

2010-2011 Updates



**The
Hidden Story of
Cancer**

**Find Out Why Cancer has
Medical Science on the Run
and How a Simple Plan Based on
New Science Can Prevent It**

By

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Table of Contents

Appendix X, 2010 (Excerpts)

- *Genetic markers fail to predict cardiovascular risk in women.....* 595
- *Cancer as a metabolic disease* 603
- *How Asbestos Causes Cancer: Mystery Unraveled.....* 609

Appendix XII, 2010: Cancer Prevention and the 15-Lipoxygenase Pathways (Especially for Cancer Researchers and Cancer Physicians)

- *Lipoxygenase Enzymes and Their Relationship to Colorectal and Prostate Cancer* 615
- *Lipoxygenase Enzymes and Their Relationship to Pancreatic Cancer* 617
- *Lipoxygenase Enzymes and Their Relationship to Gastric cancer* 619
- *Lipoxygenase Enzymes and Their Relationship to Human Breast and Esophageal Cancer.....* 620

Appendix XIII: DHA and Fish Oil Shown Completely Worthless — Five More Fish Oil Failures

- *Fish Oil Increases Platelet Aggregation.....* 623
- *Fish Oil Worthless in Decreasing Abnormal Heartbeat....* 624
- *Fish Oil Impairs Regulation of Insulin Secretion* 624
- *Fish Oil Increases Risk of Colon Cancer.....* 625
- *Fish Oil Worthless with Alzheimers* 628

Appendix XIV: Everyone Can Now Rest Easy—It's RARELY Genetic...

- *Genetics accounts for no more than 10% (the vast MINORITY) of disease.....631*
- *"... the genome contains far more inconvenient truths than was supposed a decade ago. The very idea of what we inherit and what we pass on has changed."634*

Appendix X

2010 Updates (Excerpts)

2010 Newsflash: WOMEN—Genetic markers FAIL AGAIN!

The February 16, 2010 cardiology article titled “Genetic Markers Fail to Predict Cardiovascular Risk in Women,” makes it clear¹:

- “Creation of a **genetic risk score comprised of multiple genetic markers associated with cardiovascular disease (CVD)** was *not associated with significant improvement in CVD risk prediction* in a study that included more than 19,000 women, according to a study published in the February 17 issue of *JAMA*.
- “The study included 19,313 initially healthy white women in the Women’s Genome Health Study, followed up over a median of 12.3 years.
- “A total of **101 single nucleotide polymorphisms (SNPs)** reported to be associated with cardiovascular disease or at least 1 intermediate cardiovascular disease phenotype were identified and risk alleles were added to create a genetic risk score.

1 Ref.: February 17, 2010 issue of *JAMA*.

The Hidden Story of Cancer

- “After analysis, the researchers found an absolute cardiovascular disease risk of **3% over 10 years in the lowest tertile** of genetic risk (73-99 risk alleles) **and 3.7% in the highest tertile** (106-125 risk alleles).
- ““Our study finds **no clinical utility in a multilocus panel of SNPs for cardiovascular risk** based on the best available literature. Second, our data confirm the utility of intermediate phenotypes such as *total cholesterol, high-density lipoprotein cholesterol [HDL], and blood pressure inasmuch as genetic risk scores were no longer significant* after adjustment for these phenotypes,” the researchers wrote.” (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

Once again, we see the utter failure of “genetic markers;” this time, in the prevention of cardiovascular disease because the solution is entirely *epigenetic* – originating in the cellular environment with lack of fully functional PEOs as CVD’s prime cause.

• • •

2010: DNA Mutations Explained — Important Newsflash: Warburg Confirmed AGAIN

The superb 2010 paper titled, “Cancer as a metabolic disease,” published by professor of biology at Boston College, Thomas N. Seyfried, confirms many of Prof. Warburg’s seminal discoveries. Here is what you need to know¹:

- “Emerging evidence indicates that *impaired cellular energy metabolism is the defining characteristic* of nearly all cancers regardless of cellular or tissue origin.
- “However, the **mutation rate for most genes is low** making it unlikely that the numerous pathogenic mutations found in cancer cells would occur sporadically within a normal human lifespan. This then created another paradox. **If mutations are such rare events**, then how is it possible that cancer cells express so many different types and kinds of mutations?

1 Seyfried, Thomas N, “Cancer as a metabolic disease,” *Nutrition and Metabolism*, January 27, 2010, *Nutrition & Metabolism* 2010, 7:7 doi:10.1186/1743-7075-7-7, <http://www.nutritionandmetabolism.com/content/7/1/7>, pages 1-52.

The Hidden Story of Cancer

- “[N]early all cancers *express aerobic glycolysis, regardless of their tissue or cellular origin.*
- “...[A]erobic glycolysis can arise in tumor cells from **damaged respiration.**
- “**Aerobic glycolysis, arising from *damaged respiration,* is the *single most common phenotype found in cancer.***
- “In brief, **damage to cellular respiration precedes and underlies the genome instability that accompanies tumor development.**
- “A major difference **between normal cells and cancer cells is in the origin of the energy produced rather than in the amount of energy produced** since approximately -56 kJ/mol is the amount of energy required for cell survival regardless of whether cells are quiescent or proliferating or are mostly glycolytic or respiratory. *It is important to recognize, however, that a prolonged reliance on substrate level phosphorylation [anaerobic glucose metabolism] for energy production produces genome instability, cellular disorder, and increased entropy, i.e., characteristics of cancer.*
- “...[M]itochondrial uncoupling can give the false impression of functional respiratory capacity. Oxygen uptake and CO₂ production *can occur in mitochondria that are uncoupled and/or dysfunctional.*
- “Besides glucose, *glutamine can also serve as a major energy metabolite* for some cancers. Glutamine is often present in high concentrations in culture media and serum. **Energy obtained through this pathway could give the false impression of normal oxidative phosphorylation, as oxygen consumption and CO₂ production can arise from glutaminolysis and uncoupled oxidative phosphorylation.**
- “Recent evidence also shows that the *in vitro* growth environment *alters the lipid composition of mitochondrial membranes* and electron transport chain function. The bioenergetic capacity of mitochondria is dependent to a large extent on the content and composition of mitochondrial lipids.

- **“Alterations in mitochondrial membrane lipids** and especially the inner membrane enriched lipid, **cardiolipin, disrupt the mitochondrial proton motive gradient** thus inducing protein-independent **uncoupling** with concomitant **reduction in respiratory energy production. Cancer cells contain abnormalities in cardiolipin content or composition,** which are associated with electron transport abnormalities. **Cardiolipin is the only lipid synthesized almost exclusively in the mitochondria.**
- **“Besides altering the function of most electron transport chain complexes including the F1-ATPase, abnormalities in cardiolipin content and composition can also inhibit uptake of ADP** through the adenine nucleotide transporter thus **altering the efficiency of oxidative phosphorylation.**
- **“Abnormalities in the content and composition of cardiolipin** will also **prevent oxidation of the coenzyme Q couple thus producing reactive oxygen species during tumor progression.** Increased ROS production can *impair genome stability, tumor suppressor gene function, and control over cell proliferation.* Hence, abnormalities in CL can alter cancer cell respiration in numerous ways.
- **“Mitochondrial dysfunction also increases levels of cytoplasmic calcium, the multi-drug resistance phenotype, production of reactive oxygen species, and abnormalities in iron-sulfur complexes, which together would further accelerate aberrant RTG signaling and genome mutability. Chronic tissue inflammation could further damage mitochondria,** which would accelerate these processes. Considered collectively, these findings indicate that the *integrity of the nuclear genome is dependent to a large extent on the functionality and energy production of the mitochondria.*
- **“Even different tumors within the same cancer type could appear to represent different diseases when evaluated at the genomic level. When evaluated at the metabolic level, however,**

The Hidden Story of Cancer

most cancers and tumors are alike in expressing mitochondrial dysfunction and elevated substrate level phosphorylation. Emerging evidence suggests that mitochondrial dysfunction underlies the mutator phenotype of tumor cells.

- **“Impaired mitochondrial function can induce abnormalities in tumor suppressor genes and oncogenes.**
- **“Considered collectively, these observations suggest that the bulk of the genetic abnormalities found in cancer cells, ranging from point mutations to gross chromosomal rearrangements, can arise following damage to the structure and function of mitochondria.**
- **“The accumulation of mitochondrial damage over time is what ultimately leads to malignant tumor formation.** In other words, the well-documented **tumor-associated abnormalities in oncogenes, tumor suppressor genes, and chromosomal imbalances can arise as a consequence of the progressive impairment of mitochondrial function.**
- **“It is well documented that tumorigenicity can be suppressed when cytoplasm from enucleated normal cells is fused with tumor cells to form cybrids, suggesting that normal mitochondria can suppress the tumorigenic phenotype.**
- **“...[R]ecognizing that proliferation rather than quiescence is the default state of both microorganisms and metazoans. The cellular default state is the condition under which cells are found when they are freed from any active control.**
- **“As p53 function is linked to cellular respiration, prolonged damage to respiration will gradually reduce p53 function thus inactivating the negative control of p53 and of other tumor suppressor genes on cell proliferation.**
- **“Tumor cells will continue to evade apoptosis as long as they have access to glucose and glutamine, which are required to maintain substrate level phosphorylation.**

- “Vascularity is necessary in order to provide the tumor with essential energy nutrients to include glucose and glutamine... Hence the **sustained vascularity of tumors can be linked mechanistically to the metabolic requirements of substrate level phosphorylation necessary for tumor cell survival.**
- “**Uncorrected mitochondrial damage will require a continuous compensatory energy response** involving substrate level phosphorylation in order to maintain the free energy of ATP of approximately -56 kJ/mol for cell viability. *Tumor progression is linked to a greater dependence on substrate level phosphorylation, which eventually becomes irreversible.* As the integrity of the nuclear genome is dependent on the efficiency of mitochondrial energy production, **the continued impairment of mitochondrial energy production will gradually undermine nuclear genome integrity leading to a mutator phenotype and a plethora of somatic mutations.** Activation of oncogenes, inactivation of tumor suppressor genes, and aneuploidy will be the *consequence of protracted mitochondrial dysfunction. These gene abnormalities will contribute further to mitochondrial dysfunction* while also enhancing those energy pathways needed to up-regulate and sustain substrate level phosphorylation [increased glycolysis]. **The greater the dependency on substrate level phosphorylation over time the greater will be the degree of malignancy.** Damage to the respiratory capacity of tissue myeloid cells can also produce invasive and metastatic properties according to the macrophage hypothesis of metastasis. **This metabolic scenario can account for all major acquired characteristics of cancer to include the Warburg effect.**
- “**Glucose reduction** not only **reduces insulin**, but also **reduces circulating levels of IGF-1**, which is *necessary for driving tumor cell metabolism and growth.*
- “Metabolism of ketone bodies for energy will maintain mitochondrial health and efficiency thus reducing the incidence of cancer.” (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

This article affirms what I have already stated elsewhere in this book, but which, because of its significance, bears repeating: *Any genetic alternations in the DNA are a RESULT of the decreased cellular oxygen—NOT its CAUSE.* As an example, suppose someone severely burned their arm. Do you think the DNA would remain the same? No, because there would be permanent scarring. The scarred tissue would never return to normal. This is exactly what happens with cellular hypoxia.

Furthermore, the *mitochondria control cell proliferation.* As Dr. Warburg clearly stated, oxygen supplies the intelligence of cellular control, and without it the mitochondria's control mechanism via p53 is impaired and cell growth runs rampant. Why would uncontrolled cellular growth be a primitive condition? Likely, because when life began, growth was the most important thing. Survival was an end in itself. It took time for increased intelligence to be incorporated into many life forms.

We are told how cancer researchers have been misled because energy from glutamine can be mistaken for normal oxidative phosphorylation. This mistake has had tragic consequences in preventing greater understanding and dissemination of Dr. Warburg's seminal discoveries.

Even different tumors within the same cancer type could appear to represent different diseases when evaluated only to the genomic level. *However, when evaluated at the metabolic level it is evident that they all have impaired cellular respiration.*

2010 Newsflash: Confirmed that Asbestos Causes Cancer via Inflammation

Chronic inflammation (irritation) as caused by asbestos is cancer-causing because the body will divert all excess available oxygen to the inflamed area, thereby reducing critical oxygen levels elsewhere. *Medical News Today*, has this to say²:

- “For the past 40 years researchers have tried to understand **why asbestos causes cancer**.
- “This research **emphasizes the role of inflammation in causing different types of cancers...**(Emphasis added.)

► *Life-Systems Engineering Science Commentary*

Once again, in 2010, we see the inflammation/cancer causal link. Unfortunately, this research group also fails to incorporate Dr. Warburg’s seminal cellular oxygen deprivation insight, so they have an incomplete understanding of cancer’s direct origination.

2 “How Asbestos Causes Cancer: Mystery Unraveled,” *Medical News Today*, June 30, 2010, Cancer/Oncology -- Article URL: <http://www.medicalnewstoday.com/articles/193306.php>.

Appendix XII

2010: Cancer Prevention and the 15-Lipoxygenase Pathways (Especially for Cancer Researchers and Cancer Physicians)

I thank Robert J. Rowen, M.D., Editor-in-Chief of *Second Opinion* – from my perspective, the nation’s top medical newsletter – for the wealth of information presented here. He is the reason that I have pursued the biochemistry of this particular anti-cancer modality, further substantiating the immense power of additional pathways that utilize unadulterated, fully functional parent omega-6.

Lipoxygenase enzymes and their relationship to colorectal and prostate cancer

Lipoxygenases (LOX) are important enzymes in lipid metabolism that convert the polyunsaturated fatty acids, arachidonic acid and linoleic acid, to their corresponding metabolites and are classified with respect to their position of oxygenation of arachidonic acid. Lipoxygenases have many significant roles in human disease, including cancer.

Human 15-lipoxygenase-1, which *metabolizes polyunsaturated fatty acids*, is a highly regulated, tissue-specific, *lipid-peroxidating enzyme* that functions in *physiological membrane* remodeling and in the pathogenesis of atherosclerosis, inflammation, and carcinogenesis.

Eicosanoid mediators have been implicated in the development and progression of many cancers including colorectal cancer (CRC).

The Hidden Story of Cancer

The cyclooxygenase (COX) and lipoxygenases (LOX) pathways are the two major enzyme systems involved with the *metabolism of polyunsaturated fatty acids*. LOX-1 is a multifunctional membrane receptor that binds and internalizes oxidized LDL (oxLDL). There are also other physiologic functions.

The most well-known system, but hardly the most important, involves the COX-2 enzyme, which is primarily responsible for the conversion of arachidonic acid to prostaglandin E2 (PGE2), which has been implicated in colorectal tumor growth and proliferation.

Although COX-2 overexpression is associated with increased tumor growth in a number of different histologies, this pathway will not be the focus of this section. The LOX pathway is independent of COX-2 actions.¹ The more important LOX pathways will be the focus.

In essence, 15-LOX-1, an enzyme critical to maintaining normal rates of apoptosis (programmed normal cell death), preferentially metabolizes linoleic acid (parent omega-6) to 13-(S)-HODE while 15-LOX-2 metabolizes arachidonic acid to 15-(S)-HETE but poorly metabolizes linoleic acid. A balance between these two opposing effects of 15-LOX metabolites may determine their role in preventing prostate cancer.²

“Human prostate tumors have higher expression of 15-LOX-1 compared with normal adjacent tissue and this expression corroborates with the Gleason score of the cancer.”

► *Life-Systems Engineering Science Commentary*

If the parent omega-6 is not fully functional, then its associated 15-LOX-1 metabolites won't be fully functional, either. As an analogy with the naivety of merely measuring amounts of LDL-cholesterol,

1 Shureiqi, I., et al., “15-Lipoxygenase-1 Mediates Nonsteroidal Anti-Inflammatory Drug-induced Apoptosis Independently of Cyclooxygenase-2 in Colon Cancer Cells,” *Cancer Research*; 60, 6846–6850, December 15, 2000.

2 His, L., et al., “Opposing Effects of 15-Lipoxygenase-1 and -2 Metabolites on MAPK Signaling in Prostate,” *The Journal of Biological Chemistry*, Vol. 277, No. 43, October 25, pp. 40549–40556, 2002.

it's not the amount but the structure that counts. All insight lies in *epigenetics* (environmental conditions based on dietary food intake), not a genetic-based condition.

It is important to note that the decrease in both of these metabolites is significant in progressing from adenomas (benign) to carcinomas (malignant).*

* The first paper showing decreased 15-LOX-1 in adenomas progressing to carcinomas in the colon and its role as a prognostic factor for survival was published by Martin Hesler, M.D., et al., in the journal article, "Tumor-Associated Down-Regulation of 15-Lipoxygenase-1 Is Reversed by Celecoxib in Colorectal Cancer," *Annals of Surgery*, Volume 241, Number 6, June 2005.

Lipoxygenase enzymes and their relationship to pancreatic cancer

Pancreatic cancer has an extremely low prognosis as the following article details. An omega-6-based metabolic pathway provides much needed help in increasing treatment effectiveness:³

- "Pancreatic cancer patients have an abysmal prognosis because of late diagnosis and lack of therapeutic options.
- "Here we provide evidence that 15-lipoxygenase-1 (15-LOX-1) expression and activity may exert antitumorigenic effects in pancreatic cancer.
- "**15-LOX-1** was strongly stained in **normal ductal cells**, tubular complexes, and centroacinar cells, but no staining was seen in

3 "15-Lipoxygenase-1 Production is Lost in Pancreatic Cancer and Overexpression of the Gene Inhibits Tumor Cell Growth," Hennig, R. et al., *Neoplasia*, Vol. 9, No. 11, 2007.

islets, cancer cells, PanIN lesions, or in tumor cells in lymph node metastases, indicating that 15-LOX-1 expression is **lost during tumor development in human pancreas**.

- “Thus, **induction of 15-LOX-1 expression** may be an **attractive option for the prevention and treatment of pancreatic cancer**.
- “At present ... **no effective treatment is available**. Even small resectable tumors *recur in most cases within two years after primary surgery or they have metastasized*, which becomes evident months or years later. *Chemotherapy only prolongs life by a matter of weeks, maintaining the unchanged abysmal prognosis of pancreatic cancer*.
- “The *connection between inflammation and cancer* goes back to Virchow in 1863 when he **noted a lymphoreticular infiltrate in neoplastic tissues**.
- “**Significant growth inhibition and induction of apoptosis occurred** after restoration of 15-LOX-1 activities. *These results are in line with findings in colonic, gastric, and esophageal cancers where either 15-LOX-1 expression is down-regulated in cancer cells and 13(S)-HODE [an omega-6 derivative directly influenced by 15-Lox-1] or upregulation of 15-LOX-1 causes growth inhibition by inducing apoptosis*.
- “It was recently shown that **downregulation of 15-LOX-1 is an early event in the adenoma to carcinoma sequence in colonic cancer development**. We observed similar findings in pancreatic carcinogenesis, because **15-LOX-1 expression is lost in advanced PanINs as well as cancer cells**, but not in PanIN-1 lesions.
- “These findings provide evidence that loss of **15-LOX-1 may play an important role early in pancreatic carcinogenesis**, possibly as a tumor suppressor gene. Thus, 15-LOX-1 may be an **attractive target for the prevention and treatment of pancreatic cancer**.” (Emphasis added.)

► Life-Systems Engineering Science Commentary

A diagnosis of pancreatic cancer is typically a death sentence. There is a very special feedback system between the prostaglandins directly from arachidonic acid (AA) and other omega-6 long-chain prostaglandins not derived from AA. Once again we see that utilization of unadulterated, fully functional parent omega-6 is the anti-cancer solution.

Lipoxygenase enzymes and their relationship to gastric cancer

The next journal article we discuss involves non-steroidal anti-inflammatory drugs and the attempt to arrest the growth of gastric cancer with them. Once again, the critical omega-6 pathways are utilized, and once again the authors likely are unaware of how the critical parent omega-6 is so often highly adulterated and therefore non-functional.⁴

- “[Non-Steroidal Anti-Inflammatory Drugs] NSAIDs can alter the production of **different metabolites of polyunsaturated fatty acids** (e.g. **linoleic acid** [LA] and arachidonic acid) by modulating the activity of lipoxygenases (LOXs) and COXs. *In the classic pathway involving only arachidonic acid* (the eicosanoid-generation pathway), arachidonic acid is a substrate for both LOX and COX enzymes to form various metabolites, such as 5-, 8-, 12- and 15-S hydroxy-eicosatetraenoic acids (HETEs) and prostaglandins. *However, linoleic acid, the predominant polyunsaturated fatty acid in the human diet, can undergo oxidative metabolism, mainly limited to the 15-LOX-1 pathway, which produces 13-S-hydroxy octadecadienoic acid (13-S-HODE).*

⁴ “15-Lipoxygenase-1 mediates cyclooxygenase-2 inhibitor-induced apoptosis in gastric cancer,” Wu, J., et al., *Carcinogenesis*, vol.24 no.2, pp.243–247, 2003.

The Hidden Story of Cancer

The expression of 15-LOX-1 is down-regulated in colorectal cancer. Indomethacin treatment decreases the expression of COX-2 and significantly increases the expression of 15-LOX-1 during apoptosis and cell differentiation in sodium butyrate-treated colorectal cancer cells.”

- **“Basal 15-LOX-1 expression is absent in all four human gastric cancer cell lines tested, suggesting that 15-LOX-1 down-regulation occurs in tumorigenesis... it is clear that the effects of 15-LOX-1 up-regulation on apoptosis are mediated through 13-S-HODE. These findings agree with previous reports that linoleic acid is the preferred substrate for human 15-LOX-1, which predominantly produces 13-S-HODE.”** (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

Once again, it is made clear the critical value of unadulterated, fully functional parent omega-6 and its multi-dimensional pathways in preventing and treating gastric cancer.

Lipoxygenase enzymes and their relationship to breast and esophageal cancer

How about their function in combating breast cancer? The following journal article describing their immense power in combating human breast cancer says it all.¹

- **“13-HODE [omega-6 derivative] is a known anti-motility and anti-invasion eicosanoid in cancer cells.**

1 Jiang, W., et al., “Reduction of isoforms of 15-lipoxygenase (15-LOX)-1 and 15-LOX-2 in human breast cancer,” *Prostaglandins, Leukotrienes, and Essential Fatty Acids*, 74 (2006) 235-245.

- “This suggests that synchronous **damages to the 15-LOX** and PPAR pathways may **result in accelerated cell growth and more aggressive breast tumours**.
- “(15-LOX)...has two isoforms...and converts arachidonic acid to eicosanoids **including the anti-cancer 13-HODE**.
- “Both [15-]LOX-2 and [15-]LOX-1 were found to be at **significantly lower levels in tumours from patients who developed metastasis**.
- “**Both isoforms** of 15-lipoxygenase have a **tumour suppressing role in breast cancer**.
- “Although 15-LOXs are able to convert arachidonic acid to a number of downstream eicosanoids, the well-characterized ones are 15-HETE (predominately converted by 15-LOX-2) and 13-HODE (product of LOX-1). 13-HODE has been widely shown to be an **anti-cancer lipid**, which is able to inhibit the migration and invasion of cancer cells. 15-HETE is also known to induce **apoptosis** in cancer cells such as **colon cancer cells**. Thus **the loss** of 15-LOX, through which anti-cancer eicosanoids such as 13-HODE and 15-HETE are insufficiently synthesized (within the tumour tissue), may **lead to the gain of the invasive and motile nature of cancer cells which, in turn, result in aggressive growth of breast tumours**.
- “This current study has provided evidence that both isoforms of **15-LOX expression are aberrant** at both the protein and mRNA levels **in human breast cancer**. This aberration is linked to a *poor clinical outcome and poor long-term survival*.”

[Emphasis added.]

► Life-Systems Engineering Science Commentary

It can't be any clearer. Insufficient fully functional, unadulterated parent omega-6 weakens your defense against breast cancer. Even

The Hidden Story of Cancer

esophageal cancers possess the 13(S)-HODE defect found in breast cancer.² Indeed, all cancers suffer from the same *prime* cause that Nobel Prize-winner Otto Warburg, M.D., Ph.D. made so clear decades ago.

2 Shureiqi, I., et al., "Nonsteroidal Anti-Inflammatory Drugs Induce Apoptosis in Esophageal Cancer Cells by Restoring 15-Lipoxygenase-1 Expression," *Cancer Research*; 61, 4879-4884, June 15, 2001.

Appendix XIII

The Coffin is Closed: DHA and Fish Oil Shown Completely Worthless — Five More Fish Oil Failures:

- **Increased Platelet Aggregation,**
- **No Help with Abnormal Heartbeat**
- **Decreased Glucose Tolerance**
- **Increased Risk of Colon Cancer**
- **No Help with Alzheimers**

WARNING: Fish Oil Increases Platelet Aggregation!³

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

► *Life-Systems Engineering Science Commentary*

You recall prostacyclin (PGI₂) is the body’s natural blood thinner and keeps platelets apart naturally. The last thing a CVD patient needs is a reduction in this critical substance. CVD patients require more, NOT less PGI₂. Decreased PGI₂ significantly increases, not decreases, the severity of heart attack — the opposite effect.

3 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.

2010 Newsflash: Fish Found Worthless in Decreasing Abnormal Heart Rhythm (Atrial Fibrillation—AF)⁴

Contrary to many report claims, the *American Journal of Cardiology* reported in 2010 that eating lots of fish did nothing to help an abnormal heartbeat. However, in contrast to omega-3's failure, parent omega-6 is effective. Riemersma, RA, et al., state in the medical journal article, "Dietary fatty acids and ischemic arrhythmias," *Lancet*, 1988;i:285-6, that parent omega-6 did help reverse AF.

Fish Oil Impairs Regulation of Insulin Secretion, Not Good for Diabetics

And if you'd like even more confirmation of fish oil's negative effect on blood glucose impairment, here it is. In a 1988 experiment reported in the *Annals of Internal Medicine*, it was clearly shown that fish oil is awful for a diabetic and for anyone not wishing to become diabetic.⁵

- "A significant deterioration of these patients' diabetic state (a 19% rise in the fasting glucose level and a rise in 24% mean glucose levels during a mixed meal occurred within one month of omega-3 fatty acid [fish oil] supplementation...."
- "**Glucose tolerance** during the mixed meal profile also **deteriorated significantly**."

4 Berry, J, et al., "Dietary Fish Intake and Incident Atrial Fibrillation (from the Women's Health Initiative)," *The American Journal of Cardiology*, Vol. 105, Issue 6, Pages 844-848 (15 March 2010).

5 "Adverse Metabolic Effect of Omega-3 Fatty Acids in Non-Insulin Dependent Diabetes Mellitus," Gluaber, H. et al., *Annals of Internal Medicine*, 1988; 108:663-668.

- "...Thus, there is a hypothetical mechanism whereby omega-3 fatty acid [overdoses of derivatives] **incorporated into** plasma and [**cellular**] **membrane** lipids might impair the regulation of insulin secretion." (Emphasis added.)

► *Life-Systems Engineering Science Commentary*

The body cannot deal with harmful, pharmacological overloads of omega-3 derivatives from fish oil supplements, and is forced to "shove them" into the cell abnormally or alter normal physiologic processes. One must ask what besides insulin is negatively impacted, too. Fortunately, this deterioration REVERSED when the fish oil supplements were STOPPED.

2010 Newsflash: Fish Oil Increases Risk of Colon Cancer

An October 7, 2010 article entitled "Link Between Fish Oil And Increased Risk Of Colon Cancer In Mice," published in *Medical News Today* and the medical journal *Cancer Research*, revealed some startling information from a group at Michigan State University led by Jenifer Fenton, a food science and human nutrition researcher:⁶

- "'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**, it **only took four weeks** for the tumors to develop."

6 "Link Between Fish Oil And Increased Risk Of Colon Cancer In Mice," *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 Res* 0008-5472.CAN-10-1396; Published *OnlineFirst* August 26, 2010; doi:10.1158/0008-5472.CAN-10-1396. *Cancer Res*; 70(20); 7960-9.

The Hidden Story of Cancer

- “... **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 that feeding fish oil enriched with DHA to mice would decrease the cancer risk; **we actually found the opposite.**”
- ““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.”
- “ [Fenton] said people already receiving enough omega-3 fatty acids through their normal diet and foods have no need for added supplementation.” (Emphasis added.)

► Life-Systems Engineering Science Commentary

Once again we see confirmation of the horrific effects that we have been reporting from fish oil supplementation. Of significant note is the fact that Fenton and her fellow researchers *fully expected the fish oil to have the opposite and extremely positive effect of reducing cancer risk – not increasing it!*

This result is directly aligned with the fact that this cancer, adenocarcinoma, occurs in epithelial-based tissue (such as the lining of the colon). You will already understand, if you have read my work, that there is no omega-3 component to epithelial tissue (just as with skin). That means that omega-3 supplementation cannot benefit epithelial tissue. *Quite the opposite; a pharmacological overdose of omega-3 derivatives is predicted to harm such tissue.* This study has now demonstrated pharmacological overdoses of omega-3 derivatives from fish oil do harm such tissue by causing an *increase* of cancer.

Appendix XIII: Five More Fish Oil Failures

Fish oil supplement manufacturers often recommend “high dose” amounts. But the prevalent pharmacological overdoses of DHA and EPA from fish oil supplements range from **20–200-fold** overdoses of DHA to **250–500-fold** overdoses of EPA – far more than your body would ever produce on its own. **Even so-called “low dose” fish oil supplementation approaches these overdose values.**

In her experiment, Fenton comments, “Currently, there is a call by academics and the food industry to establish dietary guidelines for omega-3 consumption....” *It is high time a researcher questioned utilizing such arbitrary, haphazardly dangerous overdose amounts.*

It is also significant that Fenton points out that her findings “**support a growing body of literature implicating harmful effects of high doses of fish oil consumption in relation to certain diseases.**” Finally an increasing number of negatives about fish oil supplementation are being exposed and published in the medical journals, and the truth is slowly being recognized by more of the medical and nutritional community.

In fact, *the negative results of fish oil supplementation and its increased risk of cancer were already discussed and published back in 2000* at 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 – if anyone had cared to look!

In contrast, we have long maintained that parent essential oils derived from specific plant-based, organically grown and processed seed sources, termed “PEOs,” can never lead to an overdose such as occurred in this study.

As always, we caution that one should be wary of animal studies, as they often do not translate consistently to results in humans. However, while mice are herbivores, if challenged they will eat anything (omnivore physiology). Therefore, we agree that this result showing increased colon cancer from fish oil needs to be taken very seriously.

Newsflash 2010: DHA and Fish Oil Shown Completely Worthless with Alzheimers

Once again, the main “active ingredient” in fish oil, DHA, has failed to cure or prevent disease. Researchers recently concluded that DHA (also found in krill oil, algae, mussels, etc.) failed to improve cognitive impairment in Alzheimer disease victims. Previously this year, it was reported that fish oil failed to reverse heart disease in diabetic women; and if failure was not bad enough, another 2010 study showed that fish oil actually promoted aggressive colon cancer in mice.

This latest, very well-designed study published in *JAMA*, one of America’s top medical journals, dispels the naive notion that DHA and therefore fish oil is beneficial in cognitive disorders.¹

- “Conclusion: Supplementation with **DHA compared with placebo did not slow the rate of cognitive and functional decline in patients with mild to moderate Alzheimer disease.**
- “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 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.” (Emphasis added.)

1 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.

► *Life-Systems Engineering Science Commentary*

Once again, a carefully controlled study shows that the active ingredient in fish oil has little benefit for a human being. The EFA derivative docosahexaenoic acid (DHA) is the most abundant long-chain polyunsaturated fatty acid in the brain. Alzheimer sufferers were given the DHA supplement (algae-based) for 18 months. If fish oil were beneficial we should certainly see a positive result in Alzheimer's patients. This length of time is sufficient to see an improvement. However, there wasn't any improvement whatsoever. This negative result shows that the problem has nothing to do with the EFA-derivatives "solving the problem."

There is recent confirmation of this finding of the vast superiority of parent essential oils over fish oil with its derivatives DHA and EPA. Here is what was stated:²

- "...[A] higher level of α -linolenic acid (ALA; 18:3n-3) [parent omega-3] **significantly decreased the risk of mild dementia...."**
- "**However, erythrocyte levels of docosahexaenoic acid (22:6n-3) [DHA] and eicosapentaenoic acid (20:5n-3) [EPA] were not significantly related** with the risk of mild dementia and MMSE-K score."
- "In conclusion, **ALA derived from plant sources of n-3 PUFA, but not eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA] from fish**, decreased the risk for mild dementia among the Korean elderly."

You have already discovered that the body makes EFA derivatives from the parent PEOs on an "as needed" basis. When will physicians understand that because the brain has a 100:1 parent omega-6 to parent omega-3 ratio, the key is all in the important, essential PEOs – the

2 Malgeunsinae, K., et al, "Erythrocyte α -linolenic acid is associated with the risk for mild dementia in Korean elderly," *Nutrition Research*, Volume 30, Issue 11, November 2010, pages 756-761.

The Hidden Story of Cancer

EFA “parents,” – not the much less important EFA- derivatives. How much longer and how many more fish oil failures will it take before the medical community begins to comprehend this physiologic fact?

Contrary to fish oil’s failure, we have seen PEO’s incredible success from the recent landmark IOWA study. PEOs in the correct physiologic quantities and ratios provide rapid improvement to the cardiovascular system, increasing arterial compliance REGARDLESS of the subject’s condition.

Appendix XIV

Everyone Can Now Rest Easy— It's RARELY Genetic...

The fledgling field of *epigenetics* (environmental factors) is now showing how two organisms with **identical genetic sequences** can have **different characteristics** because of **heritable *non-DNA factors* (like methyl groups)**, which are common reactive chemical entities that *alter the behavior of genes*.

The search for simple “genetic mutations” as the cause of disease **predictably fails again**. As you shall discover, in contrast to getting better and allowing better explanations with time, the “genetic theory of disease” gets worse with time. Even the most brilliant researchers will never accomplish their goals because they all start in the wrong place. Mutations are *caused by* epigenetic adulteration (environmental causes altering the behavior of genes but not necessarily the structure). The *prime* cause of cancer is decreased cellular oxygen, which, of course, also alters the genetic material.

2011—More Major Embarrassment and FAILURE—Traits *aren't* handed down in the manner described by Mendel. The so-called “ruined genetics” account for no more than 10% (the vast MINORITY) of disease.

The Hidden Story of Cancer

The first 2011 article³ clearly shows how the “promise” of looking at genetics fails and worries its scientists, as the whole field is looking worse and worse for actually finding solutions. It states:

“Evidence is growing that *your DNA sequence does not determine* your entire genetic fate. ...

“**Large-scale genomic studies over the past five years or so have mainly failed** to turn up common genes that play a major role in complex human maladies. ...

“More than **three dozen specific genetic variants** have been associated with type 2 diabetes, for example, but together, they have been found to **explain about 10 percent of the disease’s heritability** [90% *failure*] – the proportion of variation in any given trait that can be explained by genetics rather than by **environmental influences**. ...

“...That shouldn’t have affected the daughter mice at all, because females don’t inherit the Y chromosome. But the presence of that **uninherited DNA** in the previous generation exerted a profound effect on many of the more than 100 traits tested in the two sets of female offspring, whose own DNA was exactly the same.

“... In a separate but similarly **unsettling line of experiments**, Nadeau and his collaborators are finding that the **impact of any given gene depends on all the other genes surrounding it**. Nadeau is hardly the only scientist to identify these complex gene-gene interactions. ...

“Nadeau recalled giving a talk about all this at a conference several years ago and discovering afterward that a... **prominent Ivy League geneticist** in attendance, whom he declined to name, *simply couldn’t get the heretical ideas out of his head*. ‘He came up to me after the talk,’ Nadeau recalled, ‘and said, “**This can’t be true** in humans.” ’ ...

3 Stephen S. Hall, “The Genome’s Dark Matter,” *Technology Review*, January/February 2011, pages 53-57.

“I ran into him at breakfast the next day and he said, ‘*This can’t be true in humans.*’ And then when the meeting was over, I ran into him at the airport, and he came up to me and said, ‘*This can’t be true in humans.*’ ...

“Or as another leading genome scientist once told Nadeau at a meeting in Europe, ‘**If transgenerational⁴ [non-Mendelian] effects happen in humans, we’re screwed.**’

...

“That is to say, discovering that his findings apply to humans **would decouple a person’s DNA sequence from her or his traits, calling into question much of the work scientists have done to find the genetic sources of complex diseases** and develop drugs that target them. ...

“...The group analyzed **54 recently identified genetic locations** that statistical analysis suggested were the main contributors to height **and discovered that all of them together accounted for only 4 to 6 percent [94% failure] of the height variance** in thousands of subjects. ...

“The reason is not known, but the larger message is that *the effect of any variant seems to depend on its genetic surroundings.* ‘We see that effect all the time,’ Nadeau says. ‘*All the time! Everywhere, in every trait we look at.*’

...

“It may sound like a dramatic break, but Nadeau says **these exceptions to Mendelian patterns** should come as no surprise. ‘Mendel picked the traits where he would get simple genetics,’ he explains. ‘What Mendel said is true. But it’s **not the whole truth.**’”

(Emphasis added.)

4 Transgenerational traits appear in DNA of parents or grandparents but not in current generation, yet can be influential and can be passed to subsequent generations. This runs counter to Mendel’s patterns of inheritance.

2011 Revelation: “So indeed, **the genome contains far more inconvenient truths** than was supposed a decade ago. ***The very idea of what we inherit and what we pass on has changed.***”⁵

The next article, titled “The human genome a decade later,”⁶ states:

“In June 13, 2010, the *New York Times* ran a front-page story about the hyping of genomics. Headlined “A Decade Later, **Gene Map Yields Few New Cures....**”

“Recent studies, however, have emphasized the extraordinary power of **DNA regions that do not hold the code for a protein itself but, rather, control** the on/off switches that direct gene ‘**expression,**’ or the extent to which that protein is actually produced. ...

“The fledgling field of *epigenetics* is showing how two organisms with **identical genetic sequences** can have **different characteristics** because of **heritable non-DNA factors like methyl groups**, which are common reactive chemical entities that **alter the behavior of genes.** ...

“So indeed, **the genome contains far more inconvenient truths** than was supposed a decade ago. ***The very idea of what we inherit and what we pass on has changed.*** ...

“... Duke University geneticist David Goldstein argued in a critique published in the April 23, 2009, issue of the *New England Journal of Medicine*, that “*common variation is packing much less of a phenotypic punch than expected.*” A study on height, Goldstein noted, had *found 20 variants* that

5 Jon Cohen, “The Human Genome, a Decade Later,” *Technology Review*, January/February 2001, pages 40-44.

6 Cohen, pages 40-44.

together explained only about 3 percent [97% *failure*] of the variation found in humans. These sorts of results have led some researchers to scratch their heads about “missing heritability” and the “dark matter” of the genome. ...

“ ‘This “omic” science has corrupted us,’ says Brenner, who won a Nobel Prize in 2002 for leading a project that four years earlier completed the first entire sequence of a multicelled organism, the worm *Caenorhabditis elegans*. **‘It has created the idea that if you just collect a lot of data, it will all work out** [like genome mapping].’ ...

“ ‘I think we should be doing *genetics, not genomics,*’ says Brenner. ‘When you do genetics, you are focusing on function. When you do genomics, these are just letters and numbers. *Nobody bothers about the connections.*’ ...

“ ‘Let’s start with the patient and work backward,’ says Altshuler. ‘Something that has profoundly diminished the biomedical impact of [genomic] work is the *unquestioned faith that everything can be learned in reductionist approaches and model systems.*’ ”

(Emphasis added.)

● Life-Systems Engineering Science Commentary

The great news is that heredity plays a much smaller role than we’ve been led to believe, and (shockingly to many) genes can be altered by the environment (both in positive and negative ways). That alteration can affect the **traits** of generations to come, although it might not appear in the **structure** of the genes themselves. *Conclusion: our behaviors, exposures, and remedial actions can affect our children.* This may seem like a hereditary factor, though not in the way Mendel describes – there can be external causes to current and future traits that we can take responsibility for (like PEO deficiency and PEO supplementation).

We can be comforted as well that *inheritance is a very minor cause of disease.* The statement, “The very idea of what we inherit and what we pass on has changed,” tells it all. **In contrast to getting better and allow-**

The Hidden Story of Cancer

ing better explanations with time, the “genetic theory of disease” gets worse with time; the opposite of any valid scientific theory.

The scientists focusing on genetics were misled. The promise of the genome and the entire field of genetics are based on fallacies. The scientific truth was already given to us decades ago by the medical genius Otto Warburg, MD, PhD. *Life-Systems Engineering Science* makes significant discoveries that lead to practical solutions because this science does indeed connect all the dots, like the Nobel Prize-winner Sydney Brenner stresses.