

Vitamin Research News

Dedicated to the Scientific Pursuit of Better Health

July 2004, Vol. 18, Number 6

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Selenium is the important co-factor of the powerful antioxidant enzyme, glutathione peroxidase. Methylselenocysteine has been shown to be the safest and most bioavailable form of selenium commercially available.

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In various clinical studies, NKO™ has been shown to be a more effective source of EPA and DHA than conventional fish oils, and is far more resistant to rancidity than conventional fish oils.

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The President's Desk

When is Science Bogus Science?

So often we see the headlines: the media report on a study that has negative results in regard to a particular nutrient. But how many of those studies are sponsored by corporations with a stake in the results? In how many of those studies is the methodology lacking to produce accurate results?

At Vitamin Research Products, we dig deep into the outcome of a study to provide our customers with an honest, accurate and knowledgeable overview of the medical literature and to produce products based on sound, valid science.

A study published in the January 2004 Journal of Clinical Nutrition provides a perfect example of a bogus study appearing under the guise of science. At a first, brief glance, this study, titled "Vitamin E bioavailability from fortified breakfast cereal is greater than that from encapsulated supplements," seems perfectly valid—until one reads beyond the title. Two of the study authors work for the cereal manufacturer General Mills. What's more, the subjects consumed the 400-IU vitamin E capsule or the cereal with fat-free milk.

What's wrong with this picture? Vitamin E is a fat-soluble vitamin. In cereal, however, the vitamin E is pre-emulsified, which means the vitamin E is essentially preabsorbed and not in need of fat for proper bioavailability. Vitamin E capsules, on the other hand, which are not pre-emulsified, must be consumed with fat for proper effectiveness. Consequently, the researchers compared apples to oranges.

At Vitamin Research Products, it is our firm commitment to formulate our supplements based on real science—not a bogus impostor. After all, research is our middle name.

Robert Watson
President/CEO

Anti-Aging Nutrient Review and Update, Part 2

CoQ10 Plays Many Roles As Anti-Aging Nutrient

by Ward Dean, MD

Coenzyme Q10, also known as CoQ10, is a normal and essential component of the membranes of mitochondria—the intracellular organelles that manufacture ATP, the basic energy molecule of the cell. CoQ10 plays a critical role in the production of energy in nearly every cell of the body, and is found in most living systems.

For this reason, it was named ubiquinone (for ubiquitous quinone) by its discoverer, R.A. Morton. CoQ10 has been shown to improve many conditions associated with aging, and to extend the lifespan of several organisms.

Mechanisms of Action

CoQ10 is a vitamin-like fat-soluble nutrient (quinone) and functions as an important component of the mitochondrial energy electron transduction chain (oxidative phosphorylation) and in the production of adenosine triphosphate (ATP).

CoQ10 is a powerful antioxidant, capable of inhibiting lipid peroxidation in mitochondrial membranes (its chemical structure is similar to that of Vitamin E). It has cardioprotective, cytoprotective, and neuroprotective properties, is a calcium channel blocker and membrane stabilizer, modulates prostaglandins, inhibits intracellular phospholipases and preserves myocardial NaK-ATPase.¹

Clinical Uses of CoQ10

Congestive Heart Failure

A number of studies have confirmed CoQ10's effectiveness in improving cardiac function in cases of cardiomyopathy and congestive heart failure. In one six-year study of 122 patients with New York Heart Association classes II, III and IV chronic dilated cardiomyopathy, subjects were treated with 100 mg CoQ10 each day. At the beginning of the study, the mean ejection fraction—a measure of ventricular blood flow—was 41 percent. Ejection fraction increased to 59 percent after only six months on CoQ10. Eighty-seven percent of the participants experienced significant improvement in their ejection fraction during this time, and improved by one or two New York Heart Association classes. Those in class II achieved the greatest benefit. All of these subjects became asymptomatic after CoQ10 administration. Despite these positive results, the authors suggested that the 100 mg dose of CoQ10 was too low, and the subjects might have done even better on higher doses.²

Angina Pectoris

Angina pectoris is chest pain caused by decreased blood flow (and decreased oxygenation) of the heart. In an early Japanese study, 12 patients (average age 56 years) with stable angina were given 150 mg of CoQ10 per day for four weeks. CoQ10 reduced anginal frequency and nitroglycerin use and increased exercise time and time to ST-segment depression.³ (ST-segment is an abnormality on an electrocardiogram indicating the heart is not getting enough oxygen, presenting an

increased risk of heart attack.) These results were confirmed in more recent studies, using doses ranging from 30 to 600 mg per day.⁴

Hypertension

CoQ10 is also helpful in hypertension. In mild or borderline cases, it may be all that is needed. For example, in one study conducted at the Department of Veteran's Affairs Medical Center in Boise, Idaho, physicians administered 120 mg of CoQ10 to 46 male and 37 female hypertensive patients (mean age was nearly 70 in both cases). Systolic blood pressure was reduced by an average of 17.8 mmHg.⁵ Australian researchers obtained similar blood-pressure-lowering results with a dose of 200 mg CoQ10 in 74 hypertensive diabetics.⁶

Protection Against Statin Toxicity

Statins, or 3-hydroxy-3-methylglutaryl Co A (HMG-CoA) reductase inhibitors, are effective drugs for lowering cholesterol. However, published data confirm that statins can cause myopathies (muscle disease), rhabdomyolysis (muscle damage), and renal failure. In May 2000, the FDA warned about liver failure with regard to statin drugs. Statins have been associated with an increased incidence of cataracts, cancer, peripheral neuropathies and some psychiatric disturbances.

Statins lower CoQ10 levels in the heart, skeletal and liver tissues. In mice, statins lower ATP levels, and impair energy metabolism. Although package inserts and marketing material do not mention the statins-CoQ10 link, two U.S. patents filed in January and February of 1989 and granted in 1990 describe a method for counteracting statin-associated myopathy and potential liver damage by concurrent administration of the statins with CoQ10. Both of these patents were assigned to Merck & Co.

However, for more than 14 years, the producers of statins have not acted upon this information and failed to reveal the statin-CoQ10 relation to millions of statin users and to the medical community.

The concern by physicians and scientists regarding the depletion of CoQ10 by the statins is growing from a level of concern to one of alarm.

With ever higher statin potencies and dosages, and with a steadily shrinking target LDL cholesterol, the prevalence and severity of CoQ10 deficiency is increasing noticeably.

An estimated 36 million Americans are now using statin drug therapy. Statin-induced CoQ10 depletion is well documented in animal and human studies with detrimental cardiac consequences in both animal models and human trials. This drug-induced nutrient deficiency is dose-related and more notable in settings of pre-existing CoQ10 deficiency such as in the elderly and in heart failure.

We are currently in the midst of a congestive heart failure epidemic in the United States, and this epidemic may be due, in large part, to the excessive use of statin drugs and inadequate use of CoQ10.⁷

Neurologic and Neurodegenerative Disorders

CoQ10 has the potential to be a beneficial agent in any neurodegenerative disease which is characterized by impaired mitochondrial function and/or excessive oxidative damage. CoQ10 has been found to be protective against neuronal toxins in animal models of Parkinson's disease, amyotrophic lateral sclerosis and Huntington's

disease.⁸

Recent clinical trials in these disorders demonstrate that supplemental CoQ10 can slow functional decline—particularly of Parkinson’s disease.⁹ Parkinson’s is a degenerative neurological disorder for which no treatment has been shown to slow its progression. Nevertheless, in a large, multicenter study, 80 subjects with early Parkinson’s were administered CoQ10 at dosages of 300, 600 or 1,200 mg per day. They were followed for 16 months. Those who received CoQ10 experienced less disability than those who received the placebo, and the benefit was greatest in those who received the highest dosage.¹⁰

A short confirmatory study was conducted by scientists in Germany, giving CoQ10 in daily doses of 360 mg for four weeks to 28 Parkinson’s patients. CoQ10 supplementation provided a mild but significant symptomatic benefit on Parkinson’s symptoms and a significantly better improvement in performance compared with placebo.¹¹

Diabetes

Australian researchers recently reported a reduction of glycosylated hemoglobin (HbA1c—an indicator of blood sugar control) in a large group of hypertensive diabetics who received a daily CoQ10 dosage of 200 mg per day.⁶

Cancer

In 1994, a dramatic report of two cancer patients treated with CoQ10 was published.¹² The first was of a 59-year-old woman who had intraductal carcinoma (cancer) of the left breast, which resolved completely on a daily dose of 390 mg CoQ10. A second patient, after breast surgery, was found to have residual intraductal carcinoma in the tumor bed. She refused additional surgery, and was placed on 300 mg CoQ10 per day.

Over a year later, there was no evidence of residual tumor or metastases. Despite practicing oncology for 35 years, over which he had treated about 200 cases of breast cancer per year, one of the authors commented that he had “never [before] seen a spontaneous complete regression of a 1.5-2.0 cm breast tumor and ha[d] never seen a comparable regression on any conventional anti-tumor therapy.”

A more recent study indicated that breast cancer is associated with a decrease of plasma CoQ10 levels, and the worse the cancer, the greater the decrease in CoQ10.¹³

Periodontal Disease

CoQ10 is dramatically effective in treating periodontal disease, a common malady of aging. In an early study, eight patients received CoQ10 at 25 mg, twice a day, versus a placebo group. The results showed a reduction in pocket depth with photographic evidence of improved gingival health.¹⁴ In a more recent study, CoQ10 was used topically, with significant improvement.¹⁵

Renal Failure

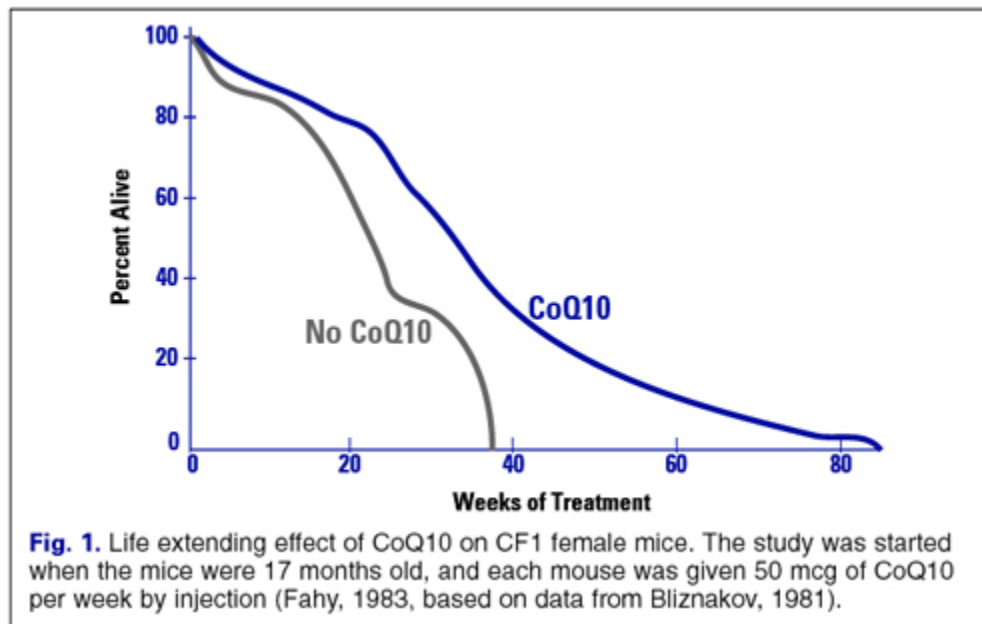
Dr. Ram B. Singh of the Heart Research Laboratory and Center for Nutrition in Moradabad, India, reported on a new indication for CoQ10 therapy, in a patient with acute glomerulonephritis, renal failure and high levels of lipid peroxides. He administered 180 mg per day of CoQ10, with a resultant lowering of lipid peroxides and significant improvement in renal function.¹⁶

In a follow-up study of 11 patients with chronic renal failure (serum creatinine levels were 5 mg/dL or above) who were on intermittent dialysis, Dr. Singh again administered CoQ10 at a dosage of 180 mg per day. After four weeks of treatment, the patients experienced significant reductions in serum creatinine and blood urea and significant increases in creatinine clearance and urine output, and fewer subjects required dialysis.¹⁷

Life Extension Research with CoQ10

Dr. Bliznakov's Mouse Experiment

One of the earliest anti-aging experiments with CoQ10 was conducted in 1980 by Dr. Emile G. Bliznakov, a pioneering CoQ10 researcher. Bliznakov divided middle-aged (16 to 18 months) white mice into two groups: one group received regular injections of CoQ10 while the other received a placebo injection (salt water). Bliznakov found that all of the mice in the saline (control group) died within eight months after the start of the experiment, whereas the CoQ10 animals survived to significantly greater maximum lifespan (up to 12 more months). The mean survival time was extended by 56 percent (Fig. 1).¹⁸



Dr. Fahy's Mouse Experiment

Similar results were also reported in an unpublished study by Dr. Gregory M. Fahy, then Director of the Organ Cryopreservation Laboratory at the American Red Cross Jerome Holland Laboratory in Rockville, Md.¹⁹ I recently discussed Dr. Fahy's findings with him at the 33rd Annual Meeting of the American Aging Association in St. Petersburg, Fla. Dr. Fahy confirmed his observations, in female mice of a different strain, although no effect on the mean lifespan of male mice was seen (probably because only five male mice were examined).

Drs. Harris and Coles' Mouse Experiments at UCLA

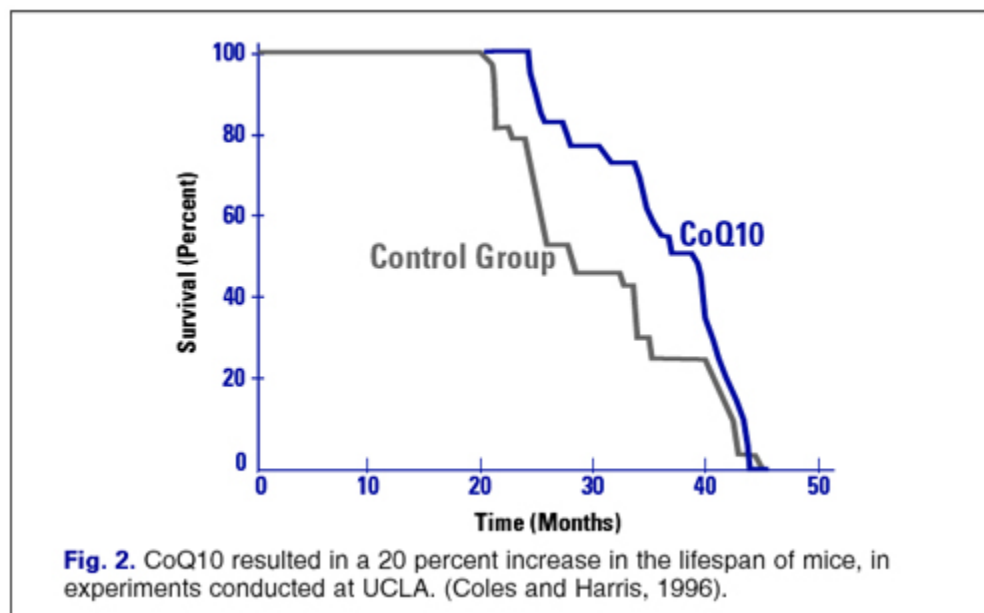
Another longevity study with CoQ10 was completed in 1994 by Dr. Steven Harris, at Dr. Roy Walford's Laboratory at the UCLA Medical Center in Los Angeles. Walford's lab was especially suited for longevity experiments because of his pioneering work

in the study of the anti-aging effects of caloric restriction. Dr. Walford (see box at right) was one of the first scientists to document the delay in the decline of the immune system in calorie-restricted mice.

Two groups of female mice were used. The first group was placed on a mildly restricted diet to ensure against obesity (which happens in this strain with ad lib feeding), and to mimic the average nonobese health-conscious consumer. The second group was given this diet with 0.1 percent CoQ10 added by weight of food. This dosage was equivalent to a human dosage of approximately 750 mg of CoQ10 per day.

At 39 months of age (which is close to the 42-month maximum lifespan for these mice), 50 percent of the CoQ10 treated mice were still alive, compared with only 25 percent of the control animals. The average lifespan for the controls was 30.8 months, whereas the average lifespan of the CoQ10 group was 37.0 months (a 20 percent increase).

A few days after the 43-month mark, however, all animals in both groups were dead, demonstrating that although CoQ10 achieved a significant rectangularization of the survival curve, a true right shift or extension of the entire curve to the right was not achieved (Fig. 2). The last CoQ10 treated animal only outlived the last control by four days.



The scientists reported that animals that received CoQ10 appeared to be much healthier than the control animals. The most spectacular difference between the two groups, however, was the far greater level of activity in the CoQ10 animals at about the 50 percent survival point than in the control animals (which appeared to be close to death in many cases).

Coles and Harris suggest that CoQ10 may be able to slow some deteriorative processes that are associated with aging in laboratory mice, although there was no retardation of the increase in the mortality rate.

Most significantly, although the maximum lifespan of the animals was not increased, the average lifespan of the animals was increased by 20 percent. To fully appreciate the significance of a 20 percent increase, consider that scientists have estimated that a total cure of all cancers would result in an increase in the average human lifespan of only 4 percent.²⁰

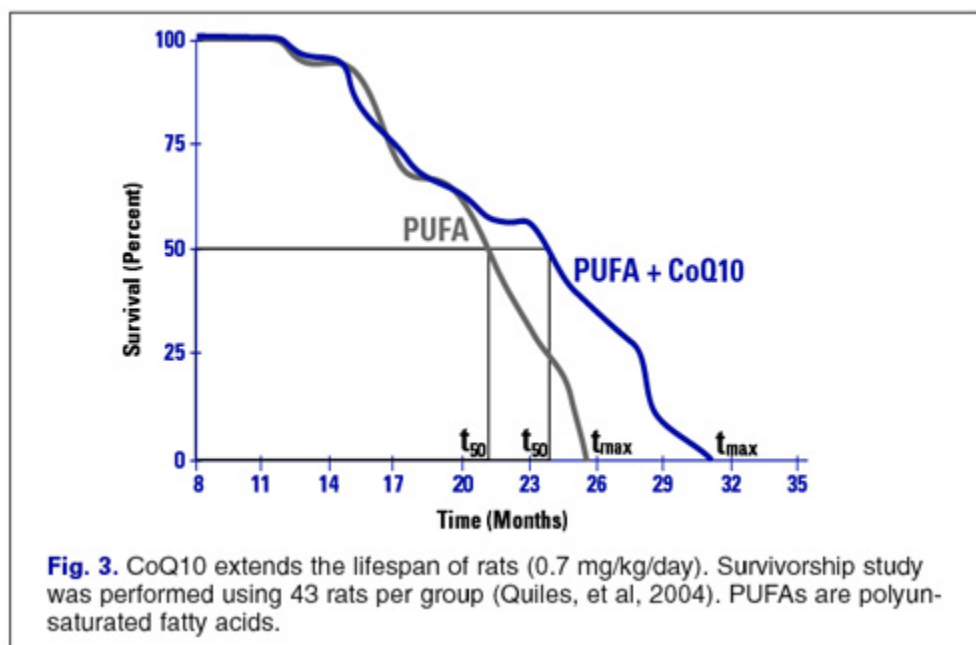
Finnish Rat and Mouse Experiment

A subsequent study in 1998 by scientists in Finland produced disappointing results, however.²¹ The scientists administered CoQ10 to rats and mice, at a dose of 10 mg/kg/day. This dose is roughly comparable to the dosage used by Drs. Coles and Harris in the UCLA study previously described. The results indicated that CoQ10 had no significant effect, positive or negative, on the lifespan of either the rats or mice.

Spanish Rat Experiment

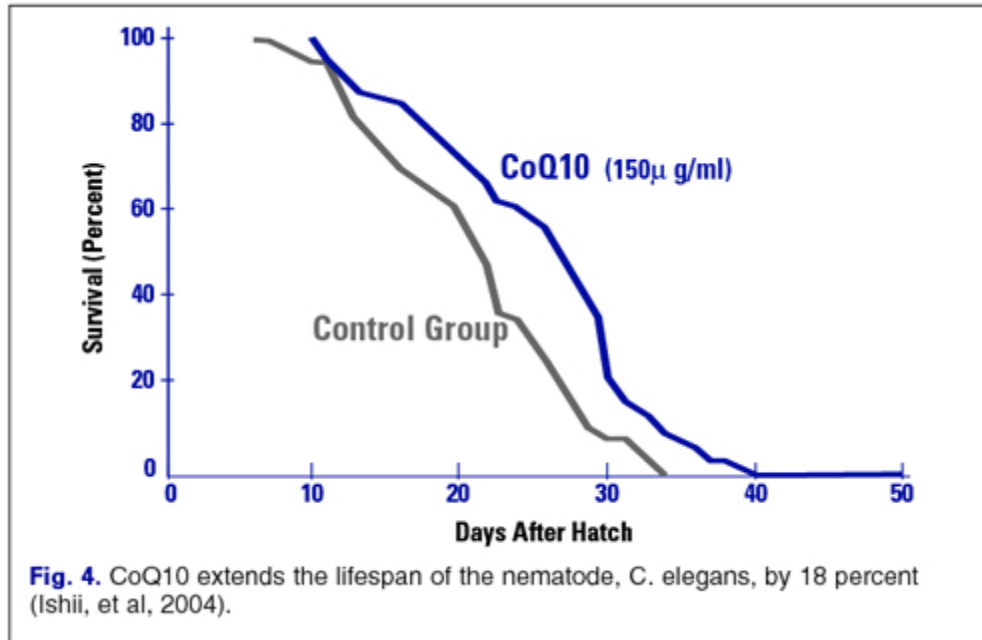
Scientists in Spain recently published another report of the lifespan-extending properties of CoQ10 in rats. In their study, they used two groups of rats fed a diet that contained 8 percent fat (containing 61 percent polyunsaturated fatty acids).

The experimental group's diet was supplemented with 0.7 mg/kg/day of CoQ10. The supplemented animals reached a significantly higher mean lifespan (2.5 months, or 11.7 percent higher than the controls) and a significantly higher maximum lifespan (six months, or 24 percent higher) (Fig. 3).²²



Japanese Nematode Experiment

Finally, another positive study has just been published indicating that CoQ10 extends the lifespan of nematodes (Fig. 4).²³ The scientists used CoQ10 concentrations of 50 and 150 mcg/ml of distilled water. Admittedly, extending the lifespan of a nematode is less exciting than extending the lifespan of a mammal, but it is a further indication that something positive is going on at a fundamental level.



Conclusion

CoQ10 appears to qualify as a highly beneficial “anti-aging” nutrient, based on its multiple mechanisms of action, its broad range of effects on a number of life-threatening or debilitating clinical conditions, its lifespan-extending properties in more than one species, and complete absence of adverse effects. Beneficial effects have been demonstrated in some conditions with as little as 30 to 60 mg per day.

Of the known lifespan studies with CoQ10 (four mouse, two rat, and one nematode), two of the mouse studies (Bliznakov’s and Fahy’s) resulted in increased maximum lifespan, one resulted in increased mean lifespan (Cole’s and Harris’), and one showed no change; one rat study showed no change; and one rat and nematode study each resulted in increased mean and maximum lifespan.

The more serious the condition, the greater the dose that should be taken. As research continues to accumulate, it appears that the higher the dosage the greater the benefit (as evidenced by the 390 mg dose in breast cancer, and 1,200 mg dose in Parkinson’s disease), and that the only limiting factor on CoQ10 dosage is the cost.

Criteria for Selecting Anti-Aging Nutrients for This Series

For this series of articles reviewing top anti-aging nutrients, Ward Dean, MD, has selected substances based on several criteria:

1. The mechanism by which the substance is believed to act. Most substances discussed are involved in one or more theories of aging (i.e., antioxidants/free radical theory; cross linkage inhibitors/cross linkage theory; hormone receptor sensitizers/neuroendocrine theory, etc).
2. The health-enhancing effect of the substance.
3. Whether the substance has shown the capability to reverse or restore a biomarker

to a more youthful state.

4. Has the substance demonstrated the ability to extend the maximum lifespan of one or more experimental organisms?

5. Practical considerations: An individual's "pill capacity"—how many capsules/tablets is a person willing to take? Cost and availability—for example, some substances are beyond the reach of many people due to high cost or other impediments (i.e., legal issues, availability, requirement for a prescription, etc.).

Based on these criteria, the series of articles presents what Dr. Dean considers to be the most effective anti-aging/life extending substances readily available today. The substances featured are presented in no particular order. The first article in the series focused on DHEA, appearing in the June 2004 issue.

Remembering Dr. Roy Walford

Dr. Roy Walford, one of the great pioneers of life-extension and antiaging medicine, died April 27, 2004, from complications of amyotrophic lateral sclerosis (commonly known as ALS, or Lou Gehrig's Disease). He was 79 (he would have been 80 June 29).

Dr. Walford observed that caloric restriction was the most effective means of extending the lives of experimental animals; he wrote four books on the subject. He practiced what he preached and applied his calorically restricted diet to himself. His colleagues joked, "Roy may not live any longer, but it will sure seem like it."

We invited Dr. Walford to speak at the Monaco Anti-Aging Conferences, but unfortunately his failing health prevented him from making the trip. Nevertheless, he believed his restricted diet enabled him to survive with his disease much longer than had he followed a more traditional diet.

We'll miss you, Roy. Rest in peace. (For information about Dr. Walford go to www.walford.com.)

Ward Dean, M.D.

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Samento®: New Remedy For An Ancient Enemy—Lyme Disease

by James South, M.A.

Lyme disease was first recognized around 1975, when a mysterious outbreak of juvenile rheumatoid arthritis occurred around Lyme, Connecticut.¹ In 1982, the causative agent of Lyme disease was discovered by Willy Burgdorfer. It turned out to be a spirochete (spiral-shaped bacterium) from the genus *Borrelia*, subsequently

named *Borrelia burgdorferi* (Bb).¹

As Lyme disease expert Jo Anne Whitaker, M.D., notes: “Lyme disease is called the ‘New Great Imitator’ because, like syphilis [the original ‘Great Imitator’], it attacks multiple organ systems and mimics many diseases. Both diseases are caused by spirochetes.”² Originally believed to be spread only through bites by the tiny deer tick (*Ixodes dammini*), it is now known to be potentially spread by many tick species, as well as bot-flies, mosquitoes and fleas.^{3,4}

And in a recent article with 224 references, physicians W.T. Harvey and P. Salvato have offered persuasive evidence that Lyme disease is transmitted sexually and congenitally (by birth from an infected mother), as well as through breastfeeding.^{1,4} They also provide evidence that Lyme disease may be a hidden epidemic, affecting as much as one-sixth of the human race, if not more.⁴ By 1994, Lyme disease experts Brian Fallon and Jenifer Nields could already state: “Now the most common vector-borne [spread by ticks and insects] infection in the United States, Lyme disease is increasing in incidence and geographic spread.”⁵

Lyme: One Disease, Many Symptoms

Lyme disease is believed to cause, mimic, manifest as, be misdiagnosed as, or contribute to more than 300 conditions and diseases.⁶ About 60 percent of those bitten by Bb-infected ticks or insects will develop a characteristic “bull’s-eye” rash (erythema migrans), yet many confirmed Lyme disease patients never develop such a rash.¹³

There may be few initial symptoms other than a flu-like syndrome, yet within weeks to years a diversity of symptoms may occur. These may include fatigue, low grade fevers, night sweats, migrating joint pains or arthritis, muscle pains, sleep disturbances, frequent and/or severe headaches, numbness or tingling in hands or feet, nerve pains, brain fog, hypersensitivity to lights, sounds, tastes or smells, memory and concentration problems, speech difficulties, depression, irritability, mood swings,⁷ heart, eye, respiratory and gastrointestinal problems,² to name just a few. Symptoms may come and go, varying in intensity. The Bb spirochete may penetrate into the brain as early as three weeks after infection.⁸

Lyme: Difficult and Controversial Diagnosis

Lyme disease has become a surprisingly controversial disease.⁹ Even famed novelist Amy Tan has been drawn into the controversy, after a belated Lyme disease diagnosis in her own case. She complained about being tested even for syphilis and ALS (Lou Gehrig’s disease) before anyone thought to test her for Lyme disease.¹⁰

Why the controversy? The CDC (United States Centers for Disease Control and Prevention) has set up a rather strict formal set of criteria for Lyme disease diagnosis. The CDC is not directly involved in disease treatment. Its criteria are designed as part of its mission to track and assess disease patterns in the United States.

Many conservative physicians use the CDC’s Lyme disease surveillance criteria as clinical diagnostic criteria. A key part of the CDC criteria is a requirement for laboratory confirmation through ELISA (enzyme-linked immunosorbent assay) and/or Western blot antibody testing.⁸

Yet as Lyme disease expert Brian Fallon has written in America’s most prestigious psychiatric textbook: “Although laboratory testing is a valuable component of the

diagnostic assessment, negative test results cannot be used to exclude Lyme disease in a patient with typical clinical features and a history of exposure to a Lyme disease endemic area.... Because the laboratory tests for chronic Lyme disease are not sufficiently reliable to document the presence or absence of persistent infection, decisions regarding treatment should be based primarily upon the physician's clinical judgment."⁸

For those wishing an accurate laboratory confirmation of Lyme disease, Dr. Jo Anne Whitaker, M.D., has developed a new "Quantitative-Rapid Identification of *Borrelia burgdorferi*" test (QRIBb©). Using a fluorescent antibody technique, Whitaker has confirmed Lyme disease in 3,500 blood specimens from ill patients.

Her results in many cases were checked by world-renowned microbiologist Dr. Lida Mattman, who was able in every case to culture and identify live Bb spirochetes from the blood samples the QRIBb test had already certified as Bb-positive. Dr. Whitaker can be reached at 727-937-9077.

She has found many patients were given a false diagnosis (e.g., ALS) who turned out to have Lyme disease, and in many cases recovered from "incurable" ailments after antibiotic treatment.¹¹

Lyme: Treatment Controversies

When Lyme disease is diagnosed, it is normally treated with antibiotics. Fallon states that for early Lyme disease without central nervous system (CNS) involvement, three to four weeks of oral doxycycline, amoxicillin or cefuroxime is recommended.⁸ For Lyme disease with CNS involvement, a four-to-six-week intravenous treatment with ceftriaxone or cefotaxime is recommended.⁸ Fallon recommends that for relapsing patients, longer and repeated courses of antibiotic treatment may be useful.⁸ He notes, "Failure to treat Lyme disease early in its course or for a sufficiently long duration may lead to a chronic illness characterized by persistent waxing and waning neuropsychiatric disturbances, arthralgias [joint pains], myalgias [muscle pains], sensory-hyperacuities, and severe fatigue."⁸

Yet many conservative physicians treating Lyme disease give only a two-to-three-week course of antibiotics, frequently only orally. Because intravenous antibiotic care may cost tens of thousand of dollars, medical insurers and medical benefit managers often discourage or deny such treatment.

Not everyone approves of massive antibiotic treatment for Lyme disease. Dr David Jernigan, co-author with his wife of a recent book on Lyme disease, observes that "It is not enough simply to take an antibiotic: even intravenous antibiotics will only kill 85 percent of the bacteria at best, leaving 15 percent alive and now antibiotic resistant.... Most people with chronic Lyme disease have already used many antibiotics with limited success or may be intolerant and allergic to them."¹²

Fallon and Nields point out "*B. burgdorferi* has been shown to be capable of persisting in human hosts despite extensive antibiotic treatment.... Several features are known to contribute to an organism's resistance to standard lengths of antibiotic treatment. These features include an intracellular location, long replication time, genetic variability, and the ability to become sequestered in difficult-to-penetrate sites. *B. burgdorferi* appears to possess all of these characteristics."⁵ Bb has been shown able to live inside various cells, including fibroblasts, macrophages, and endothelial cells, as well as in antibiotic- and immunologically-privileged sites, such as CNS, joints and the interior chamber of the eye, which protect it from immune

cells and antibiotics.⁵

Samento®

Given the recognized difficulty of successfully treating Lyme disease with standard antibiotic therapy, an alternative treatment that is natural, nontoxic, well-tolerated, effective, and can be taken orally for as many months or years as needed, would be a welcome remedy in the Lyme war.

Fortunately, such a remedy has been available since 2001. It is an herbal extract called “Samento,” made from a Peruvian vine called “Uncaria tomentosa,” also known as “cat’s claw,” “una de gato,” and “Vilcacora.”¹⁴ Samento is made from a rare chemotype of *U. tomentosa* that is rich in pentacyclic oxindole alkaloids (POA) and is guaranteed free of tetracyclic oxindole alkaloids (TOA). It is the TOA-free nature of Samento, combined with its POA potency, that gives Samento its unique effectiveness.

Oxindole Alkaloids

Most cat’s claw products on the market contain a mixture of POA and TOA, in unknown proportions. Yet K.-H. Reinhard has noted “...the root of *Uncaria tomentosa* is a valuable drug only when its pentacyclic chemotype is used without admixture of the tetracyclic chemotype. The pentacyclic oxindole alkaloids act on the cellular immune system. They raise the rate of phagocytosis [germ-killing] by granulocytes [a type of white blood cell]... and they induce the release of a factor from endothelial cells [which line the heart, blood and lymph vessels] that regulates the proliferation of lymphocytes [germ-killing white cells].... The secretion of the factor was effected by the pentacyclic alkaloids but not by the tetracyclic alkaloids. Rather, it was shown that the tetracyclic alkaloids act antagonistically on the release of the factor.”¹⁵

Falkiewicz and Lukasiak report that the POA-stimulated endothelial factor activates normally inactive B and T lymphocytes in humans, increasing germ-killing power.¹⁴ K. Keplinger and colleagues found that in humans, the POAs increased lymphocyte counts when they were too low, and lowered them when too high. Thus, the POAs are both immuno-stimulating and beneficially immunoregulating.¹⁶

Samento: More than POA

The water-alcohol Samento extract also contains many other beneficial components. Multiple quinovic acid glycosides are present as well. “These compounds are what the latest generation of quinolone antibiotics (such as Cipro®) are based on. The natural compounds provide safe and significant direct antimicrobial effects on Lyme disease.”¹⁷ The quinovic glycosides also have shown antiviral activity against rhinoviruses (cold viruses) and vesicular stomatitis virus (oral cold sores).¹⁴

Samento also contains the triterpenes oleanolic and ursolic acid. These have been shown to have liver-protective, anti-inflammatory, antiviral, antibacterial, anti-ulcer, immunostimulating/modulating and blood sugar-lowering properties.¹⁴ Catechin polyphenols, including epicatechin, with anti-inflammatory and blood sugar-lowering effects, are also present in Samento.¹⁴

Samento: Powerful Anti-Inflammatory

Cat’s claw extracts have been shown to have powerful anti-inflammatory effects. A 1998 study verified these effects through multiple in vitro and in vivo experiments.¹⁹ The cat’s claw extract reduced the production of toxic peroxynitrite, stimulated by a bacterial toxin, and reduced subsequent cell death. Mice given Samento plus the

NSAID (non-steroidal anti-inflammatory drug) indomethacin suffered no intestinal lining damage, yet control mice given the same dose of indomethacin without Samento suffered complete destruction of their intestinal lining. The study's authors concluded: "Cat's claw protects cells against oxidative stress and negated the activation of NF-kB [a powerful pro-inflammatory chemical whose production is stimulated by toxins]."

These studies provide a mechanistic evidence for the widely held belief that cat's claw is an effective anti-inflammatory agent.¹⁹ Bb is known to shed membranous materials from its surface that stimulate powerful inflammatory, autoimmune reactions.⁵ In a subsequent study, the same research group found that cat's claw extract reduced TNF-alpha expression stimulated by a bacterial toxin 65 to 85 percent, at only nanogram levels of cat's claw. A nanogram is one-thousandth of a microgram! TNF-alpha is one of the most powerful pro-inflammatory cytokines released (often to excess) by white blood cells when challenged by germ toxins.²⁰

Samento: Clinical Use

John Kule, M.D., began using Samento in his practice in March 2002. After treating 60 patients with it, he wrote a report for the British Naturopathic Journal. He used it to treat a broad range of conditions, including chronic fatigue, fibromyalgia, hypertension, irritable bowel syndrome, candidiasis, gastritis, rheumatoid and osteoarthritis, Lyme disease and benign prostatic hypertrophy. Fifty-nine out of 60 showed distinct clinical improvement.

Frequent findings were increased energy, enhanced sense of well-being, lifting of "brain fog," decreased inflammation, decreased blood pressure in hypertensives, decreased fasting blood sugar in diabetics, reduced fluid retention, and reduced blood pressure medication in hypertensives.²¹ He found only few, mild and transient side effects. Several patients did experience the Herxheimer reaction (explained later in this article).

Dr. David Jernigan, D.C., of Witchita, Kansas, uses Samento extensively for Lyme disease and other infections. He has gotten excellent results, and has found it to be nontoxic, active, highly energetic and synergistic with other remedies. It is a key component for his comprehensive program of treating Lyme disease without antibiotics.¹²

Dr. Lee Cowden, M.D., of Fort Worth, Texas, used Samento along with diet, detoxification and supplements with 13 patients with documented Lyme disease, while leaving 14 Lyme disease patients in the control group on their regular antibiotic regimen. Three of the control group members became slightly better, three became worse and eight were unchanged. All of the Samento group experienced dramatic improvements, with 11 of 13 testing negative for Bb at the end of the study.^{17, 22}

Dr. Stephen Sinatra has found Samento useful for quickly aborting bouts of the flu, as well as preventing it.¹⁸ Samento is known to contain antiviral triterpenes and quinovic acid glycosides, which may account for this benefit.

Samento: Slow But Steady

Due to the unique life cycle of Bb, a quick complete elimination of Bb is unrealistic to expect, whatever germ-killers are used. Because Bb hides inside cells, often in a dormant, cyst form, it spends much of its life cycle sequestered from antimicrobial compounds. When cells die naturally, or from the intracellular presence of Bb, the cysts are released into tissue fluids or blood, where they become a spirochete once

again. It is then that they are most vulnerable to antibiotics or Samento.

Since the various cells that hide Bb will typically have lifespans ranging from two to three weeks up to six to eight months, it may take six to eight months for even one generation of Bb to become exposed to Samento for elimination. Thus it may take eight to 16 months to gradually kill the Bb hiding in several generations of cells. Since Samento is extremely nontoxic,^{14,16} it can be safely taken daily for the “long haul” necessary to thoroughly eradicate Bb from an infected body.

Samento: Cautions

Because Samento empowers the immune system, it should not be used by those on immunosuppressive drugs, e.g. to prevent transplant rejection, nor should it be taken by those who are soon to undergo organ or bone marrow transplants.¹⁵ Since Samento has been shown to lower blood pressure and blood sugar, those with severe low blood pressure or hypoglycemia should use Samento very cautiously.²¹ Pregnant or nursing mothers, as well as very young children, should not use Samento unless advised by a physician.¹⁵ Anyone taking Samento should start with a low dose (one drop in four ounces of water twice daily) and slowly work up to five drops two or three times daily, taken on an empty stomach. Because Samento enhances immune activity and directly kills germs, it may trigger a Herxheimer reaction, especially if started at too high a dose or with too rapid dose increase.

The Herxheimer reaction may include headache, muscle pain, nausea, diarrhea, or flu-like symptoms. It is thought to be due to toxins released from the mass death of microbes killed through treatment, as well as to the immune system’s inflammatory overreaction to the germ toxins. Drinking lots of water and taking fiber and liver support supplements (silymarin, dandelion root extract, lipoic acid) may reduce the risk or severity of a Herxheimer reaction.

If such a reaction occurs when taking Samento, cease its use temporarily and restart later at a lower dose. Those known or suspected to suffer from Lyme disease or other serious infectious illness should ideally use Samento under the care of a doctor or other health care professional.

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Methylselenocysteine: The Superelenium

by James South, M.A.

Selenium is an essential trace mineral, with a recently set RDA (Recommended Dietary Allowance) of 55 mcg.¹ Selenium is known to be essential to activate various key enzymes, such as the antioxidant glutathione peroxidase, the metabolic enzyme thioredoxin reductase, and the thyroid-hormone-activating enzyme iodothyronine deiodinase.²

Although selenium is essential for life and health, it is also a potentially toxic mineral. Selenium expert Dr. R. Passwater notes "...organic forms of selenium [such as selenomethionine] are toxic at levels in the vicinity of 3,500 micrograms (3.5 milligrams) daily. Inorganic forms of selenium [such as sodium selenite/selenate] may be toxic at one-third that level."³ Passwater notes, however, that many Japanese average 600 mcg daily from their diet, and Greenlanders may ingest as much as 1,300 mcg daily, without apparent ill effects.³

Selenium: The Anticancer Mineral

A connection between selenium intake and cancer has been known for decades. In 1969 Dr. R. Shamberger reported that his cancer patients averaged selenium blood levels only 60 to 80 percent as high as non-cancer patients.⁴

Shamberger and Frost found U.S. breast cancer rates were low in areas where selenium crop levels were high, and high where selenium crop levels were low.⁵ Shamberger and Frost also showed soil selenium levels (which dictate food selenium levels) were inversely connected to cancer death rates. Low selenium soil led to 516 deaths per 100,000 people; medium selenium soil led to 450; high

selenium soil led to 430; and very high selenium soil correlated with 392 cancer deaths per 100,000.⁶

Schrauzer and colleagues found that annual selenium intake was strongly inversely correlated with breast cancer deaths in 27 countries studied, with the lowest selenium-intake countries (including the United States) having the highest breast cancer death rates, and the highest selenium-intake countries having the lowest death rate.⁷

A host of animal studies has also shown selenium to experimentally reduce cancer incidence. Harr and colleagues gave four groups of mice a carcinogen (FAA) for 210 days. Group I had no selenium added to its diet. Group II had 0.1 PPM (parts per million) added to its diet; Group III received an extra 0.5 PPM and Group IV had 2.5 PPM selenium added.⁸

After 210 days, 80 percent of Group I and II mice developed cancer; 10 percent of Group III had cancer; and only 3 percent of Group IV (highest selenium) developed cancer.

Schrauzer and Ishmael were able to reduce breast cancer incidence in female mice spontaneously prone to develop breast cancer from 82 percent in the control mice to 10 percent in the mice given 2 PPM selenium in their drinking water.⁹

Selenium vs. Cancer: Toxicity Concerns

The experimental animal cancer studies raised a major concern: the most powerful selenium anticancer effects often occurred at doses not far below the potentially toxic doses. This led researchers to study the biochemistry of selenium's anticancer action in more detail, in an attempt to surmount the potential toxicity problem.

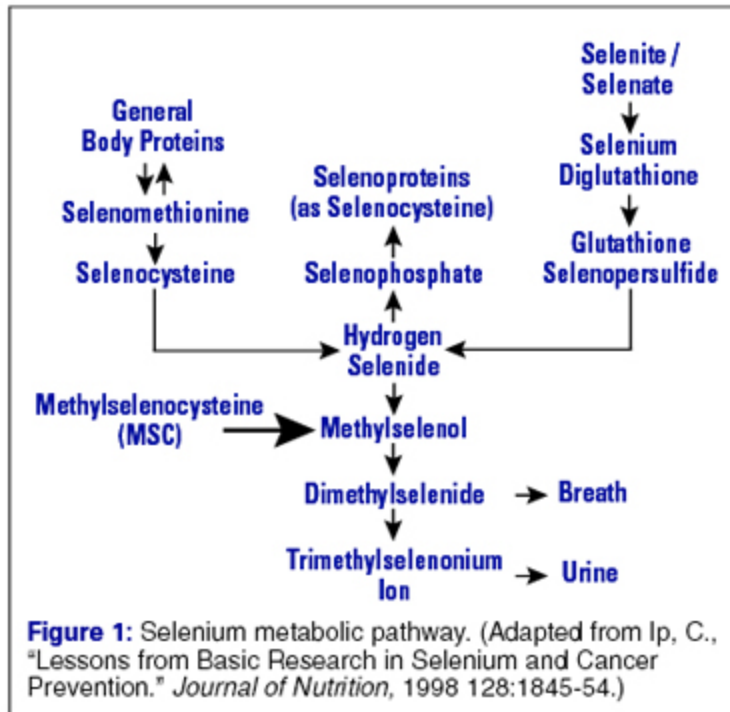
In the early years of selenium-cancer research, it was assumed that selenium provided anticancer protection through enhancing cell and liver antioxidant defenses (glutathione peroxidase) or through promoting enhanced detoxification of environmental carcinogens (glutathione S-transferase).¹⁰ Yet research from New Zealand in 1999 indicated that the selenoprotein enzymes were typically saturated (activity maximized) at dietary intakes of only 90 mcg selenium per day, far below optimal anticancer doses.¹¹ A landmark U.S. study by L. Clark and colleagues found that a 200 mcg daily supplement of selenium from yeast given for four years or more led to a significant reduction in cancer deaths and incidence of prostate, lung and colorectal cancer compared to placebo, with prostate cancer levels reduced more than 60 percent.^{12,13} The diet provided an average 85 mcg of selenium, enough to saturate selenoprotein enzymes without further supplementation. Clearly other selenium effects than optimizing selenoprotein enzymes were at work.

Selenium: One Mineral, Many Forms

Throughout the 1980s and '90s researchers discovered that different forms of selenium had different effects in the body. Inorganic forms, such as selenite/selenate, were more effective at fighting cancer than the commonly used organic form, selenomethionine, yet selenomethionine was more effective at increasing selenium tissue levels and glutathione peroxidase activity.^{14,15}

By 1998 a major selenium/cancer researcher, Clement Ip, was able to explain the different metabolic pathways taken by selenite/selenate and selenomethionine (Fig. 1).¹⁶ Because cells cannot distinguish selenomethionine from the essential amino acid methionine, some selenomethionine became incorporated into general body

proteins, increasing tissue selenium levels.



Yet the selenomethionine general proteins have no anticancer activity. Selenite/selenate are more frequently metabolized to the toxic metabolite hydrogen selenide (H₂Se). Hydrogen selenide does have anticancer effects,¹⁷ but it is more toxic than selenomethionine.³ Its primary mode of killing cancer cells (and at high levels, normal cells as well) is through a process called "necrosis."^{17,18}

In necrotic cell death, cells disintegrate in a way that ruptures the cells' DNA and membranes, and this may trigger an inflammatory reaction which can spread to nearby cells. Necrosis is a messy and toxic way to kill unwanted cells (i.e., cancer cells). Since far less selenomethionine ends up as hydrogen selenide, it is both less toxic, but also less anticancer than selenite/selenate.

Methylselenocysteine (MSC)

Selenium anticancer research of the past 15 years has focused heavily on a novel form of selenium: MSC. A relatively simple organic selenium compound, MSC is formed naturally in various plants, including garlic, wild leeks, onions and broccoli grown on high selenium soil,¹⁹ and methylselenocysteine rich foods have shown good anticancer activity, without excess tissue accumulation or toxicity.²⁰

Methylselenocysteine is easily converted to methylselenol (Fig. 1) by an enzyme, beta-lyase, widely distributed in the body.² Methylselenol has been shown to be an effective anticancer form of selenium,^{2,21} that kills cancer cells through a process of "programmed suicide," called "apoptosis." Apoptosis is a generally controlled, orderly, careful process of cellular self-destruction that can be triggered by various stimuli, including methylselenol.²

Cancer cells killed by apoptosis don't leave a messy residue to trigger inflammation and spread cell death, as occurs in necrosis. Methylselenol is also known to inhibit angiogenesis in beginning cancer tumors.¹⁷ Angiogenesis, the creation of new blood

vessels, is necessary for cancer cells to grow into a tumor, because cancer cells need a far greater blood supply than normal cells to survive.¹⁷

Methylselenocysteine: The Nontoxic Anticancer Selenium

Once methylselenol is formed from MSC, it is soon transformed into dimethylselenide, which is rapidly excreted in the breath.² Any dimethylselenide not so excreted is converted to trimethylselenonium, which is thoroughly excreted in the urine.²

Thus, since MSC goes directly into the methylated selenium pathway, it cannot easily build up to toxic levels in the body. And because it is directly converted in cells to the preferred apoptotic selenium form methylselenol, methylselenocysteine has a powerful and safe anticancer effect.^{2,17,21}

As Medina and colleagues concluded: “[Selenium] methylselenocysteine ... holds promise as a true second-generation selenium chemoprevention [of cancer] compound because of its superior in vivo efficacy, virtually nonexistent toxicity, low body accumulation, and simple formulation.”² Methylselenocysteine represents the safest and most effective anticancer form of selenium available today.

Methylselenocysteine: Safe Use

In their cancer prevention trial, Clark and colleagues found selenium (as high selenium yeast) safe at both 200 and 400 mcg/day levels, even when taken for years.¹ Various peoples around the world routinely get 400 to 600 mcg selenium daily from their diet without ill effect, although Americans and Europeans typically get 100 mcg selenium or less from their daily diet.

Therefore, without knowledgeable medical supervision, selenium supplement intake, even in the form of safe methylselenocysteine, should be limited to 200 to 400 mcg daily for maximum safety. Nutritionally-oriented physicians may use as much as 900 to 2,000 mcg selenium daily as part of a comprehensive cancer treatment protocol.²²

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NKO™: The Ultimate (Non-Fish) Fish Oil

by James South, M.A.

Neptune Krill Oil (NKO™) is the “new kid on the block” among omega-3 oil products. Yet it has already gone “toe to toe” with the market standard—fish oil caps—in a published double-blind controlled study¹ and soundly beaten the current favorite.

NKO is about to usher in a new era in effective omega-3 (EPA/DHA) supplementation. Derived from Antarctic krill, hardy shrimp-like organisms, NKO has many beneficial differences from fish oil supplements.

The EPA/DHA in fish oils are in triglyceride form, while NKO's EPA/DHA are present as phospholipids, primarily phosphatidyl choline.²

Not only are DHA phospholipids key components of human cell membranes, especially nerve cell membranes,³ but they are far better absorbed than triglycerides. Indeed, the soy-derived phospholipid lecithin is often used to emulsify other lipid compounds to enhance their digestive absorption.

NKO is also amazingly stable, strongly able to resist turning rancid. In the oil stability test, NKO is able to maintain the extremely low peroxide (rancid fat) level of less than 0.05 mEq for over 50 hours at 97.8 degrees C (208 degrees F),⁴ while fish oil goes rancid within 8 hours at 80 degrees C (176 degrees F).⁵ This is a major point in NKO's favor, since the notorious proneness of fish oil caps to turn rancid (EPA and DHA are the most rancidity-prone of all polyunsaturated fatty acids) has always been the main drawback to their safe use.

NKO: Unique Antioxidant Profile

NKO contains a unique combination of antioxidants integrated into its phospholipids structures. Small amounts of vitamins A and E, along with the powerful carotenoid antioxidant astaxanthin, help provide the unique stability to NKO.⁶ A novel flavonoid, the first ever found outside the plant kingdom, also enhances NKO's stability.⁶

Brunswick Laboratories of Wareham, Mass., performed a series of tests to measure NKO's antioxidant power and compare it to other nutrients. Using the standard ORAC (oxygen radical absorbance capacity) analysis, which compares the antioxidant power of a test substance to a reference standard (Trolox, a synthetic vitamin E analog), the lab found vitamins A and E have ORAC values of 1.25; lutein and fish oil, 8; CoQ10, 11; astaxanthin, 51; and lycopene, 58. NKO has an amazing ORAC value of 378!⁷

Thus, not only is NKO superstable, but it's a superantioxidant. And since the multiple antioxidants that create NKO's super ORAC value are integral to its DHA/EPA phospholipids, it is reasonable to assume that NKO's antioxidants will increase the stability of whatever membranes they become part of.

NKO Reduces Animal Skin Cancer

Ninety-six Nude Congenic mice were treated with either NKO or placebo by various routes. They were exposed to both "normal" and "high" levels of skin cancer-promoting UVB radiation. Comparing 48 NKO mice to 48 placebo mice after 20 weeks (including both high and normal UVB exposure), 16.7 percent of the NKO mice developed skin cancer, 27.1 percent developed premalignant lesions, and 56.3 percent remained normal.

Of the placebo mice, 37.5 percent developed cancer, 20.8 percent had premalignant lesions, and 41.7 percent remained normal. Looking just at the mice that received "normal" UVB exposure, 13.3 percent of the NKO mice developed cancer, 6.6 percent had premalignant lesions and 80.1 percent remained normal. Of the placebo mice, 26.7 percent developed skin cancer, 20 percent had premalignant lesions, and 53.3 percent remained normal.⁸ Clearly NKO proved effective at reducing UVB-induced skin cancer.

NKO: Clinical Trials

In 2003 the results of a clinical trial testing NKO against fish oil to manage premenstrual syndrome (PMS) and dysmenorrhea (painful menstruation) were published.¹

Conducted at medical centers in Quebec, Canada, 36 women were given NKO and 34 received fish oil over a 90-day trial in a double-blind study. Questionnaires assessing 10 different symptoms on a 0-to-10 scale (0 = no problem, 10 = unbearable) were filled out at day 0, day 45 and day 90 of the trial.

Two grams of NKO or fish oil were given daily the first month, then two grams daily for eight days before and two days after the onset of menstruation during the second and third months.

By day 45, the NKO women had highly statistically significant reductions in symptom severity for all 10 symptoms: breast tenderness, feeling overwhelmed, stress, irritability, depression, joint pain, weight gain, abdominal pain, swelling and bloating. The fish oil group had statistically significant reductions only in weight gain and abdominal swelling at day 45.

By day 90 the NKO group had achieved further reductions in severity score for all 10 symptoms, while the fish oil group had statistical improvements only in weight gain, abdominal pain and swelling. The NKO final scores for those three parameters were lower than each of the corresponding fish oil scores.

The study also measured analgesic (pain killer) use of the 70 women. At baseline, women in both groups took an average of 1.2 grams ibuprofen or 2.5 grams acetaminophen daily during PMS/menstruation.

By day 45, the NKO group took only 0.9 grams ibuprofen or 1.5 grams acetaminophen. The fish oil group took on average 0.9 grams ibuprofen or 1.65 grams acetaminophen by day 45.

By day 90, the NKO group took 0.6 grams ibuprofen (50 percent drop from baseline) or 1.0 grams acetaminophen (60 percent drop). The fish oil group had lowered to 0.8 grams ibuprofen (33 percent drop) or 1.48 grams acetaminophen (41 percent drop) by day 90. Sixty-four percent of the fish oil group complained of unpleasant fishy taste/odor regurgitation, while none of the NKO group had that problem.

An added plus to the study findings was that "...the NKO subjects reported an increase of alertness, energy, and well-being."¹

Acute Toxicity Study

An acute toxicity study with NKO was conducted for two months. Twenty-two men and women, ages 25 to 53, took two grams NKO three times daily. An exhaustive series of laboratory tests measuring more than 20 parameters was analyzed by an independent laboratory. No noticeable physical or laboratory adverse events occurred. There were significant reductions in total blood cholesterol, triglycerides and LDL cholesterol, with a significant increase in HDL ("good") cholesterol.

Study subjects reported such benefits as increased ability to concentrate, decreased seasonal allergy symptoms, increased skin hydration, improved hair texture, decreased joint discomfort and reduced PMS symptoms.⁹

NKO vs. Hyperlipidemia

One hundred twenty patients with mild to severe hyperlipidemia (high blood cholesterol/triglycerides) were studied in four groups of 30 for three months in a randomized, controlled study.

Thirty received 1.5 grams NKO once daily, 30 received 3 grams NKO once daily, 30 were given 3 grams fish oil once daily, and 30 received 1.5 grams placebo once daily. At baseline and 90 days later cholesterol, triglycerides, LDL and HDL levels, and cholesterol/HDL ratios were measured. At the end of 90 days, all parameters had worsened in the placebo group.

Among the fish oil group, total cholesterol had dropped 1.8 percent, triglycerides dropped 4.4 percent, LDL dropped 2.5 percent, HDL increased 4.7 percent, and the cholesterol/HDL ratio improved 5.8 percent.

Among the 1.5 gram NKO group, total cholesterol dropped 13.6 percent, triglycerides fell 11.5 percent, LDL dropped 33.9 percent, and HDL rose 43.5 percent. The cholesterol/HDL ratio improved 13.4 percent.

Among the 3 gram NKO group, total cholesterol dropped 17.9 percent, triglycerides fell 27.4 percent, LDL went down 22.8 percent, and HDL rose 36.4 percent. The cholesterol/HDL ratio improved 19.3 percent.¹⁰ Thus, even the 1.5 gram NKO dose was dramatically more effective in improving blood lipid profiles than the 3 gram/day fish oil dose.

NKO: Final Comments

NKO has been shown to be a safe and more effective EPA/DHA source compared to fish oil, even though it has a lower percentage of EPA/DHA (24 percent, compared to 30 to 50 percent for most fish oils).

The combination of phospholipid-enhanced absorption and superantioxidant protection of NKO's omega-3 oils doubtless makes up for that difference.

Because NKO is derived from a seafood, those known to be seafood-allergic should use NKO only with caution, or after suitable allergy testing. Those taking anticoagulant medication, or who suffer from extreme bleeding conditions, should use NKO only with medical supervision.

It is suggested that 1 to 2 grams (2 to 4 capsules) NKO be taken daily for one to two months, then a maintenance dose of 0.5 to 1 gram (1 to 2 capsules) be taken daily, dose depending on health issues and body size.

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Coconut Oil: The Healthy Fat

by James South, M.A.

Coconuts and coconut oil have been staple foods of the people of Asia, Africa, Central America and the Pacific Islands for thousands of years. Among South Sea Islanders, coconuts often provide as much as 34 to 63 percent of total caloric intake.¹ As coconut oil proponent Bruce Fife, N.D. notes: "The early explorers who visited the South Sea Islands in the 16th and 17th centuries described the Islanders as being exceedingly strong, vigorously built, beautiful in body, and kindly disposed.

The Islanders gained a reputation for their beauty, excellent physical development, and good health."² Yet a campaign begun in the United States in the 1980s, spearheaded by the American Soybean Association and the Center for Science in the Public Interest, has succeeded in virtually removing coconut oil from the American food supply, claiming coconut oil is rich in "artery-clogging" saturated fats that (allegedly) cause high blood cholesterol and heart disease.³ A wealth of scientific data shows this to be a classic case of the "big lie" propaganda technique.

Coconut Oil and Cholesterol

A classic study of two remote South Sea Island peoples—the Pukapuka and Tokelau Islanders—found no evidence of high blood cholesterol from a high coconut oil diet. The Pukapukans, who got 35 percent of their calories as fat, mostly coconut oil, had low blood cholesterol levels: an average of 170 mg/dL for men, 176 mg/dL for women.

The Tokelauans, who got over 50 percent of their calories from fat, mostly coconut oil, had average cholesterol levels of 208 mg/dL for men, 216 mg/dL for women.¹ Blood triglyceride levels, another key heart disease indicator, were also very low, averaging less than 50 mg/dL for both the Pukapukans and Tokelauans. The study authors concluded: “Vascular disease is uncommon in both populations and there is no evidence of the high saturated fat intake having a harmful effect in these populations.... Certainly, there is no reason based on this report, to alter the diet patterns of coconut eating groups in order to reduce coronary risk.”¹

Other studies have found similar results. Comparing a soybean oil vs. coconut oil diet, Mendis and Kumarasunderam concluded that “[coconut oil] saturated fatty acids have a neutral effect on serum cholesterol.”⁴ Sircar and Kansra concluded that “...there are numerous research data now available to indicate that the sole use or excess intake of [polyunsaturated] vegetable oils are actually detrimental to health and switching to...traditional cooking fats like ghee, coconut oil and mustard oil would actually reduce the risk of dyslipidaemias [high blood cholesterol/triglycerides], AHD [atherosclerotic heart disease] and Type-2 DM [diabetes mellitus].”⁵ Kumar compared 32 coronary heart disease (CHD) patients from India with 16 age/sex-matched, healthy controls. “Consumption of coconuts and coconut oil was found to be similar in both groups.... The results imply no specific role for coconuts or coconut oil in the causation of CHD.”⁶

Coconut Oil: Nature’s Germ-Fighter

Medium chain triglycerides (MCTs) comprise about two-thirds of coconut oil. The chief fatty acids in these MCTs are lauric acid (48 percent), capric acid (7 percent) and caprylic acid (8 percent).⁷ These fatty acids and their monoglycerides are extremely powerful antimicrobial agents, effectively killing a broad range of pathologic bacteria, viruses, fungi/yeasts and protozoa.^{3,8}

It is no coincidence that human and mammalian breast milk are rich in these fatty acids. Studies have shown that breast milk medium chain saturated fatty acids and monoglycerides provide immune protection to babies, whose immune systems are still developing.^{3,8} When nursing mothers include coconut oil in their diet, the level of these fatty acids in their breast milk can triple.

Coconut Oil: The Anti-Fat

Various studies have found that coconut oil MCTs increase “diet-induced thermogenesis.” This is an increase in the rate of burning calories to make heat or energy caused by food intake. Kasai and colleagues compared MCTs to LCTs (long chain triglycerides) from canola/soybean oil. They found that “...in healthy humans, the intake of 5 to 10 gm of MCT causes larger diet-induced thermogenesis than that of LCT...”⁹ St-Onge and coworkers studied healthy but overweight men for 28 days, feeding MCTs or olive oil as the chief fat source. They concluded: “Consumption of a diet rich in MCTs results in greater loss of AT [adipose (fat) tissue] compared with LCTs, perhaps due to increased energy expenditure and fat oxidation observed with MCT intake.

Thus, MCTs may be considered as agents that aid in the prevention of obesity or potentially stimulate weight loss.”¹⁰ Han and coworkers fed MCTs to rats. They found that “MCT-fed animals had smaller fat-pads,” and “A number of key adipogenic [fat-promoting] genes were down-regulated...”

“We also found reduced adipose tissue lipoprotein lipase [the enzyme that pulls fat from the blood into fat cells] activity and improved insulin sensitivity and glucose tolerance in MCT-fed animals.”¹¹ St-Onge and Jones compared MCT to LCT in terms of promoting increased energy expenditure (EE) and reduced food intake in animals and humans. They concluded that “...greater EE and lower food intake with MCT compared with LCT suggest replacing dietary LCT with MCT could facilitate weight maintenance in humans.”¹² It is important to note that all these studies involved replacing other dietary fats with MCTs (coconut oil is two-thirds MCT), not adding them on to an already typical high-fat American diet.

There are numerous other benefits and uses of coconut oil. It makes a great skin or hair conditioner. It's great for cooking, baking, and light frying. It makes the world's best popcorn. The interested reader is directed to references 2, 3 and 8 for further details.

Organic Virgin Coconut Oil

There are basically two grades of coconut oil available: RBD (refined, bleached, deodorized) and virgin coconut oil. RBD oil is subjected to high heat, filtered through clays to remove impurities, and processed with sodium hydroxide to prolong shelf life, due to the lesser quality dried coconut (copra) used to make it. A properly produced RBD oil is an acceptable form of coconut oil. It is a basic, no-frills product.

The ultimate coconut oil is organic, virgin coconut oil. VRP is pleased to now offer Tropical Traditions™ organic, virgin coconut oil. It is produced through a traditional, low heat process from freshly harvested, hand-selected organically grown coconuts.

It is not mass produced, but is made by hand in the rural Philippines, just as it has been done for hundreds of years. The lauric acid content typically runs 50-55 percent. Lauric acid is the most powerful antimicrobial of the MCTs. The oil has a distinct coconut smell and taste. RBD oil is usually bland, with no coconut taste or odor.

For the health food connoisseur, Tropical Traditions organic virgin coconut oil is the ultimate coconut oil. For those who don't like the taste or smell of coconuts, VRP's basic coconut oil is a good alternative.

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Customers' Corner

by Ward Dean, MD

VRP Medical Director

Hypothyroidism & Osteoporosis

Dear Dr. Dean,

I am a 75-year-old male; tall, thin and athletic. Two weeks ago I had to see an endocrinologist. After a detailed history and blood work, he diagnosed a chronic thyroid inflammation (many years ago I did have a bout of thyroiditis). At the same time a bone density test revealed osteoporosis of the hip.

For the thyroid, the doctor prescribed 75 mcg Synthroid® (in this instance he preferred Synthroid to the Armour product).

In addition, he suggested Fosamax® (after reading the insert, I think I'll wait a while with this). For the osteoporosis the doctor prescribed Citracal® Plus D. Should I not be also taking additional magnesium to balance the 1,500 mg. calcium? Also, with regard to the Fosamax, is there a safer alternative? Thanks for your usual terrific suggestions. — Mr. H.

Dear Mr. H.,

What do you mean, "in this instance he preferred Synthroid to the Armour product?" Ask him when he would ever prescribe Armour® Thyroid? I'll bet he never prescribes it. Tell him you want the Armour product. It's cheaper, and I'll bet you'll do better with it. I'd also suggest UniZyme™ to help reduce the inflammatory aspects.

For osteoporosis, I'd recommend a broad spectrum mineral replacement like Essential Minerals (or Advanced Essential Minerals), both formulated based on the recommendations of Dr. Alan Gaby, in his book, Reversing Osteoporosis.

In addition, I'd add extra vitamins D and K, Xylitol (40 gm/day) to stop bone loss, and Strontium to increase bone formation. See my recent article on Strontium (on VRP's website at <http://www.vrp.com/art/1193.asp>). Strontium is far cheaper and more effective than Fosamax. Osteoflavone Complex may also help.

Ward Dean, MD

Herpes, Lupus & BHT

Dear Dr. Dean,

I was diagnosed with Herpes 2 about 10 years ago, and have frequent outbreaks. I was recently diagnosed with Lupus (SLE) and it is active in my kidneys.

I'd like to try the BHT but wanted to make sure it would not affect my Lupus in any way. Could you give me some ideas on dosage and if there is something that I should take with it if is safe to use? — Ms. C.

Dear Ms. C.,

It is always difficult to recommend anything for folks with Lupus, as they respond so differently to many substances. Nevertheless, I think you should give BHT a try. You will probably know immediately whether it is causing you any problems, as most Lupus patients that I know are very sensitive to the effects of drugs and nutrients (both good and bad effects). I suggest following the instructions on the package insert that comes with the BHT. Since BHT is fat soluble, it is not likely that it will effect your kidneys in any way.

Also, if you are not already taking it, I suggest you take DHEA. DHEA has been shown to be very helpful for patients with Lupus. The dosage of DHEA for Lupus patients is much higher than for “normal” people—usually in the range of 50 to 100 mg for women.

Ward Dean, MD

Melatonin and CRP

Dear Dr. Dean,

I'm a 56-year-old man and vegetarian. I have no serious problems except for insomnia. To enjoy good health, I'm taking some of VRP's products such as Extend Core, Coenzyme Q10 (75 mg), Turmeric Extract (150 mg), Melatonin (1.5 mg) and two capsules of UniZyme™.

In an article of yours concerning C-Reactive Protein (CRP), I read that one of the causes of CRP elevation is hormone replacement therapy. Is Melatonin one of those hormones? Does Melatonin have any side effects if taken for a long period of time?

Additionally, can you suggest another product for good health apart from the above mentioned? Thank you for your help. — Mr. K.

Dear Mr. K.,

I don't recall ever saying anything about CRP and hormone replacement therapy (HRT). In fact, I just reviewed my article on CRP and could not find anything about HRT. At any rate, melatonin is not known to adversely affect CRP. The only long-term side-effect of Melatonin taken for a long time that I am aware of is that it may increase your lifespan.

I can certainly suggest a number of products—however, I'd suggest that you select what you think might be best for you based on your personal condition and situation. If you have not been following our newsletter for a long time, I'd suggest reviewing a lot of what has "gone before," by reading the VRP Newsletter Anthologies for 2000 and 2001, and the Anthologies for 2002 and 2003 when they become available.

Ward Dean, MD

Hysterectomy—Nutritional Support

**Dear Dr. Dean,
I'm scheduled for a hysterectomy and was wondering what vitamins would you suggest for after I come home from the hospital?**

I currently take Turmeric, HerBalance I™ and II™, HerBalance Cream™, Lycopene, GLA, CoQ10, LipiControl®, BioDIM®, Detox-FiberPlex and B-Complex.

Do I need to stop taking any of these after my surgery? Also, I have read a lot of articles about problems with infections and adhesions and was wondering if there is a natural approach to treating them?

Thank you for all the advice you have given me in the past. I have taken it to heart and it has helped me greatly! Thank you! — Ms. N.

Dear Ms. N.,

The only substance that I have a question about going into your surgery is Turmeric Extract—and that is because it may prolong bleeding by reducing your fibrinogen levels. However, if your pre-surgical fibrinogen levels are within the normal range, then I would continue to take it, as its anti-inflammatory properties will probably help to speed healing, and reduce the likelihood of adhesions.

I would also recommend a broad-spectrum multinutrient like Optimum 6, as well as additional UniZyme™ after surgery. UniZyme is a powerful anti-inflammatory that should complement the anti-inflammatory effects of Turmeric.

Are you taking thyroid hormone? One of the most common side effects of thyroid deficiency is "female problems." Many of my hypothyroid patients have had hysterectomies. I wonder how many of them could have avoided this surgery if their hypothyroid condition had been addressed in a timely manner. Please read my article on hypothyroidism on VRP's website (<http://www.vrp.com/art/561.asp>).

Ward Dean, MD

Hypothyroid Symptoms

**Dear Dr. Dean,
I have hypothyroidism and am currently taking 112 mcg of Synthroid® and have no energy, am tired all the time, can't lose weight, mind is fuzzy. My**

doctor says that I'm "in range" on my thyroid but I still don't feel right. My height is 6'1" and weight is 270 lbs. Any suggestions?

Also, I can't find the Armour® Thyroid in your catalog. — Thanks, Mr. S.

Dear Mr. S.,

My first suggestion is to find a new doctor—one who will treat you, not your blood test. Contact VRP Customer Service for the name of a physician in your area who will prescribe an adequate dosage of Armour® Thyroid for you.

You can't find Armour Thyroid in VRP's catalog because it is a prescription medication. Your physician will need to prescribe it for you.

Ward Dean, MD

Bipolar Disorder & Cardiovascular

Dear Dr. Dean,

I had been taking prescription lithium for bipolar disorder and had to stop because it was causing my pulse rate to slow down.

I have had two stents put into my heart, and am currently on Lipitor® and Plavix® and nitroglycerin for heart pains. My doctor placed me on Zyprexa® for my bipolar disorder, which helped my pulse return to normal; however, I am now having side effects to this medicine. Can I safely use Lithium Orotate with my heart condition? — G.

Dear G.,

You should be able to safely take Lithium Orotate with your heart condition. I don't know of any side effects with it. The dose that is required for your bipolar condition is a fraction of the prescription dose that you were taking.

However, I don't think you can safely take Lipitor. I believe it is a bad drug. See the information about statin toxicity in the article on CoQ10 in this issue. I suggest a combination of Niacin (about 1,500 mg daily), plus Turmeric (1 gram twice daily). Please see my article on Fibrinogen to gain an appreciation of the importance of Turmeric. (The article is on VRP's website at <http://www.vrp.com/art/463.asp>.)

For the angina, in addition to your nitroglycerin, I suggest CoQ10 (150 mg twice daily). This is especially important if you continue to take Lipitor. Oral ChelatoRx would also be beneficial over the long run. Let me know how you do.

Ward Dean, MD

Niacin and Histamine Release

Dear Dr. Dean,

I am concerned about niacin and the release of histamine because I have urticaria pigmentosa (subcutaneous) and am allergic to all histamine-producing drugs. When I have to take a narcotic, I take a H1H2 blocker to stop

the itching that would result.

Now that I am in the generation using statin drugs, my doctor wants me to take Lipitor® and niacin. From my research it looks like IHN (inositol hexanicotinate) could be an alternative form of niacin for me. Could you please tell me if it releases histamine in any amount and any other information regarding this issue that may be helpful. Thank you in advance. — Mrs. R.

Dear Mrs. R.,

I agree that IHN may be a better choice for you. IHN does not usually result in a histamine release. I strongly urge you to avoid Lipitor or other statin drugs.

You might try Policosanol along with IHN. If the combination of IHN or Policosanol does not normalize your lipid profile, you might consider VRP's LipiControl®, plus CoQ10. LipiControl contains IHN, plus other lipid lowering substances, including Red Yeast Rice Extract (RYRE). RYRE is the natural prototype of the statin drugs, and has a much lower side effect profile than the drugs.

Ward Dean, MD

Elevated Liver Enzymes

Dear Dr. Dean,

After routine blood work at the time of an annual physical, the doctor's office called to report that my son's liver enzymes are elevated, and asked that I have him tested again in three months.

My son has Down's Syndrome, is 43 years old, and the prescribed medications he takes are Synthroid®, 75 mcg per day, and Zanax® 0.5 mg per day at bedtime.

He takes a good quality vitamin-mineral supplement each day. Which of the liver support products from VRP might benefit him at this time? Thank you for providing this service. — Mr. P.

Dear Mr. P.,

I suggest HepatoGen™ plus N-Acetyl Cysteine. Let me know how he does.

Ward Dean, MD

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