Nutritional & Flexoelectric Breakthroughs for the Relief of Prolonged Inflammation

As scientists delve deeper into the root causes of illnesses they are finding that inflammation – the body’s first defense against infection – when chronic rather than transitory – leads to a host of diseases. Chronic and prolonged inflammation destabilizes cholesterol deposits in the coronary arteries leading to heart disease, chews up the brain cells of Alzheimer’s victims, fosters carcinogenesis and causes serious and permanent damage to the organism.

Pharmaceutical researchers are jumping on the anti-inflammatory bandwagon to understand and treat the cascade of events that fuels prolonged inflammation. Millions of people rely on non-steroidal anti-inflammatory (NSAIDs) and the so-called Cox-2 inhibitors for pain relief (the most widely prescribed drugs worldwide with sales now exceeding six billion dollars a year).1,2

Despite over a decade of effort and research, there is still only limited and inclusive evidence on the long-term safety of these drugs. In fact, an excellent review article by McTaggart cited a significant body of research published in prestigious medical journals (JAMA, New England Journal of Medicine, Lancet, and Annals of Internal Medicine) revealing that NSAIDs and Cox-2 inhibitors have side effects: internal bleeding, kidney dysfunction, gastrointestinal ulcers, increased risk of heart disease, cardiac arrest, and sudden and unexpected death.3

Indeed, consumers of Cox-2 drugs were outraged to find out that a JAMA study of 8,076 patients reported a 238% higher risk of having a cardiovascular event (heart attack, angina attack, blood clot, cardiac arrest, sudden or unexpected stroke or a mini-stroke of the brain, and death) while taking these pain-relieving drugs. While patients may enjoy relief from pain on these drugs, they are beset with GI ulcers that commonly require the use of another medication to alleviate gastric pain and the side effects caused by the original drugs they were taking for their condition.4

Despite their initial promises, drug companies have failed to develop better treatments for disorders of inflammation. To our great satisfaction, though, a chance discovery was made that has led to what promises to be a successful nutritional manipulation of prostaglandin (PG) pathways in the direction of supplementing with nutrient precursors and co-factors that support and nourish the anti-inflammatory PG pathway (PG1). Using a mix of scientific evidence with good old-fashioned intuition and biochemical ingenuity led to the development of a novel organic compound.5 In time, our insights and those of others may lead to further advances in the treatment of pro-inflammatory disorders.

Understanding the Biochemistry of Prolonged Inflammation

Stopping prolonged inflammation requires a brief review of nutritional biochemistry. The normal orchestration of PGs is highly complex and involves myriad compounds to thwart inflammation. For example, in recent years, nutritional scientists have pinpointed key nutritional deficiencies involved in the inflammatory cascades that lead to an increase in cancer, cardiovascular disease, and neurodegenerative disorders. While the picture of the role of nutrition in inflammation is not yet completely clear, there’s enough new information to make alternative treatments more specific and effective.

Before discussing nutritional solutions, it’s useful to understand that currently available NSAIDs (including Cox-2 inhibitors) block both Cox-1 and Cox-2 enzymes.6-7 While most pharmacologists and nutritionists believe selectively blocking Cox-2 is the answer in halting prolonged inflammation, the core issue in Cox-2 overexpression is the depletion of key nutrient precursors and co-factors that maintain the delicate balance of the prostaglandins. Furthermore, since both Cox-1 and Cox-2 have physiological functions in modulating PGs to counter inflammation when there is trauma or infection, it makes no sense to inhibit them for long periods of time (cox-1 is the housekeeping enzyme that maintains normal circulation and prevents mucosal damage to the gastrointestinal tract).

Silencing Cox-1 and Cox-2 delays the healing of gastric and duodenal ulcers and inflammation. The result: prolonged states of gastritis and duodenitis inhibit the digestion, absorption and assimilation of nutrients, leaving the patient malnourished and deficient. In fact, when inflammation is chronic, the only nourishment these patients can easily absorb and assimilate is empty calories that increase abdominal fat and lower the metabolic rate. In these cases, nutritional lacks weaken cell-mediated immunity causing deep-rooted infections and deficiencies of the precursors/co-factors needed to support the natural cycles of inflammation, repair and regeneration. Clearly, blocking cox enzymes is responsible for the disastrous side effects reported in the literature. To complicate matters further, the increase in Body Mass Index has quadrupled from 1986 to 2000 with mounting evidence documenting that abdominal fat is an active metabolic organ that pumps out powerful pro-inflammatory mediators that induce and promote inflammation.8 Could the rise in pain syndromes be related to the fact that 64% of Americans are overweight or obese or struggling to conquer their expanding waistlines?

In answering this question, it is important to note that the PG pathways are central to overall health and to the proper, short-term expression of pro-inflammatory mediators.
Nutritional manipulation of this pathway may provide a safe and effective treatment strategy for inflammatory disorders and metabolic syndromes associated with insulin resistance and low endocrine function, especially of the thyroid gland. When there is prolonged inflammation, the liver fails to convert T4 to the active hormone T3 causing a mitochondrial energy crisis and an increase in abdominal fat that further fuels the cycles of prolonged inflammation. Furthermore, it is well known that environmental stressors, both xenobiotic and microbial have increased dramatically in the past few decades, putting abnormal amounts of pressure on the PG pathways.

**Nutritional Goals to Support PGI**

The nutritional goals of supporting and nourishing the body’s natural anti-inflammatory and repair mechanisms are:

1. to systematically, and in the proper sequence, remove stressors that induce and activate the enzyme phospholipase A2 that cleaves arachidonic acid (AA) from the phospholipids of cell membranes. Since endotoxins and cortisol are the key stressors that induce phospholipase A2 raising AA, it is critical to systematically remove microbial stressors. By lowering blood levels of converted AA the Cox enzymes will not be overexpressed. In addition, since nearly all potent mediators of inflammation are derivatives of AA, reducing the conversion of AA to leukotrienes will undoubtedly prove to be beneficial in treating inflammatory disorders.

2. to increase the levels of Gamma-linolenic acid (GLA) and dihomogamma-linolenic acid (DGLA). After decades of clinical research, we discovered that the missing link in facilitating transport and utilization of GLA, was that it had to be in a non-oily, plant form and bound to a metalloprotein carrier, specifically histidine and organic zinc.

3. to inhibit the overexpression of delta-5-desaturase (D-5-D), the enzyme that has hierarchy over cox enzymes and that is maintained by the insulin-glucagon ratio. GLA is precursor to DGLA. DGLA is converted to arachidonic acid (AA) through the enzyme action of D-5-D which, in turn, triggers the production of pro-inflammatory leukotrienes. However, when histidine is organically fused with plant-based GLA and organic zinc, this nutritional compound becomes a powerful leukotriene synthesis inhibitor, inhibiting D-5-D enzyme activity. This novel histidine-GLA-zinc (HGZ) complex – operating via known biochemical pathways of H2 histamine receptors – provides the safest and most effective anti-inflammatory actions in the body. When utilized long enough to correct deficiency states, HGZ can safely and effectively shift fatty acid metabolism away from pro-inflammatory mediators (preventing the excessive conversion of GLA into DGLA) avoiding the whirlpool of pro-inflammatory mediators that underlie prolonged inflammation. Protein-bound organic zinc efficiently corrects zinc deficiencies that are known to be involved in insulin sensitivity, prostaglandin synthesis and immune function.

4. improve the conversion of alpha-linolenic acid (ALA) to eicosapentaenoic acid (EPA) and docosahexaenoic acid.

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(DHA). Studies now estimate that only 0.2% of ALA is converted into DHA and EPA. With HGZ, the conversion of ALA to DHA is enhanced between 25 and 30%. It is well known that poorly metabolized oils increase free radical damage and have been linked to prostate cancer, arteriosclerosis, and a wide spectrum of inflammatory disorders.

Nutritionists are unaware that Omega-6 essential fatty acids can actually whirl the wheels of inflammation faster and propel inflammation deeper into the organism. Flax, borage, black current seed oil, evening primrose oil yield poor clinical results when HGZ is deficient. Why? HGZ is needed to aid in the proper metabolism and utilization of EFAs. Moreover, silent chronic infections lie at the root of inflammatory disorders and generate endotoxins that trigger excessive cortisol, thereby accelerating inflammation throughout the entire organism.

To make matters worse, excess cortisol from stressors (emotional, microbial, xenosterogens, endotoxins, dental focal toxicosis, etc) blocks the action of the enzyme, histidine decarboxylase, which slows the degradation of histidine and prevents conversion to histamine causing poor gastric digestion and low hydrochloric acid levels and deficiencies of histadine, GLA and zinc. Nickel toxicity is the most powerful inhibitor of this enzyme. And, since histidine is the most powerful chelator of nickel and heavy metals, it makes sense that deficiencies of zinc and histidine will cause ongoing metal-induced toxicity and oxidative stress, and trigger progressive inflammation.

Moreover, since histamine2 receptors regulate the immune system and cyclic AMP energizes T-cells and B-cells functions, it becomes apparent why nutritional support of the body’s anti-inflammatory and digestive functions is critical to curb inflammatory cascades.

Flexoelectric Goals to Re-establish Coherence

HGZ is an organic, non-covalent, metalloprotein matrix with plant-based GLA. The organic fusion of HGZ is accomplished via flexoelectric technology12 that synchronizes quantum fluctuations and creates a coherent, amplified crystalline resonant field to propel these nutrients deep within the cells of the body, thereby upregulating nutrient uptake. We utilize other flexoelectric technology, specifically the Quantum Coherence Generator™, to enhance electron transfer functions, stabilize molecular defenses to reduce oxidative stress in the sub-molecular realm, and regulate meridian energy flow and cranial-sacral rhythms. By reestablishing cellular resonance and eliminating chaos we are able to clear out toxins that induce inflammation. Moreover, this technology is designed to correct faulty brain-proprioceptive feedback, a factor in all prolonged neuromuscular pain and inflammation. As an added bonus, our preliminary findings show that the flexoelectric technology may adjust the pineal gland’s resonance to metabolize fat without the negative effects of lipid peroxidation.

Poor nutritional status of the host wrecks the immune response. Prolonged inflammation, a process that locks microbial infections deep within the extracellular matrix or ground regulation system of the body, causes immunological unresponsiveness. Plus, the effect of a nutritional deficiency on the pathogen itself is interesting as a nutritional lack causes a virulent strain to become more virulent as the virus itself aggressively proliferates when there is active inflammation.13

In summary, it seems certain that a solution to unwanted inflammation should leave physicians with an unprecedented window into disease mechanisms and how the body works. Despite popular beliefs, the solution to prolonged inflammation will not be found in genomics but in the: 1. correction of nutritional deficiencies using unique carrier proteins and channel proteins to effectively gate nutrients into the cell, thereby shifting fatty acid metabolism away from pro-inflammatory mediators,5 and 2. removal of the inducers and promoters of prolonged inflammation in the proper sequence.15

Pain, due to prolonged inflammation, can be debilitating and discouraging. Chronic inflammation is a result of the body being at war when inflammatory fires flare up for prolonged periods causing permanent damage to the organism. Medical science has little to offer pain sufferers in the way of a cure. In fact, many researchers have raised a cautionary flag regarding long-term use of NSAIDs and Cox-2 inhibitors. Instead of treating the symptoms, pharmacologists seem to be ignoring the nutritional biochemistry necessary for the body to thwart prolonged inflammation and pain. The force of destruction and the force of prevention, maintenance and repair are regulated by PGs. By modulating PG1 and re-establishing coherence, we set the body’s maintenance levels higher so that it counterbalances the force of destruction.

*The flexoelectric prototypes used by the authors were donated by www.ewater.com/quantum. The statements made in this article regarding these types of products have not been evaluated by the Food and Drug Administration. Furthermore, the concepts and ideas presented and tested in this column reflect clinical research and is not intended to treat, cure, or prevent disease. This clinical research was done without compensation from any funding source or private/professional corporation and neither author is employed nor is a consultant of any company that sells flexoelectric technology.

References


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