

Chapter 6: Scientific Support

Testimonial from Robert L. Kagan, Medical Director of MRI Scan and Imaging Center about results of PEO regiment



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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)

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Testimonial from Robert L. Kagan, page 2

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

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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/fm

Scientific Support for Chapter 6

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

“Professor Peskin and Dr. Rowen have added solid proof to previous work (Peskin) that Parent Essential Oils [PEOs] are the way to go when considering fatty acid supplementation.”

– Rob Krakovitz M.D. -- Preventive Medicine

“Hi , my name is Diane , I am 50 years old and **have had eczema all my life** , especially on my hands. I **have seen numerous dermatologists**, who prescribed all sorts of medications. **Occasionally I would get temporary relief** and thought I had found the “cure.” My **symptoms would always return**. A little over a year ago my husband started me on an EFA supplement following Prof. Peskin’s recommendations that has the proper ratio of Parent omega-6 to Parent omega-3. **He explained to me that my body’s skin requires mainly Parent omega-6 , which I never heard from any medical doctor**. I always felt that I needed to improve my skin from the inside out, but I didn’t know what my body required to achieve that. **Following the Professor’s recommendations has cleared my skin--I would say 95 % better than before. *I know for a fact that the PARENT ESSENTIAL OIL is what is making my skin improve because when we run out the eczema starts to return.*** I think if I can improve my diet I can improve my skin and my general health even more. Thank you Prof. Peskin for your honesty and courage.

Very sincerely,

Diane J. (e-mail: 2013)

Fats fulfill our appetite

2008 Newsflash: Just like fruits fulfill our natural “sweet tooth,” fats fulfill our appetite. Fats—not protein or carbohydrates.¹

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)**...

1 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.

- “...[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.**
- “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 polyunsaturated 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 *unadulterated* 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 states²:

“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

2 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 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!

“I can attest to the the power of PEOs again. I stopped taking all pills because of I have been very sick and trying to get two major assignments done I **just forgot to take them.** Today, I had brain fatigue. This is where your brain is exhausted and highly limits as to what you can do for the day. **I took the PEOs and the feeling disappeared in 30 minutes.”**

—John M. -- Canada (e-mail:2014)

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*:³

“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 gastrointestinal 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.

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

“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.’*”

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- ▶ “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.*]
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“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.]
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“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 PGE1 levels in rats. There can therefore be no doubt that GLA enhances the rate of formation of this very desirable substance.

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- ▶ **“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.

“I’m 17-years old. Never in my life have I ever gotten a compliment on my skin (besides; omg ur so tan!) as I did a couple weeks ago. **Completely cleared my skin acne**

scars and all. I have not gotten a pimple or any of that mess in months”

– Andrew F. -- Canada (e-mail: 2014)

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.**”⁴ 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:

- “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**...
- “Wealthier peoples are becoming increasingly sensitive 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.

4 April 7, 1956, pages 381–383.

- “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:⁵

“...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.*”

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- ▶ “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.**”
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5 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.”]

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 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.

Newsflash 2014: PEOs don't contributed to excess bodyfat and significantly increase muscle formation. This new study confirms what Dr. Kasper understood and published back in 1973. (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.)

Abdominal fat accumulation prevented by unsaturated fat

Date: February 24, 2014

Source: Uppsala University

New research from Uppsala University shows that saturated fat builds more fat and less muscle than polyunsaturated fat. This is the first study on humans to show that the fat composition of food not only influences cholesterol levels in the blood and the risk of cardiovascular disease but also determines where the fat will be stored in the body. The findings have recently been published in the American journal *Diabetes*.

The study involved 39 young adult men and women of normal weight, who ate 750 extra calories per day for seven weeks. The goal was for them to gain three per cent of their starting weight. The project received considerable attention when it started in 2011, partly because the extra calories were ingested in the form of muffins with high fat content, baked in the lab by Fredrik Rosqvist, a doctoral candidate and first author of the study.

One half of the subjects were random to eat surplus calories from polyunsaturated fat (sunflower oil), while the other half got their surplus calories from saturated fat (palm oil). Both diets contained the same amount of sugar, carbohydrates, fat, and protein; the only difference between muffins was the type of fat.

The increase in body fat and the distribution of fat in the body was measured using a magnetic resonance imaging (MRI scans) before and after the weight gain, as was the muscle mass in the body. Gene activity was measured in the abdominal visceral fat before and after the weight gain with the help of a gene chip that studies several thousand genes at a time.

Despite comparable weight gains between the two diet groups, the surplus consumption of saturated fat caused a markedly greater increase in the amount of fat in the liver and abdomen (especially the fat surrounding the internal organs, visceral fat) in comparison with the surplus consumption of polyunsaturated fat. Moreover the total amount of body fat was greater in the saturated fat group, while, on the other hand, the increase in **muscle mass was three times less for those who ate saturated fat compared with those who ate polyunsaturated fat [PEOs]**. Thus, gaining weight on excess calories from polyunsaturated fat caused more gain in muscle mass, and less body fat than overeating a similar amount of saturated fat. Since most of us are in positive energy balance, and consequently gain weight slowly but gradually over time, the present results are highly relevant for most Western populations.



Despite comparable weight gains between two diet groups in this study, the surplus consumption of saturated fat caused a markedly greater increase in the amount of fat in the liver and abdomen (especially the fat surrounding the internal organs, visceral fat) in comparison with the surplus consumption of polyunsaturated fat.

Credit: © Ljupco Smokovski / Fotolia

Scientific Support for Chapter 6

<http://www.sciencedaily.com/releases/2014/02/140224110017.htm>

"Liver fat and visceral fat seems to contribute to a number of disturbances in metabolism. These findings can therefore be important for individuals with metabolic diseases such as diabetes. If the results regarding increased muscle mass following consumption of polyunsaturated fat can be confirmed in our coming studies, it will potentially be interesting for many elderly people, for whom maintaining muscle mass is of great importance in preventing morbidity," says Ulf Risérus, associate professor at the Department of Public Health and Caring Science and director of the study.

When it comes to the risk of developing diabetes and cardiovascular diseases, it seems more important where in the body the fat is stored than how much fat the body has. Visceral fat, along with a high proportion of fat in the liver, is closely associated with increased risk for developing type-2 diabetes. These fat depots are therefore important targets for new drugs and dietary strategies. A number of studies have indicated that a higher intake of polyunsaturated fats from plant oils and nuts is associated with a decreased risk of type-2 diabetes, but the reasons for this remain unclear.

The present study proposes a potential explanation for such an association, showing that polyunsaturated fatty acids can affect fat distribution in the body more favorably than saturated fats, probably by regulating increased energy combustion or decreased storage of visceral fat in connection with calorie-rich diets.

The researchers were also able to see that over-consumption of saturated fats seems to be able to "turn on" certain genes in fatty tissue that increase the storage of fat in the abdomen and at the same time hamper insulin regulation. Polyunsaturated fats, instead, can "turn on" genes in visceral fat that in turn are linked to reduced storage of fat and improved sugar metabolism in the body. However, more research is required to understand how this occurs in humans.

The discovery may also be a contributing factor regarding the tendency of some individuals to accumulate fat in the liver and abdomen. The new findings suggest that the fat composition of the diet, in the long term, might play a role in preventing obesity-related disorders, like type-2 diabetes, at an early stage, before overweight develops.

"This is of great interest, as we lack preventive treatments for fatty liver and visceral fat today. The new findings also support international dietary recommendations including the new Nordic nutritional recommendations, which, among other things, recommend replacing some saturated fat from meat, butter, and palm oil, for example, with unsaturated fats from plant oils and fatty fish," says Ulf Risérus.

The next step is now to find out in greater detail what happens in the body when we eat the respective fats and to study what the effects are in overweight individuals with elevated risk of type-2 diabetes.

Story Source:

The above story is based on [materials](#) provided by [Uppsala University](#). *Note: Materials may be edited for content and length.*

Journal Reference:

1. Fredrik Rosqvist, David Iggman, Joel Kullberg, J Jonathan Cedernaes, Hans-Erik Johansson, Anders Larsson, Lars Johansson, Håkan Ahlström, Peter Arner, Ingrid Dahlman, and Ulf Risérus. **Overfeeding Polyunsaturated and Saturated Fat Causes Distinct Effects on Liver and Visceral Fat Accumulation in Humans**. *Diabetes*, February 2014 DOI: [10.2337/db13-1622](https://doi.org/10.2337/db13-1622) [1939-327X](https://doi.org/10.2337/1939-327X)

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Uppsala University. "Abdominal fat accumulation prevented by unsaturated fat." ScienceDaily. ScienceDaily, 24 February 2014. <www.sciencedaily.com/releases/2014/02/140224110017.htm>.

Peskin Protocol: Adjunct Therapy for Use with Chemotherapy and Radiation

May 2008 it was brought to my attention by the superb radiologist, Robert Kagan, M.D., Medical Director of MRI Scan & Imaging Centers in Ft. Lauderdale, Florida, that **increased cellular oxygen increases the effectiveness of both chemotherapy and radiation treatments in destroying cancer cells.**

It was extremely gratifying to learn of this, since the Peskin Protocol is designed to increase cellular oxygen throughout the body, including at cancerous sites.

James B. Mitchell, Ph.D., head of the tumor biology (NCI-radiation biology branch) section at the National Cancer Institute, reported in an article published by Radiological Society of North America (4/23/2008):

· "...[T]hey were able to successfully measure oxygen levels in tumors,' which could be important because **'tumors with higher concentrations of oxygen [are] more susceptible to radiation.'**

· **"Lower oxygen level 'in the tumor allows tumor cells to survive more easily by making the DNA destruction more difficult.'**

· **"'Chemotherapy drugs also don't work as well when tumors have less oxygen.'" (emphasis added)**

I immediately began searching medical journal articles to see if this critical concept was well-understood. The following comments comprise a (small) representative sample of what I found:

RADIATION ADJUNCT THERAPY:

· **"A large body of published evidence points to tumor hypoxia as a major obstacle to effective treatment of tumors using ionizing radiation^{a,b} because cells exposed to radiation under hypoxic conditions are approximately thrice [3 times] more resistant than when treated under aerobic conditions.^c (emphasis added)**

· **"Despite significant evidence of a role of hypoxia [low cellular oxygen] in cellular resistance to ionizing radiation-induced toxicity, the underlying molecular mechanisms remain unclear. This study focused on the influence of hypoxia on radiation-induced signals in TK6 human lymphoblastoid cells.^d (emphasis added)**

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CHEMOTHERAPY ADJUNCT THERAPY:

- “Solid tumors frequently contain **large regions with low oxygen concentrations** (hypoxia). The hypoxic microenvironment induces adaptive changes to tumor cell metabolism, and this alteration can further distort the local microenvironment. The net result of these tumor specific changes is a microenvironment that **inhibits many standard cytotoxic anticancer therapies [“chemotherapy”] and predicts for a poor clinical outcome.**^e (emphasis added)
- “**Hypoxia** and anemia (which contributes to tumor hypoxia) can lead to ionizing radiation and **chemotherapy resistance** by depriving tumor cells of the oxygen essential for the cytotoxic activities of these agents. Hypoxia may also **reduce tumor sensitivity** to radiation therapy and **chemotherapy** through one or more indirect mechanisms that include proteomic and genomic changes.^f (emphasis added)

^a Eric, E., “The oxygen effect and reoxygenation.” In: *Radiobiology for the radiologist*. Philadelphia: JB Lippincott Co.; 1994. p. 133–52.

^b Samuni, A., et al., “Effects of Hypoxia on Radiation-Responsive Stress-Activated Protein Kinase, p53, and Caspase 3 Signals in TK6 Human Lymphoblastoid Cells,” *Cancer Res* 2005; 65(2): 579-86.

^c Brown, J., “Tumor microenvironment and the response to anticancer therapy,” *Cancer Biol Ther*; 2002;1:453–8.

^d Samuni, A., et al., “Effects of Hypoxia on Radiation-Responsive Stress-Activated Protein Kinase, p53, and Caspase 3 Signals in TK6 Human Lymphoblastoid Cells,” *Cancer Res* 2005; 65(2): 579-86.

^e Cairns, R., et al., “Metabolic targeting of hypoxia and HIF1 in solid tumors can enhance cytotoxic chemotherapy,” *Proceedings of the National Academy of Science*, May 29, 2007; vol. 104, no. 22: 9445–9450.

^f Harrison, L., Blackwell, K., “Hypoxia and Anemia: Factors in Decreased Sensitivity to Radiation Therapy and Chemotherapy?,” *The Oncologist* 2004;9(suppl5):31-40.