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The NUTRITION REPORTER™

THE INDEPENDENT NEWSLETTER THAT REPORTS VITAMIN AND MINERAL THERAPIES © APRIL 1998 VOL 9 NO 4 BY JACK CHALLEM

TMJ Pain May Be Aggravated by Free Radicals, Relieved Partly by Antioxidants

Temporomandibular joint (TMJ) disease affects an estimated 10 million Americans. The joint, located on each side of the head where the temporal bone of the skull meets the lower jaw (mandible), is considered the most complicated joint in the body. It moves backward and forward, opens and closes like a hinge, and is subject to enormous pressure during chewing. Not surprisingly, then, the cause of TMJ disease is often mechanical, the result of malocclusion (bad bite). TMJ disease can also be a manifestation of arthritic inflammation.

For someone with TMJ disease, chewing food causes pain, which can radiate outward and affect nearby tissues, resulting in toothache, headache, muscle tightness in the face, clicking or popping noises, and neck and shoulder pain. Conventional treatments can include bite plates, orthodontics, and prescription muscle relaxants.

Free radicals – the same molecules implicated as a cause of cancer and heart disease – may play a major role in exacerbating TMJ disease symptoms, according to a recent article by Stephen B. Milam, DDS, PhD, and his colleagues at the University of Texas Health Sciences Center, San Antonio, Texas. If free radicals do contribute to TMJ disease, then antioxidants may provide a therapeutic option.

Milam believes that the mechanical stress of the TMJ generates free radicals through a number of mechanisms, triggering a cascade of biochemical reactions. The end result amplifies the tissue damage and pain associated with TMJ disease.

"One can envision that a disease state could result from an accumulation of such radicals by either increased production of such radicals, overwhelming local antioxidant defenses, or by a deficient production of free radical scavengers," he wrote.

• Physical and mechanical stresses, such as shearing, can generate free radicals. This, wrote Milam, has been demonstrated in blood cells and fingernails exposed to shearing stresses. Grinding and shearing may occur with movement of the temporomandibular joint.

• Hypoxia-reperfusion injury can also generate free radicals. Unbalanced joint pressure can temporarily induce localized oxygen starvation. When oxygen is restored, cell metabolism can shift and increase production of free radicals.

• Hemoglobin, the iron-containing protein in blood is a potent source of free radicals and inflammation. Microbleeding can deposit hemoglobin into joint tissues, enabling its iron to generate hydroxyl radicals, which can damage collagen, elastin, and other types of tissues, Milam wrote.

• Breakdown of arachidonic acid, an essential fatty acid, increases production of free radicals that attack cell membranes. Some cytokines (peptide hormones) can also generate free radicals.

"Free radicals generated by these mechanisms in normal TMJs may not lead to a pathologic state if endogenous free radical scavenging mechanisms prevent their accumulation," Milam wrote. "However, if the scavenging capacity of affected articular tissues is exceeded by an overwhelming production of free radicals, significant tissue damage could occur. Also, if the scavenging capacity of the affected tissue is compromised (eg, genetic deficiency, nutritional deficiency, compromised synthesis by mechanically stressed cells), then tissue damage could result from a modest production of free radicals."

Milam and his colleagues cautiously approached the use of antioxidants in ameliorating TMJ disease symptoms – dentistry has been slower than medicine to recognize the therapeutic potential of antioxidants. Milam cited one study showing that superoxide dismutase (an antioxidant enzyme produced by the body) reduced TMJ disease symptoms. Other antioxidants, including glutathione, vitamins C and E, and melatonin are well established as free radical scavengers.

Milam SB, Zardeneta G, Schmitz JP, "Oxidative stress and degenerative temporomandibular joint disease: a proposed hypothesis," *Journal of Oral and Maxillofacial Surgery*, 1998;56:214-223.

Vitamin Supplements Protect Against Sunburn from the Inside Out

Taking supplements of vitamins C and E can help protect against sunburn, according to a new study by German physicians.

Previous research has shown that topical applications of vitamins C and E have a weak sun-blocking effect. This study is the first showing that oral intake of these vitamins bolsters the skin's internal resistance to sunburn.

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At the beginning of the study, Bernadette Eberlein-König, MD, and her colleagues from the dermatology clinic at the Technical University of Munich exposed 20 men and women to ultraviolet (UV) light. They then measured the UV dose needed to cause sunburn (minimal erythema dose, or MED) in the subjects. The researchers also measured the subjects' blood flow in skin exposed to UV and protected from UV.

Next, Eberlein-König gave half of the subjects either a daily placebo or supplements containing 2,000 mg of vitamin C and 1,000 IU of natural vitamin E daily for eight days. Then, portions of the subjects' skin were again exposed to UV light.

People taking vitamins C and E had an average increase of 20 percent in MED, indicating greater resistance to sunburn. In contrast, patients taking the placebo had an average 14 percent decrease in MED – that is, they were more sensitive to UV exposure.

Also, people taking vitamins had a decrease in blood flow in skin exposed to UV rays, another sign that they were more resistant to reddening. People taking the placebo showed an increased sensitivity to UV after eight days, suggesting that skin had become "primed" to respond more intensely to UV light.

"This study shows for the first time that systemic administration of vitamins C and E reduces the sunburn reaction in humans....Systematic photoprotecion is convenient and could provide a desirable basic UV shield for the entire body surface..." write Eberlein-König.

Excessive UV exposure can set the stage for skin cancer, in part by suppressing the immune system's response to tumors, according to Dr. Helen K. Gensler of the University of Arizona, Tucson. In a recent study, Gensler found that the topical application of niacinamide (a form of vitamin B3) prevented UV-induced immune suppression in mice. Animals receiving topical niacinamide had a 43 percent reduction in skin tumors.

References: Eberlein-König B, Placzek M, and Pryzybilla B, "Protective effect against sunburn of combined systemic ascorbic acid (vitamin C) and d-a-tocopherol (vitamin E)," *Journal of the American Academy of Dermatology*, 1998;38:45-8; Gensler HK, "Prevention of photoimunosuppression and photocarcinogenesis by topical nicotinamide," *Nutrition and Cancer*, 1997;29: 157-162. □

Is Olive Oil An Antiinflammatory?

Monunsaturated fatty acids (MUFAs), such as those found in olive oil, can reduce the risk of coronary heart disease and some cancers. New research shows that MUFAs work, at least in part, by turning off some of the cells that promote inflammation – extending the number of conditions that MUFAs may benefit.

"Less attention has been paid to the effects of MUFAs on the immune system, yet cells of the immune system are an inherent part of the inflammatory events involved in atherosclerosis and several animal studies showed that olive oil has some potent immunomodulatory actions," wrote Parveen Yaqoob, PhD, of the University of Southampton, England.

In a recent double-blind study, Yaqoob asked 30 middle-age men to eat a high-MUFA diet for two months, then compared various aspects of their immune systems to those of men who ate a conventional diet.

Men eating the high-MUFA diet had adhesion cells – a type of cell involved in promoting inflammatory and allergic reactions – that were about 20 percent less active.

"An important finding of the study was a decrease in the expression of IACM-1 with the MUFA diet," Yaqoob wrote in the *American Journal of Clinical Nutrition*. "ICAM-1 is a member of the immunoglobulin superfamily of adhesion molecules..."

MUFAs also resulted in a modest decrease in the activity of another type of adhesion cell, called Mac-1.

The implications were "exciting," Yaqoob wrote. That's because the low incidence of heart disease and inflammatory diseases, such as rheumatoid arthritis, among Mediterranean populations may be related to their high MUFA consumption.

Although MUFAs decreased the activity of some adhesion cells, they "do not appear to involve a general suppression of immune cell functions," Yaqoob wrote.

Reference: Yaqoob P, Knapper JA, Webb DH, et al., "Effect of olive oil on immune function in middle-aged men," *American Journal of Clinical Nutrition*," 1998;67:129-35. □

Supplements of Coenzyme Q10 Can Strengthen a Failing Heart

Coenzyme Q10, a vitamin-like substance, can strengthen the hearts of patients with congestive heart failure. The condition, caused by a lack of energy in heart muscle, interferes with the heart's ability to pump blood.

CoQ10, synthesized by the body and found in foods, serves two cellular functions: it is essential for bioenergetics – that is, the production of energy in the form of adenosine triphosphate (ATP) – and it is also an antioxidant.

"Since all cellular function depends on an adequate supply of ATP, CoQ10 is an essential component of life itself," wrote Stephen T. Sinatra, MD, a cardiologist at Manchester Hospital, Manchester, Conn., in *Connecticut Medicine*.

Sinatra described three of the patients he treated with CoQ10.

• LG was diagnosed at age 60, in 1977, with congestive heart failure. In 1984, her ejection fraction (a measure of

the heart's ability to pump blood) was 35 percent of normal. By 1994, she was suffering from severe heart failure with an ejection fraction of 10-15 percent, "barely enough to support her bed-to-chair existence," Sinatra wrote. She was given 90 mg/day of CoQ10 in addition to conventional therapy. In March 1995, she inadvertently began taking 300 mg of CoQ10 daily and, in four weeks, had a significant improvement in her health. By October, she was visiting relatives and shopping. "In addition to her conventional medical therapy, she has been successfully maintained on 300 mg of CoQ10 daily."

• HD had a history of emphysema and heart problems, along with a diagnosis of congestive cardiomyopathy several years ago. He was given a multivitamin/multimineral supplement plus 90 mg of CoQ10 daily. With an ejection fraction of only 15 percent, his CoQ10 was increased to 120 mg daily and later to 300 mg daily. Now with near-normal heart function, he is able to go for long walks on the beach. He has also been able to reduce, though not cease, other heart medications.

• LH, age 79, had been hospitalized with pneumonia and congestive heart failure. Her condition rapidly deteriorated, and her family arranged for her to be transferred to a hospital where she could obtain a combination of CoQ10 and conventional treatment. Her estimated ejection fraction was 50 percent. She initially received 450 mg of CoQ10 daily through a nasogastric tube. She is "enjoying a good quality of life" on 300 mg of CoQ10 and angiotensin converting enzyme (ACE) inhibitors.

"Although CoQ10 can be synthesized in the body, situations may occur in which the body's capacity to produce CoQ10 is insufficient to meet its requirements," Sinatra wrote. "Therefore some patients may be deficient in this vitamin."

Sinatra ST, "Coenzyme Q10: a vitamin therapeutic nutrient for the heart with special application in congestive heart failure," *Connecticut Medicine*, 1997;61:707-711. □

Vitamin E May Help Arthritics and Ease the Inflammatory Response

Natural vitamin E supplements can ease the pain and stiffness associated with rheumatoid arthritis, according to a report in *Annals of the Rheumatic Diseases*.

A team of British and German researchers investigated the effect of vitamin E because previous studies had shown that the vitamin has some antiinflammatory properties.

While the researchers were not able to document any antiinflammatory properties for vitamin E, they did provide a scientific framework for why vitamin E would help arthritics.

The researchers asked 20 subjects with rheumatoid arthritis to take 600 mg of natural vitamin E twice daily and 22 others to take placeboes for 12 weeks. The subjects kept a daily diary describing the intensity of early morning stiffness, evening pain, and pain after routine daily activities.

On average, arthritic pain decreased by about one-half among the patients taking vitamin E supplements. Furthermore, a larger percentage of patients taking vitamin E improved, compared with placebo. "According to the patients' global assessment of efficacy, 60% of the patients improved with alpha-tocopherol compared with 31.8 percent for placebo," wrote Dr. P. G. Winyard of the Royal London School of Medicine and Dentistry.

Winyard noted that nitric oxide (NO), a free radical, is involved in nerve transmission of pain. Vitamin E is known to quench nitric oxide radicals. "The interaction of vitamin E with NO is a mechanism by which it could exert its analgesic effect," the researchers wrote.

Meanwhile, in an animal study at the University of Washington, Seattle, researchers found that vitamin E could ease an overly aggressive immune response to bacterial toxins. Eileen M. Bulger, MD, noted that a systemic (body-wide) inflammatory response is a major cause of complications and death among intensive-care hospital patients.

"The underlying pathologic mechanism involves the development of an uncontrolled overexpression of the inflammatory response, leading to the self-destruction of host tissues and resultant multiple organ failure," she and her colleagues wrote in the *Archives of Surgery*.

Oxidative stress, or excess of free radicals, is particularly high in patients with a systemic inflammatory response. "Antioxidant therapy, therefore, seems a logical approach in these patients," Bulger wrote.

In the experiments, she gave one of three different forms of vitamin E—alpha-tocopherol, alpha-tocopheryl acetate, and alpha-tocopherol succinate—or two types of placeboes to laboratory rats. Afterwards, Bulger measured how macrophages (a type of white blood cell) from the different mice reacted to a bacterial toxin called lipopolysaccharide.

The toxin triggered the macrophages' production of tumor necrosis factor, a compound that promotes inflammation. However, macrophages from mice given any of the three different forms of vitamin E had virtually no increase in tumor necrosis factor.

References: Edmonds SE, Winyard PG, Guo R, et al., "Putative analgesic activity of repeated oral doses of vitamin E in the treatment of rheumatoid arthritis. Results of a prospective placebo controlled double blind trial," *Annals of the Rheumatic Diseases*, 1997;56:649-655; Bulger EM, Helton WS, Clinton CM, et al., "Enteral vitamin E supplementation inhibits the cytokine response to endotoxin," *Archives of Surgery*, 1997;132:1337-1342. □

Quick Reviews of Recent Research

• FOS enhances calcium absorption

People who have part of their stomach surgically removed suffer a subsequent loss in bone density, and calcium supplementation is not sufficient to maintain bone density. In an experiment with laboratory rats undergoing a similar surgery, researchers found that fructooligosaccharides (FOS) prevented calcium loss and enhanced calcium absorption. FOS is an indigestible sugar that some intestinal bacteria use as a food source.

Ohta A, et al., *Journal of Nutrition*, 1998;128:106-110.

• Vitamin E protects against cholesterol

Researchers fed rabbits a high-cholesterol diet, then treated them with either natural vitamin E or the cholesterol-lowering drug probucol. Vitamin E inhibited the activity of protein kinase C (PKC), an enzyme that promotes the growth of smooth muscle cells, which are associated with an increased risk of cardiovascular disease, whereas probucol did not. In addition, vitamin E "fully prevented cholesterol-induced atherosclerotic lesions," which probucol did not. Vitamin E's benefits appeared related more to its influence over gene expression than to its antioxidant properties.

Ozer NK, Sirikci O, Taha S, et al., *Free Radical Biology & Medicine*, 1998, 24:226-233.

• Carotenoids help smokers

Researchers asked 22 smokers and nonsmokers to eat a selection of carotenoid-rich foods: carrots (for beta-carotene), pear tomatoes (for lycopene), and French beans, cabbage and/or spinach (for lutein). The mix of carotenoids (approximately 30 mg/day for two weeks) increased blood carotenoid levels by 23 percent in smokers and 11 percent in nonsmokers. Furthermore, the higher carotenoid levels delayed oxidation of the low-density lipoprotein form of cholesterol by 28 percent in nonsmokers and 14 percent in smokers. These changes in carotenoid levels and LDL oxidation may lower the risk of coronary heart disease.

Hininger I, et al., *European Journal of Clinical Nutrition*, 1997;51:601-606.

• Beta-carotene reduces cancerous cell changes

Researchers exposed laboratory rats to a cancer-causing chemical, then fed them beta-carotene, vitamin A, or corn oil for five weeks. Rats receiving beta-carotene had a much lower incidence and number of precancerous liver lesions. Two-thirds of the rats getting beta-carotene developed precancerous lesions, compared with all of the rats receiving vitamin A and 90 percent of those receiving corn oil. Overall, rats receiving beta-carotene had only 380 precancerous lesions, compared with 504 in the vitamin A group and 1,541 in the corn oil group.

Rizzi MB, et al., *International Journal of Vitamin and Nutrition Research*, 1997;67:415-22.

• *Ginkgo biloba* extract protects against aging

Many researchers believe that the aging process begins in mitochondria, the energy-producing structures in cells. Mitochondria generate large numbers of free radicals, which can damage genes and cell membranes. In a study with laboratory rats, researchers noted that older rats had much more free radical damage to mitochondrial DNA (deoxyribonucleic acid) than did young rats. Treatment with *Gingko biloba* extract EGb 761 reduced the amount of free radical damage in brain and liver cells of old rats.

Sastre J, et al., *Free Radical Biology & Medicine*, 1998, 24:298-304.

• Carotenoids protect mice against DNA damage

Damage to DNA is the fundamental cause of most cancers and other degenerative diseases, as well as of the aging process itself. Researchers fed laboratory mice either a diet with supplemental palm oil, rich in alpha- and beta-carotene, or a diet without the carotenoids. They then exposed all of the mice to x-ray radiation. Mice receiving the carotenoids had increased levels of these nutrients in bone marrow and the liver. The x-rays induced DNA damage in the bone marrow and reduced white blood cell counts in all of the mice, but the effects were attentuated in the carotenoid-fed mice.

Umegaki K, et al., *Carcinogenesis*, 1997;18:1943-1947.

• Soy prevents diet-induced heart disease

Researchers fed male rhesus monkeys a high-cholesterol diet, plus supplemental soy protein with phytoestrogens (isoflavones) intact, soy protein with most phytoestrogens extracted, or milk protein for 14 months. Animals eating the soy protein with phytoestrogens (equivalent to 143 mg/day in a human) developed the smallest number of coronary artery lesions—50 percent fewer than the other soy protein group and 90 percent fewer than the milk protein group.

Anthony MS, et al., *Arteriosclerosis, Thrombosis and Vascular Biology*, 1997;17:2524-2531.

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Aloha, OR 97006-5505 USA

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