WARNING: FISH OIL CONTAINS NO TRUE EFAS—PHYSICIANS MAY BE UNKNOWINGLY PRESCRIBING THE WRONG SUBSTANCE TO PATIENTS CAUSING GREAT HARM—PEOS SOLVE THIS PROBLEM

© *By Prof. Brian Peskin, B.Sc., USA*

Abstract

Physicians and their patients are being misled. Tragically, physicians are prescribing what they think are essential fatty acids (EFAs) to their patients, but they aren't; instead, they are unknowingly prescribing "derivatives" of EFAs, consisting of enormous supra-physiologic overdoses of EPA and DHA.

Aside from the brain and nervous system, which comprise only 3% of total body weight, there are normally only small, trace amounts of these derivatives in the plasma, cellular membranes, and tissues of the human body. Fish oil supplements, however, supply EPA and DHA in supra-physiologic amounts often in excess of 100-fold or even 500-fold amounts more than the body would ever naturally produce on its own. This mistake of recommending a derivative when the "parent" EFA—Parent Essential Oil or (PEO)— is necessary is why fish oil supplements consistently fail to prevent cardiovascular disease (CVD), fail to prevent cancer, and significantly worsen diabetic patients by raising blood sugars and blunting the insulin response.

To the contrary, organic, unprocessed, fully functional parent essential oils, linoleic acid (LA) and alpha linolenic acid (ALA) in the correct physiologic ratio of 1:1–2:1 (LA:ALA)—containing more parent omega-6 than parent omega-3—can both prevent and reverse existing cardiovascular disease, as evidenced by the landmark IOWA study; prevent and slow existing cancerous tumor growth; and also significantly enhance cellular insulin sensitivity.

Glossary

Essential fatty acids (EFAs): Essential fatty acids are those fatty acids (FAs) that cannot be synthesized by the body's tissue; i.e., linoleic acid and linolenic acid.

Omega-6 fatty acids: These constitute a family of unsaturated fatty acids all of which have a final carbon–carbon double bond in the n-6 position—i.e., the sixth bond, counting from the methyl group of the molecule.

Omega-3 fatty acids: These constitute a family of unsaturated fatty acids all of which have a final carbon–carbon double bond in the n-3 position—i.e., the third bond, counting from the methyl group of the molecule.

Derivatives (omega-6 and omega-3 fatty acids): Derivatives are molecules synthesized from linoleic or linolenic acid that contain more carbon–carbon double bonds and a higher number of carbon atoms (in units of two). The most commonly known omega-3 derivatives are DHA (docosahexaenoic acid) and EPA (eicosapentaenoic acid).

Parent essential oils (PEOs): These are unadulterated oils (meaning no chemical processing or excessive heat treatments) containing those fatty acids that are parents of all the omega-6 and omega-3 fatty acids—linoleic acid and linolenic acid.

Peskin Protocol PEOs: A formulation based on PEOs in which the ratio of linoleic to linolenic acid is balanced for proper physiologic tissue functionality.

Introduction

Quantum mechanics in physics was invented to explain just one experiment. As Nobel Prize winner in physics Richard Feynman aptly stated, "The sole test of the validity of any idea is experiment…. We choose to examine a phenomenon which is impossible, *absolutely* impossible,

to explain in any classical way, and which has in it the heart of quantum mechanics."[1] Similarly, the notion that fish oil improves cardiovascular health is destroyed with one recent seminal experiment with physiologic parent essential oils (PEOs), called "Investigating Oils With respect to Arterial blockage (IOWA)."

Parent Essential Oils (PEOs) are Physiologic; DHA and EPA are not Physiologic: They are EFA Derivatives

Because fish oil supplies not parent EFAs but omega-3 series derivatives, with DHA and EPA being the significant components, in pharmacological overdoses—100-fold to 500-fold—fish oil cannot prevent or reverse disease via established metabolic pathways. Fish oil's consistent, multiple failures to prevent or treat disease has been published extensively in the world's leading medical journals, for those who care to look: failure of fish oil to prevent or reverse cardiovascular disease,[2] failure to prevent or reverse cancer,[3] and failure in treating diabetes.[4] In fact, *fish oil makes these three medical conditions worse*! The Centers for Disease Control and Prevention estimate 14% of Americans are now diabetic (diagnosed and undiagnosed). Is the rampant use of fish oil supplements in part to blame? Absolutely.

Physiologic Parent Essential Oils vs. Non-Physiologic EFA Derivatives Like DHA and EPA

Why does fish oil consistently fail in medical experiments? Because its constituents are not physiologically required. There are only two essential fatty acids: the parent omega-6, linoleic acid (LA) and the parent omega-3, alpha linoleic acid (ALA); both must come from food.[5] To work properly, they must be fully physiologically functional, i.e., not heated, not chemically processed, and from organically produced sources to guarantee full physiologic functionality.

Only LA and ALA are essential; all other EFA-derived substances are correctly termed EFA "derivatives." This includes the most common derivatives: DHA, EPA, GLA, AA, etc. Most physicians do not understand that derivatives are made in the body from the parent EFAs on an "*as-needed basis*" in *extremely limited quantities*. Consumption of derivatives from food is therefore unnecessary. Fish oil's "active ingredients" consist entirely of DHA and EPA in supra-physiologic overdoses, thereby overdosing the patient and causing damage instead of health.

A Prime Cause of Disease: *Adulterated* PEOs—in Particular, Parent Omega-6 (LA)

The use in fast foods of *adulterated, non-functional* EFAs that can no longer be termed fully functional parent essential oils is a prime cause of our nation's ill health.

How and Why PEOs Work When Omega-3 Derivative Fish Oil Fails

Physiology is the prime science utilized to determine tissue amounts of parent omega-6, parent omega-3, and their derivatives. Once these values are known, biochemistry may then be utilized. Fortunately, these physiologic values have already been determined, making it immediately obvious why fish oil can't possibly work as claimed. First, aside from the brain and nervous system, the body has little use for EPA/DHA. The prime overlooked issue is that the body can easily support the brain and nervous system requirements for these derivatives with adequate PEOs without the risk of overdosing on EPA/DHA. Contrary to what physicians have been told, the elongating delta-6 and delta-5 desaturase enzymes are not impaired in the vast majority of patients. Furthermore, the physiologic amounts of EFA-derivatives are naturally extremely small. In fact, over 95%-99% of parent EFAs stay in parent form. *There is a maximum of < 2% natural conversion of ALA to the derivatives EPA and DHA and this conversion is easily achieved in the general population.*[6] *Even babies adequately convert PEOs into derivatives.*[7] With the great fish oil fallacy, these significant physiologic facts have been obscured.

Parent PEOs are considerably more important compared to derivatives, and no one until now has focused on them. One hundred trillion cell membranes contain an abundance of PEOs in significant favor of parent omega-6 with no EFA derivatives.[8] Furthermore, as Nobel Prize winner Otto Warburg, M.D., Ph.D., discovered, the structural functionality of cellular membranes are key,[9] and *unadulterated, fully functional* LA (parent omega-6) directly incorporates and allows oxygen into each cellular membrane as well as their trillions of cellular mitochondria.[10] There is much less cellular ALA than LA. The tables below exemplify key tissue and plasma physiology.

Few, if any, physicians ask to see the "normal standard" values of physiologic EPA/DHA amounts in tissue and plasma compared to the parent PEO amounts in tissue and plasma. When they discover the truth, they are often shocked and dismayed that they have been (unknowingly) harming their patients, and wish to correct their recommendation based on human physiology.

Derivative EFAs Are Only Made in Extremely Small Amounts

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

Table 1. Omega-6/-3 ratios for various body tissues.[12]

Ratio of Tissue Composition			
Tissue	Percentage of Total Body Weight	Omega-6 PEO	Omega-3 PEO
Brain/Nervous System	3	100	1
Skin*	4	1000	1
Organs and Other Tissues	9	4	1
Adipose Tissue (body fat)	15-35	22	1
Muscles	50	6.5	1

* There is virtually NO omega-3 in skin tissue.

22:6n-3 [DHA]." Steady-state homeostatic plasma conditions were used in this excellent analysis. Mean transit times and half-lives of the individual fatty acids were also utilized.[11]

When tissue amounts and ratios are calculated, we see that humans require at least a 11:1 LA:ALA ratio, highly in favor of parent omega-6 (*Table 1*).

With all the focus on omega-3 fatty acids today, it is significant to note that the free fatty acids in human plasma ordinarily are composed of about 15% LA (linoleic acid, parent omega-6) and just 1% of ALA (alpha linolenic acid, parent omega-3), a 15:1 ratio (*Table 2*). Derivative amounts are so insignificant that they are not listed. As seen above, highly detailed experiments show a miniscule 0.046% net production of DHA from parent substrate ALA,[13] i.e., a minimum of a *2,600-fold greater amount of parent ALA than its metabolite, DHA*.

LDL-Cholesterol Transports PEOs

Cholesterol esters and plasma phospholipids have ratios of 100:1 in favor of parent omega-6. Esterified cholesterol is the transporter of PEOs. *The entire issue with LDL-cholesterol lies in the adulteration of these PEOs—in particular, LA, not the cholesterol structure itself.*[14] Physicians need to know these important and critical facts. *Also, all the food processing and adulteration leading to impaired PEO functionality is directed solely at the parent omega-6 cooking oils; the parent omega-3 oils* aren't used by food processors, because they are far too reactive.

Amounts of EPA/DHA in Fish Oil—Pharmacological Plasma Overdoses of Each

An average 1,000 mg, health-food-grade fish oil capsule contains approximately 180 mg EPA and 120 mg DHA. Pharmaceutical-grade versions contain higher doses. As an example, using the USDA food composition research formulas covered earlier, if you consumed a supplement of 600 mg of parent ALA, you would naturally convert it to EPA by no more than the (generous) factor of 0.25% = 1.5 mg EPA and 1.5 mg x 0.63 x 0.37 = 0.35 mg to DHA in your plasma. Therefore, just one capsule provides the following amounts, and many people are overdosing even more by taking 2 to 4 fish oil capsules each day, likely in part because the American Heart Association recommends "EPA + DHA ranging from 0.5 to 1.8 grams per day." This equates to the following *plasma overdoses*: EPA = 180 mg/1.5 mg = *120 times overdose; DHA* = 120 mg/0.35 mg = *340 times overdose*.

As a specific example, [redacted] a well-known brand of fish oil, contains [redacted] mg of EPA and [redacted] mg of DHA, with insignificant amounts of other omega-3 derivatives. The company recommends two capsules each day for a total of [redacted] mg EPA and [redacted] mg DHA each day.

In comparison, 3 grams of Peskin Protocol PEOs per day is the general prophylactic dosage. The amount of parent omega-3 (ALA) contained is approximately 1,000 mg. Given the (generous) theoretical maximum 2% conversion into the omega-3 derivative DHA means the body *naturally converts* only a maximum of 20 mg to DHA. Contrast this to the fish oil dosage of 450 mg, i.e., *a DHA pharmacologic overdose by a factor of at least 20-fold, if not the 200-fold amount from the USDA analysis above!* The

Table 2. Percentages of linoleic and alpha linolenic acid in plasma.[15]

Percentages of Linoleic Acid (LA) & Alpha Linolenic Acid (ALA) in Plasma and Classes of Lipids				
Fatty Acid	Plasma % (Unesterified)	Plasma % Triglycerides	Plasma % Phospholipids	Plasma % Cholesterol Esters
LA (parent omega-6)	17	19.5	23	50
ALA (parent omega-3)	2	1.1	0.2	0.5
Parent omega-6: Parent omega-3 Ratio	**8.5:1**	**17.5:1**	**115:1**	**100:1**

EPA overdose is even worse, as only a (generous) theoretical maximum of 0.26% of ALA is normally converted. Of the 1,000 mg of ALA in Peskin Protocol PEOs, just 2.6 mg is converted to EPA, whereas the fish oil supplement provides 650 mg or a *250-fold* pharmacological overdose of EPA. Would you ever consider giving a patient 250 aspirin tablets? Of course not!

Krill Quickly Decomposes

Krill tastes awful and is not normally human food but is still promoted by supplement manufacturers. Why is krill oil the current "oil du jour?" Could it possibly be it is cheap and plentiful, which has little to do with what a human being actually requires for good health? The presentation delivered at the 99th American Oil Chemists' Society (AOCS) stated, "Krill decompose very quickly, so the current thinking is either to dry them aboard the vessel and bring the powder back to a land-based plant for oil extraction or to enzymatically digest the krill and then separate the oil." While krill oil has less of the overdose amounts (approximately 130 mg EPA and 70 mg DHA per capsule), it is still excessive and potentially harmful.

Given these facts, is it any wonder that fish oil categorically fails in experimental medical tests? Recommending derivative EFA overloads without compensating PEOs or omega-6 based derivatives is even worse, because of the gross disparity between the critical omega-6 and omega-3 series derivative balance. Fish oil is harmful to most patients and the *IOWA study confirms fish oil's enormous negative effect in the cardiovascular area*.

Fish Does Contain Significant EPA/DHA Yet Does Not Stop Disease

Unlike fish oil, fresh fish contains significant amounts of both EPA and DHA. However, fresh fish is unprocessed and unadulterated. Regardless, fish's derivative EPA/DHA overload is physiologically problematic. Fish consumption is not required in the human diet, and there are cultures that eat no fish, such as the Hunza, who exist isolated from the rest of the world in the Himalayan Mountains in Pakistan.

The problem with fish is that even though the majority of *wild* species contain the proper PEO balance of more parent omega-6 than parent omega-3, *fish often contains far too many omega-3 series derivatives* as shown at the USDA Database: http://www.nal.usda.gov/fnic/foodcomp/search/. This derivative overdose is one of the primary reasons that consuming fish does not prevent cancer, as evidenced by Japan's skyrocketing cancer rate.

If more fish was the answer to better health then countries consuming the most fish, like Japan, should see significantly less disease rates. Unfortunately, this is not true. Their cancer rates are higher, not less. Japanese consume the ideal wild, unprocessed fish— sushi. *Yet, in spite of this, Japan cancer ranks first in leading cause of death since 1981.* In 2002 cancer accounted for over 30% of their total number of deaths, as verified from Vital Statistics of Japan, Statistics and Information Department, Minister's Secretariat, Minister of Health, Labour and Welfare. Heart disease and cerebrovascular disease was next. *In 2002 Japan had 241/100,000 population cancer deaths and America had 194/100,000 population—a 24% greater rate /100,000 population more cancer deaths than Americans from cancer!* Fish consumption is not the anti-cancer answer.

Clinical Application: Landmark IOWA Study—Pulse Wave Velocity/Photoplethysmography (PTG) and Arterial Compliance

Hardening of the arteries, i.e., arteriosclerosis, is a prime cause of cardiovascular disease and patient death.[16] Just like a scale directly measuring weight requires no interpretation, patient arterial compliance (flexibility) measurement, termed *photoplethysmography,* requires no interpretation.

This technology is now available yet underpublicized because successful intervention and metabolic treatment did not exist until now. Beta-blockers and ACE inhibitors do not increase arterial compliance. A 2007 *Clinical Medicine* article points the way to better clinical treatment of CVD, stating: "Arterial stiffness measured by pulse wave velocity (PWV) is an *accepted, strong, independent predictor of cardiovascular events and mortality*."[17]

Anesthesiologists are well aware of this technology, used for monitoring purposes. While pulse oximetry became standard in the operating room and in other critical care areas as a detector of inadequate oxygen levels in the blood (hypoxemia)—all pulse oximeters are fundamental photoelectric plethysmographs—PWV has been largely ignored. This is unfortunate, as noninvasive PWV (plethysmographic) information itself may provide important clues regarding the CV condition of the patient.[18] Digital pulse analysis (DPA) based on photoplethysmography (PTG) is the next evolution in pulse wave velocity (PWV), and is based on the measurement of reflected infrared light (IR). PTG has been validated for accurately calculating systemic arterial compliance (flexibility).[19]

The recent 2010 article, "Arterial Stiffness and Cardiovascular Events: The Framingham Heart Study," published by Gary F. Mitchell, MD and his colleagues, makes the case for a new intervention, stating: "In this study, we assessed the incremental value of *adding pulse wave velocity [PWV]* ... to a risk model that includes standard risk factors for a first cardiovascular event…. Adding pulse wave velocity led to significant reclassification of risk and improvement in global risk prediction. … [W]e need to *focus our efforts on identifying and implementing interventions that can prevent or reverse abnormal aortic stiffness* in order to prevent a marked increase in the burden of disease potentially attributable to aortic stiffness."[20] PEOs are the new proven answer to reversing hardening of the arteries, as seen below.

Anti-Aging Cardiovascular Effect—IOWA Study[21]

Short-term (3-month) PEO use

The effects of short-term PEO supplementation were evaluated in 16 subjects with a daily dosage of 2,900 mg PEO formulation. The sub-groups were as follows: 7 male subjects and 9 female subjects, aged 46-84, with a *median age of 64 years old*, utilizing the formulation of a median of 2.5 months usage (half of the subjects with less duration and half of the subjects with more duration) and mean average of 3 month's usage. Minimum PEO formulation usage was 1 month and the maximum subject usage was 8 months PEO usage. Vascular assessment was made via photoplysmography measuring arterial flexibility.

Overall short-term improvement = 43% effectiveness—highly significant

Of the 16 subjects in the trial, 7 improved. *This corresponds to a forty-three per cent (43%) measurable effectiveness rating over a very short period of time*. The average improvement in arterial flexibility was 7.2 years, meaning the average subject utilizing the PEO formulation had a cardiovascular system with the arterial flexibility of a younger subject by 7.2 years (p = .001; *Figure 1*). This is a significant anti-aging effect.

NNT effectiveness = 2.3—a "remarkable" result

The number needed to treat (NNT) is calculated as follows: 16 subjects/7 improved subjects = *2.3*. NNT quantifies how many patients have to be treated to obtain one successful outcome. An NNT of less than 50 is considered effective in the pharmaceutical industry. An outcome of 2.3 is outstanding.

Comparison to Statins

As a comparative example, statins, as reported by the pharmaceutical industry, have NNTs > 80 in preventing a cardiovascular event. This means a minimum of 80 patients would need to be treated to see a single positive outcome. In contrast, the PEOs improve a *much more direct physiologic measurement*, i.e., arterial flexibility, in a profound way, resulting in the *remarkable 2.3 NNT*.

Next, we looked at subjects previously consuming fish oil supplements before replacing them with PEOs.

Subjects Discontinued Fish Oil Supplementation, Replacing it with PEO Formulation

PEOs versus fish oil

The effects of the PEOs were evaluated in subjects who

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

Short Term Results — With Baseline

IOWA: Investigating Oils With Respect to Arterial Flexibility
Significant differences in biological age compared to physical age (short-term)
Brian Peskin, BSEE: Founder: Life-Systems Engineering Science with David Sim, M.D., Interventional Cardiologist
(Based on 16 patients using the PEO formulation 1 month - 8 months)

Age: 46-84	**Median age: 64**	9 females, 7 males

Paired t-test. Median: 2.5 months PEO formulation use / Mean: 3 months PEO formulation use

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

Note: This experiment has a 99.00% accuracy—5 times more accurate than the 5% standard error used in most clinical trials. Therefore, this result is *not* due to possible error and is significant with patient CV health 7.2 years better than physical age predicts.

Analysis by Alex Kiss, Ph.D. (statistics) — March 26, 2010
Analysis Variable : agediff

N	**Mean**	Std Dev	Pr > |t|
16	**-7.24**	10.19	0.0099

Figure 1. Results of the IOWA study.

ceased fish oil supplementation, replacing it with a daily dosage of 2,900 mg PEO formulation. The effects of the PEO formulation were measured in 15 subjects: 7 male subjects and 8 female subjects aged 46-74, with a *mean age of 60 years old*, utilizing the formulation of an average duration of 3.5 months. Vascular assessment was made via photoplethysmography measuring arterial flexibility.

Overall Improvement

Of the 15 subjects in the trial, 13 improved with the PEOs for an *87% effectiveness* rating and an NNT of 15/13 = 1.2. *Improvement (anti-aging effect) was 11.1 years* as measured by standard population samples.

On average, the PEO formulation quickly improved the cardiovascular system's arterial flexibility by over 11 years (younger) in the subjects. Thirteen subjects improved; one (1) subject remained the same, one (1) subject worsened by 1 year. Results were highly statistically significant *($p = 0.0001$)—99.99% accuracy* (*Figure 2*).

Subjects with "high cholesterol"

Of the 7 subjects previously diagnosed with high cholesterol levels, replacing fish oil supplements with the PEO formulation resulted in improvement in cardiovascular biological ages in 6 subjects. This translates to an *NNT of 7/6 = 1.2* for improvement in cardiovascular system compliance in subjects with high cholesterol manifestations of heart disease.

Subject with both diabetes and "high cholesterol"

One subject having both diabetes and high cholesterol diagnosis also improved.

Comparison to statins

In contrast to the NNTs > 80 of statins in preventing a cardiovascular event, PEOs in this experiment improved arterial flexibility — a *remarkable 1.2 NNT*.

Statin user improvements

Two patients were taking statins and both subjects improved their biological age by 20 years for an *NNT = 1 in those patients taking statins*.

PEOs trump fish oil supplements

Arterial compliance with PEOs increased in the fish oil pre-treatment group compared to the group taking nothing before PEO implementation. *Note 2* gives four first-rate experiments, published in world-class medical journals, in which fish oil failed to either prevent or reverse cardiovascular disease. To the contrary, fish oil often accelerated disease progression. Therefore, *since the positive difference was greater in those taking fish oil prior to taking the PEOs*, the only possible *conclusion is that treatment with fish oil decreases arterial compliance (harmful)*, increasing "hardening of the arteries," i.e. arteriosclerosis, making your patient's probability of CVD greater, not

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

Analysis by Alex Kiss, Ph.D. (statistics) — August 20, 2010

Mean of BIO_AGE_W_FO variable		Mean of BIO_AGE_PEO variable	
Analysis Variable: BIO_AGE_W_FO		Analysis Variable: BIO_AGE_PEO	
Mean	Std Dev	Mean	Std Dev
49.20	11.33	38.07	8.12

Paired t-test run: mean change (FO - PEO) was found to be 11.1 (sd=8.4).
This was statistically significant (p=0.0001)

Analysis Variable: diff

Mean	Std Dev	t value	Pr > |t|
11.13	8.37	5.15	0.0001

Figure 2. Biological age, fish oil versus PEO.

less. Fish oil is once again, proven harmful.

In view of these findings, the IOWA results clearly show that the PEO formulation is superior to fish oil supplements in preventing and reversing cardiovascular disease—a remarkable anti-aging effect. In fact, as this experiment definitely shows, fish oil worsens arterial compliance because the improvement is greater with fish oil taken before PEO implementation than taking nothing!

PEOs—the Answer to Diabetes and Its Complications

Diabetes has become an American epidemic over the last 60 years. Clearly, too many processed carbohydrates are one cause but there has to be more to this puzzle. *Insulin resistance* in all diabetics is directly caused by an impacted cell membrane, and PEOs positively impact this impairment.[22] Cardiovascular disease is significantly increased in diabetics and PEOs are a beneficial treatment for the reasons discussed above.

Prostacyclin, an omega-6 metabolite from arachidonic acid (AA), is decreased in diabetics and this decrease accelerates platelet adhesion and aggregation.[23] Neuropathy and capillary abnormalities such as retinopathy are all improved with increased blood flow and increased oxygenation, alleviating cellular hypoxia. Prostaglandin E1 (PGE_1), an omega-6 metabolite, is the body's most potent natural anti-inflammatory. PGE_1 increases a diabetic's (impaired) membrane fluidity, and increases membrane insulin sensitivity. Diabetic patients benefit from numerous important metabolic pathways: in particular, through the omega-6 series PEOs and their metabolites.[24]

PEOs—the Anti-cancer Answer

Nobel Prize winner Otto Warburg, MD, PhD, conclusively proved that cancer's *prime* cause is *always* lack of cellular oxygen. A sustained hypoxic 35% cellular oxygen decrease always induces cancer.[25] This was verified by American physicians and scientists back in 1953 and again in 1955.[26] Causes of chronic inflammation all relate back to the *lack of cellular oxygen*. Today, we know how to solve the cellular oxygen impairment caused by common, everyday food processing, with PEOs, as proved by the following clinical *in vivo* experiment in mice.

Clinical Experimental Proof of PEO Effectiveness in Slowing Tumor Growth

In 2004 we commissioned an experiment with mice at an independent laboratory experienced in oncology studies. The purpose of the experiment was to show whether pretreatment with organic raw parent EFA oils in the ratios and amounts comparable to our human recommendations, prior to *implantation of breast cancer tumors* in the mice, affected the growth of the cancer cells in any way. A breast cancer strain was chosen because breast cancer is the number one worldwide cause of death by cancer in women. Mice were used because they respond very similarly to humans regarding EFA metabolism and because their shorter lifespan allows all effects and results to occur more quickly.

One group of mice was pretreated with the PEO oils for 4 weeks prior to tumor implantation (Group 2, showing the greatest inhibition of tumor growth at the bottom of the graph in *Figure 3*), followed by a daily dose

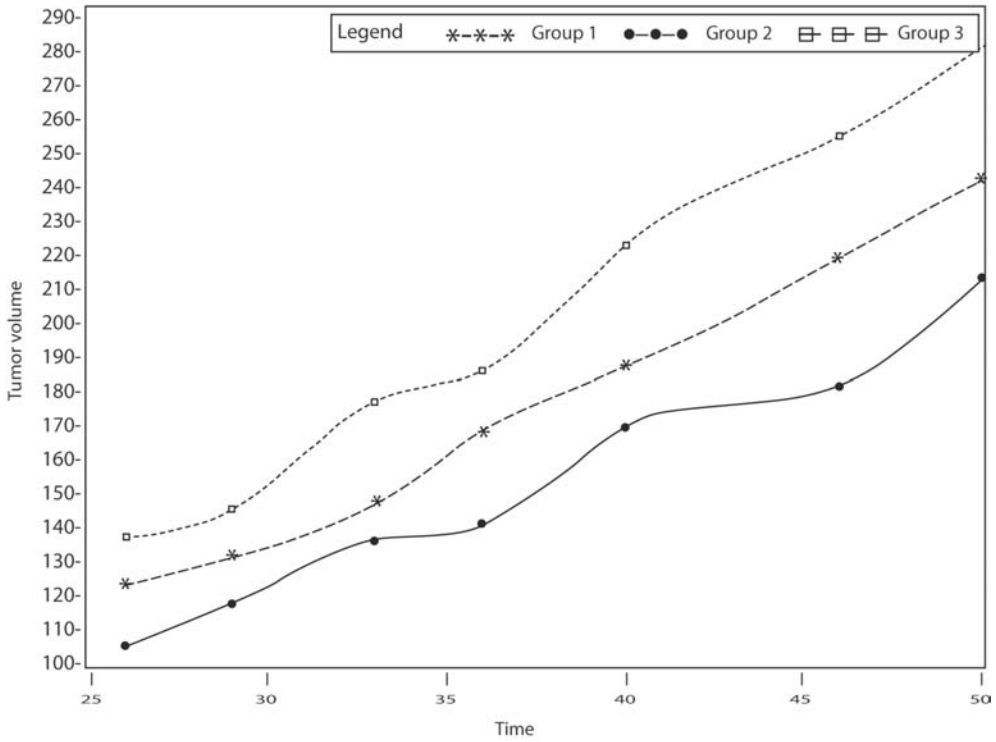

Figure 3. Plot of tumor volumes over the course of the experiment for the three groups.

(5 days a week) for 50 days after implantation of the cancer tumors. A second group was pretreated with PEO oils for 2 weeks prior to implantation (Group 1, showing less tumor growth at the middle of the graph) and then given the daily dose for the rest of the 50 days. The third group, the control group (Group 3, showing uncontrolled tumor growth at the top of the graph), was not pretreated or given any PEOs at all.

Tumors consisting of 2 million breast cancer cells each were implanted in the mice and measurement began. The statistical analysis focused on the period from day 26 to 50 to ensure that any hormonal changes and other transitory effects that occurred after implantation had stabilized (as recommended by Dr. Warburg). An independent expert in statistical analysis calculated and reported the results.

The statistical *F* test was used to determine the significance. The experiment showed that although the tumors continued to grow in all mice, there was a highly significant 24% smaller tumor size (growth) in the longer 4-week pretreatment mice than in the control mice that received no PEO oils at all. This result occurred consistently upon measurements at the day 26 endpoint, the final endpoint (day 50), and at every intermediate point. This same effect occurred with the 2-week pretreatment group, although to a lesser extent than the 4-week pretreatment group, as would be expected. Note: All mice responded positively to PEO treatment, and no mice were eliminated from the study for any reason.

Additionally, in the last 10 days of the experiment, *there was a 42.8% lower growth volume of the tumors in the 4-week pretreated mice than in the tumors in the untreated mice*.

These results clearly demonstrate and prove PEOs' value increases with longer pretreatment. A logical conclusion from this result would be that the EFA oils are modifying the cells' internal structure, making them more cancer resistant.

Clinically, *Which PEO Ratio Is Best?*

What PEO ratio is best? A balanced physiologic blend of PEOs that allows the body to make EFA derivatives "as needed."

Organic flax oil is appropriate for the parent omega-3 component, but offers insufficient parent omega-6. High linoleic (not high oleic) strains of sunflower and safflower are fine for parent omega-6; high oleic strains cannot be used because there will not be a sufficient quantity of LA. Detailed analysis and calculations show that the parent omega-6/omega-3 ratio must be in the range of *2:1 to 1:1 in favor of parent omega-6*.[27] No omega-3-based derivative fish oil is to be used, few EFA derivatives, and only a conservative amount of GLA from evening primrose oil. Note: organic evening primrose oil produces approximately 15 times more arterial outflow of the powerful anti-inflammatory PGE_1 from its GLA content than borage oil.[28]

This unique, plant-based, patent-pending combination is termed "Peskin Protocol PEOs." Clinicians can use this new physiologic approach to both prevent and treat cardiovascular disease, cancer, and diabetes in their patients regardless of patient diet or existing complications.

Look to future Explore! issues for other articles in our continuing series on Essential Fatty Acids (EFAs).

ABOUT THE AUTHOR

Prof. Brian Peskin, B.Sc., is a world-leading scientist specializing in physiologic parent EFAs—termed *PEOs*—and their direct relationship to the prevention of both cancer and cardiovascular disease. He graduated from M.I.T. with a degree in electrical engineering, and received an appointment as an adjunct professor at Texas Southern University in the Department of Pharmacy and Health Sciences (1998-1999). The former president of the University said of Brian's discoveries: "... His nutritional discoveries and practical applications through Life-Systems Engineering are unprecedented."

Prof. Peskin's current work, grounded strictly in state-of-the-art science—in particular, physiology—can be found in his seminal work *The Hidden Story of Cancer* and peer-reviewed medical journal articles. Clinical physicians throughout the world rely on and have validated Prof. Peskin's PEO recommendations.

While advancing the scientific understanding of the role of essential fatty acids in the body's metabolic pathways, he has concurrently developed a means for alleviating cancer's *prime* cause, as postulated by Nobel Prize winner Otto Warburg, M.D., Ph.D., by increasing cellular oxygenation. Amazingly, there is a fundamental cancer/heart disease connection, in which the same physiologic solution, parent essential oils, PEOs, solve both conditions. This information leads to a new understanding of how to treat and prevent both cancer and heart disease.

In the most exciting development to date, Brian's theoretical conclusions were recently completely validated in a physiological experiment by precise instrumentation capable of measuring arterial compliance. This experiment, Investigating Oils With respect to Arterial flexibility (IOWA study) provides the first conclusive clinical proof and validation of Prof. Peskin's theory. Peskin Pharmaceuticals has a patent pending on the medicament that embodies this development.

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NOTES

1. Feynman, R. Six Easy Pieces: Essentials of Physics Is Explained by its Most Brilliant Teacher. New York, NY: Addison-Wesley, 1995, 36, 117.
2. Sacks F. M., P. H. Stone, C. M. Gibson, D. I. Silverman, B. Rosner, and R. C. Pasternak 1995. Controlled trial of fish oil for regression of human coronary atherosclerosis. *J Am Coll Cardiol* 25: 1492-8; von Schacky, C., P. Angerer, W. Kothny, K. Theisen, and H. Mudra. 1999. The effect of dietary omega-3 fatty acids on coronary atherosclerosis: A randomized, double-blind, placebo-controlled trial. *Ann Intern Med* 130: 554-62; Angerer, P., W. Kothny, S. Störk, and C. von Schacky. 2002. Effect of dietary supplementation with omega-3 fatty acids on progression of atherosclerosis in carotid arteries. *Cardiovasc Res* 54: 183-90; Knapp, H., I. A. Reilly, P. Alessandrini, and G. A. FitzGerald. 1986. In vivo indexes of platelet and vascular function during fish-oil administration in patients with atherosclerosis. *New Engl J Med* 314: 937-42.
3. Calder, P. C. 2000. Omega-3 polyunsaturated fatty acids, inflammation and immunity. International Society for the Study of Fatty Acids and Lipids (ISSFAL), 4th Congress, June 9, 2000, Tsukuba, Japan; MacLean, C. H., S. J. W. A. Mojica, et al. 2006. Effects of omega-3 fatty acids on cancer risk: A systematic review. *JAMA* 295: 403-15; National Cancer Institute. Omega-3 fatty acids unlikely to prevent cancer. 2006. *NCI Cancer Bull* 3 (Jan. 31).
4. Stacpoole, P., A. Alig, L. Ammon, and E. Crockett. 1989. Dose-response effects of dietary marine oil on carbohydrate and lipid metabolism in normal subjects and patients with hypertriglyceridemia. *Metabolism* 38: 946-56; 2003. Delarue, J., F. Labarthe, and R. Cohen. Fish-oil supplementation reduces stimulation of plasma glucose fluxes during exercise in untrained males. *Br Med J Nutr* 90: 777-86.
5. Burr, G. O., and M. O. Burr. 1929. A new deficiency disease produced by the rigid exclusion of fat from the diet. *J Biol Chem* 82:345-67.
6. Sinclair, A. J., N. M. Attar-Bashi, and D. Li. 2002. What is the role of alpha-linolenic acid for mammals. *Lipids* 37: 1113-23; Salem, Jr., N., J. Yuhong Lin, T. Brenna, R. J. Pawlosky. Alpha-linolenic acid conversion revisited. PUFA Newsletter. Available at http://www.fatsoflife.com/article.php?nid=1&id=15&issueid=31&edition=arch; Crawford, M. A. 2000. Commentary on the workshop statement. Essentiality of and recommended dietary intakes for omega-6 and omega-3 fatty acids. *Prostaglandins Leukot Essent Fatty Acids* 63: 131-4; Barceló-Coblijn, G., E. J. Murphy, R. Othman, M. H. Moghadasian, T. Kashour, and J. K. Friel. 2008. 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. *Am J Clin Nutr* 88: 801-9; Hussein, N., E. Ah-Sing, P. Wilkinson, C. Leach, B. A. Griffin BA, and D. J. Millward. 2005. Long-chain conversion of [13C]linoleic acid and alpha-linolenic acid in response to marked changes in their dietary intake in men. *J Lipid Res* 46: 269-80; Demmelmair, H., B. Iser, A. Rauh-Pfeiffer, and B. Koletzko. 1999. Comparison of bolus versus fractionated oral applications of [13C]-linoleic acid in humans. *Eur J Clin Invest* 29: 603-9.
7. Carnielli, V. P., D. J. Wattimena, I. H. Luijendijk, A. Boerlage, H. J. Degenhart, and P. J. Sauer.1996. The very low birth weight premature infant is capable of synthesizing arachidonic and docosahexaenoic acids from linoleic and linolenic acids. *Pediatr Res* 40: 169-74.
8. Murray, R. K., D. K. Granner, P. A. Mayes, and V. W. Rodwell. Harper's Illustrated Biochemistry. 26th edition. New York, NY: McGraw-Hill: 2003, 418; Sinclair, H. M. 1980. Prevention of coronary heart disease: the role of essential fatty acids. *Postgrad Med J* 56: 579-84; Meisenberg, G., and W. H. Simmons. Principles of Biomedical Chemistry. First edition. New York: Mosby, 1998, 226.
9. Warburg, O. The Metabolism of Tumours: Investigations from the Kaiser Wilhelm Institute for Biology. F Dickens (trans). London: Constable & Co Ltd., 1930, 56.
10. Campbell, I. M., D. N. Crozier, and R. B. Caton. 1976. Abnormal fatty acid composition and impaired oxygen supply in cystic fibrosis patients. *Pediatrics* 57: 480-486; Murray, R. K., D. K. Granner, P. A. Mayes, and V. W. Rodwell. Harper's Illustrated Biochemistry. 26th edition. New York, NY: McGraw-Hill: 2003, 93; Ibid., 97; Ibid., 191; Guyton, A., and J. Hall. 9th ed. Textbook of Medical Physiology. Philadelphia, PA, WB Saunders, 1996, 16; 861-2.
11. Pawlosky, R. J., J. R. Hibbeln, J. A. Novotny, and N. Salem Jr. 2001. Physiological compartmental analysis of alpha-linolenic acid metabolism in adult humans. *J Lipids Res* 42: 1257-65.
12. Spector, A. A. 2001. Plasma free fatty acids and lipoproteins as sources of polyunsaturated fatty acid for the brain. *J Mol Neurosci* 16: 159-65; discussion 215-21; "Most of the plasma free fatty acid (EFA) is derived from the triglycerides stored in the adipose tissue [body fat]." Note: Organs, including the brain, use these EFAs for structural incorporation; Chapkin, R. S., V. A. Ziboh, C. L. Marcelo, and J. J. Voorhees. 1986. Metabolism of essential fatty acids by human epidermal enzyme preparations: evidence of chain elongation. *J Lipid Res* 27: 945-54; Makrides, M., M. A. Neumann, R. W. Byard, K. Simmer, and R. A. Gibson. 1994. Fatty acid composition of brain, retina, and erythrocytes in breast- and formula-fed infants. *Am J Clin Nutr* 60:189-94; Andersson A, Nälsén C, Tengblad S, Vessby B. 2000. Fatty acid composition of skeletal muscle reflects dietary fat composition in humans. *Am J Endocrinol Metab* 279: E744-51.
13. See note 11.
14. Peskin, B. S., and D. Sim. 2008. Vytorin failure explained—A new view of LDL. *Townsend Lett Phys* (June): 101-112.
15. Waddington, E., K. Sienuarine, I. Puddey, and K. Croft. 2001. Identification and quantification of unique fatty acid and oxidative products in human atherosclerotic plaque using high-performance liquid chromatography. *Anal Biochem* 292: 234-44.
16. Peskin, B. S., and R. J. Rowen. 2010. Breakthrough in clinical cardiology: In-office assessment with pulse wave velocity (PWV) and digital pulse analysis (DPA). *Townsend Lett Phys* (May): 80-86.
17. Khoshdel, A. R., Sl L. Carney, B. R. Nair, and A. Gillies. 2007. Better management of cardiovascular diseases by pulse wave velocity: combining clinical practice with clinical research using evidence-based medicine. *Clin Med Res* 5:45-52.
18. Nijboer, J. A., J. C. Dorlas, and H. F. Mahieu. 1981. Photoelectric plethysmography—some fundamental aspects of the reflection and transmission method. *Clin Phys Physiol Meas* 2: 205-15; Shelley, K. H., M. Dickstein, and S. M. Shulman. 1993. The detection of peripheral venous pulsation using the pulse oximeter as a plethysmograph. *J Clin Monit* 9: 283-7.
19. Cohn, J., S. Finkelstein, G. McVeigh, et al. 1996. Noninvasive pulse wave analysis for the early detection of vascular disease. *Hypertension* 26: 503-8.
20. Mitchell, G. F., S. J. Hwang, R. S. Vasan, et al. 2010. Arterial Stiffness and Cardiovascular Events: The Framingham Heart Study," *Circulation* 121: 505-11.
21. Full details of the landmark IOWA study are available at http://brianpeskin.com/BP.com/studies-experiments/IOWA-Study-Results.pdf.
22. See note 9.
23. Siegel, G., F. Schnalke, K. Rückborn, J. Müller, and R. Hetzer. 1992. Role of prostacyclin in normal and arteriosclerotic human coronary arteries during hypoxia. *Agents Actions Suppl* 37: 320-32.
24. Horrobin, D. F. *Omega-6 Essential Fatty Acids: Pathophysiology and Roles in Clinical Medicine.* New York: Wiley-Liss, 1990: 497-503; 506-510.
25. Warburg, O. The Metabolism of Tumours: Investigations from the Kaiser Wilhelm Institute for Biology. F Dickens (trans). London: Constable & Co Ltd., 1930, 56; Warburg, O. 1956; On the origin of cancer cells. *Science* 123: 309-14; Peskin, B. S. The Hidden Story of Cancer. Houston, TX: Pinnacle Press, 2009; Peskin, B. S., and M. J. Carter. 2008. Chronic cellular hypoxia as the prime cause of cancer: What is the de-oxygenating role of adulterated and improper ratios of polyunsaturated fatty acids when incorporated into cell membranes? *Med Hypotheses* 70: 298-304.
26. Goldblatt, H., and C. Cameron. 1953. Induced malignancy in cells from rat myocardium subjected to intermittent anaerobiosis during long propagation in vitro. *J Exp Med* 97: 525-52; Malmgren, R. A., and C. C. Flanigan. 1955. Localization of the vegetative form of Clostridium tetani in mouse tumors following intravenous spore administration. *Cancer Res* 15: 473-8.
27. Peskin, B. S. *The Scientific Calculation of the Optimum PEO Ratio.* Houston, TX: Pinnacle Press, 2008.
28. Horrobin, D. F. Omega-6 Essential Fatty Acids: Pathophysiology and Roles in Clinical Medicine. New York: Wiley-Liss, 1990: 44-5.