

# Cancer and Mitochondria Defects: New 21st Century Research

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I recently presented at the 17th Annual World Congress on Anti-Aging Medicine in Orlando, Florida (April 2009), utilizing new research from 2007–2009 to identify the prime cause of cancer. Predictably, these findings caused quite a sensation. Numerous physicians met with me afterwards to applaud the presentation and discuss direct patient application in their practices. This article addresses the major points of my presentation, with a focus on clinical application. While environmental pollution and other factors certainly play a role in the cancer causing process, they are a topic unto themselves. “Connecting the dots” with this new information leads to a startling conclusion that we will now explore.

## What Cancer Isn't

First, let me state what cancer is *not*. Cancer is not an invader in our bodies like a virus or bacterial infection, nor is it a genetic distortion determined to kill us. Cancer is the body, at the cellular level, attempting to allow the injured tissue or organ to survive by reverting to a primitive survival mechanism. This definition is the result of embracing the latest research from a variety of disciplines whose practitioners include epidemiologists, geneticists, and oncologists in the forefront of their fields.

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**More than 1.5 million Americans will be diagnosed with cancer this year, so effective prevention is vital.**

## 2008/2009 Cancer Research Breakthrough

In remarkable research sponsored by the National Cancer Institute published in 2008 and 2009, researchers found major abnormalities in content or composition in a complex lipid called *cardiolipin* (CL). These abnormalities are “found in all tumors, linking abnormal CL to irreversible respiratory injury.”<sup>1</sup> Cardiolipin is a fat-based complex phospholipid found in all mitochondrial membranes, almost exclusively in the inner membrane, and is intimately involved in maintaining mitochondrial functionality and membrane integrity. It is used for ATP (energy) synthesis, and consists roughly of 20% lipids.<sup>2</sup>

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## Abnormalities in CL impair mitochondrial function.

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CL serves as an insulator and stabilizes the activity of protein complexes important to the electron transport chain. It also “glues” these protein complexes together.<sup>3</sup> While most lipids are linked together in the endoplasmic reticulum, cardiolipin is synthesized in the mitochondria.

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## In mammals, the main substrate in CL is parent omega-6 (LA) with virtually no parent omega-3 (ALA) or its derivatives.<sup>4</sup>

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This means that humans require plenty of functional omega-6 – the opposite of what many cancer

researchers and physicians believe; they think it is cancer-causing (which it is if adulterated).

Breakthrough research in 2006 by Valeria Fantin and colleagues at Harvard University showed that although mitochondria may be intact in cancerous cells, they don't function properly because their membranes have a high potential, not a low potential as they should.<sup>4</sup> Why does this physiologic change occur? The key is unadulterated, fully functional parent omega-6.

## Major American Heart Association Reversal: Omega-6 Is Good

For over a decade, virtually all cancer researchers and physicians have scorned omega-6; however, in 2009 the American Heart Association started championing parent omega-6 because: “[O]mega-6 PUFAs also have powerful anti-inflammatory properties that counteract any pro-inflammatory activity.”<sup>5</sup>

## The Cancer/Inflammation Connection

Inflammation plays a large part in the development of cancer. This is exactly the condition that renowned cancer researcher Dr. Robert Weinberg of the Massachusetts Institute of Technology (MIT) spoke of in 2007: “The connection between inflammation and cancer has moved to center stage in the research arena.”<sup>6</sup> It is fortunate that a cancer researcher of Weinberg's stature has been influential in refocusing the research community's attention. What does chronic inflammation cause? Massive



amounts of oxygen deployment to the inflamed tissue, if enough oxygen is available.

### **Respiration vs. Glycolysis (Fermentation)**

Over 80 years ago, medical physicist, physiologist, and Nobel Prize-winner Otto Warburg, MD, PhD, proved that a 35% reduction in oxygen causes any cell to either die or turn cancerous. Meticulous (American) experiments performed by renowned researchers from 1953 to 1955 also confirmed the result. While it is understood that heart attacks can stem from lack of oxygen, this is also true of cancer. Most normal, healthy cells get the majority of their energy by using oxygen – in a process called cellular respiration (oxidative phosphorylation) that takes place in the mitochondria. However, cells can also utilize energy without oxygen, and this metabolic process is termed *glycolysis*. This energy method is useful for short-term energy expenditure, such as lifting a weight, but not for long-term energy requirements like running a marathon – it is too energy inefficient. Cancers live and ultimately thrive on the energy from glycolysis, and that is why they need such large vascular networks providing tremendous amounts of carbohydrates. Glycolysis is also a much simpler biochemical process, compared with cellular respiration (oxidative phosphorylation). In the presence of oxygen deficiency, cells that can't obtain enough energy through glycolysis perish. But the cells that succeed in utilizing glycolysis exhibit their innate will to survive; these are the ones that don't die from the oxygen deficiency.

But there is a huge price to be paid for lack of oxygen: lack of cellular intelligence – these cells have the intelligence of “dumb yeast.” In

essence, cancer is the “idiot cell” that can survive but do little more than reproduce more “idiot cells” with no fully functional mitochondria.

### **Recently Released Research Changing Minds**

Most members of the medical and research professions, including those physicians who treat cancer, still mistakenly believe that the answer to the cancer puzzle will be found in oncogenes – genes that predispose the individual toward cancer. The following 2009 statement should give you pause:

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**“There is very little reason to be encouraged that prevention strategies can be revolutionized with what we've discovered so far [on the genetic basis of common diseases].”**

**David Goldstein, Director  
Center for Population Genomics and Pharmacogenetics  
Duke University, Durham, North Carolina**

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### **Geneticists Misled ...**

In 2008, *Scientific American* published an article describing how cancer researchers had been led astray by renowned geneticist Lawrence Loeb's claims of cancer's 10,000 to 100,000 mutations per cell. The reality was that there were only 65 to 475 mutations per cell – not enough mutations to cause cancer!<sup>7</sup> That is why “more research” in this area often yields little, except to motivate the well meaning to contribute more money to finance those researchers' wrong path.

### **Cancer is a Systemic Problem – Not Just a Local One**

Many physicians approach cancer as a localized issue, meaning that they focus only on the affected tissue as the problem because the genes have been ruined there. While this

concept has been advocated in the past, the most recent noteworthy research suggests that the cancerous tissue is the most oxygen-deprived tissue; that's why that particular tissue became cancerous.<sup>8</sup> Once the cell is chronically oxygen deprived, the genetic material does change, but that is solely a consequence, not a cause. You've got much more to worry about than one cancerous area, since many tissues are oxygen deprived along with the cancerous ones. They just haven't reached the critical 35% cellular oxygen deficiency threshold yet.

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**“Breast cancer is not a local problem. It is a systemic [whole body] disease.”<sup>9</sup>**

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I'd like to acknowledge the extraordinary insight of the above 2009 statement by Homer Macapintac, MD, chair and professor of nuclear medicine at the University of Texas M. D. Anderson Cancer Center. The proof of his statement follows.

In 2007 it was reported that tumor-free breast tissue manifests precancerous *epigenetic* changes: “A new study using mastectomy tissue shows that precancerous changes can occur in normal-appearing areas of the breast as distant as two inches from a tumor's edge.”<sup>10,11</sup>

Relying solely on genetics to explain this is difficult at best. With the wealth of new scientific information that has become available over the last two and a half years, it is reasonable to conclude that there has to be a physiologic (epigenetic) cause changing the distant tissue, not vice versa.

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**Nature provides a means to escape quick organ death caused by lack of oxygen by allowing anaerobic glycolysis, but at a price – that is, if the problem isn't fixed, the benign tissue becomes malignant (cancerous) and ultimately destroys its host.**

## Major News Flash in 2007: Physiologic Environment Triggers Cancer

Genetic factors may be less important than initially thought. In 2007 it was also reported that scientists discovered a new type of cell that plays a significant role in the development of cancer – a highly volatile, precancerous stem cell (pCSC) that can either remain benign or become malignant, depending upon environmental cues. “[I]t appears that pCSCs require some sort of signal, or cue, from their immediate environment that directs them to become benign or malignant.”<sup>12</sup> These cancer researchers are on the right path, and by now “connecting the dots” we understand that this environmental cue is lack of oxygen (hypoxia).

### Why Cancers Are Highly Resistant to Treatment Once They Return

Oncologists already know that when cancer returns, chemotherapy often won't work again. The reason for the returning cancer's virulence requires understanding the following three key points:

1. Chemotherapy and radiation kill both respiring (normal) and cancerous (fermenting) cells. If respiration (oxygen transfer) falls below a specific minimum, even for a cancer cell, that cell will die. Normal cells survive chemo and radiation better than cancer cells, because they start with a better respiration; therefore they have stronger residual respiration after chemo/radiation treatments.
2. Oncologists understand that after chemo and radiation treatments, many normal cells are killed and many new cancerous cells are created in which glycolysis takes the place of the cells' ruined respiration. These surviving descendants of normal cells compensate for decreased respiration with increased glycolytic capability. Therefore, the cells that live and haven't been killed are now prime candidates for a continued oxygen-deficient environment. Hypoxia

won't kill these cells, because they already thrive in a deoxygenated environment, making them harder to treat.

3. Therefore, over time, these concentrated groups of functional hypoxic cells can easily become fully cancerous, capable of metastasis; they possess the exact conditions needed to cause more cancer in the future. Chemo and radiation will be much less effective the next time around because we have created (through “treatment”) a more efficient cell with decreased respiration capability that can better utilize glycolysis in a hypoxic environment; that is, cancer.<sup>13-17</sup>

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### **Our cells are struggling to stay alive to keep the oxygen-deprived hypoxic organ alive, but they have a handicap and can't get the necessary oxygen for respiration.**

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#### **2009 Revelation: Number One Cancer in Men Depends on Oxygen Level**

Very recently, a group of investigators studying prostate cancer reported: “Hypoxia, or reduced oxygen levels, in prostate tumors significantly predicts a poor long-term biochemical outcome, regardless of other prognostic factors.... We have followed the patients now for 8 years and it turns out that the patients who had low prostate tumor oxygen levels had much worse outcomes and much more biochemical failures than patients who had normal or higher levels of oxygen in their tumors.”<sup>18</sup> This is a problem because oxygen delivered to a tumor is critical to the treatment for many cancers. For example, radiation therapy creates free radicals that damage DNA in tumors, and oxygen acts as the mediator

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that perpetuates the free radicals. This finding, along with many other medical journal reports concerning the hypoxia/cancer connection in nonprostate cancers, confirms that the greater the oxygen deficiency, the more virulent the cancer.

### **A Possible Cause of Widespread Cellular Oxygen Deficiency**

Cellular oxygen deficiency occurs with long-term consumption of adulterated oils and fats courtesy of the food processing industry, crossing all socioeconomic barriers. Normal but harmful processing and refining ruin omega-6-containing oils, such as canola, safflower, and sunflower oils, and even many olive oils found in supermarkets.

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### **Tragically, we are unknowingly increasing the risk of contracting cancer by eating processed foods.**

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The creation of trans fats by stopping the oxygenation capability of vital oxygenating fats is only one method used by food processors to obtain long shelf life. All commercial cooking oils have significantly impaired oxygen transferability.<sup>8</sup>

Nature in her wisdom has also provided us with an opportunity to fix this problem. Because full-blown cancer takes years to develop, often decades, we have the opportunity to remedy the cells' oxygen deficiency. The great news is that it has already been proven that these precancerous cells can be kept in check so that they either stay benign or are killed as a result of the resupply of cellular oxygen.

### **Identifying the Appropriate Omega-6:-3 Ratio**

My research emphasis over the past 15 years has been on deducing the appropriate supplemental ratio of



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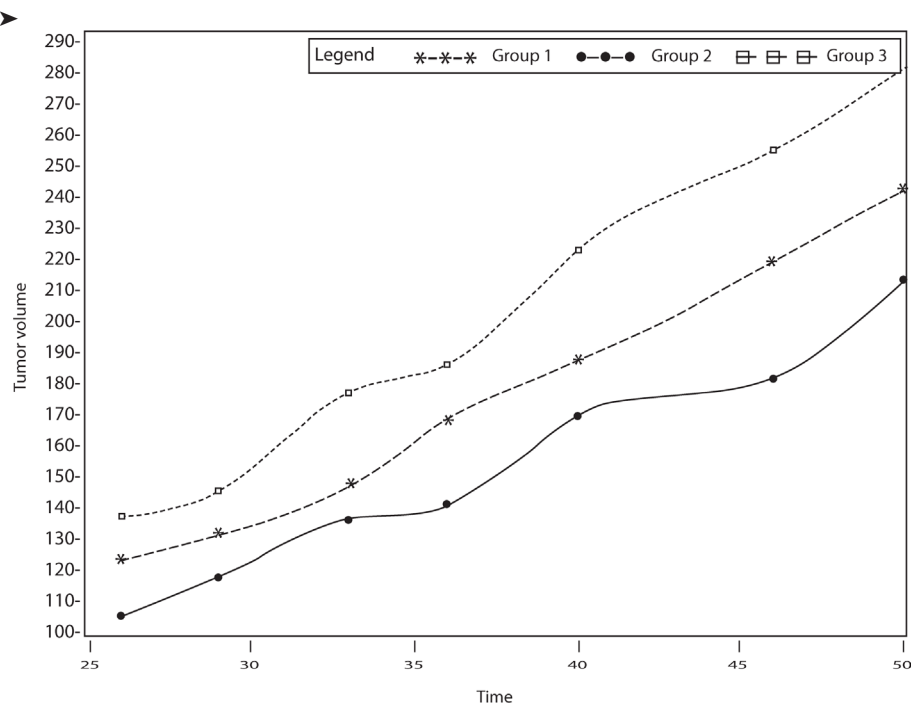


Figure 1: Group 2 (bottom) was pretreated with PEO formulation for 4 weeks prior to tumor implantation. Group 1 (middle) was pretreated for 2 weeks prior to tumor implantation. Group 3 (top) is the control.

physiologic omega-6:-3 to maximally oxygenate cells and keep their mitochondria optimal. Parent omega-3 (not fish oil derivatives, such as EPA) is required in each cell, although we require much more unadulterated parent omega-6.<sup>19</sup> Most physicians think that the majority of “parents” automatically become transformed into “derivatives,” so fish oil makes an appropriate supplement. This is questionable at best when referring to the most current research. It was published in 2008 that EFA derivatives (including DHA and EPA) are made “as needed” by the body and a maximum of only 1% to 5% of parents become derivatives; the majority, over 95%, remain as parents in the cell.<sup>20</sup> Other journal articles report less than 1% normal conversion amounts.<sup>21</sup> In view of these new findings, fish oils give a pharmacologic overload of derivatives. Consequently, practitioners may need to reevaluate their recommendations.

In my research, I commissioned and directed an experiment with mice to study the relationship between cancer

growth rates and supplementation with Peskin Protocol PEOs.<sup>22</sup> Mice metabolize EFAs as humans do, so these experimental results are directly applicable to humans. This seminal experiment showed that, in spite of tumor implantation with 2 million cancer cells at once, there was a statistically significant 24% reduction in tumor size (growth) in the longer 4-week pretreated mice compared with the control mice that received no PEO supplementation. 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 compared to the untreated mice. These results clearly show the increasing value of a longer pretreatment period of PEOs, and that PEO-based oils are modifying the cells’ internal structure in an epigenetic fashion, making them more cancer resistant.

## Notes

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