

Focus

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The 21st Century Vitamin E?

• Delta-Tocotrienol Proves Surprisingly Potent in Fighting Cholesterol, Cardiovascular Disease and Cancer

“The tocotrienol subfamily of natural vitamin E possesses powerful neuroprotective, anticancer, and cholesterol-lowering properties.”

–Ohio State Medical University, 2007.

Turn to page 2 for more on Delta Tocotrienol.

• Interview with biochemist Barrie Tan, Ph.D.

“Tocotrienols can be really optimal support compounds for cardiovascular disease, both for protection and treatment.”

Turn to page 4 for more with biochemist Barrie Tan, Ph.D.

Mastic Gum: Its Applications Go Far Beyond the Treatment of Ulcers

• Interview with Leo Galland, M.D.

“Since there are virtually no side effects, I’ve begun to incorporate mastic gum in my protocols for any patient of mine with a GI complaint, from gastritis to ulcers to inflammatory bowel disease.”

Turn to page 8 for more on Mastic Gum: Its Applications Go Far Beyond the Treatment of Ulcers.

• From Folklore to Good Science: New Uses for Mastic Gum

New studies find two powerful mechanisms by which mastic gum works: by inhibiting bacterial growth and reducing inflammation, in conditions ranging from Crohn’s disease to dental caries.

Turn to page 10 for more on Mastic Gum: From Folklore to Good Science.

Glutathione: New Research Reveals New Benefits

The newest research continues to uncover the significance of glutathione in health, and offers promising routes to boosting glutathione through novel, targeted supplements.

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The 21st Century Vitamin E?

Delta-Tocotrienol Proves Surprisingly Potent in Fighting Cholesterol, Cardiovascular Disease and Cancer

Vitamin E—the golden capsule, our most potent fat-soluble antioxidant—has a secret. It turns out to be a many-faceted nutritional gem, with a highly specific form called delta-tocotrienol that offers profound cholesterol-lowering properties, potent cardiovascular benefits, and in laboratory studies, verifiable anticancer effects. In new research, delta-tocotrienol proved 40 to 60-fold more powerful as a free radical scavenger than alpha-tocopherol, the most commonly available Vitamin E in supplements.

Vitamin E in the form of delta-tocotrienol can lower cholesterol by an astonishing 15-22%, and markedly improve LDL/HDL ratios, while preserving and even increasing Coenzyme Q10. Unlike the “blockbuster” statin drugs, which deplete CoQ10 and lead to a host of uncomfortable side effects including muscle deterioration and pain, Vitamin E in the form of delta-tocotrienol is nontoxic, CoQ10 preserving, and serves as a powerful antioxidant that protects lipoproteins. It also has a markedly beneficial effect on cardiovascular function.

In fact, delta-tocotrienol may be one of the most significant, new heart-protective nutrients available today. This is important news, since more than 102 million Americans suffer from high cholesterol (over 200 mg/dL) and cardiovascular disease remains our country’s top killer.

In addition, there is an impressive array of laboratory studies demonstrating that delta-tocotrienol markedly inhibits Chlamydia infection (associated with heart disease); and, that both

delta- and gamma-tocotrienol are potentially potent anti-cancer nutrients.

Delta-tocotrienol is present at best in only trace amounts in Vitamin E supplements, while tocopherols dominate. Tocopherols are found in corn, soybean and olive oils—staples of the American diet. In contrast, oils like palm, annatto and rice are rich in the tocotrienols—and rarely used in America. And yet, according to the latest scientific research, the tocotrienols, especially delta, have disease-fighting properties that may herald a new golden age for the golden capsule. In brief:

- Tocotrienols are absorbed better

Vitamin E in the form of delta-tocotrienol can lower cholesterol by an astonishing 15-22%, and markedly improve LDL/HDL ratios, while preserving and even increasing Coenzyme Q10

than tocopherols, probably because of their unique shape and tail, which allows them to move throughout the entire cell.

- Tocotrienols are a far stronger class of antioxidants than tocopherols—they can be up to 40 to 60-fold more powerful as a free radical scavenger and up to 70-fold more bio-available. And delta-tocotrienol, in particular, is four times as powerful as the three other tocotrienols.

- Two of the tocotrienols, delta and gamma, can lower cholesterol by 15-22% and improve LDL/HDL ratios.

- Delta- and gamma-tocotrienols lower triglycerides.

- Delta-tocotrienol may be highly pro-

TECTIVE in atherosclerosis—more so than alpha-tocopherol. It can do a much better job at reducing the fatty streaks that form on artery walls (a first step in atherosclerosis), it helps stop monocytes from sticking to blood vessel lining, and inhibits “platelet aggregation” (sticky platelets that clump together).

- Delta-tocotrienol may fight Chlamydia infection. Chlamydia is implicated in heart disease, and delta-tocotrienol stops Chlamydia from entering cells via “lipid rafts” (yes, literally, little boats of fat).

- Alpha-tocopherol, the “famous” vitamin E, can actually compete with all the other vitamin Es (tocopherols and tocotrienols) and block their health-promoting effects. Worse, alpha-tocopherol accelerates the breakdown of tocotrienols and tocopherols. In fact, alpha-tocopherol can increase cholesterol if given in high doses.

- Delta-tocotrienol functions as a highly protective antioxidant for neurons.

- Laboratory and animal studies on tocotrienols offer promising early results in the treatment of cancer. For instance, delta-tocotrienol can inhibit lung, liver, breast, pancreas, skin, and prostate cancer in laboratory experiments on mice and on human cancer cells.

The New Face of Vitamin E

We often think of Vitamin E as alpha-tocopherol, which is by far the most studied form (just as we often focus on beta-carotene when there are over six hundred identifiable, natural carotenoids). In truth, alpha-tocopherol’s main function may be simply as an

antioxidant, as the title of a review of alpha-tocopherol by specialist Maret G. Traber of Oregon State University puts it, “Vitamin E, antioxidant and nothing more.” Traber, who identified the alpha-tocopherol transport protein, writes, “Virtually all of the variation and scope of Vitamin E’s biological activity can be seen and understood in the light of protection of polyunsaturated fatty acids.” In that antioxidant capacity, of course, alpha-tocopherol is important, because it protects the lipid cell membrane. It is the major lipid-soluble antioxidant in the body.

But Vitamin E is far more than alpha-tocopherol. Vitamin E is really a generic term for eight common but different vitamin variations—four tocopherols and four tocotrienols (alpha, beta, gamma and delta). In this special report, we introduce new research demonstrating the unique benefits of delta-tocotrienol, an unusually potent antioxidant. As researchers at the Laboratory of Molecular Medicine at Ohio State Medical University put it in a 2007 research article: “The abundance of alpha-tocopherol in the human body and the comparable efficiency of all vitamin E molecules as antioxidants led biologists to neglect the

non-tocopherol vitamin E molecules as topics for basic and clinical research. Recent developments warrant a serious reconsideration of this conventional wisdom. The tocotrienol subfamily of natural vitamin E possesses powerful neuroprotective, anticancer, and cholesterol-lowering properties that are often not exhibited by tocopherols... A rapidly expanding body of evidence supports that members of the vitamin E family are functionally unique.”

Delta-tocotrienol proved 40 to 60-fold more powerful as a free radical scavenger than alpha-tocopherol

One reason the benefits of delta-tocotrienol have remained a bit of a secret is that they were expensive to source. That is no longer true. Biochemist Barrie Tan, Ph.D., formerly of the University of Massachusetts, and a world expert on tocotrienols, recently discovered that South American annatto is abundant in easily sourced tocotrienol that is already tocopherol-free. (See interview p. 4)

“Carotene is my love but tocotrienol is my fate,” confesses Dr. Tan. “I went to Malaysia and discovered that natural palm oil is a rich orange color. That orange is carotene—and the signature

is almost analogous to that of a carrot! But lo and behold, when I extracted the carotene, I found that the fraction of palm oil without the color had potent antioxidant properties. It looked like Vitamin E, and it turned out to be tocotrienol. Years later I stumbled onto annatto, a South American and true Amazon Rainforest plant with a red pod rich in carotenoids. Once again it was the beautiful carotene that attracted me, and once again when I tried to

find out what was protecting that carotene from decomposing, it turned out to be tocotrienols, and nearly exclusively delta-tocotrienol. I became very

interested in this potent antioxidant. What’s even better is, in annatto we have discovered a natural source of tocotrienol that is tocopherol-free and needs no chemical manipulation.”

The timing was perfect, as tocotrienols have been gaining increasing recognition for their cardiovascular benefits in general, and their unique cholesterol-lowering properties in particular.

Powerful And Safe Cholesterol Buster

Delta-tocotrienol has proved effective at lowering cholesterol in laboratory,

Another Vitamin E Gem: Gamma-Tocopherol

Each fraction of vitamin E has its own power, but recent research shows that gamma-tocopherol shines as a scavenger of one of our body’s most harmful free radicals, peroxynitrite. This molecule is formed when white blood cells (such as leukocytes, monocytes and macrophages) are activated to fight infection. Nitric oxide reacts with superoxide, forming peroxynitrite, and can cause significant cellular damage and chronic inflammation. Martin Pall, Ph.D., School of Molecular Biosciences, Washington State University, has identified the NO/ONOO- cycle - the stress-induced increase in the levels of nitric oxide and its oxidant product peroxynitrite, and the biochemical vicious cycle that keeps these levels chronically elevated - as a key mechanism underlying oxidative stress. Gamma-tocopherol is one of a number of key nutrients that together may assist in down-regulating this NO/ONOO- cycle mechanism.

Gamma-tocopherol is also a potent antioxidant, and may have anticoagulant, immunomodulating, and cardiovascular effects. It decreases oxidation of cholesterol, and increases activity of the free-radical scavenger superoxide dismutase. Gamma-tocopherol also protects polyunsaturated fatty acids (PUFAs) in cell membranes and in cholesterol.

Low levels of gamma-tocopherol may be linked to coronary artery disease, according to a recent study. In laboratory studies of cells, gamma-tocopherol seemed to inhibit the activation of one of the body’s most powerful inflammatory molecules—NF-Kappa B. And, in a study of human prostate cancer cells in the laboratory, gamma-tocopherol was a more powerful inhibitor of cancer than alpha-tocopherol.

Though about 70% of Vitamin E in our diet is in the form of gamma-tocopherol, it is much less present in the blood than alpha-tocopherol, and seems to be more rapidly taken up and turned over in tissues (as is delta-tocotrienol). Gamma-tocopherol is found in seeds and nuts, including soybeans, corn and walnuts.

animal and human studies. *In vitro*, in liver cells, delta-tocotrienol showed a 30-fold greater inhibition of cholesterol than alpha-tocotrienol. When animals' diets were supplemented with delta- and gamma-tocotrienol, their cholesterol decreased by a total of 32%, and their LDL cholesterol decreased 66%. The ratio of HDL to LDL improved by up to 150%.

In the first human study by researchers at the University of Wisconsin, pub-

lished in 1992 and mentioned above, four weeks of supplementation with delta- and gamma-tocotrienol at 100 milligrams a day reduced cholesterol by 15-22% and decreased LDL cholesterol by 10-20%. According to Dr. Tan, there was some hope at that time among researchers that tocotrienol might be a drug candidate for cholesterol reduction. Instead, today, we have the statins, all of which reliably lower cholesterol, but also produce myopathy and raise liver enzymes, among

other side effects. Delta-tocotrienol has no such side effects.

In open trials in humans, fasting blood lipids were measured before and two months after daily supplementation with tocotrienols from annatto (67 milligrams of delta-tocotrienol and about 8 milligrams of gamma-tocotrienol). In both studies, total cholesterol levels dropped 13%, LDL dropped between 9-15%, and HDL increased by 4-7%. The LDL/HDL ratio improved by up to 21%.

“Tocotrienol Is My Fate”

An Interview with Barrie Tan, Ph.D.

Introduction: Biochemist Barrie Tan, Ph.D., has published over sixty papers in his field. He was a researcher and professor at the University of Massachusetts for twelve years, and first began working with tocotrienol in 1984. He isolated it from palm oil in 1988, and from rice in 1994. Most recently, he isolated it from annatto in 2002.

Focus: *Why did you start working with tocotrienol?*

Tan: Tocotrienol is a much stronger antioxidant than tocopherol, and delta-tocotrienol is the strongest of all, and is able to reduce cholesterol. Tocotrienols can be really optimal support compounds for cardiovascular disease, both for protection and treatment.

Focus: *Can you explain why the effective doses of tocotrienol are relatively low?*

Tan: Both cell-line and animal studies suggest that about 100-200 milligrams works well. In cancer, studies thus far suggest that you may need 200-500 milligrams, but those studies are still in their initial stages. Also, palm oil extract contains 75% tocotrienol and 25% tocopherol. Tocopherol competes with tocotrienol. Annatto tocotrienol is pure, so when we studied it we were confident that we would need less and still be just as effective. One study on arteriosclerosis had used 25, 50, 100 and 200 milligrams per day, and found that 100 milligrams was highly effective for lowering cholesterol, while 200 milligrams was not any more effective.

Focus: *What drew you to tocotrienol research in the first place?*

Tan: Tocotrienol must be my fate. I first encountered it in Malaysia in palm oil. I went to Malaysia to visit a friend and saw palm oil the color of squeezed carrot juice, from the natural carotene. I thought it was terribly wasteful to bleach out this healthy carotene and all I wanted to do at first was extract that beautiful color. I wasn't thinking about tocotrienol at the time. Tocotrienol is the colorless, potent antioxidant that protects the carotene in palm. I would call carotene my unrequited love, it captured my imagination from the start. But tocotrienol is my fate.

Focus: *What is special about annatto?*

Tan: Annatto is a pod with a red color on the head. Incas used annatto to make red markings on their body. The red is a carotenoid. I found that fascinating because annatto is phototropic, it follows the sun like a sunflower, and carotenoids are usually unstable. Something was seriously protecting the carotenoids from decomposing, and it turned out once again to be tocotrienol. But in this case it was of a natural origin, tocopherol-free, unlike palm oil. When I analyzed it, it had one super-dominant peak that was delta-tocotrienol (90%), and a smaller peak that was gamma-tocotrienol (10%). I'd never seen a natural source with such a huge amount of this active ingredient, delta-tocotrienol. And we are able to extract it without solvents, through a simple, physical method.

Focus: *What are you working on now with tocotrienol?*

Tan: We are doing a placebo, double blind study on men with metastatic prostate cancer. Cancer cells use 10-100 times more cholesterol, and for whatever reason, 10 times more tocotrienol as well. And in recruiting tocotrienol, the cancer cell stifles its synthesis of cholesterol and its ability to procreate. I was not confident at first that neoplastic cells could be changed back to normal cells with tocotrienols, but cell studies have shown that it can indeed reverse neoplasms. This is amazing. Medicine generally does not accept that yet and contends that you have to kill a cancer cell, not revert it to normal.

Can delta-tocotrienol be used on its own, or in conjunction with statins to potentiate their effect? Yes, according to molecular geneticists at Texas Southwestern Medical Center. Delta-tocotrienol is unique, compared to all other forms of vitamin E, in its ability to reduce cholesterol, according to their 2006 study. Thus it might “potentiate the therapeutic effectiveness of statins or, in some cases, provide an alternative therapy.” (In fact, as will be explained later in this article, other research suggests that statins and tocotrienol together could offer a novel approach to cancer.)

Finally, tocotrienol from annatto appears to preserve or increase Coenzyme Q10, which is depleted by statins. Patients given a daily dose of 75 milligrams of tocotrienols had their fasting CoQ10 measured after two months of supplementation. CoQ10 rose about 19% overall. “Further research will shed more light on the tocotrienol-CoQ10 connection,” says Tan.

Delta Benefits Diabetes and the Heart

Nearly 47 million American adults suffer from “metabolic syndrome”—a constellation of high triglycerides, high blood pressure, high blood sugar, and insulin resistance. These individuals

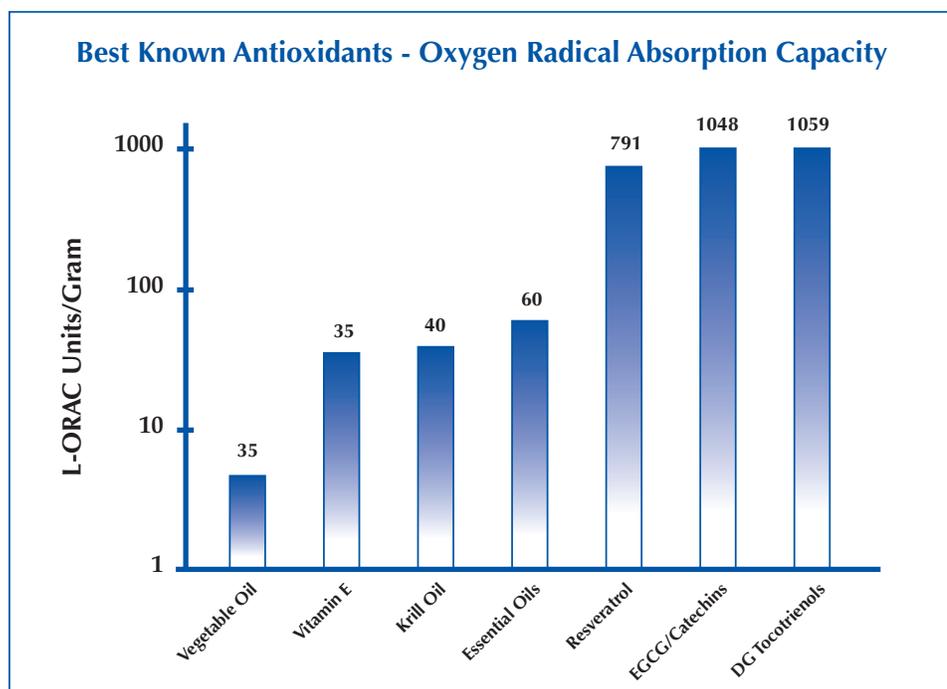
are at increased risk for heart disease and diabetes. Delta- and gamma-tocotrienol are able to help protect against metabolic syndrome in a number of remarkable ways. First, they increase the heart’s vascular integrity by preventing cross-linking of proteins and sugars; cross-linking stiffens tissues and decreases their function. Second, they decrease blood glucose and help control blood sugar. Third, they reduce triglycerides in the blood and the liver. In clinical studies with both Type 1 and Type 2 diabetics and metabolic-syndrome patients, even small amounts of tocotrienol (from rice bran) were beneficial in all three ways. In a 2005 study of Type 2 diabetics, (80% of deaths in type 2 diabetics are related to cardiovascular complication) delta-tocotrienol decreased serum total lipids by 23 percent, total cholesterol by 30 percent and LDL cholesterol by 42 percent (plummeting from 179 mg/dL to 104 mg/dL) within a mere 60 days.

Delta-tocotrienol helps stop the fatty streaks that form on artery walls—the first step in atherosclerosis. These fatty streaks form when white blood cells (monocytes) stick to an inflamed blood vessel lining. The monocytes are trying to fight inflammation, but by sticking to the artery walls they reduce blood flow. Tocotrienol stops the monocytes from being so sticky. Compared to oth-

er forms of vitamin E, delta-tocotrienol has the most profound inhibitory effect on monocyte “stickiness”, according to a 2005 study from the Kyoto Prefectural University of Medicine. Delta-tocotrienol accumulates in heart endothelial cells up to 95-fold more than alpha-tocopherol. The researchers suggest that tocotrienol’s power comes from its ability to accumulate inside the heart cells.

Delta-tocotrienol also helps prevent platelets from clumping, another step in atherosclerosis. Plaques form when platelets clump on the inner, inflamed blood vessel walls, form clots and then reduce blood flow. In a double-blind crossover study, delta-tocotrienol proved a potent inhibitor of such clumping—as much as 71% percent (compared to between 5 and 37 percent with other tocotrienols.) When mice were fed a diet designed to create atherosclerosis, those mice receiving tocotrienols had a 60% lower cholesterol level than a control group. Atherosclerotic lesion size was reduced ten-fold. Alpha-tocopherol, in contrast, had no such effect. In another mouse study, atherosclerotic lesions diminished by 92-98% with tocotrienol compared to alpha-tocopherol or control mice.

Tocotrienol even reverses carotid artery arteriosclerosis. In a four year clinical study, patients received tocotrienol supplements derived from palm (for the first three years) and rice bran (for the fourth year). The rice bran oil also contained other useful sterols. Arteriosclerosis stabilized or even regressed in an astonishing 88% in the tocotrienol/tocopherol group arteriosclerosis. Yet in a placebo group, 60% deteriorated, and only 8% improved. In that last year, the rice bran oil group also saw a 14% decrease in total cholesterol and a decrease in triglycerides as well. This drop in cholesterol and triglycerides was most likely due to other micronutrients such as rice plant sterols and oryzanol, the latter of which is only available from rice. As the researchers note, “Tocotrienols and tocopherols... represent a promising adjunct therapy in cardiovascular patients, especially the post stroke patient.”



A Highly Potent Bioavailable Antioxidant

Tocotrienol from annatto has a far greater L-ORAC (Oxygen radical absorption capacity) value than alpha-tocopherol, and is more potent than resveratrol and green tea catechins. It is up to 60-fold more potent and 70-fold more bioavailable than alpha-tocopherol. (see graph p. 5)

Tocotrienol has proven its antioxidant power in several studies. A 2006 study found that delta-tocotrienol had the most potent antioxidant properties of all the tocotrienols, and all the tocotrienols were more efficient antioxidants than tocopherols. Tocotrienol-rich Vitamin E protects against oxidation from ultraviolet radiation of the skin, and is more potent than alpha-tocopherol alone. In a 2003 study, tocotrienol was more potent than tocopherol at preventing cellular death from selenium deficiency. Another year-long study of 50 patients with high cholesterol found that tocotrienol (from rice bran) not only lowered total cholesterol levels but was able to reduce a sign of lipid peroxidation called TBARS (thiobarbituric acid-reactive substances). And a six month double-blind study of tocotrienol-rich Vitamin E found that 118 mg/day of tocotrienols (80 mg of delta- and gamma-tocotrienol) and 42 mg/day of alpha-tocopherol was able to prevent oxidative damage to DNA in older adults. In a study in rats, tocotrienols protected the brain against stroke- and glutamate-induced damage.

Delta Wags Its Handsome Tail

Tocopherols have a long tail and tocotrienols a short one. And therein lies a major difference between the two, with significant consequences for health. First, a shorter tail allows delta to move freely throughout the cell, rendering it more bioavailable to tissue—as much as 70-fold.

Second, of course, is delta-tocotrienol's impact on cholesterol. The pathway that leads to cholesterol also leads to Coenzyme Q10, heme (in blood), and

other very important molecules. Statin drugs inhibit an enzyme at the center of this pathway, HMG CoA reductase (HMG-CoAR). Statins inhibit HMG-CoAR directly by blocking it through "competitive inhibition." When they do this, they also block other products dependent on HMG-CoAR, and this includes Coenzyme Q10 and other important molecules. This can lead to statin side effects including myopathy, iron-unrelated anemia, fatigue, and heart problems.

Delta-tocotrienol, on the other hand, downregulates HMG-CoAR in an entirely different way that actually preserves CoQ10. It does so through its unique, short tail, which is "farnesylated" (contains a farnesyl group). In general, its small tail allows it to move easily through the cell and thus be far more bioavailable and potent than the tocopherols. This tail also helps regulate HMG-CoAR in a healthy fashion, by releasing farnesol (alcohol form of farnesyl). As farnesol increases, HMG-CoAR automatically decreases in re-

In a 2005 study of Type 2 diabetics, delta-tocotrienol decreased serum total lipids by 23 percent, total cholesterol by 30 percent and LDL cholesterol by 42 percent (plummeting from 179 mg/dL to 104 mg/dL) within a mere 60 days.

sponse in a feedback manner. This "dial down" feedback effect is gentler than a simple "shut down" effect, and does not deplete CoQ10.

This short farnesyl tail of tocotrienol causes it to be less anchored in the cell membrane, allowing it to move faster and cover a greater area over a shorter period of time. It also has a unique "head". Its head has fewer methyl groups attached, and so is smaller, and also has greater access to repair damaged membranes in tissue. All this may explain why delta-tocotrienol is a potent and highly bioavailable antioxidant.

Fighting Infection: A New Weapon Against Chlamydia

Intracellular Chlamydia bacteria have been implicated in atherosclerotic

plaques as well as Alzheimer's disease, asthma and respiratory tract infections. According to research by Dr. Elizabeth Stuart at the University of Massachusetts, tocotrienols inhibit Chlamydia. They do so by interfering with Chlamydia's use of cell membrane cholesterol, so that it can't infect cells as easily. Chlamydia may enter the cell through a "lipid raft", taking cholesterol and using it to survive and replicate. Tocotrienols from annatto, which contain high amounts of delta and gamma, may help prevent this. *In vitro*, when cells were incubated with delta-tocotrienol before being infected with Chlamydia, their infection levels were 50% less than untreated cells. There were also changes in the Chlamydia bacteria that rendered it less infectious overall in delta-tocotrienol cells.

What Lies Ahead: Tocotrienols and Cancer

Tocotrienols have repeatedly been shown to inhibit proliferation and induce cell death in cancer cells. They may be doing so in multiple ways: by inhibiting angiogenesis, through free-radical quenching, by interfering with tumor pathways (through HMG-CoAR suppression), and more. In laboratory experiments with breast cancer cell lines, tocotrienols inhibited growth irrespective of estrogen receptor status.

Delta- and gamma-tocotrienol were the most potent. In mouse studies from Kyoto Prefectural University of Medicine, delta-tocotrienol "resulted in significant suppression of liver and lung carcinogenesis." In cancer cell lines, delta-tocotrienol was found particularly effective against prostate cancer cells. According to the researchers at East Tennessee State University, delta-tocotrienol "may prove very useful as chemotherapeutic or chemopreventive agent for treating prostate cancer." In a nother study from Case Western Reserve University, tocotrienol-rich extracts of palm oil inhibited human prostate cancer cell growth and even encouraged cell death. Other *in vitro* laboratory studies have found that both delta- and gamma-tocotrienol inhibit pancreatic tumor growth and reduce cervical cancer growth.

In fact, a 2007 study from Texas Woman's University suggests that tocotrienols may potentiate the anticancer effect of statins. The problem until now has been that the high dose of statins required to inhibit cancer is toxic. The doses required lead to myalgia, muscle weakness, anorexia, lesions, nausea, diarrhea, fatigue and elevated creatine phosphokinase activity. However, using statins and tocotrienols "may lead to synergistic impact on tumor HMG-CoA reductase activity and consequently tumor growth," they speculate. This may allow medicine to use lower doses of statins that are better tolerated, and "offer a novel approach to cancer chemoprevention and/or therapy." Melanoma cell lines were markedly inhibited by a combination of lovastatin and tocotrienol 48 hours after treatment. Cells shrunk and died. In follow-up research, melanoma tumors were implanted into mice that were either supplemented with nothing (control), lovastatin alone, tocotrienol alone, or a blend of lovastatin and delta-tocotrienol for 22 days. Tumors in the "blend" group were smaller than the other groups. "Our study has shown for the first time that lovastatin and delta-tocotrienol, two suppressors of HMG-CoA reductase, synergistically suppress tumor growth *in vivo*," they conclude.

In fact, according to a 1990 review article in *Nature*, regulating the pathway involving HMG-CoA reductase may have major implications for both heart disease and cancer. This review was written by the Nobel laureates of 1985, Dr. Joseph Goldstein and Dr. Michael Brown, for their discovery of the LDL receptor. With all this good news, delta-tocotrienol may turn out to be our 21st century vitamin E.

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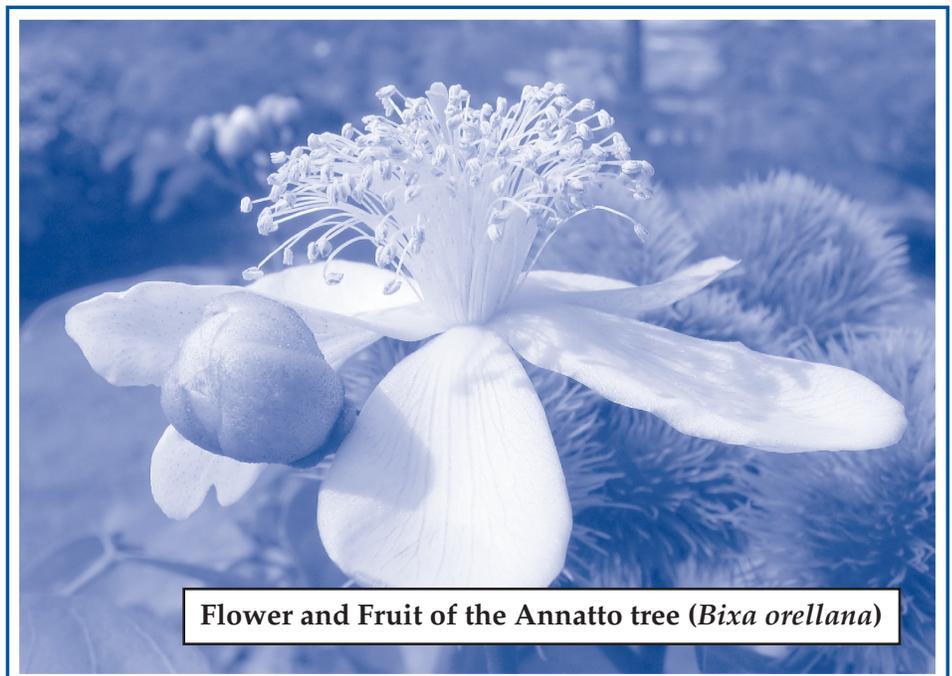
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Flower and Fruit of the Annatto tree (*Bixa orellana*)

Mastic Gum: Its applications Go Far Beyond the Treatment of Ulcers

Interview with Leo Galland, M.D.

Introduction: Leo Galland, M.D. is Director of the Foundation for Integrated Medicine, and author most recently of *The Fat Resistance Diet*. He has written many articles on the gastrointestinal ecosystem and novel approaches to gastrointestinal disorders. Here he discusses the latest research on mastic gum, and his protocol for the use of mastic gum not only in ulcers, but in other GI tract disorders.

Focus: You began using mastic gum in your practice years ago for stomach ulcers and found it highly effective. Can you briefly review mastic gum for us here?

Galland: Mastic gum has a long history of traditional use in the Mediterranean for digestive disturbances. Because of this a study was done that found it was able to kill *H. pylori*, the causative agent of stomach ulcers, *in vitro*. Before capsules were available, I actually found chunks of mastic gum in a middle eastern store in Brooklyn, and had my patients suck on it or chew it. Once capsules became available I started treating patients.

Focus: What is your preferred treatment regimen?

Galland: First, I test patients for *H.*

pylori antigen in the stool, and for *H. pylori* antibodies in the blood. These are both noninvasive and effective screening methods. If the patient is positive and symptomatic, with gastritis or ulcers, I prescribe 1000 milligrams of mastic twice a day for thirty days. Then I add in 10-14 days of a combination of antibiotics, along with acidophilus culture, and lactoferrin. I use 200 milligrams of lactoferrin twice a day, because it is immune stimulating and controlled studies have shown that it enhances the response to antibiotic therapy. After that, I continue with the probiotic alone.

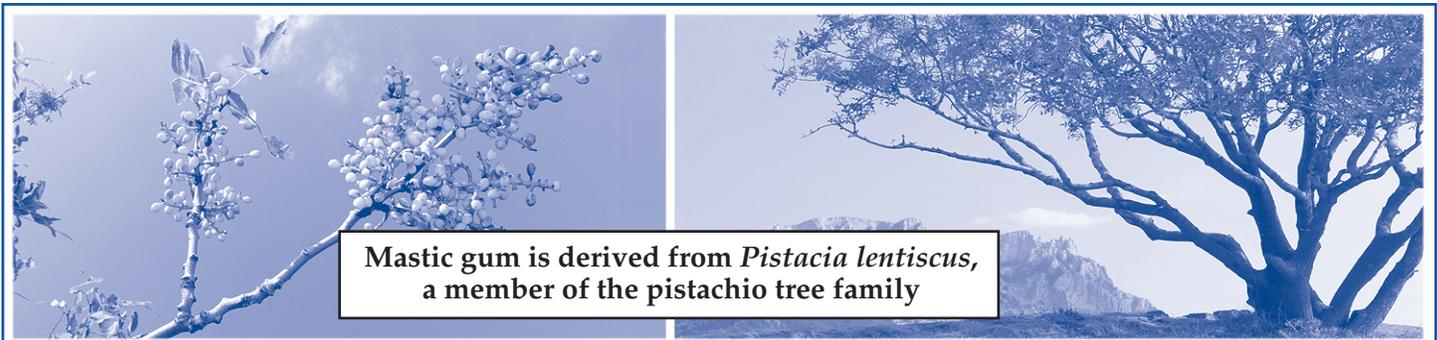
Focus: How many patients improve on this regimen?

Galland: At least 75% recover, become antigen- and antibody-negative and their symptoms clear up. I see only about a 10% relapse rate. But I've begun to be fascinated by other uses of mastic gum for gastrointestinal disorders. I had a patient with inflammatory bowel disease—with both gastritis and ulcerative colitis—who was *H. pylori* positive. I put her on mastic gum and her colitis cleared up. It was quite dramatic. She was on the mastic gum for six months total, and her symptoms have not returned. That was pretty amazing. I wasn't sure if this was a fluke,

but there have been recent studies with mastic gum that show it is quite helpful in a number of different gastrointestinal disorders. A 2007 study from the University of Athens showed that mastic actually altered the function of certain immune cells in patients with active Crohn's disease. Another 2007 study showed that in patients with active Crohn's disease, mastic gum downregulated inflammatory markers like NF-Kappa B and interleukin-6, as well as C-reactive protein. Their Crohn's symptoms also improved. Research last year even showed that mastic gum can inhibit salivary bacteria.

Focus: So what do you think is the future for mastic gum?

Galland: Since there are virtually no side effects, and it is used as a food throughout the Mediterranean, I've begun to incorporate mastic gum in my protocols for any patient of mine with a GI complaint, from gastritis to ulcers to inflammatory bowel disease. Wherever there is gut inflammation, mastic gum may be helpful. One study even showed that mastic has anti-proliferative activity in human colon cancer cells. I like mastic gum, and I use it a lot, and I believe its applications go far beyond the treatment of ulcers and *H. pylori*.



Mastic gum is derived from *Pistacia lentiscus*, a member of the pistachio tree family

— Abstracts —

Kaliora AC, Stathopoulou MG, Triantafillidis JK, Dedoussis GV, Andrikopoulos NK. Alterations in the function of circulating mononuclear cells derived from patients with Crohn's disease treated with mastic. *World J Gastroenterol.* 2007 Dec 7;13(45):6031-6.

AIM: To assess the effects of mastic administration on cytokine production of circulating mononuclear cells of patients with active Crohn's disease (CD). **METHODS:** The study was conducted in patients with established mildly to moderately active CD, attending the outpatient clinics of the hospital, and in healthy controls. Recruited to a 4 wk treatment with mastic caps (6 caps/d, 0.37 g/cap) were 10 patients and 8 controls, all of who successfully completed the protocol. Interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), monocyte chemotactic protein-1 (MCP-1), macrophage migration inhibitory factor (MIF) and intracellular antioxidant glutathione (GSH) were evaluated in peripheral blood mononuclear cells (PBMC) before and after treatment. **RESULTS:** Treating CD patients with mastic resulted in the reduction of TNF-alpha secretion (2.1 +/- 0.9 ng/mL vs 0.5 +/- 0.4 ng/mL, $P = 0.028$). MIF release was significantly increased (1.2 +/- 0.4 ng/mL vs 2.5 +/- 0.7 ng/mL, $P = 0.026$) meaning that random migration and chemotaxis of monocytes/macrophages was inhibited. No significant changes were observed in IL-6, MCP-1 and GSH concentrations. **CONCLUSION:** This study shows that mastic acts as an immunomodulator on PBMC, acting as a TNF-alpha inhibitor and a MIF stimulator. Although further double-blind, placebo-controlled studies in a large number of patients is required to clarify the role of this natural product, this finding provides strong evidence that mastic might be an important regulator of immunity in CD.

Kaliora AC, Stathopoulou MG, Triantafillidis JK, Dedoussis GV, Andrikopoulos NK Chios mastic treatment of patients with active Crohn's disease. *World J Gastroenterol.* 2007 Feb 7;13(5):748-753

AIM: To evaluate the effectiveness of mastic administration on the clinical course and plasma inflammatory mediators of patients with active Crohn's disease (CD). **METHODS:** This pilot study was conducted in patients with established mild to moderately active CD, attending the outpatient clinics of the hospital, and in healthy controls. Ten patients and 8 controls were recruited for a 4-wk treatment with mastic caps (6 caps/d, 0.37 g/cap). All patients successfully completed the protocol. CD Activity Index (CDAI), Nutritional Risk Index (NRI), C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), monocyte chemotactic protein-1 (MCP-1), and total antioxidant potential (TAP) were evaluated in the plasma at baseline and at the end of the treatment period. Results were expressed as mean values +/- SE and $P < 0.05$ was considered to indicate statistical significance. **RESULTS:** Patients exhibited significant reduction of CDAI (222.9 +/- 18.7 vs 136.3 +/- 12.3, $P = 0.05$) as compared to pretreatment values. Plasma IL-6 was significantly decreased (21.2 +/- 9.3 pg/mL vs 7.2 +/- 2.8 pg/mL, $P = 0.027$), and so did CRP (40.3 +/- 13.1 mg/mL vs 19.7 +/- 5.5, $P = 0.028$). TAP was significantly increased (0.15 +/- 0.09 vs 0.57 +/- 0.15 mmol/L uric acid, $P = 0.036$). No patient or control exhibited any kind of side effects. **CONCLUSION:** The results suggest that mastic significantly decreased the activity index and the plasma levels of IL-6 and CRP in patients with mildly to moderately active CD. Further double-blind, placebo-controlled studies in a larger number of patients are required to clarify the role of this natural product in the treatment of patients with CD.

Aksoy A, Duran N, Toroglu S, Koksall F. Short-term effect of mastic gum on salivary concentrations of cariogenic bacteria in orthodontic patients. *Angle Orthod.* 2007 Jan;77(1):124-8.

OBJECTIVE: To determine antibacterial activity of chewing mastic gum against the salivary levels of *Streptococcus mutans*, the total number of viable bacteria, and lactobacilli in patients undergoing therapy with fixed orthodontic appliances. **MATERIALS AND METHODS:** In this study, the levels of *S mutans*, lactobacilli, and total cultivated bacteria were measured before and after chewing mastic gum. The antibacterial effects of chewing mastic gum against these microorganisms in saliva were compared with a placebo gum. The counts for orthodontically treated patients were evaluated before chewing gum; just after chewing gum; and after 45, 75, 105, and 135 minutes. Saliva samples taken from the patients were inoculated onto trypticase-yeast-cystine-bacitracin agar for *mutans streptococci* and onto Rogosa agar for lactobacilli. The agar plates were incubated for 48 hours anaerobically at 37 degrees C. The total number of viable bacteria was then counted. **RESULTS:** Just after chewing the mastic gum for 15 minutes, a significant decrease of total bacteria and *S mutans* was observed ($P < .001$). The reduction in lactobacilli was not significant at later first stage ($P > .05$). However, at the end of 135 minutes, there were significantly fewer *S mutans* ($P < .001$), total viable bacteria ($P < .001$), and lactobacilli ($P < .001$) in the oral cavity after chewing mastic gum than after chewing paraffin ($P < .001$). The results show that chewing mastic gum decreased the total viable bacteria, *S mutans*, and lactobacilli in saliva in orthodontically treated patients with fixed appliances. **CONCLUSION:** Chewing mastic gum might be useful in preventing caries lesions.



From Folklore to Good Science: New Uses for Mastic Gum

Mastic gum, a resin from an evergreen shrub common in the Mediterranean and Mideast, has long been revered in folk medicine as a unique treatment for digestive disorders. The gum was used by Greeks, Egyptians and Babylonians. Though mastic trees are found throughout the Mediterranean islands, the tree that produces the healing resin is particular to the island of Chios. It has a lemony balsam-like scent that wafts through the warm, mild island air.

In the 2nd century B.C., Galenus suggested mastic was useful for bronchitis and improving the condition of the blood. In the 15th century, Christopher Columbus wrote that mastic gum was antibacterial and even used against cholera. And Thomas Fuller's *Pharmacopoeia extemporanea*, published in 1710, includes mastic. In Europe, mastic gum has been used in toothpaste and mouthwash, and pharmaceutical companies use it in self-absorbing surgical threads as well as bandages for surgical wounds.

Scientific research backs up and echoes the folk wisdom: it turns out that mastic gum is so effective in healing the gut that in studies utilizing the resin, the original site of an ulcer has been completely replaced by healthy epithelial cells. In fact, researchers at the University of Nottingham found that just a gram of mastic gum per day for two weeks markedly reduced pain and resulted in rapid resolution of peptic ulcers.

Over the last few decades, research has demonstrated two powerful mechanisms by which mastic gum works: by inhibiting bacterial growth and reducing inflammation. Particularly well studied is mastic's deadly impact on *H. pylori*, the infamous microbe implicated in ulcers. Most pathogenic bacte-

ria cannot penetrate the protective lining of the stomach and small intestine, but *H. pylori* first alters the pH of the lining, and then drills into it, causing gastritis and ultimately ulcers. Nearly 90% of duodenal ulcers test positive for *H. pylori*, while 70% of gastric ulcers test positive.

Mastic's moment arrived in 1998, when a landmark study in the *New England Journal of Medicine* demonstrated that the resin effectively inhibited the growth of seven different strains of *H. pylori*—a standard reference strain and six fresh clinical isolates. Three of the isolates were sensitive to the most common antibiotic treatment, metronidazole, and three were resistant. Mastic killed all seven strains. The minimal bactericidal concentration at 24 hours for all seven strains was 0.06 mg of crude mastic per milliliter on an agar plate. At lower concentrations, bacterial growth was significantly inhibited. Transmission electron microscopy showed obvious structural changes in the organisms.

A 2007 study from the University of Athens backs up the *New England Journal of Medicine* research: mastic gum given to mice over a 3-month period resulted in a 30-fold reduction of colonization with *H. pylori*.

Since that time, mastic gum has been studied in everything from Crohn's disease to dental caries. It seems to have a potent anti-inflammatory mechanism of action. In Crohn's disease, according to 2007 research from the University of Athens, mastic gum lowered levels of the inflammatory molecule tumor necrosis factor-alpha (TNF-alpha), and increased the beneficial macrophage migration inhibitory factor (MIF). In prostate cancer research from the University of Shanghai in 2007, mastic

gum suppressed NF-kappaB activity in the prostate cancer cell. The NF-kappaB signaling pathway leads to many inflammatory molecules.

Even dental caries might be reduced with mastic gum, new research shows. In research from Süleyman Demirel University in Turkey, chewing mastic gum inhibited the common *Streptococcus mutans* bacteria known to cause most tooth caries. Orthodontically treated patients with fixed appliances were evaluated before chewing gum; just after chewing gum; and then after 45, 75, 105, and 135 minutes. Just after chewing the mastic gum for 15 minutes, a significant decrease of total bacteria and *S. mutans* was observed ($P < .001$). At the end of 135 minutes, there were significantly fewer *S. mutans* ($P < .001$), as well as total viable bacteria ($P < .001$), and even lactobacilli ($P < .001$) in the oral cavity. The researchers concluded that mastic gum's powerful antibacterial effect might help prevent cavities. Even bad breath might be eased, according to a 2006 study from the Hebrew University's Hadassah School of Dental Medicine. By measuring volatile sulfide levels in the mouth, researchers found that a number of herbs were helpful (echinacea, elder, mastic gum, marigold, sage, lavender, thyme, and chamomile). Another study from the same department tested mastic gum alone on *Porphyromonas gingivalis*, a bacterium implicated in gingivitis. Paper discs impregnated with mastic gum methanolic extract produced large inhibition zones without showing signs of hemolysis, and so the researchers concluded that "mastic gum may be used as a potential non-toxic local agent in treating oral malodor and gum disease." And finally, researchers at Meikai University School of Dentistry, in Saitama, Japan, com-

pared mastic gum to a placebo gum in a double-blind trial, and found that over a seven day period, mastic-gum chewers had significantly less bacteria in their mouths and a significantly reduced plaque index.

The benefits of mastic gum, a naturally occurring resin, are truly being rediscovered for both antimicrobial and immune modulating effects.

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Glutathione: New Research Reveals New Benefits

Precursors May Be Key To Restoring Health

Glutathione is our body's most important water-soluble antioxidant and is found in impressively high concentrations inside cells. Since oxidative stress can disrupt normal cell function and even lead to cell death, it's not surprising that low levels of glutathione are implicated in a surprisingly wide array of disorders—Parkinson's, Alzheimer's, autism, asthma and cystic fibrosis, cardiovascular disease, cancer, immune dysfunction, mercury toxicity, diabetes, macular degeneration, chronic fatigue syndrome and the aging process itself. The newest research continues to uncover the significance of glutathione in health, and offers promising routes to boosting glutathione through targeted supplements, such as whey protein and concentrated whey protein that are rich in precursors, or extracts such as curcumin, which protects and preserves glutathione in the body.

Glutathione exists in reduced (GSH) and oxidized (GSSG) states. In healthy cells and tissue, more than 90% of the

total glutathione pool is in the reduced form (GSH) and less than 10% exists in the disulfide form (GSSG). The consequences of low glutathione include:

- A reduced ability to detoxify environmental toxicants
- A reduced ability to chelate and clear heavy metals
- Increased gut permeability
- Increased Th2 (pro-inflammatory t-cells)
- Reduced levels of the potent antioxidants, Vitamin C and Vitamin E, both of which depend on glutathione

Here's a look at the latest, cutting-edge research on glutathione and health.

- **Parkinson's Disease.** Oxidative stress can promote neurodegeneration in Parkinson's disease, and has been correlated to the severity of the condition. The disease is also associated with loss of glutathione, and in-

travenous glutathione administration has been used clinically with success, according to researchers at the Robert Wood Johnson Medical School. In one Italian study of nine Parkinson's patients, intravenous glutathione given once a day for a month decreased their symptoms by 42%, a therapeutic effect that lasted up to four months. In a 2008 study, depletion of glutathione was found in the neurons of the substantia nigra—the part of the brain affected in Parkinson's disease.

- **Alzheimer's Disease.** A study on Alzheimer's patients found that the worse their cognitive ability, the lower their levels of glutathione. Beta amyloid (which leads to the plaques that are a signature of the disease) have been shown to increase oxidative stress and lipid peroxidation. Glutathione has been shown to protect cells in culture against beta amyloid.

- **Immune Function.** White blood cells produce more antibodies in the presence of glutathione. The immune sys-

tem's t-cells, b-cells, macrophages and neutrophils rely on glutathione, and a deficiency is linked to higher levels of the cytokines interleukin-8 and -4, and even t-cell inactivation.

- **Lung Diseases.** The lung is a primary site for glutathione, and the level of extracellular glutathione in the lung's epithelial lining fluid (ELF) is 140 times that in blood plasma. Cystic fibrosis is marked by a severe deficiency of glutathione in the ELF. According to a 2008 study, pulmonary fibrosis (IPF) is characterized by a huge alveolar oxidant burden and a deficiency of glutathione, a major antioxidant, in the pulmonary epithelial lining fluid (ELF). When 1.8 grams of N-acetylcysteine (NAC), a building block for glutathione, was given intravenously to eight pulmonary fibrosis patients and six healthy patients, total glutathione in the patients increased markedly, while it remained unchanged in their healthy counterparts. Higher doses of NAC had no additional effect on the patients.

- **Asthma.** Asthma, too, is marked by low and oxidized glutathione. A 2008 study from the Kurume University School of Medicine in Japan notes that inflammatory cells (particularly eosinophils) in the airways of asthmatics release large amounts of harmful free radicals. According to another 2008 study of the sputum of 44 asthmatics and 31 healthy individuals from the University of Newcastle in Australia, oxidized glutathione and alpha-tocopherol are elevated in asthmatics. And an animal study from Gunma University in Japan found that glutathione status regulates airway hyperresponsiveness and airway inflammation. Type 2 t-cells and cytokines are characteristic of asthma and inflammation. In this study, a membrane-permeating glutathione precursor was used to elevate intracellular glutathione (GSH). In response, the infiltration of allergenic cells (eosinophils) was suppressed. The authors conclude, "These findings suggest that changing glutathione redox balance, increase in GSH level, and the GSH/GSSG ratio... ameliorate bronchial asthma... by suppressing chemokine production and eosinophil migration itself."

- **Liver Function.** Our livers are rich in

glutathione, which exports glutathione to other organs, and liver cells synthesize glutathione from its precursors, and recycle it after it is oxidized. In patients with liver damage, from cirrhosis to viral hepatitis, glutathione is often abnormally low and can be a predictor of inflammation and fibrosis.

- **Cardiovascular Disease.** Glutathione improves symptoms in patients with clogged arteries who find walking painful. In a double blind Verona University study, 40 patients suffering from stage II peripheral artery disease were studied. Twenty were given intravenous glutathione twice a day, and the other twenty were given saline. There were significant increases in pain-free walking, and in blood flow measured with laser Doppler flowmetry after a treadmill test, in the glutathione group. Other research published in the New England Journal of Medicine in 2003 found that low levels of glutathione in red blood cells were linked to an increased risk of heart problems among cardiovascular patients. Patients who have had heart attacks have lowered levels of glutathione, and patients with peripheral artery disease showed improvement in microcirculation after glutathione treatments.

- **Autism:** Recent research in children with autism reveals a distinct pattern of abnormalities indicating oxidative stress. Their blood levels of key molecules such as methionine, cysteine, adenosine, SAH (S-adenosylhomocysteine), SAME (S-adenosylmethionine), and homocysteine are all abnormal, according to a 2004 study in the American Journal of Clinical Nutrition. These substances are important in the synthesis of glutathione, which was found to be as much as 80% depleted in autistic children. They have far lower levels of GSH and higher levels of oxidized glutathione (GSSG).

- **Chronic fatigue syndrome and aging.** A 1999 article in Medical Hypotheses suggested that since glutathione is important both in the functioning of lymphocytes, and in skeletal muscle, low levels might be implicated in chronic fatigue syndrome. The authors note, "As an antioxidant, glutathione (GSH) is essential for allowing the lymphocyte to express its full potential

without being hampered by oxiradical accumulation... Because GSH is also essential to aerobic muscular contraction, an undesirable competition for GSH precursors between the immune and muscular systems may develop. It is conceivable that the priority of the immune system for the survival of the host has drawn to this vital area the ever-diminishing GSH precursors, thus depriving the skeletal muscle of adequate GSH precursors to sustain a normal aerobic metabolism resulting in fatigue and eventually myalgia." In addition, a 2007 article points out that glutathione, with its key role in oxidative stress, is a significant factor in aging and age-related diseases.

How to Boost Glutathione: Specialized Precursors

Vitamin C, N-acetylcysteine, whey protein and concentrated whey protein, as well as curcumin, have all been proven in research to reliably increase and preserve intracellular glutathione.

- **Vitamin C:** A 2006 study found that Vitamin C increases glutathione levels in those with ascorbate deficiency. The effect of vitamin C supplements was determined before supplementation, after 13 weeks of vitamin C supplements (500 or 1000 mg/d), and after 13 weeks of matching placebo. The supplementation group was selected on the basis of low plasma ascorbate (<33 mmol/L) and consisted of 48 healthy men and women, smokers and nonsmokers, aged 25–64 years. Ascorbate and glutathione were measured in purified lymphocytes. On supplementation with vitamin C, lymphocyte ascorbate increased by 51% and was accompanied by an increase of lymphocyte glutathione by 18%.

- **Curcumin.** According to a 2008 study in Free Radical Biology & Medicine, curcumin treatment alleviates the effects of glutathione depletion both *in vitro* and *in vivo*. Curcumin is a natural polyphenol derived from turmeric, and treatment of mouse neurons with curcumin restores depleted glutathione levels, protecting against oxidation and preserving mitochondrial activity.

- **N-acetylcysteine.** According to a 2007 article from Stanford University

School of Medicine, supplementing with NAC, a cysteine “prodrug” or cysteine-based glutathione precursor), is safe, well-tolerated and successfully treats GSH deficiency in a wide range of infections, genetic defects and metabolic disorders. “Over two-thirds of 46 placebo-controlled clinical trials with orally administered NAC have indicated beneficial effects,” researchers conclude. Intracellular levels of the amino acid cysteine are rate-limiting for glutathione synthesis. Increasing cysteine is one mechanism by which cells can meet the demand for glutathione. Neurons are highly dependant on cystine and cysteine uptake for glutathione synthesis, and are very vulnerable to heavy metal-induced oxidative stress.

- Concentrated whey protein. Whey protein has been shown to be a potent building block that increases levels of intracellular glutathione. Whey proteins have captured the attention of many physicians, and have been found clinically extremely useful in chronic fatigue syndrome and chronic illness. A 2006 study on whey protein and asthma tested eleven children before and after one month of 10 grams of whey protein daily. IgE significantly decreased. However, to obtain significant levels, large amounts of whey protein need to be taken daily. In contrast, whey protein extract/filtrate (WPEF) is a hydrolysed whey protein powder that is highly concentrated and specifically designed to support glutathione production in the liver. A tablespoon can be taken daily. The key active ingredients in WPEF are cysteine-containing peptides that are easy for the body to absorb. In an informal study, eleven volunteers taking WPEF reported improved energy, motivation, sleep and mental alertness. In an independent animal study, rats were fed a standard diet in which the supply of glutathione precursors was increased by 40%, using WPEF. In the WPEF group almost two times more GSH was synthesized compared to controls. In a second study, researchers examined the effect of WPEF on liver glutathione levels after toxic and oxidative stress. In rats given WPEF after oxidative insult, high baseline glutathione levels were re-established.

Finally and most significantly, a 21-day

placebo controlled, double blind study of WPEF demonstrated remarkable benefits. Individuals consumed 40 grams of alcohol daily (2 glasses of red wine) while on a controlled diet. Alcohol presents a challenge to the liver, and thus was chosen as a model for this particular study on oxidative stress. One group received 3.4 grams of WPEF (one level tablespoon) daily, while the placebo group received 3.4 grams of an amino acid mixture that looked identical to WPEF but did not contain the key, cysteine-containing peptides. Three weeks later, urine markers of lipid peroxidation were found to be significantly reduced in the WPEF group, and blood levels of inflammatory C-reactive protein, were reduced.

Conclusion

We can clearly see the utility and even necessity of restoring adequate glutathione levels in a host of chronic illnesses. Though there are no magic bullets in medicine, glutathione is one of our body’s most important antioxidants and can help protect cells across a stunningly diverse range of illnesses and disorders.

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New Sales and Marketing Manager

We are also pleased to announce that Jeffrey Lipsius was hired on April 28, 2008 to fill the newly created position of Manager of Sales and Marketing. This new management structure will combine the National Accounts Manager and Customer Service Manager positions.

Reesa Sokoloff resigned her position as National Accounts Manager effective May 15, 2008 to return to practice as a licensed nutritional clinician in New York City. The former Customer Service Manager left the company in March 2008. The combination of these two management positions will allow us to transition our Customer Service and Inside Sales Department into to one cohesive sales group.

Jeffrey comes to Allergy Research Group with 28 years of success in marketing and selling dietary supplements to the natural foods industry and to health care professionals. He has extensive experience managing all aspects of marketing and selling products to the alternative healthcare industry. Jeffrey received his Bachelor of Arts in Psychology from Albright College in Reading, Pennsylvania.

“We are pleased to have Jeffrey fill this important new consolidated position. This is a positive step toward boosting our sales and marketing efforts and to improve service to our distributors, retail and professional customers,” said President, Fred Salomon.

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