

# Vitamin Research News

Dedicated to the Scientific Pursuit of Better Health

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# The President's Desk

## Part II: Is Conventional Medicine (CM) Evidence-Based?

Last month we featured excerpted highlights of a paper titled, "Is Conventional Medicine (CM) Evidence-Based?" by recognized hormone replacement researcher, John Lee MD. This month we continue with Dr. Lee's observations on the qualitative differences between Conventional Medicine (CM) and Alternative Medicine (AM), based on his collection of high quality references that refute the notion that conventional medicine is truly evidence-based. These references focus on hormones and their application to health, which has been the focus of study by Dr. Lee for over 23 years.

- *Conventional Medicine promotes the notion that there is no proof that estrogen causes breast or other cancers, despite evidence showing that estrogen is involved in these cancers, and that estrogen-progestin replacements are even worse.*<sup>16-23</sup>
- *Conventional Medicine doesn't recognize that progesterone protects against breast cancer, or that this benefit can be provided by transdermal application. Conversely, the evidence shows that a deficiency of progesterone leads to a greater than five-fold increase in the incidence of breast cancer. Furthermore, there is good evidence that transdermal progesterone greatly reduces human breast epithelial cell proliferation, the hallmark of impending breast cancer. At least eight different metabolic mechanisms of progesterone are known to be protective against breast cancer. Overall survival of node-positive patients 20 years after breast surgery is 50 percent less in women with low progesterone levels on the day of surgery than in women with normal progesterone levels on the day of surgery.*<sup>24-29</sup>
- *Conventional Medicine believes that "normal" hormone levels are, in fact, healthy. The truth is that laboratory ranges of hormone levels merely represent statistically common levels found in a certain population. When US ranges of estrogen are compared to world-wide ranges, estrogen levels in the US and industrialized nations are abnormally high prior to menopause. Experts, such as Dr. Peter Ellison, believe that this is a major factor in the higher breast cancer incidence found in industrialized countries.*<sup>30</sup>
- *Conventional Medicine has long argued that birth control pills are not a significant cause of breast cancer. The evidence is that oral contraceptives prior to 1976 significantly increased the risk of breast cancer among women with a strong family history (sisters and mothers) of breast cancer.*<sup>31</sup>
- *Conventional Medicine believes that oral contraceptives do not cause cervical dysplasia. The evidence is that they do.*<sup>33</sup>
- *Conventional Medicine accepts the fact that estrogen is the only known cause of endometrial cancer but doubts that progesterone will prevent endometrial cancer. The evidence is that oral progesterone (200 mg/day) prevents endometrial cell proliferation and atypical hyperplasia in postmenopausal women receiving estrogen therapy. There is good evidence that topically applied progesterone does the same thing.*<sup>34-36</sup>

- *Conventional Medicine believes that estrogen falls essentially to zero after menopause. The evidence is that at least two-thirds of postmenopausal women continue to produce sufficient estradiol years after menopause. The problem is that, in the absence of sufficient progesterone, sex hormone binding globulin (SHBG) rises and inhibits estrogen function.*<sup>37</sup>
- *Conventional Medicine believes that serum levels of hormone are accurate measurements of bioavailable hormone. The evidence is that serum levels are irrelevant unless one also knows the concurrent concentration of sex hormone binding protein (SHBG) and can somehow calculate its inhibiting effect.*<sup>37-39</sup>
- *Conventional Medicine believes that low endogenous estrogen after menopause is the cause of osteoporosis. The fact is that bone mass begins to fall at age 36 in US women, a time when estrogen levels remain high. Even after menopause, the evidence is that two-thirds of US women produce sufficient endogenous estrogen for optimal estrogen bone benefit. The problem is that, when progesterone is deficient, SHBG levels rise and inhibit estrogen bioavailability. Among 107 postmenopausal women who are truly estrogen-deficient and given estradiol in doses of 1 mg, 0.5 mg, or 0.25 mg, the 0.25 mg/day dose is optimal for inhibiting bone resorption. Conventional Medicine customarily prescribes 0.5–2 mg doses, which are 2-8 times higher than needed.*<sup>37,40</sup>
- *Conventional Medicine prescribes estrogen supplements to postmenopausal women in the belief they need it to prevent memory loss and decline in cognitive function. The evidence is that estradiol does help brain function but the optimal concentration, measured as the “free” non-protein-bound fraction, is often within the range of that found in postmenopausal women, and not at the higher levels found in premenopausal women. Serum levels that do not distinguish between protein-bound and “free” estradiol are irrelevant. Estrogen supplements are needed only in those women who are truly estrogen deficient!*

Thank you Dr. Lee! More to come next month.

**Robert Watson**  
President/CEO

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## A Nutritional Approach: **Controlling Seizures**

by Ward Dean, MD

Seizures can be attributed to a number of causes, including metabolic abnormalities, infections, nutritional deficiencies, or trauma. Emotional stress also increases the frequency of seizures. But most seizures occur due to unknown reasons.

In the 1920s, before anti-convulsant medications were available, high fat diets were used to control seizures in epileptics. Clinical trials are now confirming that high-fat diets work “better than any other regimen” according to Dr. John M. Freeman, Director of the Pediatric Epilepsy Clinic at Children’s Center in Baltimore, Maryland. He recommends a stringent diet consisting of high fat, low protein, and low carbohydrate foods. Some experts estimate that this diet can lead to a 50 to 70 percent reduction of seizures—a record that few drugs can claim. Dr. Freeman has written a book titled, *The Epilepsy Diet Treatment: An Introduction to the Ketogenic Diet*, Demos Publications, 1994, New York (Maltz, 1994).

Gamma-aminobutyric acid (GABA) —the brain’s major inhibitory neurotransmitter—tends to occur in lower than normal levels in seizure-prone rats<sup>1</sup> and humans with epilepsy.<sup>2</sup> Seizure-prone preeclamptic patients (hypertensive condition during late pregnancy) also have decreased brain GABA concentrations.<sup>3</sup> Brain GABA levels depend on both zinc and vitamin B6. Zinc is involved in the maintenance of pyridoxal phosphate concentrations by the activation of pyridoxal kinase. Pyridoxal kinase is important in decarboxylation, and lack of this enzyme results in lowered brain levels of GABA. Consequently, zinc deficiency may increase the risk of pre-eclamptic seizures by reducing brain GABA concentrations and by lowering the seizure threshold. Unfortunately, plasma pyridoxal phosphate measurements alone do not appear to accurately reflect vitamin B6 status or true tissue pyridoxal phosphate levels.<sup>3</sup>

Glutamate concentrations in the brain are higher in some seizure patients, and these concentrations can increase to potentially neurotoxic concentrations during seizures. Thus, it appears that a rise in brain glutamate may precipitate seizures. These concentrations may reach levels capable of causing cell death.<sup>2</sup> The importance of relative concentrations of glutamate, gamma-aminobutyric acid, and pyridoxal-5-phosphate with respect to seizures is illustrated by a 33-month-old male seizure patient whose cerebro-spinal fluid glutamate levels were 200 times normal! When he was given vitamin B6 at a dose of 5 mg/kg body weight per day (350 mg), his EEG normalized and his seizures stopped—but the CSF (cerebral spinal fluid) glutamate concentration was still 10 times normal. With a higher dose of B6 (10 mg/kg bw/d-700 mg), the CSF glutamic acid normalized. These results indicate that the optimal dose of B6 for epileptics should be the dose that normalizes CSF glutamate levels—not just the control of seizures.<sup>4</sup>

Dr. Stephen Lasley<sup>1</sup> found that brains of rats that are genetically prone to seizures also have reduced levels of taurine as well as increased levels of aspartate. Therefore, I believe that avoidance of aspartame should be a key element in an anti-seizure diet. Also, taurine, in doses of 1 to 3 grams per day may be helpful.

In addition to vitamin B6, magnesium and dimethylglycine have also frequently resulted in a rapid—sometimes overnight—appearance of speech in formerly non-speaking autistic children. Magnesium, vitamin B6 and dimethylglycine all have strong anti-seizure properties and can be effective even when other anti-seizure medications fail.<sup>5</sup> The deficiency of another member of the B-complex—B1—has also been reported as a cause of epileptic seizures.<sup>6</sup>

Vitamin E has been helpful in patients with complex partial seizures, which are often resistant to drug therapy, and may compensate for vitamin E deficiencies induced by anticonvulsant medications. Dr. Sheldon Levy<sup>7,8</sup> believes that vitamin E, although not an anticonvulsant or an antiepileptogenic agent, plays a useful role in anticonvulsant therapy as an adjunctive therapy which compensates for anticonvulsant-induced vitamin deficiencies.

Carnitine is an amino acid that is excreted in large amounts when anti-seizure medications like valproic acid (Depakote™) or Tegretol™ are taken. Depakote is a very effective antiepileptic drug but has limited use due to risk of fatal hepatotoxicity. The hepatotoxicity is probably due to valproate-induced carnitine deficiency. Carnitine transports long chain fatty acids into the mitochondria. Valproic acid treatment results in a reduction of free carnitine levels. Carnitine is supplied both by the diet and by endogenous biosynthesis from lysine. Carnitine's primary metabolic role is to transport 12-20 carbon long-chain fatty acids into the mitochondria where they are catabolized to acetyl-CoA for synthesis of mainly citrate and acetoacetate. Carnitine also is involved in a variety of fatty acid and organic acid transacylation reactions, where the acyl moieties of acetyl-CoA esters are transformed to or from carnitine.

There are four metabolic actions of carnitine that have been utilized as therapeutic rationales: (1) to correct an absolute relative carnitine deficiency, (2) to enhance fatty acid oxidation, (3) to accept and shuttle unmetabolized acyl groups from the mitochondria and (4) to increase levels of free unesterified coenzyme A and thereby increase the intracellular free-CoA/acetyl-CoA ratio, an important regulator of enzyme activation/deactivation.<sup>9</sup> Carnitine supplementation is effective in reducing valproic acid-associated hyperammonemia.<sup>10</sup> Recommended dosages for carnitine replacement are 50-100 mg/kg/day in children, and 1 to 3 gm per day for adults in 2 or 3 divided doses.<sup>11</sup>

In many cases of epilepsy, there is an association with celiac disease and cerebral calcifications. Gluten-free diets—a mainstay in the treatment of celiac disease—often reduce the incidence of seizures—especially if the diet is started soon after the onset of seizures. The efficacy of the gluten-free diet in epilepsy appears to be inversely related to the duration of epilepsy before the diet, and to the age at the beginning of the diet.<sup>12</sup> The possibility of celiac disease should be investigated in all cases of epilepsy—especially if cerebral calcifications are identified.

In this regard, Dr. A. Ventura<sup>13</sup> reported on 2 females—5 and 23 years old—who had focal occipital epilepsy with cerebral calcifications and who were not responding well to anti-epileptic therapy. Both females also had celiac disease as well as documented folic acid deficiency. It is well known that antiepileptic drugs may induce a folate deficiency. The patients were placed on gluten-free diets with supplementary folic acid (dosage unknown). This led to complete normalization of the EEG in the five year old and a cessation of seizures. The 23 year old's EEG improved significantly and seizure frequency was reduced. Folic acid levels returned to the normal range within several months. This report suggests that there is an association between folic acid deficiency and neurologic diseases such as epilepsy. Dr. Ventura believes that the mucosal abnormalities of celiac disease may have caused the folate deficiency, which precipitated the seizures.<sup>13</sup> The causative relationship of cerebral calcifications to seizures is unknown, but this may be a condition that may be helped by EDTA chelation therapy. EDTA chelation is probably the treatment of choice for metastatic calcification in any tissue. Whether resolution of cerebral calcification would help in reducing seizures is unknown, but it certainly wouldn't hurt.

Magnesium sulfate is standard therapy for pregnancy-induced hypertension (eclampsia and pre-eclampsia) to prevent seizures. 10 gm of magnesium are administered intramuscularly initially, followed by 5 gm intramuscularly every 4 hours. If administered intravenously, a 6 gm bolus over 15 minutes is given, followed by 1 to 3 gm per hour. In a comparative study, Dilantin™ was compared to magnesium in preventing seizures and reducing blood pressure. The investigators found no differences in the patients' tolerance, adverse reactions or outcomes between the 2 groups. The authors then made the amazing conclusion that Dilantin "is a well tolerated alternative to magnesium sulfate for seizure prophylaxis in patients with mild pregnancy-induced hypertension."<sup>14</sup> My question is, "what about magnesium as a well-tolerated alternative to Dilantin?"

Seizures may also result from glutathione peroxidase deficiency, which could be from lack of bioavailable selenium.<sup>15</sup> Selenium supplementation in children resulted in a reduction in seizures and improvement in EEG recordings after 2 weeks. Selenium is important in the formation of glutathione peroxidase, which may play a role in protecting neuronal cells against oxygen radicals and peroxidative damage. Selenium deficiency in the brain of patients with epilepsy may be an important triggering factor for the origin of intractable seizures and subsequent neuronal damage.<sup>16</sup>

Recently, a colleague related a story of a patient with a history of multiple, intractable, daily grand mal seizures for over 50 years. Because of the frequency of her daily seizures, the patient has been unable to attend school, and is illiterate. She was treated with pregnenolone, with immediate and near-total resolution of her seizures, being reduced in frequency from several each day to less than one per month. She repeats over and over that pregnenolone has finally given her a life. Although this anecdotal report is without precedent or confirmation, pregnenolone certainly seems to be worth trying. I recommend starting with 10 mg each morning for one month, increasing the dose to 30 mg, then to 100 mg, at monthly intervals.

In summary, for seizure disorders I recommend using a nutritional "shotgun" therapy, which includes:

<b>Magnesium</b>	500-1,000 mg/day
<b>Selenium</b>	100-200 mcg/day
<b>Taurine</b>	1-3 gm/day
<b>L-Carnitine</b>	1-3 gm/day
<b>GABA</b> (gamma-amino-butyric acid)	500-1,000 mg/day
<b>Vit. B complex, w/special emphasis on</b>	
• <b>B1</b>	50-100 mg/day
• <b>B6</b>	200-500 mg/day
• <b>Folic Acid</b>	400-1,000 mcg/day
<b>Vitamin E</b>	400-800 IU/day
<b>DMG</b> (dimethylglycine)	50-200 mg/day
<b>Pregnenolone</b>	100-500 mg/day

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# Protecting Memory and Enhancing Cognitive Function

by **Jim English**

The human brain is the most elegant —and complex—structure ever devised. Comprising some ten billion neurons and supportive cells, no other organized structure—organic or silicon— can begin to match the sheer complexity and processing power of the human brain. It regulates virtually all life systems while simultaneously generating the thoughts, dreams and feelings that define us and shape our perception of reality. Every thought, concept, opinion, belief and emotion arises from the untold millions of chemical and electrical reactions that occur in the brain every second. And to power all of this activity the brain places a huge demand on the body's energy reserves. Though it accounts for a mere two percent of body's weight, the brain greedily consumes more than twenty percent of the body's available energy in the form of oxygen and glucose.

## **The Aging Brain**

Time takes a terrible toll on the human brain. Many tissues, such as the skin and liver cells, have the capacity to regenerate, but this trait is not shared by brain cells— once a brain cell is lost, it is gone forever. By age seventy most people lose ten percent of their original brain cells to the effects of "normal" aging. This continual loss of brain cells is further aggravated by damage from other age-related conditions, such as hypertension, arteriosclerosis (hardening of the arteries), diabetes, and cerebrovascular diseases (CVD) such as cerebrovascular insufficiency, strokes and multi-infarct dementia (MID).

## **Dementia**

Dementia is defined as the loss of cognitive or intellectual functions. Unlike occasional forgetfulness, dementia is marked by a profound impairment of memory as well as the loss of additional, complex abilities required for problem-solving, decision making, spatial orientation, and even the ability to put simple words together to communicate. Dementia is a permanent, progressive disease that mostly affects the elderly, who, over time may lose the ability to function normally and require round-the-clock care. It is estimated that up to 8 percent of all people over 65 suffer from some form of dementia, and that the number of cases doubles with every additional five years, leading to the estimate that anywhere from 20 percent to 50 percent of people in their 80s suffer from dementia. There are close to fifty different causes of dementia, including neurological disorders (Alzheimer's disease), vascular disorders (multi-infarct disease), inherited disorders (Huntington's disease), and infections (viruses such as HIV).

A common factor shared by all of these disorders is the reduction in the flow of blood and oxygen to the brain. Reduced blood flow, aside from starving brain cells of needed fuel, also increases the production of free radicals that further damage cell membranes and accelerate brain cell death. As the number of lost brain cells grows—from the ravages of age or the debilitating effects of degenerative diseases such as Alzheimer's—mental deterioration continues. Memories begin to fade and the ability to form new thoughts and solve problems is further reduced. Depression, incontinence, disorientation, speech disturbances, tremor, muscle weakness, tinnitus (ringing in the ears), and loss of both visual acuity and coordination also increase as the conditions progress.

### **Alzheimer's Disease**

Alzheimer's disease (also called "senile dementia of the Alzheimer type") is a chronic and progressive degenerative neurological condition. Alzheimer's currently afflicts over four million people in the United States, and accounts for up to 60 percent of all cases of dementia. Alzheimer's commonly appears after age fifty, and from age sixty-five on, the risk of developing the disease doubles with every additional five years of age. As if these numbers weren't bad enough, they are expected to almost double in the coming decades, placing a further drain on health care resources, and leaving almost no family untouched.

While there is currently no cure for Alzheimer's disease, exciting new research shows that several nutrients may help to halt the destructive progression of dementia and improve cognitive function of patients suffering from Alzheimer's and other forms of dementia.

### **Hope for Aging Brains**

While medical researchers and pharmaceutical companies race to patent new (and profitable) treatments for Alzheimer's and other degenerative brain conditions, a number of existing nutritional compounds have already been proven to safely support healthy brain function while protecting and prolonging cognitive ability. Supported by numerous double-blind controlled trials in Europe and the US, these supplements can slow down the age-related loss of higher-level cognitive functions that can appear in healthy individuals as young as fifty years of age.

### **Huperzine**

Alzheimer's is characterized by the destruction of nerve cells in key areas of the brain devoted to higher mental function. Most noticeable is the loss of presynaptic cholinergic neurons that results in a dramatic decrease in brain levels of acetylcholine, a neurotransmitter involved in memory and intracellular communication. Research has shown that levels of acetylcholine are deficient in the brains of patients with Alzheimer's disease, and what little acetylcholine is produced is quickly broken down by the enzyme, acetylcholinesterase (AChE), contributing to the loss of memory and other cognitive functions.

Huperzine is a nutritional supplement that readily crosses the blood-brain barrier to prevent acetylcholinesterase (AChE) from destroying acetylcholine. By inhibiting AChE and increasing acetylcholine concentrations in the brain, Huperzine A has been shown to be effective in alleviating some of the symptoms associated with acetylcholine deficiencies.

Researchers have demonstrated that patients suffering from Alzheimer's and various other memory disorders gain significant benefit from huperzine, both in terms of memory and life quality. In one study, researchers found that 58 percent of Alzheimer's patients experience significant improvement in both cognitive and memory function when given 200 mg of Huperzine per day. Huperzine's memory-enhancing properties suggest that it may be an effective agent for improving memory and learning in healthy humans as well. These findings suggest that Huperzine not only protects from the effects of Alzheimer's and senile memory deficits, but also provides a unique and exciting supplement for supporting memory in the healthy aging human as well.

### **Vinpocetine**

Vinpocetine is a powerful memory-enhancing nutrient that facilitates cerebral metabolism by improving cerebral microcirculation, stepping up brain cell ATP production, and increasing utilization of glucose and oxygen. Vinpocetine also selectively increases blood flow to the brain, particularly to impaired areas, without affecting blood flow to the rest of the body.

Because of its selective effects on improving cerebral circulation, vinpocetine is often used for the treatment of cerebral circulatory disorders such as memory problems, acute stroke, aphasia (loss of the power of expression), apraxia (inability to coordinate movements), motor disorders, dizziness and other cerebrotic (inner-ear) problems, and headache.

In studies involving 882 patients with neurological disorders ranging from stroke to cerebral insufficiency, vinpocetine was found to confer significant improvements in 62 percent of the patients. In one of the studies, cerebral insufficiency patients were asked to memorize a list of ten words. Without vinpocetine the subjects were able to memorize an average of six words. After a month of treatment the average went up to ten words.

Reactive oxygen species (ROS) are believed to play a crucial role in the neuronal damage occurring in ischemic injury (stroke) and neurodegenerative disorders. In studies designed to examine the antioxidant effects of vinpocetine to prevent the formation of ROS and lipid peroxidation in brain synaptosomes, researchers found that vinpocetine significantly decreased oxidative stress and inhibited ROS formation up to 83 percent. The researchers concluded that the antioxidant effects of vinpocetine contributed to reducing neuronal damage in pathological situations.

### **Pyroglutamate**

Pyroglutamate (2-oxo-pyrrolidone carboxylic acid, or PCA) is an amino acid found in vegetables, fruits, dairy products, and meats. Pyroglutamate is also present in large amounts in the human brain, cerebrospinal fluid, and blood.

Pyroglutamate is known to have a number of remarkable cognitive-enhancing effects. After oral administration, pyroglutamate passes into the brain through the blood-brain barrier and stimulates cognitive functions. Pyroglutamate improves memory and learning in rats, and has anti-anxiety effects in rats.

Pyroglutamate has also been shown to be effective in alcohol-induced memory deficits in humans and, more recently, in people affected with multi-infarct dementia. In these patients, the administration of pyroglutamate brought about a significant increase of attention and an improvement on psychological tests investigating short-term retrieval, long-term retrieval, and long-term storage of memory. A statistically significant improvement was observed also in the consolidation of memory.

In human subjects, pyroglutamate was compared with placebo in a randomized double-blind trial for assessing its efficacy in treating memory deficits in 40 aged subjects. Twenty subjects were treated with pyroglutamate and 20 with placebo over a period of 60 days. Memory functions were evaluated at baseline and after 60 days of treatment by means of a test made up of six memory tasks. The results show that pyroglutamate is effective in improving verbal memory functions in subjects affected by age-related memory decline.

### **Choline**

Choline is a precursor to acetylcholine, a cholinergic neurotransmitter that declines with advancing age. Individuals predisposed to Alzheimer's disease and other dementias, infants and children, diabetics, and athletes (who often have reduced plasma-choline levels after training or competition) may be at increased risk of choline deficiency.

Choline has been shown to have considerable potential for preserving the integrity of neuronal structures and in preventing some of the alterations in the central nervous system during aging. Choline supplementation appears to prevent the age-induced decline of the dendritic network composed of neurons that fire impulses to the cells. Choline increases the number of dendritic spines in the cerebral cortex of old mice and improves the animals' learning performance.

Under conditions of increased demand for acetylcholine production, excess choline availability becomes a limiting factor for acetylcholine synthesis. When this additional, exogenous choline supply is

unavailable, cholinergic neurons are able to use free choline taken from a choline “reservoir” to continue the synthesis of acetylcholine. This process, termed “autocannibalism,” can lead to a decrease in the quantity and quality of membrane in these cells. Indeed, cholinergic neurons’ ability to use this alternative source of choline appears to contribute to their vulnerability in Alzheimer’s disease. Researchers believe defects in choline-metabolism may play a central role in the development of Alzheimer’s disease as defects in choline transport exist in the cells of Alzheimer’s victims. Defects in choline metabolism may also be influential in Down’s syndrome, normal aging, Huntington’s disease, amyotrophic lateral sclerosis, the familial dysautonomias, and the post-polio syndrome.

### **DMAE**

DMAE (dimethylaminoethanol) is a nutrient found abundantly in fish and in human brains. In the brain DMAE is converted into choline, the precursor to acetylcholine. Because acetylcholine conducts nerve impulses within the brain, the increased acetylcholine synthesis seen after DMAE supplementation may improve memory and learning skills, elevate mood, prevent memory loss in elderly adults, and increase physical energy.

Studies suggest DMAE may work by inhibiting choline metabolism in peripheral tissues, causing free choline to accumulate in the blood, enter the brain and stimulate choline receptors. As the immediate precursor to choline, DMAE assists in the building and repair of cell membranes, particularly in the brain and central nervous system.

Animal studies have demonstrated that DMAE stimulates brain neurons and improves working memory performance. In one study, rats treated with DMAE demonstrated significant improvements in remembering how to negotiate a maze. In another study, mice trained to negotiate a maze demonstrated improved memory retention when treated with DMAE.

DMAE’s ability to stimulate acetylcholine synthesis has led researchers to explore its effects in senile dementia and Alzheimer’s. In a promising study, 14 senile dementia patients were treated with DMAE for four weeks. The dosage was gradually increased to 600 mg, three times daily, during the first two weeks, with no adverse effects. Although the patients experienced no improvement in cognitive function or memory, ten of the 14 patients experienced reduced depression, irritability and anxiety and increased motivation and initiative.

### **Phenylalanine**

Phenylalanine is an essential amino acid and vital precursor used by the brain to produce dopamine, epinephrine and norepinephrine, stimulatory neurotransmitters that regulate mood, sex drive, memory, alertness, and learning. L-phenylalanine is also converted, via a separate pathway, into phenylethylamine (PEA), a mood-elevating compound that occurs naturally in the brain.

Phenylalanine has been found useful for alleviating depression. In one study forty depressed patients were treated with L-phenylalanine daily for up to six months. Doses started at 500 milligrams and gradually built up to between 3 to 4 grams daily. Patients also took 100 to 200 milligrams of vitamin B6 daily to enhance neurotransmitter synthesis. Thirty-one patients (77 percent) reported positive benefits, and ten patients (25 percent) reported complete relief from symptoms of depression. The principal investigators reported that those who responded positively did so almost immediately.

### **Ginkgo Biloba**

A number of clinical studies have shown that Ginkgo biloba can protect brain cells from damage caused by free radicals while improving blood circulation and oxygen delivery, particularly through the microcapillaries. In one study, researchers measured a fifty-seven percent increase in blood flow through capillaries within sixty minutes of giving Ginkgo to volunteers. A second study by German scientists involved 60 patients diagnosed with cerebral insufficiency and depression. Patients receiving Ginkgo extract began to show marked improvement after only two weeks, with a significant reduction of many of their symptoms.

In another clinical trial of 166 patients over the age of sixty, researchers found that patients suffering

from cerebral insufficiency showed a significant improvement following three months of treatment, confirming the efficacy of Ginkgo extract in cerebral disorders due to aging.

Researchers have also found that Ginkgo can be especially helpful when given to Alzheimer's patients at the first sign of symptoms. In one published study, German scientists gave a daily dose of 120 mg of Ginkgo to twenty elderly patients exhibiting various early symptoms of dementia. The results were dramatic, and the patients receiving Ginkgo showed impressive improvements on a variety of clinical tests, as compared to patients receiving a placebo.

In one large study published in 1996, German researchers tested Ginkgo extract on a group of 222 patients, aged fifty-five or older, who were diagnosed with mild to moderate dementia caused by either Alzheimer's disease or multi-infarct dementia. Patients were given either 240 milligrams of Ginkgo biloba extract, twice a day before meals, or a placebo, for the duration of the six-month long trial. At the conclusion of the study the researchers reported that patients receiving Ginkgo showed a remarkable overall improvement in their condition, including a 300 percent increase in memory and attention as compared to those receiving the placebo pills. The researchers concluded their report by stating that, in cases of dementia, Ginkgo extract could improve a patient's quality of life while preserving independence and postponing the need (and expense) of full-time care.

### **Ginkgo Biloba and Multi-Infarct Dementia**

The second most common cause of dementia in older people is multi-infarct dementia (MID), a condition that accounts for about fifteen percent of all cases of dementia. Multi-infarct dementia usually affects people between the ages of 60 and 75, and men are more likely to have multi-infarct dementia than women. MID is typically caused by a series of mini-strokes, also referred to as transient ischemic attacks (TIAs), that can occur when an artery in the brain either becomes blocked or ruptures. Strokes are generally caused by high blood pressure, high blood cholesterol, diabetes, and heart disease. Of these causes, the most important risk factor for multi-infarct dementia is untreated high blood pressure. In fact, it is extremely rare for a person to develop multi-infarct dementia without also having high blood pressure.

While these mini-strokes may or may not be noticed at the time, the effect on the brain is the same—brain cells become damaged by a lack of oxygen and die. Over time a series of mini-strokes can begin to destroy substantial portions of the brain that control speech and visual processing.

As with Alzheimer's disease, Ginkgo has been shown to help patients suffering from MID by enhancing memory, alertness and overall quality of life. Additionally, given the underlying disorders that cause blood vessels to rupture, Ginkgo can also benefit patients suffering from MID by restoring elasticity and strength to stiff, weakened blood vessels.

### **Summary**

Baby boomers and aging adults face a loss of cognitive powers and impaired mental functions. Research supports the role of a number of potent anti-aging therapies to slow brain aging and preserve cognitive function. Rather than waiting for signs of an irreversible decline in mental abilities or other, more serious cognitive problems, it would be prudent to take steps to support the brain's ability to heal and self-repair. In short, we can take steps now to slow age-dependent brain cell changes, preserve vital functions, and maintain mental health and vigor.

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## Nutritional focus: Preventing Macular Degeneration, Diabetic Retinopathy and Related Ocular Disorders

# Preserving Vision and Preventing Degenerative Disorders

by Jim English

The structure of the human eye naturally lends itself to comparison to a living camera. Light passing through the lens is focused on the retina, a tightly packed layer of light-sensitive cells (photoreceptors) arrayed across the back of the eye. Millions of photoreceptors, called rods and cones, respond to variations in color and light intensity by transmitting electrical signals to the brain where they are processed to produce what we experience as vision.

The photoreceptors of the retina expend a great deal of energy processing light and require a constant

supply of oxygen, glucose, and other nutrients. Consequently the retina is supported by a dense tangle of blood vessels that supply one of the highest rates of blood flow found anywhere in the body. Unfortunately the retina is an extremely delicate structure that is vulnerable to damage from a number of sources, including oxidative damage from free radicals. The tissues of the retina are also rich in polyunsaturated fatty acids, which are particularly prone to damage from free radicals.

### **Macular Degeneration**

Macular degeneration is a group of disorders that involve the slow destruction of the central region of the retina known as the macula. In ARMD, the macula slowly deteriorates, eventually leading to almost complete blindness of the central visual field and leaving only the very edges of peripheral vision. Most cases of macular degeneration occur in people over age sixty and are referred to as age-related macular degeneration (ARMD). ARMD is a major cause of blindness affecting up to fifteen million people over the age of sixty.

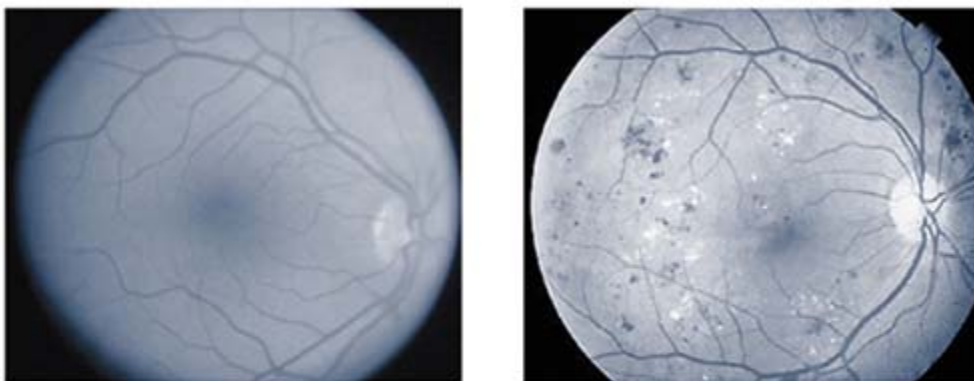
Macular degeneration causes different symptoms in different people, and in its early stages there may be few noticeable changes in vision. Often there is only loss of vision in one eye while the other eye continues to see well for many years. But when both eyes are affected, reading and close-up work can become difficult.

ARMD, like atherosclerosis, is a disease caused by poor circulation. If blood flow is affected by atherosclerosis, diabetes, or any other age-related health problem, the macula slowly atrophies and dies. This process is further hastened by the accelerated production of free radicals that accumulate in the retina when there is reduced blood flow.

Smoking contributes to the progression of ARMD by reducing the supply of blood, narrowing the blood vessels, and thickening the blood. A high-fat, high-cholesterol diet leading to fatty plaque deposits in the macular vessels also hampers blood flow. Additionally, a shortage of antioxidants may also increase the tendency for ARMD.

### **Diabetic Retinopathy**

While people with diabetes are at increased risk of developing serious eye problems, such as cataracts and glaucoma, diabetic retinopathy is the number one vision threat. Diabetic retinopathy is a serious complication of diabetes that damages the small blood vessels of the retina (Fig. 1). Diabetic retinopathy affects half of all Americans diagnosed with diabetes, and if left untreated, about half of those with the advanced form, proliferative retinopathy, become blind within five years, compared to just five percent of those who receive treatment.



**Fig 1.** Healthy retina (left) and retina damaged by diabetic retinopathy (right).

In the early stages of the disease, non-proliferative, or background retinopathy, the small blood vessels of the retina weaken and develop bulges (micro-aneurysms) that can leak blood into the surrounding tissues. Vision is rarely affected during this stage of retinopathy. In the advanced, proliferative stage, impaired circulation caused by damaged and narrowed blood vessels deprives the retina of oxygen. To cope with this problem the circulatory system attempts to maintain adequate oxygen levels by growing new, fragile blood vessels on the retina that can extend into the vitreous (the jelly-like substance inside the back of the eye).

These fragile vessels can rupture and release blood into the interior of the eye, leading to blurred vision or temporary blindness. This results in the formation of scar tissue that eventually pulls the retina away from the back of the eye (retinal detachment), and leads to permanent vision loss. An additional condition called macular edema can occur at any time, causing severe blurring of vision as fluid accumulates around the macula.

While all diabetics are at risk for developing diabetic retinopathy, pregnant women with diabetes are more susceptible and may require dilated eye examinations each trimester to protect their vision.

### **Glaucoma**

According to the American Academy of Ophthalmology, over one million people in the United States are at risk for going blind because they don't know they have glaucoma. Glaucoma is a condition marked by damage to the optic nerves (the bundle of nerve fibers that carries information from the eye to the brain) caused by elevated pressure inside the eye. It is estimated that about fifty million people worldwide suffer impaired vision, if not complete blindness, caused by glaucoma. In the United States, about 300,000 new cases are diagnosed each year, adding to the more than three million cases already on record.

Glaucoma is called the "sneak thief of sight" because it strikes without obvious symptoms. People with glaucoma are usually unaware of it until they have a serious loss of vision. In fact, about half of those who have glaucoma do not know it. Currently, that damage cannot be reversed.

While there are usually no warning signs, some symptoms may occur in the later stages of the disease, such as a loss of peripheral vision, difficulty focusing on close work, seeing halos around lights, and frequent changes of prescription glasses. Unfortunately, though, once the vision is lost, it is gone forever.

African Americans are at a higher risk of developing glaucoma than other racial groups. Others at risk include:

- Anyone with a close relative who has glaucoma;
- Seniors;
- People with diabetes;
- People taking steroid medications for extended periods of time.

### **UV-Induced Eye Damage**

Exposure to ultraviolet (UV) radiation produced by the sun can damage the cornea, leading to a painful condition known as photokeratitis. Ultraviolet radiation also contributes to the development of other serious eye disorders, including cataracts, degenerative corneal changes, and skin cancer around the eye.

UV actually refers to three types of ultraviolet light — UV-A, UV-B, and UV-C. The milder form of radiation, UV-C rays, are normally screened out by the ozone layer and don't present much of an immediate health threat. The more powerful UV-A rays are composed of longer wavelength radiation that causes skin tanning and premature skin aging. UV-A rays can reach the retina, and long-term exposure may greatly increase incidence of macular degeneration. UV-B light, the active, shorter wavelengths of radiation, are responsible for blistering sunburns and skin cancer, and cause the greatest damage to eyes.

Cataracts, caused when the lens becomes cloudy, occur over a period of many years and are a major

cause of visual impairment and blindness worldwide. Studies have implicated UV radiation in the development of cataracts, and have also shown that certain types of cataracts are linked to a history of higher exposure to UV rays, especially UV-B radiation.

Since the human lens absorbs UV radiation, individuals who have cataract surgery are at increased risk of retinal damage from sunlight. And people with retinal dystrophies or other chronic retinal conditions may be at an even greater risk since their retinas may be less resilient to normal exposure levels to begin with.

### **Protecting Your Eyes**

Age-related cataracts, glaucoma, macular degeneration, and diabetic retinopathy are among the leading causes for loss of vision. Unfortunately conventional medical approaches to preserving sight haven't offered much hope for treatment of these blinding eye diseases and about the only option eye specialists can offer is sympathy and "watchful waiting," while documenting their patients' progressive loss of vision. Often laser surgery or radiation treatments are used as a last resort to temporarily stall further loss of vision once the problem has progressed to the point of imminent blindness.

As with many degenerative conditions, the best approach is a course of prevention, combined with nutrients shown in numerous clinical studies to support healthy vision. A number of nutrients have been extensively studied for their ability to treat a wide variety of vision-related conditions by preventing the damage caused by free radical activity and by enhancing the delivery of blood and oxygen to the retina to help repair tissues.

### **Taurine**

Taurine is a sulfur-containing amino acid that stabilizes membranes, protects against free radicals, and is an ocular housekeeper, helping the macrophages in the retina eliminate debris from the photoreceptor cells. Taurine is necessary for the chemical reactions that produce normal vision, and deficiencies are associated with retinal degeneration. Besides protecting the retina, taurine may help prevent and possibly reverse age-related cataracts. When taurine is deficient, physicians often observe retinal decline. In one study of patients with primary open angle glaucoma (OAG) the fluid discharge efficacy almost doubled after treatment with taurine. In addition, taurine counterbalances excessive levels of glutamate and excitatory neurotransmitters and protects ocular tissue against oxidative stress and ischemia.

### **R-Lipoic Acid**

A broad range of free radicals are rendered helpless by lipoic acid, a fat- and water-soluble antioxidant. Lipoic acid has been shown to prevent cataracts and diabetic nerve damage by eliminating oxidative stress, improving glucose utilization, and increasing blood flow to the nerves. In addition, lipoic acid recycles dietary antioxidants like vitamin E and raises glutathione levels. In one group of 45 patients with OAG, visual function and liquid drainage improved in approximately 50 percent of the subjects taking lipoic acid in comparison to controls. R-lipoic acid has been demonstrated to be 2-10 times more effective than lipoic acid. In a study designed to mimic the radiation exposure experienced by astronauts, jet crews and military personnel who have suffered a radiation accident, R-lipoic-acid was shown to protect against radiation-associated protein leakage in the eye lens.

### **Xanthophylls**

Researchers have demonstrated that when eyes are exposed to intense UV light the result is extensive damage to retinal tissues, leading to a loss of photoreceptors, fragmentation of retinal cells, and extensive cell death in the macula. Xanthophylls, such as lutein, zeaxanthin and cryptoxanthin, are carotenoids that help to protect the retina from damaging exposure to sunlight, shielding up to 40 percent of damaging UV rays to help maintain ocular health.

### **Bilberry**

Bilberry extract contains anthocyanosides, potent antioxidants that assist in maintaining the integrity of collagen structures in the eyes reducing leakage of fine capillaries in the retina. Bilberry pigment helps produce visual purple, an important chemical that helps convert light into electrical signals for the brain. Bilberry has traditionally been recommended for reducing eyestrain and improving night vision due to its

ability to enhance vision in low light conditions often encountered by pilots and military personnel. Bilberry also reduces general eyestrain, which makes it particularly beneficial for students, computer operators, and anyone who must use their eyes for long periods without rest.

### **Quercetin**

Quercetin is a plant phytochemical (flavonoid) similar to, and in some ways, more powerful than vitamin C. Quercetin prevents the vascular damage caused by LDL oxidation, shields the eye against free radicals, modulates nitric oxide production and prevents collagen breakdown.

### **N-Acetyl Cysteine**

N-Acetyl Cysteine (NAC) is the pre-acetylated form of the simple amino acid Cysteine. NAC is a powerful antioxidant and a premier antioxidant and immune support substance. In the eye NAC scavenges reactive intermediates, guards against the toxicity of nitric oxide, and lessens oxidant injury.

### **Ginkgo Biloba**

Ginkgo biloba is a potent free radical scavenger that supports healthy vision by preventing free radical damage commonly seen in eye diseases such as macular degeneration. A number of experimental studies suggest that ginkgo extracts are potentially useful for treating retinal damage induced by a variety of disorders. When German scientists tested ginkgo's protective effects on the retinas of twenty-five older people they found that the herb dramatically improved vision in all subjects after only four weeks of treatment. According to the researchers, ginkgo caused a "significant increase in retinal sensitivity."

Scientists speculated that ginkgo extract might also slow the progression of ARMD by increasing blood flow to the retina and by halting the free radical damage to the photosensitive cells. In one double-blind trial twenty volunteers were given either 160 mg of ginkgo extract, or a placebo pill, every day for six months. At the end of the study, the group receiving ginkgo showed significant improvements in their long distance visual focus. There was no improvement in the group receiving the dummy pill.

Ongoing studies also show that the greater the damage to retinal tissues, the more profound an effect the ginkgo has on improving vision. These studies show that ginkgo is not only effective in improving vision but, in cases where the vision is damaged by poor circulation, the damage can be significantly reversed.

Human studies also support the use of ginkgo extract in treating diabetic retinopathy. In one double-blind trial, researchers gave daily doses of 160 mg of a standardized ginkgo extract to a small group of people with mild diabetic retinopathy. After six months, these volunteers had a noticeable improvement of their pre-existing impaired vision. In addition, it is suspected that ginkgo's ability to inhibit the platelet-activating factor (PAF), is involved in protecting eye tissues from retinopathy, since ginkgolide B, a known PAF antagonist, has been shown to reduce experimentally induced retinal lesions in animals.

In 1999, researchers tested the therapeutic effects of ginkgo extract on people with glaucoma. Eleven healthy volunteers were treated with either 40 mg of ginkgo extract, or a placebo, three times daily for two days. By measuring blood flow in the eyes before and after treatment, the researchers found a significant increase in blood flow in the main eye artery in those receiving ginkgo, but no change was noted in the placebo group. The results indicate that ginkgo effectively increased the blood flow in the eyes, which helped lower the intraocular pressure, thereby slowing the progression of the disease.

### **Carnosine**

Carnosine is a naturally-occurring dipeptide that has demonstrated efficacy in treating a variety of ophthalmic conditions, including corneal diseases, cataracts as well as glaucoma and increased intraocular pressure. In 1997, clinical trials were conducted in 109 ophthalmic patients with carnosine-containing eye drops. The results confirmed accelerated healing of corneal erosions, trophic keratitis, post-herpetic epitheliopathy, primary and secondary corneal dystrophy and bullous keratopathy. Most striking, however, was the ability of carnosine to eliminate existing cataracts. Furthermore, carnosine is thought to function as a "molecular water pump." In earlier experiments it was

demonstrated that applying carnosine to the conjunctiva (the membrane covering the eye) caused a decrease in normal intraocular pressure and reduced prostaglandin-induced ocular hypertension (related to glaucoma).

### Conclusion

While sometimes referred to as “windows into the soul,” eyes in fact can reveal much about our general state of health. Many of the nutrients discussed here work together to offer a wide range of health benefits to protect these exquisite structures and help preserve vision—and general health—for a lifetime.

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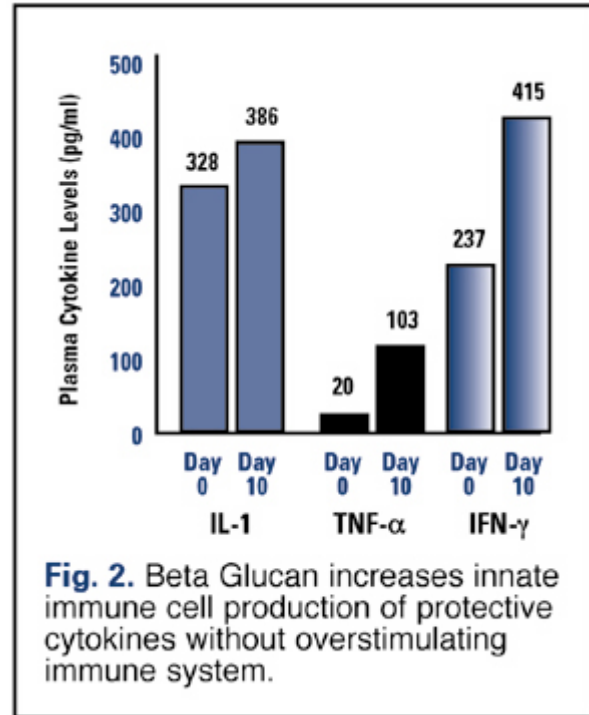
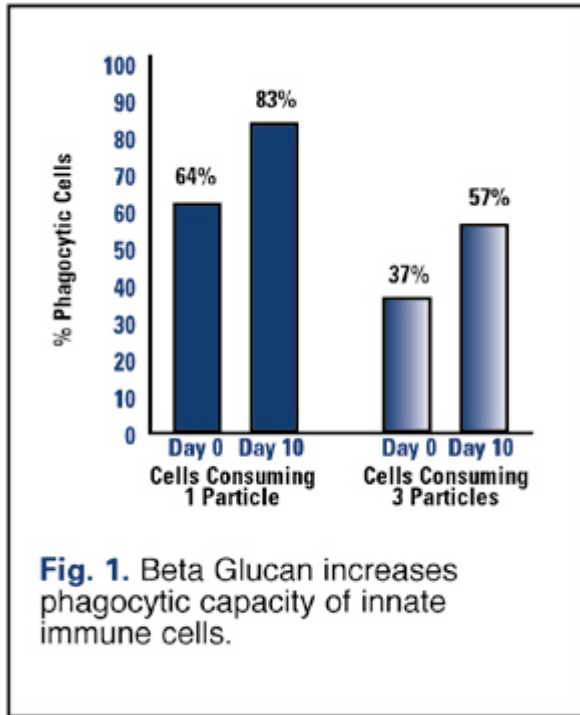
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## New Research

# Beta 1,3-D Glucan Safely Enhances Immune Function

An apple a day may keep the doctor away, but results of a recent human clinical study suggest far better results may be obtained with a dietary supplement called Beta 1,3-D Glucan. Beta 1,3-D Glucan is a highly purified and patented carbohydrate extracted from the cell walls of Baker's yeast.

An independent study conducted by Biophage Pharma Inc., a Canadian biopharmaceutical company, treated normal human volunteers with 250 milligrams of Beta 1,3-D Glucan for 10 consecutive days. The subjects were monitored for 30 days. The results of the human clinical study found that the Beta 1,3-D Glucan—the identical Beta Glucan carried by VRP—was safe and well tolerated, as evidenced by the lack of significant changes in key blood and liver enzymes. It also significantly increased phagocytic capacity—the ability of the innate immune cells to eat and destroy foreign intruders (Fig. 1).



On day ten of the study Beta Glucan had increased the percentage of immune cells able to phagocytose one target particle from 63.8 percent to 83.2 percent. The number of highly phagocytic cells—those consuming at least three particles—increased from 37.3 percent to more than 50 percent. These results show that taking Beta Glucan enhanced the human immune system to defend the body against a challenge.

“The results are consistent with the biological activity of Beta 1,3-D Glucan observed in numerous animal studies,” said Rosemonde Mandeville, Chief Scientific Officer of Biophage Pharma. In addition, researchers also found that Beta 1,3-D Glucan increased plasma cytokines INF-γ and TNF-α, which play an important role in regulating the body’s immune response. There was no significant increase in cytokine IL-1, which can cause fever, chills and muscle aches associated with other immune-enhancing supplements (Fig. 2).

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## New Improved Formula

# Oral ChelatoRx

*Oral ChelatoRx*, VRP’s classic oral chelating formula, has been improved. *Oral ChelatoRx* is a broad spectrum chelator of heavy metals, designed to be used to prevent or alleviate cardiovascular and other aging-related chronic degenerative diseases. The principal active ingredient, ethylene diamine tetraacetic acid (EDTA), is the substance used intravenously by many physicians who perform chelation therapy. Oral EDTA provides similar benefits to IV treatments, as well as unique benefits not shared by the IV treatment. In addition to EDTA, *Oral ChelatoRx* contains a number of other synergistically acting natural chelators, including garlic, chlorella, and malic acid.

The original formula also contained Bromelain, a potent anti-inflammatory, proteolytic (protein-dissolving) enzyme. In the new upgraded formula, Bromelain has been replaced by the enzyme, Serrapeptase. Serrapeptase has been used in Europe to treat arterial blockages due to its ability to alleviate arterial inflammation and dissolve blood clots and arterial plaque, to an even greater degree than bromelain. Serrapeptase has also been used to treat arthritis, fibrocystic breast disease, carpal tunnel syndrome, and other inflammatory conditions. VRP's new *Oral ChelatoRx* contains a unique, enterically coated form of serrapeptase that survives transit through the stomach to be absorbed in the intestines. We believe that the addition of this novel form of serrapeptase provides an even greater level of benefit to this unique formula.

We have also switched to a smaller capsule size, and increased the capsule count to provide a full month's supply in each bottle of *Oral ChelatoRx*. For maximum efficacy, we recommend that a broad-spectrum mineral replacement formula (like *Essential Minerals* or *Advanced Essential Minerals*) be taken when undergoing intravenous or oral chelation therapy.

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## New Improved Formula

# Polysorbate 80

Polysorbate 80 became popular based on its reputation for topically stimulating hair growth in men with male pattern baldness. Used as a shampoo surfactant and a food additive emulsifier, it is often found in mayonnaise, salad dressings, and some liquid detergents. Durk Pearson and Sandy Shaw popularized the use of polysorbate 80 for alopecia in their best-selling book, *Life Extension: A Practical Scientific Approach*. In their book, Pearson and Shaw report that use of polysorbate 80 in studies has resulted in an average of 60 percent hair regrowth after just six months of daily use.

### **Histamine-Releaser**

Although histamine is usually considered the culprit behind seasonal allergies, in actuality this substance is essential for cell growth and reproduction. In fact, since histamine is a growth factor, applying to the scalp a substance that triggers histamine release may also promote hair growth. In animals, administration of polysorbate 80 triggers the production of histamine in mast cells and plasma histamine concentrations rise after administration of polysorbate 80. In humans, the administration of polysorbate 80 causes effects in the body similar to the body's response after histamine administration. One way polysorbate 80 may result in hair regrowth may be through its ability to trigger histamine release in the scalp, stimulating cell growth.

### **Other Possible Mechanisms**

Polysorbate 80 may also remove dihydrotestosterone (DHT) from the scalp. Excessive levels of DHT are thought to cause hair loss. In addition, polysorbate 80 may affect genetic activities in the hair follicle cells due to its ability to reverse early tissue damage that occurs at the beginning of toxin-induced carcinogenesis.

### **Recommended Dosage**

In *Life Extension*, Pearson and Shaw recommend coating the scalp in the bald or balding locations with a thin film of polysorbate five or ten minutes before shampooing. Rub into the scalp with the fingertips vigorously enough for the scalp to turn somewhat red and feel warm—an indication of histamine release. Wait one to three minutes then rinse thoroughly. Within two or three months of daily use, new hairs may appear. At first bright side lighting and a black background may be required to see the thin and short hairs that first appear. In some studies, hundreds of men in their 50s and 60s reported more than 60 percent average regrowth after using polysorbate daily for a year. For the first week or two patients may

notice an increase in hair loss due to loosening of hairs in resting phase atrophic follicles.

#### References

1. Durk Pearson and Sandy Shaw. *Life Extension: A Practical Scientific Approach*. Pg 218 - 220. Warner Books, Inc. 1982.
2. Masini E, Planchenault J, Pezziardi F, Gautier P, Gagnol JP. Histamine-releasing properties of Polysorbate 80 in vitro and in vivo: correlation with its hypotensive action in the dog. *Agents Actions*. 1985 Sep;16(6):470-7.

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## Customers' Corner

by *Ward Dean, MD*

VRP Medical Director and Director, Research & Development

### ***Exercise and Sore Muscles***

Dear Dr. Dean,

I recently began weight lifting after a hiatus of several years. I currently take **Amino EDGE** once a day with a meal, and **Myo EDGE** once immediately after a workout. After only 2 weeks I've noticed muscle gains. However, I've also noticed that the left side of my chest is considerably larger than the right. It almost appears swollen. My muscles are also very sore, typical of returning to weight lifting. I first noticed this after stretching in an attempt to relieve the tightness I felt. Should I be concerned?

Mr. M.

Dear Mr. M.

I would not be concerned. Although we think we are "symmetrical," we aren't always. Also, the difference may be due to a difference in your training. You may be unconsciously working one side of your body harder than the other.

The soreness is a sign that your muscles are adapting to the heavier loads you are placing on them. I'd stop working out for several days until the soreness is relieved. This is an indication that the "injured" muscle (from working out) is recovered, and you are ready to continue your progress. **Myosin** cream may speed resolution of muscle soreness.

Ward Dean, MD

### ***Cystitis and Thrombosis***

Dear Dr. Dean,

I am from Greece and began using VRP products about three months ago, as did my wife. We currently use **Extend Core**, **Coenzyme Q10**, and **Optimum Silver**. My wife suffers from venous thrombosis on the lower limbs. I would like to know if my wife can take **Optimum Silver** and **Coenzyme Q10**, and are there any products you can recommend to prevent thrombosis?

Also, my wife has suffered from urinary tract infections since 1976. The causative organism is always E. Coli. She has tried antibiotics and **ActiBiotic™**, but unfortunately, nothing has proved effective.

I read in *Vitamin Research News* July 2003 about a product called **D-Mannose**. Will this product have

any effect on my wife's case? Does it have any after-effects in respect of thrombosis? Thank you in advance!

Yours sincerely, K.S.

Dear Mr. S.,

Your wife can take all of the supplements she asked about. **D-Mannose** may help with her urinary tract infections. In addition, I'd suggest **Culturelle**, and **Liquid Silver Mild Silver Protein**.

For her deep vein thrombosis, I suggest **Oral ChelatoRx**, **Turmeric**, 1/2 baby aspirin per day, and **UniZyme™**. VRP's new **Vein Support Formula** may also help.

Ward Dean, MD

## ***Aminoguanidine Dosage/Safety***

Dear Dr. Dean,

I am about to order some amino-guanidine (**AGEBlock**) for hypertrophic cardiomyopathy. Is this essentially the same as Alteon-711? I have been waiting for Alt-711 to come out but that is probably still a few years away. I am interested in the safety factor of aminoguanidine and its effectiveness for diastolic heart failure.

Thank you, Mr. G.

Dear Mr. G.,

Aminoguanidine is not the same as ALT-711. Unfortunately, it looks like we'll be waiting longer for ALT 711 to be available than we thought.

I have been taking six **AGEBlock** capsules almost daily ever since it came out. **AGEBlock** contains 100 mg amino-guanidine bio-equivalent per capsule. The clinical studies indicated that 300 mg per day was an effective, safe dose—but that there was some question about the safety of 600 mg.

Nevertheless, I usually try to be the company guinea pig regarding safety, because I know many life extensionists believe if a little is good, more will be better. I want to be the person to sound the warning if one is needed.

I recommend 300 mg daily for CHF (congestive heart failure). Also, I'd add several grams of Taurine as and Carnitine as well. An excellent combination formula that may benefit CHF is **CardioCare**.

Ward Dean, MD

## ***False Positive Drug Tests***

Dear Dr. Dean,

I've just ordered some supplements for my husband. He has Type II diabetes, is a truck driver, and is concerned about these supplements producing a "false positive" when he takes his Department of Transportation (DOT) physical. As you can imagine, he has to be very careful about what he takes. I have ordered **Optimum D**, **GluControl**, **Vanadyl Sulfate** and **AGEBlock**.

Thanks very much, Mrs. M.

Dear Mrs. M.,

As a former military flight surgeon, where people were also tested frequently, I can assure you that none of the ingredients in any of the products will result in a false positive on any DOT drug test. However, I suggest that since he is taking the new **GluControl** formula and **Optimum D**, he does not need the additional Vanadyl Sulfate or AGEBlock.

I used to recommend additional **AGEBlock** with **GluControl**, in order to raise the amount of standardized Goat's Rue Extract in the old **GluControl** formula to a full 300 mg/day of aminoguanidine bio-equivalent. However, 8 caps per day of the new **GluControl** formula now provides this optimum amount. Therefore, it is no longer necessary to add additional **AGEBlock**.

Also, 8 caps of the new formula provides 100 mg of **Vanadyl Sulfate**, which is within the therapeutic range of 50-150 mg. Therefore, additional Vanadyl Sulfate is also no longer recommended.

Ward Dean, MD

## **Controlling Lipid Levels**

Dear Dr. Dean,

I am on three medications for hypertension: Prinivil, Norvasc, and Atenolol (this last added in late February). In March my total cholesterol was 234, VLDL was 54, LDL was 143, HDL was 37, and triglycerides were 268.

I started taking **Inositol Hexanicotinate** (3 caps/day), Beta-sitosterol (2 caps, twice a day), and cod liver oil. I also started exercising (brisk walks) 5 times a week, and eliminated most fat from my diet (which has always been predominantly low fat vegetarian, chicken/fish once a week, no eggs, butter, cheese, etc).

I was considerably disappointed when my lipid profile on July 8 was as follows: Total cholesterol 249, VLDL and LDL not measurable, HDL 37, Triglycerides 452. Now my physician wants me to take TriCor (fenofibrate) which I do not wish to do. What would you advise?

Thanks so much, Mr. B.

Mr. W.

Dear Mr. B.,

I understand your frustration. Lipid-lowering is somewhat of an art, sometimes. Nothing works for everyone in exactly the same way.

First, I'd get back on a higher protein, higher fat diet. Start back with eggs. I think they're one of the best foods there is. Just try not to hard-cook the yolks. I usually eat 3 to 4 eggs per day. A low-fat diet is a high-carbohydrate diet. Carbohydrates break down into sugars. Sugars cause triglycerides to climb, and cholesterol follows. I think an Atkins-type diet is more likely to help normalize your lipids.

I'd suggest trying **Pressure-FX** for your blood pressure. You may be able to reduce your reliance on medications to control your blood pressure. Also, ask your physician to prescribe Metformin. Please see my article "The Metabolic Pattern of Aging" in the April, 2002 issue of *Vitamin Research News*. It explains the importance of controlling insulin to help control hypertension, hyperlipidemia, and other signs associated with aging.

If he will not prescribe Metformin, I suggest VRP's **GluControl** or **AGEBlock**. Metformin, **GluControl**, or **AGEBlock** should help with your blood pressure control, as well as have a favorable influence on your lipids.

You might also benefit by increasing your Inositol Hexanicotinate. I'd suggest doubling your current dose. If that does not result in a significant improvement, other options/additions to consider include **Oral ChelatoRx** and **Turmeric**. **Turmeric** acts as an anti-inflammatory agent, and should also reduce fibrinogen levels. Fibrinogen is an even more significant risk factor than abnormal lipids.

Let me know how you do,

Ward Dean, MD

## ***Lithium and Myelin***

Dear Dr. Dean,

I am seeing a young boy—6 years old with a myelin problem. I have read that lithium helps bipolar individuals by helping the nerves to myelinate. I am thinking about using your product **Lithium Orotate**. Have you heard of lithium helping to myelinate nerves?

Thank you, K.W., D.O.

Dear K.,

We have had several articles about the benefit of lithium in Alzheimer's disease. I wouldn't be surprised if the mechanism were not due to its remyelination of the nerves.

Lithium also acts acutely in many conditions (often, within minutes). I think it has unique nerve-stabilizing properties. Because of the increasing recognition of a number of benefits from **Lithium Orotate**, and absolute absence of toxicity in therapeutic dosages, we had considered adding it to our multi-mineral formulas (**Essential Minerals** and **Advanced Essential Minerals**), but decided against it because it is so commonly associated with manic depressive illness, and figured some people might avoid it because of this "stigma." However, I take it on a routine basis, as part of my own multi-mineral supplement program.

Ward Dean, MD

## ***Holes From Radioactive Pellets***

Dear Dr. Dean,

Last year my 61-year-old brother was treated for prostate cancer, which involved placing radioactive beads around his prostate. Now, over a year later, he has developed three holes (ulcers) in his intestines and colon. The first was dime-size and was cauterized shut. The second hole is larger than a silver dollar, and remains open. The doctors are treating him with steroids and pain meds, but they claim that the holes are not caused by the radioactive beads. I think the beads are eating through his tissues and wonder if there are any supplements he can take to support his immune system, and to help him recover. Any suggestions would be appreciated.

Thank you in advance for your help. Mrs. C.

Dear Mrs. C.,

I think you're right—destroying tissue to "cure" a disease just does not make sense. **Thymic Protein A** is my number one recommendation as an "Immune Normalizer." See the August, 2003 issue of our newsletter for an article on this subject. I recommend he start with one envelope per day until there is an improvement—although this is not assured as the cause of his problem may be his previous attempts at "cure."

Radiated tissue is essentially burned, scarred tissue, which does not heal well. With addition of more

radiation, which may be killing cancer cells, it is also more than likely burning and killing off healthy tissue as well. Like I said, I don't think radiation therapy makes much sense. Unfortunately, it may be too late to undo the damage.

Another excellent immune enhancing anti-cancer nutrient is **Beta 1, 3-D Glucan**. Studies in healthy humans with **Beta Glucan** have recently been completed which demonstrate a significant augmentation in immune-enhancing cytokines. Presumably, these same effects would probably occur in cancer patients as well.

Ward Dean, MD

## ***Mind Enhancers and B12***

Dear Dr. Dean,

I already take several of your formulas. I am 74 years old, in great shape, but occasionally forget words that I know very well. I know that this is considered to be part of the aging process, but I am looking at two of your formulas—**Extension IQ** and **Phosphatidylserine 100 Plus**.

Which do you suggest? I see that the **Extension IQ** has extra **B12**. If I took this might this not be too much in addition to the high potency B vitamins that are in my multiple?

Thanks for your exceptional helpfulness! Mr. H.

Dear Mr. H.,

If you have to make a choice, I suggest **Extension IQ**. **Extension IQ** is a broad-spectrum formula that contains **DMAE**, **Vinpocetine**, **Huperzine**, **Pyroglutamic Acid** and **Ginkgo Biloba**, plus other synergistic nutrients. This is a very powerful, comprehensive formula. It is designed for two dosage levels—four capsules per day provide a dose believed to be in the minimum effective range for each of the cognitive enhancing ingredients, while 8 capsules per day is in a documented effective dosage range (i.e., 40 mg Vinpocetine; 100 mcg Huperzine-A; 500 mg DMAE, 1 gm Choline, etc.).

There is no toxic range that I know of for **B12**. **B12** is important for protein synthesis and energy production. **B12** is one of the few vitamins that you will find (often, along with **B5** and a few others) that will be found as co-factors in VRP products.

As you surmised, we generally don't add nutrients to other formulas that are included in adequate amounts in our multi-nutrient formulas.

Ward Dean, MD

## ***Prostate Cancer After Surgery***

Dear Dr. Dean,

I am 75 years old and have been a consumer of VRP products for a long time. Five years ago my prostate was removed (not malignant). Now I have five incipient malignant tumors in the same area, caused, I've been told, by testosterone. The only choices I've been offered are the removal of my testicles or monthly injections and pills.

Do you have any suggestions for reducing the production of testosterone? I'm also diabetic and had successful surgery for a bypass and heart valve. Thank you for your time and consideration.

Mr. W.

Dear Mr. W.,

At age 75, I really doubt that you're making that much testosterone. Consequently, I think removing your testicles at this point is insane.

I think you are at greater risk from diabetes than from your incipient malignancies. I suggest for your diabetes that you take either Metformin or Phenformin (which I think you can still obtain in Mexico). Metformin was a principle element in Vladimir Dilman's anti-cancer regimens. I think controlling your diabetes will help control your cancer.

I would add as many anti-cancer nutrients as you can afford, including **Thymic Protein A**, **CelMend**, **Modified Citrus Pectin**, **Beta 1,3-D Glucan**, and **Inositol Hexaphosphate (IP6)**. **BioDim** and even **Progesterone Cream** may help.

I suggest hanging onto your testicles, and not taking the monthly injection or pills (ask the physicians to show their success rate with these treatments). A recent article showed that the drug finasteride, which is used to treat benign prostatic hypertrophy, was also effective in reducing the incidence of prostate cancer. Many of the ingredients in VRP's **ProstaCol** act by the same mechanism as finasteride, and may produce the same prostate-cancer preventive effects. Consequently, I'd add **ProstaCol** to this regimen.

Ward Dean, MD

## ***Pollen, Beta Sitosterol for BPH***

Dear Dr. Dean,

I have been taking VRP's **Beta-Sitosterol** for an enlarged prostate. It seems to be helpful. A friend has been having good results with a product made of rye pollen. I would like to try it and wondered if there is any reason why I can't take both products at the same time. What is your opinion?

Mr. H.

Dear Mr. H.,

There is excellent research on the use of flower pollen to alleviate benign prostatic hypertrophy (BPH). There is no reason not to combine pollen with beta sitosterol.

VRP's newly reformulated **ProstaCol** formula now provides a full 300 mg of beta sitosterol in each daily dose.

Ward Dean, MD

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## **Nutrition Review**

### ***Vitamins C and E Prevent Heart Disease in Children***

An estimated 50 million US children have elevated levels of cholesterol, placing them at increased risk of suffering from heart disease and heart attack. Writing in the journal *Circulation*, Marguerite Engler of the University of California San Francisco and colleagues report how vitamins C and E can reverse the damage caused to blood vessels by cholesterol.

Engler's team studied 15 children and young adults, aged 9 to 20 years. The researchers measured arterial health by monitoring signs of endothelial dysfunction, which causes blood vessels to stiffen so they cannot stretch to accommodate increased blood flow, an early sign of atherosclerosis.

Half the children received 500 mg of vitamin C and 400 IU of vitamin E daily for six weeks, while the other half got placebos. After six weeks and a six week washout period the groups were switched. The researchers found that vitamins C and E restored endothelial function to damaged arteries by interacting with the free radicals that cause atherosclerosis (hardening of the arteries). "When we gave these children moderate doses of vitamins C and E for six weeks, we saw a significant improvement in blood-vessel function, which is an important indicator of cardiovascular health," stated Engler.

"The impact was quite significant," Engler said. "These results are encouraging and, if confirmed in further studies, we may be able to improve the cardiovascular health of children with inherited lipid disorders using vitamin supplements," she added.

*Circulation. 2003 Aug 11.*

## ***New Study Finds Creatine Boosts Memory, Intelligence***

Taking supplements of creatine, a compound found in muscle tissue, can significantly boost both working memory and general intelligence, according to researchers in Australia. Creatine helps cells replenish stores of ATP, the primary source of energy for cellular energy production. Creatine is already popular with athletes for body-building and sports that require intense bursts of energy. Commenting on the amount of energy required for thinking, Catherine Rae, lead researcher at the University of Sydney, Australia, stated, "You're fuel-limited every time you're thinking."

When researchers in Australia gave creatine to a group of vegetarians they measured significant improvement in a number of memory tests. The researchers chose vegetarians since non-vegetarians already obtain creatine from their diet. Rae and her colleagues asked 45 vegetarians in their twenties to take either five grams of creatine, equivalent to about two kilograms of meat, or a placebo daily for six weeks.

The volunteers' reasoning ability and short-term memory was tested before and after the six-week period. In one test, for example, Rae's team found that those who took creatine could remember an average of 8.5 numbers compared with 7.0 for those on the placebo.

Rae says the team intends to examine the effect of creatine on meat-eaters in the future. Older people might also be candidates for study, because both creatine levels and memory decline with age.

*Proceedings of the Royal Society B, Vol 270.*

## ***DHEA Improves Arterial Function, Insulin Sensitivity***

Dehydroepiandrosterone (DHEA) has been shown effective for improving both functioning of the arteries and insulin sensitivity in men, according to a recent study published in the Journal of Clinical Endocrinology & Metabolism.

DHEA is the most abundant steroid hormone in the body. It is also one of the most significant age-related biomarkers, which predictably declines with age in even the healthiest of people. Abnormally low levels of DHEA have been reported to be related to a number of diseases, including cancer, diabetes, coronary artery disease, obesity, and Alzheimer's disease.

Researchers have hypothesized that raising DHEA levels with supplementation can restore the

protection this hormone exerts against age-related disorders. Additionally, DHEA has previously been shown to be a potential treatment for multiple sclerosis and other chronic inflammatory diseases of the central nervous system.

In the current randomized, double-blind study, researchers enrolled 24 men (average age 54) who had been diagnosed with elevated cholesterol. Following daily treatment with 25 mg of DHEA, the research team measured significant improvements in endothelial function (artery flexibility) after just four weeks. The research team also noted a significant drop in blood sugar levels without altering insulin levels.

According to the researchers, "These beneficial changes have the potential to attenuate the development of age-related disorders such as cardiovascular disease."

*Journal of Clinical Endocrinology & Metabolism, Vol. 88, No. 7, 3190-3195.*

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