High Point Health Solutions

Life Changing Care

Anti- Oxidants vs. Free Radicals

Dr. Harry Schick, DC
Highland Park, NJ 08904
732•249•9800

Free radicals are highly reactive chemicals that have the potential to harm cells. They are created when an atom or a molecule (a chemical that has two or more atoms) either gains or loses an electron (a small negatively charged particle found in atoms). Free radicals are formed naturally in the body and play an important role in many normal cellular processes. At high concentrations, however, free radicals can be hazardous to the body and damage all major components of cells, including DNA, proteins, and cell membranes. The damage to cells caused by free radicals, especially the damage to DNA, will play a role in the development of cancer, aging, chronic disease and other health conditions.

Antioxidants are chemicals that interact with and neutralize free radicals, thus preventing them from causing damage. Antioxidants are also known as "free radical scavengers."

The body makes some of the antioxidants it uses to neutralize free radicals. These antioxidants are called endogenous antioxidants. However, the body relies on external (exogenous) sources, primarily the diet, to obtain the rest of the antioxidants it needs. These exogenous antioxidants are commonly called dietary antioxidants.

Free radicals come from stresses-what you eat, drink and think.
They destroy cells!

So it is essential to make sure that you have enough of the good guys to win this daily battle.

The products, Diphasic AM and Diphasic PM are the best combination that I know of, filled with the essential nutrients, supplying the anti-oxidants. The following pages gives more details on these.

And then minimizing stress. Ah, yes. What you eat, drink, and think...oh, and minimizing EMF's-electromagnetic frequency exposure. Not easy-hence, the need for extra help.

L-Carnitine (LC)

Carnitine (LC) is a quaternary ammonium compound biosynthesized from the amino acids lysine and methionine. Vitamin C is essential to the synthesis of LC. Carnitine is synthesized in the liver, and in the brain and kidney, from lysine and methionine, with catalysts vitamins 86, 83, C, and folate. In our natural diet, carnitine is found in meat and in milk products.

- Carnitine appears to improve insulin resistance. It decreases the spike in glucose concentration after glucose administration, and decreases the associated insulin secretion.
- Carnitine improves heart muscle exercise tolerance.
- Carnitine prevents cardiac arrythmias, including the occurrence of ventricular fibrillation in the early phases of ischemia.
- Carnitine prevents angina.
- Carnitine is a vase-dilator of coronary blood vessels (and lowers blood pressure).
- Carnitine (because of its role in su.pplying fatty acids to the heart muscle) is valuable in the prevention of chronic heart failure.
- Carnitine protects membrane structures.
- Carnitine increases lymphocyte proliferation following mitogenic stimulation, and increases white blood cell motility.
- Carnitine was shown to be beneficial in reducing chronic fatigue.
- Carnit!ne causes hypertrophy of Type I muscle fibers; it increases exercise tolerance.

Regarding its role as a protector against cardiovascular disease, it has been shown that carnitine decreases lipid peroxides in the heart.

Acetyl- L- Carnitine (ALC)

- Long-term administration of ALC to aged rats restores a synaptic pattern in the hippocampus comparable to that of young rats.

Chronic Fatigue Syndrome (CFS) in the elderly: administering ALC reduced both physical and mental fatigue and improved the cognitive status as well as physical functions.

Propionyl-C-Carnitine (PLC)

- LC has a positive effect on bone mass. Administration of LC or PLC is capable of increasing serum osteocalcin that decreases with age, leading to osteoporosis.

Carnosine

Carnosine is an amino acid di-peptide (beta-alanyl-L-Histidine). It is partly hydrolyzed in the small intestine to the amino acids beta-alanine and histidine. Carnosine is found in its highest concentrations in the brain and in muscle tissue.

- powerful antioxidant - scavenges hydroxyl radicals and protects SOD against peroxidation
- protects and potentiates the immune system
- protects cellular proteins from aging
- protects against toxic carbonyl groups associated with aging
- strengthens the heart and improves the circulation
- Carnosine is not only a powerful antioxidant, one study showed it is the only antioxidant to significantly protect cellular chromosomes from oxidative damage.
- Carnosine quenches the most destructive protein oxidizing agent, the hydroxyl radical.
- Glycated proteins produce 50 times more free radicals than non-glycated proteins. Carnosine is the most effective anti-glycating agent ever found.
- Carnosine's anti-glycation benefits are particularly important for diabetic patients, since most complications of diabetes involve the formation of advanced glycation end products.
- Carnosine is an effective antioxidant in defense against malondialdehyde (MDA). MDA causes protein cross-linking and formation of advanced glycation end products. Carnosine has been shown to prevent MDA from inducing protein cross-linking.
- The reason such a high carnosine concentration is found in the brain is because there, carnosine protects against cross-linking, glycation, excito- toxic brain cell destruction, and oxidative damage.
- Carnosine can rescue neurons from zinc and copper mediated neurotoxicity, suggesting that one function of carnosine may be as an endogenous neuroprotective agent.
- In animal studies, it has been shown that carnosine protects the brain in simulated ischemic stroke.
- Carnosine not only has anti-ischemic effects in the brain, but in the

heart as well.

- Carnosine has been shown to increase the strength of heart contractility by enhancing calcium response in heart cells.
- The copper-zinc compounds that contribute to the amyloid-beta plaque formation in Alzheimer's disease are inhibited by carnosine.
- Not only does carnosine protect against the formation of amyloid-beta senile plaques, but also protects the cells that line the brain blood vessels from damage by those plaques that do form.
- Carnosine protects the brain against both lipid peroxidation and against damage from excess alcohol.
- Carnosine has been shown to rejuvenate cells approaching senescence by extending the life over which those cells will continue to divide with the frequency typical of youth. In tissue cultures supplemented with carnosine, cells retain a youthful appearance and have an extended cellular life span. This ability for carnosine to increase cellular life span holds true even for old cells. One study showed a 67% increase in cellular life span with carnosine supplementation.
- Extending the study of carnosine's life span increasing property from tissue cultures into living organisms, studies were conducted showing that mice supplemented with carnosine lived an average of 20% longer than un- supplemented mice, and were twice as likely to reach old age in a healthy state.
- In humans, carnosine levels decline with age. Muscle carnosine concentration decreases 63% from age 1O to age 70.
- Carnosine not only serves as an antioxidant in muscle, but also as a pH buffer. It protects muscle cell membranes from oxidation under the acidic conditions of muscular exercise.
- Carnosine has been shown to dramatically improve exercise recovery (but does not increase performance, which means that it is not an "ergogenic aid," but rather facilitates the anabolic response to exercise).
- Carnosine has been shown to quickly restore muscle contraction capability after fatigue.

- Carnosine has a rejuvenating effect on connective tissue cells, and has been shown to benefit wound healing.
- Because of its ability to prevent cross-linking, carnosine has been shown to be effective in the treatment of senile cataracts, and in the prevention of cataracts.
- Carnosine has immune potentiating properties. It protects the immune system from immune suppression by hydrocortisone, by anti-tumor drugs, and many other immunosuppressive drugs.
- Carnosine inhibits histamine-induced suppression of lymphocyte proliferation. Thus, it is classified among H-2 histamine blockers, which explains why it is a beneficial treatment for allergies.

Chondroitin Sulfate

Chondroitin sulfate is the patriarch of the family of compounds that once were known as muco-polysaccharides, and which have more recently been re-named glycosaminoglycans, or GAGS. It is even more absurd with chondroitin sulfate than with glucosamine that the health food industry has pigeon-holed it as an arthritis remedy when that is only one of many (and not nearly the most important) beneficial effect it offers.

Chondroitin sulfate (CS) has many important functions in human physiology. Many of those functions relate to the structure and function of joints and other connective tissues. The importance of CS to connective tissues will be discussed in future Letters.

For now, let us concentrate on the most important function of CS – <u>the protection of the cardiovascular system</u>.

- The protection of the cardiovascular system.
- CS helps maintain arterial elasticity. (Remember, arteries are largely connective tissue.)
- CS retards the arteriosclerotic and aging processes within the arterial wall.
- CS also possesses lipid clearing activity. It lowers cholesterol and triglycerides, and it normalizes the ratio between HDL, LDL, and VLDL. Most importantly, CS clears lipids not just in the serum but from within the cells as well.

- CS supplementation has also been shown to significantly reduce angina in patients with cardiovascular disease. ..
- CS has been found to protect against thrombus formation.
- The most striking statistic regarding CS supplementation shows that in cardiovascular disease patients treated with CS, the likelihood of having a myocardial infarct, suffering coronary insufficiency or myocardial ischemia, or developing congestive heart failure, is only 1/6 of that reported for control patients who receive no CS supplementation.

- In addition to the striking reduction in mortality and morbidity in patients with ischemic coronary heart disease, the Institute for Arteriosclerosis Research at Loma Lind University School of Medicine reports experimental studies showing that...

CS CAN PREVENT, AS WELL AS ACCELERATE REGRESSION AND HEALING OF, CORONARY AND AORTIC ATHEROSCLEROSIS.

- CS not only clears lipids at the cellular level, but also stimulates cellular metabolism, increases turnover of fatty acids at the cellular level, and increases RNA and DNA synthesis in tissue cultures.

The cholesterol and triglyceride lowering capability of CS is also tied in with its effect on maintaining the normal body colloid. It is only when damage to the arterial intima creates a loss of tissue membrane polarity that cholesterol, calcium, and the other components of atherosclerotic plaguing are pulled into the lesion. CS prevents the accumulation of lipids in atherosclerotic lesions. But it does even more than that - it can actually reverse these lesions. This gets into what we call "oral chelation."

Oral chelation is the process of not just preventing arterial plaguing but actually breaking up and eliminating those plaques. Chondroitin sulfate achieves this. It acts in much the same way as the chelating agent EDTA. It goes into the atherosclerotic lesion and breaks it apart by grabbing the calcium contained in the plaque.

CS is the only substance that is calcium-specific in its chelating ability. In other words, it has the ability to go in and grab calcium, rip it out of a plaque, carry it to the kidneys and eliminate it – and not grab any other important mineral nutrients at the same time.

This effect of CS to maintain normal biological membrane polarity and thus normal permeability, shows up in kidney function as well. One impact of CS on the kidneys is to help the kidneys eliminate excess sodium build up. The second interesting fact about CS as it relates to kidney function is that CS is very effective at blocking the growth of kidney stones.

In its now popular role as an arthritis cure, chondroitin sulfate does decrease the pain and inflammation of arthritis. Furthermore, this is not simply a matter of symptomatic relief, as the CS actually halts the progression of the arthritic degeneration, and actually reverses it in most cases. One important aspect of the CS impact in joints suffering from osteoarthritis is that it increases the synovial hyaluronic acid of these joints.

Chondroitin sulfate has been shown in studies on mice to be effective in decreasing motor neuron disease.

Chondroitin sulfate has been shown to regulate mammary gland development. CS is active in controlling the proliferation, the differentiation, and the involution of breast tissue. It may be that it has its effects on breast tissue by potentiating the benefits of progesterone, or by opposing the damaging effects of estrogen.

CS is also an important activator of the immune system. In particular, macrophage function is enhanced by CS supplementation. Coenzyme Q10 (Co-Q10)

Coenzyme Q-10 (CoQ) is an essential component of the mitochondrial electron transport chain, which is the fundamental unit for energy production in our cells. In addition to being essential for generating energy, CoQ is an important antioxidant. The heart, with its high energy requirements, is especially rich in CoQ.

- CoQ deficiency is a significant part of myocardial failure.
- CoQ improves cardiac response to exercise.
- End stage heart failure patients who supplement with CoQ have a 40o/o survival rate compared to a 10% survival rate without CoQ.
- CoQ lowers blood pressure.
- CoQ reduces angina.
- Co Q prevents arrhythmias.

There seems to be no end to the flood of research highlighting the cardiovascular protective effects of Co Q-10. This nutrient is turning out to be one of the most valuable clinical tools you have for patients with a diversity of health problems, but particularly for those at risk for cardiovascular disease (CVD).

A study published in Clinical Investigations, 1993; 71/8 Supplement: 8140-4 entitled, "Isolated Diastolic Dysfunction of the Myocardium, and its Response to Co Q- 10 Treatment," studied patients in the early stages of congestive heart failure and found that Coenzyme Q-10 resulted in:

- a decrease in high blood pressure in 80% of hypertensives
- an improvement in diastolic function in all patients based on endocardiograms
- a reduction in myocardial thickness in 53% of hypertensives and in 36% of those with combined mitral valve and fatigue syndrome.

A study published in Clinical Investigations, 1993; 71(8 Supplement) S116-23 entitled, "Perspectives on Therapy of Cardiovascular Diseases with Co Q-10," showed that Co Q-10 myocardial tissue levels were significantly lower in patients with more advanced heart failure compared with those in the milder stages of heart failure.

Administering Co Q-10 to these patients showed significant improvement in patients' capacity for physical activity and overall quality of life. The benefits were found to be far greater than those from treatment with traditional methods such as angiotensin converting enzyme inhibitors.

A study published in The International Journal of Tissue Reactions, 1990; 12(3):163-8 entitled, "Pronounced Increase of Survival of Patients with Cardiomyopathy when treated with Co Q-10," showed that patients with all classes of cardiomyopathy accompanied by low ejection fractions experienced dramatic improvement in ejection fractions and pronounced increase in survival, which was attributed to Co Q-10's bioenergetic activity in regard to myocardial function.

What family of drugs generates more $$$$$ for the pharmaceutical industry than any other? Anti depressants? Anti inflammatories? Antibiotics? No, no, no -- by far it is the Statin drugs to lower cholesterol --- generating nearly 20 billion dollars in annual revenues for the drug companies. If this family of drugs were to cause serious side effects, how much would the drug companies invest in terms of financial incentives and political pressure to keep the truth buried?

The truth, aggressively suppressed, is that the Statin drugs have irreversible and often fatal consequences, including cardiomyopathy, congestive heart failure, and rhabdomyolysis. Researchers have now discovered that the reason for

the deadly side effects of cholesterol lowering drugs is that they deplete the body of Coenzyme Q-10. To their credit, the Canadian government is way ahead of the US government in its resistance to drug company lobbyist pressure. In Canada, Statin drugs are required to carry a label with an explicit precautionary warning that the drug can cause Co Q-10 depletion and lead to impaired cardiac functioning in patients with congestive heart failure. Consider these studies:

Proc Natl Acad Sci USA. 190 Nov;87(22):8931-4. Lovastatin decreases Coenzyme Q-10 levels in humans. Folkers, et al.

Mol Aspects Med. 1997;18(suppl):s137-s144. Dose-related decrease of serum Coenzyme Q-10 during treatment with HMG CoA-reductase inhibitors. Mortenson et al.

Biofactors. 2003;18(1-4):113-24. Statins lower plasma and lymphocyte ubiquinol/ubiquinone. Passi, et al.

Biofactors. 2003;18(1-4):101-11.The clinical use of HMG CoA- reductase inhibitors and the associated depletion of Coenzyme Q-10. Langsjoen, et al.

In this last study the head researcher says, "The depletion of the essential nutrient Co Q-10 by the increasingly popular cholesterol-1owering drugs, HMG CoA-reductase inhibitors (Statins), has grown from a level of concern to one of alarm. With ever higher Statin potencies and doses, and with a steadily shrinking target LDL cholesterol, the prevalence and severity of Co Q-10 deficiency are increasing noticeably. We are currently in the midst of a congestive heart failure epidemic in the United States --- as physicians, it is our duty to be absolutely certain that we are not inadvertently doing harm to our patients by creating a widespread deficiency of a nutrient critically important to heart function."

Imagine --- a drug given to millions upon millions of patients with cardiovascular disease that actually causes cardiovascular disease. Imagine that this drug also causes cognitive dysfunction, memory loss, and in many cases severe muscle pain.

As an antioxidant, Co Q-10 protects against the oxidative free radical damage that often triggers a cancer. Then, Co Q-10 inhibits the aberrant oxidative metabolism of existing cancers by promoting normal mitochondrial electron transport. One interesting study showed the benefits Co Q-1O in prostate cancer. Co Q-10 supplementation significantly lowered cell growth of the PC3 cancer line without affecting non-malignant cells.

Researchers have demonstrated that Co Q-10 is present in seminal fluid, and is directly correlated to sperm motility in infertile men.

Males are not the only ones to require Co Q-10 for normal reproductive function.

Pre-eclampsia, a life-threatening disorder affecting about 7% of late-stage pregnancies, is associated with extreme edema, hypertension, and proteinuria. Serum levels of Co Q- 1O are severely depressed in pre-eclampsia patients. Free Radie Biol Med. 2003 Dec 1:35(11):1453-6. Pre-eclampsia is associated with a decrease in plasma Coenzyme Q-10 levels. Teran, et al.

These researchers showed that Coenzyme Q-10 levels should rise approximately 20% in pregnant women relative to non-pregnant women who have normal blood pressure. In women with pre-eclampsia, however, Co Q-10 drops nearly 20% to a level more than 35% lower than found in healthy pregnant women.

The eye is one of the more metabolically active tissues in the body. As such, it is subject to oxidative free radical damage. One manifestation of PUFA's destructive influence is macular degeneration. A recent research study reports that Co Q-10 may improve retinal function in patients with age-related macular degeneration by improving the performance of mitochondria in the retinal pigment epithelium.

- Iron plays an important role in Parkinson's Disease (PD) pathology. It has been demonstrated that CoQ10 has a neuroprotective role in iron-induced apoptosis in cultured human dopaminergic neurons. Iron-induced mitochondrial damage and apoptosis are characterized by ROS production, increased metallothionein and glutathione synthesis, caspase-3 activation, NF-kappa B induction. Higher concentrations of iron sulfate in mitochondria- induced CoQ1O depletion, plasma membrane perforations, mitochondrial damage, and nuclear DNA condensation and fragmentation. Iron sulfate induced deleterious changes were attenuated by pretreatment with CoQ10 and by deferoxamine, a potent iron chelator. MPTP induces striatal release of free iron and increases the expression of NF-kappa B, whereas ferritin and melanin synthesis are significantly reduced in the substantia nigra of PD model mice compared with controls. CoQ10 treatment inhibits MPTP- induced NF-kappa B induction in all genotypes. These data suggest that glutathione and metallothione in synthesis may be induced as an attempt to combat iron-induced oxidative stress, whereas exogenous administration of CoQ10 or metallothione in induction might provide CoQ10-mediated neuroprotection in PD.
- Oral CoQ1O significantly decreased elevated lactate levels in patients with

Huntington's Disease. CoQ10 is a powerful antioxidant that buffers the potential adverse consequences of free radicals produced during oxidative phosphorylation in the inner mitochondrial membrane. Oxidative stress, resulting in glutathione loss and oxidative DNA and protein damage has been implicated in many neurodegenerative disorders including Alzheimer's Disease, PD, and HD.

Alpha Lipoic Acid (ALA)

- Alpha lipoic acid is a di-thiol antioxidant. It is reduced to the thiol form intracellularly. The di-thiol (two sulfur) character of its molecular structure is what gives it its anti-anabolic, anti-reductive stress activity in your Diphasic A.M. because of its metabolically active sulfur, it has antioxidant activity as part of the glutathione system of antioxidants, as well as in the glutathione derivatives cysteine and n-acetyl-cysteine.
- Lipoic acid not only restores glutathione and glutathione peroxidase as part of your body's anti-anabolic antioxidant defense system, but is also an important part of your anti-catabolic anti-oxidant system. This anti-oxidant function is shown in the research as an amazing effect at decreasing malondialdehyde, one of the principal end products of age-related lipid peroxidation. Lipoic acid also potentiates the antioxidant enzyme systems super oxide dismutase and catalase, and glutathione reductase.
- It particularly decreases iron-dependent lipid peroxidation.
- Lipoic acid has anti-aging effects by attenuating the decrease in both enzymatic (e.g., SOD) and non-enzymatic (e.g., vitamin E) antioxidant levels with age.
- Lipoic acid has been shown to decrease oxidative stress associated with lead poisoning.
- Oxidation of hemoglobin is prevented by both lipoic acid and vitamin E (but not by vitamin C).
- Some of the most highly toxic products of lipid peroxidation inhibit mitochondrial respiration by inhibiting alpha ketoglutarate dehydrogenase and pyruvate dehydrogenase.

This toxic inhibition is associated with decreased enzyme activity, which is induced by insufficient availability of lipoic acid sulfhydryl groups.

- Lipoic acid is an anti-oxidant in both fat and water soluble media, and is active both intra- and extra-cellularly.
- Lipoic acid increases intra cellular Co-enzyme Q-10, and regenerates both vitamin C and vitamin E intracellularly.
- Lipoic acid is a hydroxyl radical quencher (due to the di-sulfate bond in the di-thiol ring).
- Lipoic acid (and sodium iodide) increases cyclo-oxygenase, which increases the oxidation of arachidonic acid, and increases the reduction of Prostaglandin PGG2 to Prostaglandin PGH2, which decreases inflammation of all types.
- Lipoic acid prevents oxidative stress in the liver, the heart, and in the gastrocnemius muscle in response to exercise.
- Lipoic acid increases energy availability to the brain and to muscles during exercise.
- Lipoic acid has been shown to improve cardiac autonomic neuropathy, which is diagnosed by reduced heart rate variability (a Sympathetic Imbalance indicator) at rest. (Tie this in with your NUTRI-SPEC clinostatic pulse response.)
- Lipoic acid, prevents atherosclerosis, and particularly lowers triglycerides.
- Endothelial migration of monocytes is one of the first steps in atherosclerosis, along with the action of vascular adhesion molecules. These two fundamentals of atherosclerosis are stimulated by glycation end products, and are reversed by lipoic acid.
- Lipoic acid given to patients with coronary artery disease and essential hypertension has been shown to have a favorable influence on the fatty acid content of the blood.
- Lipoic acid has been shown in clinical studies to decrease elevated triglycerides by as much as 45%. (Elevated triglycerides (and not elevated cholesterol) is one of the few true risk factors for heart attacks and strokes.)

- Lipoic acid is an alpha keto-acid dehydrogenation co-enzyme. It is thus the link between lipid an carbohydrate metabolism. Lipoic acid can also be considered the universal co-enzyme of alpha keto-acid oxidation.
- Upoic acid reverses the age-related decrease in hepatocyte glutathione and ascorbic acid.
- Lipoic acid has been shown to decrease cataracts.
- Lipoic acid has been shown to decrease the tendency to calcium oxalate kidney stones.
- Lipoic acid increases T-Cell function in cancer patients.
- Lipoic acid is an essential constituent of biological membranes. Another study shows that membrane fluidity and protein sulfhydryl reactivity of RBCs is decreased in diabetes, and is increased by lipoic acid supplementation.
- In its antioxidant role, lipoic acid has been shown to decrease diabetic neuropathy.
- A note on diabetic neuropathy: Studies have shown that in diabetic neuropathy the nerve is ischemic and hypoxic, with increased dependence on anaerobic metabolism. Lipoic acid increases glucose uptake and efficient oxidative metabolism and thus benefits the diabetic neuropathy.
- Type II diabetics have increased fasting lactate and pyruvate concentrations in their blood. Furthermore, the increased lactate and pyruvate concentrations double after glucose loading in obese patients, but not in lean patients. Lipoic acid was shown to decrease excessive lactate and pyruvate levels in the serum of Type II diabetics. (These are generally your Ketogenic Imbalance patients.)
- Lipoic acid has been shown to decrease age-related memory loss.
- Parkinson's Disease, ALS, Huntington's Disease, Friedreich's ataxia, and mitochondrial cytopathies and other neuromuscular diseases share to some extent the final common pathway leading to cell death through either necrosis or apoptosis. Compounds such as creatine monohydrate, and CoQ10 offer substantial neuroprotection against ischemia, trauma, oxidative damage, and neurotoxins. Miscellaneous agents, including alpha lipoic acid, beta-hydroxy beta-methyl butyrate, riboflavin, and nicotinamide, have also been shown to improve various metabolic

parameters in brain and/or muscle.

- Lipoic acid and acetyl-1-carnitine are 2 mitochondrial antioxidants studied in a chronic rotenone-induced cellular model of PD. Both nutrients were found to be protective against mitochondrial dysfunction, oxidative damage, and accumulation of alpha-synuclein and ubiquitin. Most notably, it was found that combined lipoic acid and acetyl-1-carnitine worked at 100 to 1.000 times fold lower concentrations than they did individually. Pretreatment with combined lipoic acid and acetyl-1-carnitine increased mitochondrial biogenesis and decreased production of ROS through the upregulation of peroxisome proliferator-activated receptor-gamma coactivator 1alpha as a possible underlying mechanism. This study provided important evidence that combining mitochondrial antioxidants at optimal doses might be an effective and safe prevention strategy for PD.

TOCOPHEROLS

VITAMIN E (ALPHA TOCOPHEROL) IS NOT REALLY SUCH A GREAT ANTIOXIDANT.

Its antioxidant activity is positively dwarfed by the antioxidant activity of gamma tocopherol particularly, and the other tocopherols as well. The mixed tocopherols represent the complete vitamin E family – alpha, beta, gamma, and delta tocopherols.

When you see Vitamin E on the label of a nutrition supplement, it is almost always one of the various forms of alpha tocopherol. Whether the label says alpha tocopherol,

alpha tocopheryl succinate, alpha tocopheryl acetate, vitamin E, natural vitamin E, or whatever, it is certain to be an alpha derivative.

It is interesting to note that alpha tocopherol can actually displace gamma tocopherol in living tissues. So, now think about all the people that are taking 400, 800, or more international units of vitamin E each day in the belief that they are getting antioxidant protection, when actually they are destroying the most potent (and all too rare) antioxidant of all, gamma tocopherol.

The truth about vitamin E is that its antioxidant activity can be every bit as powerful a protector of the cardiovascular system as the medical/pharmaceutical establishment feared. That is why they had to attack it so aggressively. Do even the most superficial Medline search of vitamin E, and with just a few clicks of the mouse you will find countless dozens of studies showing that even a

fragmented antioxidant such as alpha tocopheryl acetate will:

- decrease lipid peroxidation throughout the body, and particularly in the cardiovascular system
- decrease platelet aggregation ("thins the blood")
- decrease inflammation in the vasculature (---and, decreases inflammation systemically)
- lowers C-reactive protein
- decrease oxidation of LDL cholesterol
- prevent and relieve angina
- decrease incidence of strokes
- improve myocardial recovery from exercise

Vitamin E increases prothrombin time (i.e., potentiates the effect of Warfarin).