

Title:

***Nutritional Support for Sport Inflammation
(Natural Oxygenation Stimulation Formula
and additional Fatty Acids Support)***

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Abstract:

This discussion proposes the effects of a natural oxygenation formula with additional nutritional support on sport fatigue pain, and sport injury performance. The SCIO treatment provides a basic repair stimulation signal for cellular rejuvenation. Diseased tissue has a different type of electrical signature than healthy tissue. When the SCIO detects an injured tissue signal it responds with a curative stimulation electrical pattern to promote and speed healing. There are also many additional effects from the device to enhance sport performance in general.

There are many additional nutritional items that can help with sport injury repair. Most concerns of medicine are for the synthetic compounds and steroids marketed by

greedy chemical companies. Here we approach a natural path with out synthetic compounds.

Key Words:

Stimulation, Flower Pollen, Pangamic Acid, Oxygenation, Xrroid, SCIO

Since flower pollen and certain yeast's RNA and DNA components have been demonstrated to be an oxygenator supplement for stimulating the Brain. It has been used as an energy kick pill to complement sport training exercises. The B vitamins are well documented for brain Stimulation and are one of the few documented thinking enhancers.

The Russian scientists in the 1950 have shown the profound oxygenation stimulation effects of Pangamic Acid (known as B₁₅). In the 1950's there was an over reaction of the American FDA to certain B vitamins. They labeled B₁₅ as illegal to make in America. This B₁₅ formula was called liquid Oxygen by the Russian developers. The sports effects were profound and the soviets lead the world in Olympic events for the next decades. Since the wellness of any organ or organism is dependent on how well it uses oxygen, Pangamic acid has an overall tonic or panacea for any condition.

Perhaps a combination of other known oxygenators with pollen can provide a synergistic effect for cellular oxygenation. Since the action of the pollen seems to be from the nucleotides and the trace elements in the pollen, providing an extra source of nucleotide might facilitate absorption. Towards this goal, RNA and DNA from yeast sources were added to our formula. Since nucleotide absorption depends on protein-digesting enzymes (deficient in most clients), comfrey pepsin is added to the herbal base where the protease pepsin lies dormant in its protein-breaking-up action waiting for HCL in the stomach to activate it into pepsinogen. B₁₂, folic acid and most importantly, B₁₅ were also added to the formula for their strong oxygenation abilities and their methyl donor action, fortifying both lung and liver action. B₁₇ + B₁₈ + B₁₉ + B₂₀ were also added in trace amounts from herbal sources. This addition of the higher B complex (B₁₂, folic acid, B₁₅, and B₁₇) helps stabilization of nerval function as well.

Oxygenation and stabilization of blood pH is also dependent on zinc in the form of zinc anhydrase and other zinc dependent enzymes. Since the average American diet is deficient in zinc, a trace amount is added to the formula.

So our formula for oxygenation will include the following.

Formula:

- _____ RNA, DNA (yeast type)
- _____ Thiamine B₁
- _____ Riboflavin B₂
- _____ Niacin B₃
- _____ Pantothenic Acid B₅
- _____ Pyridoxine B₆
- _____ Choline B₁₁
- _____ Biotin B₁₀
- _____ Folic Acid B₉
- _____ Pangamic Acid B₁₅ (pangamate yeast carrier)
- _____ B₁₆, B₁₇, B₁₈, B₁₉, B₂₀ --all natural source
- _____ Hunzas Bee Pollen
- _____ Zinc Aspartate (chelated)
- _____ Comfrey Pepsin
- _____ Free Fatty Acids
- _____ Minerals- Calcium, Phosphorous, Potassium, Magnesium
- _____ Trace Minerals- Iron, Tin, Zinc, Manganese,

Inflammation

Systemic **inflammation** is an underlying cause of many seemingly unrelated, sport -related injuries. Systemic **inflammation** can inflict devastating degenerative effects throughout the body there can be a natural method of nutritional support for treatment. This fact is often overlooked by the medical establishment, yet persuasive scientific evidence exists that correcting a chronic inflammatory disorder will enable many of the sport injuries to be prevented or reversed.

The pathological consequences of **inflammation** are well-documented in the medical literature (Willard et al. 1999; Hogan et al. 2001). Regrettably, the dangers of secondary **inflammation** continue to be ignored, even though proven ways exist to reverse this process. The inflammatory cascade can be significantly reduced with natural methods.

Injury results in an increase of inflammatory cytokines (destructive cell-signaling chemicals) that contribute to the progression of many diseases later in life (Van der Meide et al. 1996; Licinio et al. 1999). Rheumatoid arthritis is a classic autoimmune disorder in which excess levels of cytokines such as tumor necrosis factor-alpha (TNF-a), interleukin-6 (IL-6), interleukin 1b [IL-1(b)], and/or interleukin-8 (IL-8) are known to cause or contribute to the inflammatory syndrome (Deon et al. 2001). Improper and or unnatural treatment of the sport injury at an early age can lead to a progressive disease at later life.

Progressive **inflammation** is also involved in diseases as diverse as atherosclerosis, cancer, heart valve dysfunction, obesity, diabetes, congestive heart failure, digestive system diseases, and Alzheimer's disease (Brouqui et al. 1994; Devaux et al. 1997; De Keyser et al. 1998). In aged people with multiple degenerative diseases, the inflammatory marker, C-reactive protein, is often sharply elevated, indicating the presence of an underlying inflammatory disorder (Invitti 2002; Lee et al. 2002; Santoro et al. 2002; Sitzer et al. 2002). When a cytokine blood profile is conducted on people in a weakened condition, an excess level of one or more of the inflammatory cytokines, e.g., TNF-a, IL-6, IL-1(b), or IL-8, is usually found (Santoro et al. 2002). **Preventing**

Progressive Inflammatory-Related Disease

The New England Journal of Medicine published several studies in the year 2000 showing that the blood indicators of **inflammation** are strong predictive factors for determining who will suffer a heart attack (Lindahl et al. 2000; Packard et al. 2000; Rader 2000). The January 2001 issue of Life Extension Magazine described these studies and explained how individuals could protect themselves against these inflammatory markers (such as C-reactive protein, homocysteine, and fibrinogen).

A growing consensus among scientists is that common disorders such as atherosclerosis, colon cancer, and Alzheimer's disease are all caused in part by a chronic inflammatory syndrome.

Seemingly unrelated diseases have a common link. People who have multiple degenerative disorders often exhibit excess levels of pro-inflammatory markers in their

blood. Here is a partial list of common medical conditions that are associated with chronic **inflammation**:

Diseases Related To Chronic **Inflammation**

Disease	Mechanism
Allergy	Inflammatory cytokines induce autoimmune reactions
Alzheimer's	Chronic inflammation destroys brain cells
Anemia	Inflammatory cytokines attack erythropoietin production
Aortic valve stenosis	Chronic inflammation damages heart valves
Arthritis	Inflammatory cytokines destroy joint cartilage and synovial fluid
Cancer	Chronic inflammation causes many cancers
Congestive heart failure	Chronic inflammation contributes to heart muscle wasting
Fibromyalgia	Inflammatory cytokines are elevated
Fibrosis	Inflammatory cytokines attack traumatized tissue
Heart attack	Chronic inflammation contributes to coronary atherosclerosis
Kidney failure	Inflammatory cytokines restrict circulation and damage nephrons
Lupus	Inflammatory cytokines induce an autoimmune attack
Pancreatitis	Inflammatory cytokines induce pancreatic cell injury
Psoriasis	Inflammatory cytokines induce dermatitis
Stroke	Chronic inflammation promoted thromboembolic events
Surgical complications	Inflammatory cytokines prevent healing

A critical inflammatory marker is C-reactive protein. This marker indicates an increased risk for destabilized atherosclerotic plaque and abnormal arterial clotting. When arterial plaque becomes destabilized, it can burst open and block the flow of blood through a coronary artery, resulting in an acute heart attack. One of the New England Journal of Medicine studies showed that people with high levels of C-reactive protein were almost three times as likely to die from a heart attack (Ridker et al. 1997).

IMUNE advises sport athletes current and retired to have an annual SCIO - C-reactive protein blood test to detect systemic **inflammation** that could increase the risk of heart attack, stroke, cancer and a host of age-related diseases. In fact, on January 28, 2003,

the American Heart Association and Centers for Disease Control & Prevention (CDC) jointly endorsed the C-reactive protein test to screen for coronary-artery **inflammation** to identify those at risk for heart attack.

Possible Aetiology of Elevated C-reactive Protein?

- [Elevated C-Reactive Protein and Interleukin-6 Predict Type II Diabetes](#)

While some doctors are finally catching on to the fact that elevated C-reactive protein increases heart attack and stroke risk, they still know little about its other dangers. Even fewer practicing physicians understand that pro-inflammatory cytokines are an underlying cause of systemic **inflammation** that is indicated by excess C-reactive protein in the blood.

In an abstract published in the March 6, 2002 issue of the Journal of the American College of Cardiology (JACC), tumor necrosis factor-alpha (TNF-a) levels were measured in a group of people with high blood pressure and a group with normal blood pressure (Verdecchia et al. 2002). The objective of this study was to ascertain if arterial flow mediated dilation was affected by hypertension and chronic **inflammation** as evidenced by high levels of the pro-inflammatory cytokine TNF-a.

The hypertensive subjects taking anti-hypertensive medications had about the same blood pressure as the healthy test subjects. Arterial flow mediated dilation, however, was significantly impaired in the hypertensives and this group also showed higher levels of TNF-a, indicating persistent **inflammation** despite blood pressure control. This study showed that even when blood pressure is under control, hypertensives still suffer from continuous damage to the inner lining of the arterial wall (endothelial dysfunction) caused by a chronic inflammatory insult. The doctors who conducted this study concluded by stating:

"Antihypertensive therapy alone may be insufficient to improve endothelial dysfunction in hypertensives with high plasma levels of inflammatory markers. Additional therapy to target **inflammation** may be necessary to improve endothelial function and to prevent progression of coronary atherosclerosis in high-risk hypertensives with subclinical inflammations."

A sensitive index to evaluate how much endothelial damage is occurring is the measurement of TPA (tissue-type plasminogen activator), a clot-dissolving enzyme found in the blood. This same study showed elevated TPA levels in hypertensives, indicating continued endothelial damage despite blood pressure reduction. These findings indicate that hypertensives should have their blood tested for both TNF-a and TPA to assess how much inner wall (endothelial) arterial damage is occurring (Verdecchia et al. 2002). If TNF-a and/or TPA levels are high, aggressive therapies to suppress the inflammatory cascade should be considered.

Elevated C-Reactive Protein and Interleukin-6 Predict Type II Diabetes In a study published in the July 18, 2001 issue of the Journal of the American Medical Association, a group from the famous Women's Health Study was evaluated to ascertain what risk factors could predict future development of Type II diabetes (Pradhan et al. 2001). The findings showed that baseline levels of C-reactive protein and interleukin-6 (IL-6) were significantly higher among those who subsequently developed diabetes compared to those who did not.

When comparing the highest versus lowest quartile, women with the higher IL-6 levels were 7.5 times more likely to develop diabetes while those in the higher C-reactive protein ranges were 15.7 times more likely to become diabetic. After adjusting for all other known risk factors, women with the highest IL-6 levels were 2.3 times at greater risk, while those with the highest C-reactive protein levels were 4.2 times more likely to become diabetic. It should be noted that these other diabetic risk factors (such as obesity, estrogen replacement therapy and smoking) all sharply increase inflammatory markers in the blood. The doctors who conducted this study concluded by stating:

"Elevated C-reactive protein and IL-6 predict the development of Type II diabetes mellitus. These data support a possible role for **inflammation** in diabetogenesis."

C-Reactive Protein and IL-6 Predict Death

It is well established the elevated C-reactive protein, IL-6 and other inflammatory cytokines indicate significantly greater risks of contracting or dying from specific diseases (heart attack, stroke, Alzheimer's disease, etc.).

A group of doctors wanted to ascertain if C-reactive protein and IL-6 could also predict the risks of all-cause mortality. In a study published in the American Journal of Medicine, a sample of 1,293 healthy elderly people were followed for a period of 4.6 years (Harris et al. 1999). Higher IL-6 levels were associated with a twofold greater risk of death. Higher C-reactive protein was also associated with a greater risk of death, but to a lesser extent than elevated IL-6. Subjects with both high C-reactive protein and IL-6 were 2.6 times more likely to die during follow up than those with low levels of both of these measurements of **inflammation**. These results were independent of all other mortality risk factors. The doctors concluded by stating:

"These measurements (C-reactive protein and IL-6) may be useful for identification of high-risk subgroups for anti-inflammatory interventions."

In a study of almost 5,000 elderly people, scientists discovered that frail seniors were more likely to have signs of increased **inflammation** than their more active counterparts. This study was published in the Archives of Internal Medicine (Walston et al. 2002) and showed that these frail seniors with elevated blood inflammatory markers also tended to show more clotting activity, muscle weakness, fatigue and disability than active elderly people.

Findings from these studies should motivate every health conscious individual to have their blood tested for C-reactive protein. If it is elevated, then the Inflammatory Cytokine Test Panel is highly recommended. Those who suffer from any type of chronic disease

may also consider the Inflammatory Cytokine Test Panel in order to identify the specific inflammatory mediator that is causing or contributing to their problem.

Glycation's Role in **Inflammation** Eating high temperature cooked food is another contributor in the production of inflammatory cytokines. In fact, it has been shown that eating high temperature cooked food leads to the formation of advanced glycation end (AGE) products. Glycation can be described as the binding of a protein molecule to a glucose molecule resulting in the formation of damaged protein structures. Many age-related diseases such as arterial stiffening, cataract and neurological impairment are at least partially attributable to glycation. These destructive glycation reactions render proteins in the body cross-linked and barely functional. As these degraded proteins accumulate, they cause cells to emit signals that induce the production of inflammatory cytokines.

The glycation process is presently irreversible, though an important study indicates a drug in clinical trials may be partially effective. According to a Proceedings of the National Academy of Sciences study, consuming foods cooked at high temperature accelerates the glycation process, and the subsequent formation of advanced glycation end products.

A more succinct descriptive term for "advanced glycation end products" is "glycotoxin," since "advanced glycation end products" are toxic to the body. We will use the word "glycotoxin" from here on to describe the term "advanced glycation end products."

Inflammation: Chronic

Cooking Injury and Aging Have Similar Biological Properties Cooking foods at high temperatures results in a "browning" effect, where sugars and certain oxidized fats react with proteins to form glycotoxins in the food. Normal aging can also be regarded as a slow cooking process, since these same glycotoxins form in the skin, arteries, eye lenses, joints, cartilage, etc. of our body.

The Proceedings of the National Academy of Sciences study shows that consuming foods high in glycotoxins might be responsible for the induction of a low-grade, but chronic state of inflammation. In addition, the glycotoxins in food cooked at high temperatures also promote the formation of glycotoxins in our living tissues. The implication of these findings is profound.

What one eats plays a major role in chronic inflammatory processes. Consuming low glycemic foods prevents the insulin surge that contributes to chronic inflammatory processes. It is also important to avoid over consumption of foods high in arachidonic acid (beef, egg yolk, dairy, etc.).

We now know that eating too much over-cooked food causes an increase in inflammatory cytokines. Since most "junk" foods are cooked at extremely high temperatures, it makes sense to avoid French fries, hamburgers, potato chips, fried food and other snacks. These foods not only contain lots of glycotoxins, they also create other metabolic disorders that can induce degenerative disease.

Consuming at least 1000 mg a day of carnosine, and/or 300 mg of the European drug aminoguanidine can inhibit pathological glycation reactions in the body. Eating high temperature cooked foods also induces the formation of glycotoxins. Avoiding foods cooked at high temperature not only reduces pathological glycation processes, but also prevents the formation of numerous gene-mutating toxins that are known carcinogens.

Food is cooked to destroy bacteria and other pathogens that could cause a serious illness. It is important not to eat undercooked food, but avoiding food unnecessarily cooked at higher temperatures is desirable. Certain foods (like fried foods) have to cook at high temperatures. Health conscious people are increasingly avoiding fried foods because they are associated with many health risks.

With the availability of cytokine blood profile tests, it is now possible to ascertain the underlying cause of chronic inflammatory disease. The appropriate drugs, nutrients, dietary change(s) and/or hormones can then be used to suppress the specific cytokines (such as IL-6 or TNF- α) that are promoting the inflammatory

Diet and Inflammation In addition to toxic cytokines, there are other inflammatory pathways that can be mediated via diet modification. A common problem involves overproduction of pro - inflammatory hormone-like "messengers" (such as prostaglandin E2) and underproduction of anti-inflammatory "messengers" (such as prostaglandin E1 and E3).

The good news is that omega-3 fatty acids found in fish oil help to suppress the formation of undesirable prostaglandin E2 and promote synthesis of beneficial prostaglandin E3 (Kelley et al. 1985; Watanabe et al. 2000). Gamma - linolenic acid (GLA) induces production of the anti-inflammatory prostaglandin E1 (Das et al. 1989; Fan et al. 1997). What you eat can significantly affect whether you have more of the beneficial prostaglandins (E1 and E3) as opposed to the pro-inflammatory prostaglandin E2.

Because prostaglandin E2 is a culprit in inflammation, reducing the consumption of foods that are high in omega-6 fatty acids and increasing the consumption of omega-3 rich foods, such as salmon and other fish, can be beneficial. Limiting foods that convert to arachidonic acid can help reduce inflammation. Arachidonic acid is a precursor to both prostaglandin E2 and the pro-inflammatory cytokine leukotriene B(4) (Brock et al. 1999). Another dietary factor that can lead to high levels of arachidonic acid is the overconsumption of high-glycemic index carbohydrates that cause excess production of insulin (Kreisberg et al. 1983). These quickly digestible foods include fruit juices or rice cakes. Food heavy in polyunsaturated fats or saturated fats can also increase prostaglandin E2.

Additionally, a study of elderly patients with heart disease requiring elective surgery (Tepaske et al. 2001) found that nutritional supplements containing omega-3 polyunsaturated fatty acids (as well as yeast and L-arginine) improved the outlook for high-risk patients when given a minimum of 5 days prior to surgery.

The number of inflammatory-related diseases that could be successfully treated with cytokine-lowering therapy is staggering. PTX and supplements such as fish

oil, nettle leaf, DHEA, and vitamin K possess mechanisms of suppressing inflammatory cytokines. Unfortunately, there are no side-by-side comparisons to enable us to categorically state whether PTX or natural agents (such as DHA fish oil) work better.

Foods cooked at high temperatures can produce a browning effect in which glycotoxins are formed from the reaction of sugars and oxidized fats with protein. Glycotoxins may contribute to low-grade chronic inflammation. High glycemic foods may also contribute to the inflammatory process. Dietary modifications to reduce inflammation should include elimination of foods and cooking processes that contribute to a chronic state.

For those who have multiple degenerative diseases, the cytokine profile blood test and the C-reactive protein blood test are highly recommended. This may be done through your own physician or Health care professional. If your cytokine test reveals excess levels of cytokines such as TNF-a, IL-1(b), or both, nutritional supplementation, dietary modifications, or the following

The following supplements are suggested:

- § The docosahexaenoic acid (DHA) fraction of fish oil may be the most effective nonprescription supplement to suppress pro-inflammatory cytokines. Gamma-linolenic acid (GLA) is a precursor of PGE1, a potent anti-inflammatory agent. A product called Super EPA/DHA provides 1400 mg of EPA and 1000 mg of DHA in 4 capsules.**
- § DHEA is a hormone that decreases with age. DHEA has been shown to suppress IL-6, an inflammatory cytokine that often increases as people age. Typical doses of DHEA are 25-50 mg daily, although some people take 100 mg daily. Refer to the DHEA Replacement protocol for suggested blood tests to safely and optimally use DHEA.**
- § Nettle leaf has been shown to suppress the proinflammatory cytokine TNF-a. Take 1000 mg daily.**
- § Vitamin E and N-acetyl-cysteine (NAC) are protective antioxidants with anti-inflammatory properties. Vitamin E that contains gamma-tocopherol and tocotrienols provides the most broad-spectrum protection. Take 1 capsule daily of Gamma E Tocopherols with Sesame Lignans and**

Tocotrienols with Sesame Lignans. NAC is an amino acid with antiviral and liver protectant properties. One 600 mg capsule daily is recommended.

- § Vitamin K helps reduce levels of IL-6, a pro-inflammatory messenger. Vitamin K also helps in the treatment of osteoporosis by regulating calcium and promoting bone calcification. One 10 mg capsule daily is recommended for prevention purposes. Do not take vitamin K if you are taking Coumadin or some other type of anticoagulant medicine.**
- § Consuming at least 1000 mg per day of carnosine and/or 300 mg of the European drug aminoguanidine can inhibit pathological glycation reactions in the body.**

Fatty Acid Liquescence 10 drops 3 times a day

Sport Oxygenator 5 pills at bed

Glucoseamine 1000 mg a day during inflammation

Chondrotin 1000 mg per day during inflammation

ANTI-INFLAMATION HOMEOPATHIC 6 DROPS 3/DAY 6 DROPS 3/DAY

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Inflammatory Response

The inflammatory response is a complex process through which many parts of the body overcome the stress of wounds and return the body to homeostasis. The primary function of the inflammatory response is to bring phagocytic cells (neutrophils and monocytes) to the inflamed area to destroy bacteria and rid the tissue spaces of dead and dying cells so that tissue repair can begin. The illustrations below trace the steps of the inflammatory response.

Inflammation produces four cardinal signs: redness, swelling, heat, and pain. The first three signs result from local vasodilation, fluid leakage into the extravascular space, and blockage of lymphatic drainage. The fourth results from tissue space distention caused by swelling and pressure, and from chemical irritation of nociceptors (pain receptors).

The acute phase of the inflammatory response typically lasts 2 weeks; the subacute phase (a less intense version of the acute phase), 2 weeks.

- (1) Splinter punctures epidermis
- (2) Bacteria introduced
- (3) Bacteria implanted in tissue
- (4) Injured cells release histamine and kinins, causing capillary dilation
- (5) Dilated capillaries make skin hot and red; escaping fluid from blood vessels causes swelling; edema, kinins, and other substances produce pain; neutrophils and monocytes migrate through vessel walls toward bacteria
- (6) Neutrophils and monocytes destroy bacteria by phagocytosis

