glucose concentration drops*, often resulting in *relative* hypoglycemia. *Relative* hypoglycemia results in **brain & nerve cells being deprived** of the glucose that has been 'drained' out of the bloodstream, by the insulin, for storage and use in other tissues.

► This is why the typical very poor "high/low" glycemic control in patients is disastrous. For example, a 300 mg/dl reading dropping to 120mg/dl is a definite "relative low."

In an effort to allow sufficient glucose to the brain, the body compensates, by becoming '*insulin resistant*'—the body is protecting neural synapses—but at a large price. With this mechanism, there is now a higher glucose concentration remaining in the bloodstream to help better supply the glucose-fuel needs of the brain & nerves. I term that process ***'compensatory hyperglycemia.'*** Whenever anyone trying to prevent relative hypoglycemia consumes excess glycemic carbohydrates, their blood glucose concentration rises temporarily, to an above-the-national-average concentration [transient supernormal glycemia *aka* TSG] in order to help prevent relative-hypoglycemia particularly in **type 1/ type 2** diabetics.

When anyone, especially children, eats too often, the **pancreatic beta-cells** become **'inflamed'** [less functional] in order to help **reduce insulin production**

and increase 'compensatory hyperglycemia' attempting to best protect the brain/nervous system [e.g., type 1A diabetes]. **Eating less OFTEN** increases 'localized' brain/nerve insulin production ['fine-tuning' of brain/nerve glucose metabolism] without the typically associated blood glucose "lows."

Therefore, the CURE for diabetes is to eat less OFTEN—along with optimum PEO consumption— ensuring maximum cellular insulin response. This guarantees fewer rapid reductions in brain/nerve glucose concentration and therefore **fewer episodes of *relative* hypoglycemia so the pancreatic beta cells don't become "inflamed**."

When an aging adult eats too often too, the brain/nerve cells can become chronically 'starved & inflamed' from lack of 'localized' brain/nerve insulin production, and increase 'compensatory loss-of-appetite' / 'loss-of-memory-to-eat' / 'eating less often' [type 3A diabetes], to help protect the brain from potential SEVERE chronic *relative* hypoglycemia.

Diabetes can now be seen as a protective cycle that can be controlled / mitigated and/or cured, by intermittent-fasting [which also increases liver 'digestion efficiency'], a ketogenic / low glycemic diet ensuring enough energy from patients' stored body fat, and eating high glycemic foods less frequently—all of which the **PEO Solution** assists with.

Arteries and Insulin Resistance: *Medical News Today* **reported:**[1]

> "Earlier studies showed that in the context of systemic *insulin resistance*, blood vessels become resistant, too. But it wasn't clear if arteries become diseased because ***they can't respond to insulin*** or because they get exposed to too much of it. Now comes **evidence in favor of the former explanation**.
>
> "Insulin-resistant blood vessels don't open up as well, and levels of a protein known as VCAM-1 increase, too. VCAM-1 belongs to a family of **adhesion** molecules… The animals' [mice] **insulin-resistant arteries** develop **plaques that are twice the size of those on normal arteries**."

---

1 "Arteries and Insulin Resistance," May 5, **2010**, http://www.medicalnewstoday.com/articles/187720.php.

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:[2]

> "An international team of researchers that reviewed data from several trials found that taking calcium **supplements was linked to a higher risk of** heart

2 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.

attack **and other cardiovascular events**; the authors called for new research to **re-assess** the **role of calcium supplements** in the treatment of osteoporosis.

"...[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...[3]

- "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.**

---

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

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.

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:[4]

> "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**:[5]

> "**Telomerase** is a specialized reverse transcriptase **capable of extendin**g the 3′ end of chromosomes **by adding** TTAGGG repeats.

---

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

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

"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 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."[6]

> "Telomere **shortening** is **counteracted** by the cellular **enzyme telomerase**. "[7]

The **2010** article, "Is telomere length a biomarker of aging? A review," has this to say:[8]

> "Although telomere length is implicated in cellular again, the **evidence suggesting telomere length is a biomarker of aging** in humans is **equivocal**. These

---

6 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.

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

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

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*.
