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Vitamin E: While it is not known how to increase lens SOD activity directly, animal work suggests that vitamin E deficiency decreases the levels of both SOD and glutathione reductase in the lens. Although there is evidence that long-term vitamin E supplementation is inversely associated with the mean 5-year change in lens nuclear density, two randomized trials found vitamin E supplementation to be ineffective.18

Zinc: Superoxide dismutase requires zinc for its activity. When zinc levels in lenses with mature senile cataract were compared with those in lenses with traumatic cataract, those with senile cataract had lower levels.19 Some studies, however, have found lens zinc levels to be higher in patients with senile cataract, possibly secondarily to disturbed glucose utilization in the lens due to loss of activity of key zinc-dependent enzymes.20-22

Zinc supplementation improves the impaired glucose metabolism occurring in old age, and a rabbit study found that zinc stimulates mitotic activity of the lens epithelium.22,23 Therefore, zinc supplementation may theoretically be beneficial for both prevention and treatment of senile cataract.

Thus, as is the case with other antioxidant nutrients, randomized trials have yet to offer evidence that combating cataract by aiding the lens’ antioxidant enzyme systems is an effective intervention.18

Dr. Werbach cautions that the nutritional treatment of illness should be supervised by physicians or practitioners whose training prepares them to recognize serious illness and to integrate nutritional interventions safely into the treatment plan.

Notes


Doctor Werbach's voluminous Nutritional Influences on Illness CD-ROM, with 4,200 pages of text and covering over 100 different illnesses, makes it easy to search the nutritional literature. For information on his publications or a free brochure, contact Third Line Press Inc., 4751 Viviana Drive, Tarzana, California 91356, USA; 800-916-0076; 818-996-0076; Fax: 818-774-1575; tlp@third-line.com; or http://www.third-line.com.
Nutritional Influences On Illness
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Cataract: the Antioxidant Enzymes

The development of senile cataract, especially nuclear cataract, is associated with progressive oxidative changes in the intrinsic membrane fraction and membrane-related components of the lens, which eventually extends beyond them to also involve the soluble components of lens proteins. Thus, one nutritional approach to combating cataract formation is to increase the activity of the antioxidant enzyme systems present in the lens.

Glutathione Peroxidase and Glutathione Reductase

Glutathione is the antioxidant substrate for two important antioxidant enzymes, glutathione peroxidase and glutathione reductase. While glutathione levels decrease gradually in normal lenses with advancing age, cataractous lenses contain only about one-tenth as much glutathione. Moreover, as glutathione levels fall, lens oxidation increases. In fact, nuclear glutathione appears to be the key factor in preventing lens oxidation.

In rats, supplementation with alpha-lipoic (thioctic) acid, a substance known to increase endogenous glutathione levels, protected 60% of the animals from experimentally induced cataract formation. Also, there is reason to believe that increased cysteine or methionine (the rate-limiting amino acids in glutathione synthesis) may help to prevent cataract formation by retarding the age-related decline in glutathione levels. Human studies have yet to demonstrate, however, whether increasing lens glutathione levels protects against cataract formation.

Riboflavin

There is evidence of an association between riboflavin deficiency and increased risk of presenile and senile cataracts. Riboflavin is a precursor of flavin adenine dinucleotide, which is required as a coenzyme for glutathione reductase, so a riboflavin deficiency may promote cataract formation by interfering with glutathione reductase activity. Also, galactose, a riboflavin antagonist, is a substance that may foster cataractogenesis; thus a high lactose intake may promote the development of cataracts by causing a riboflavin deficiency.

A study of 408 older women found that the geometric mean 5-year change in lens nuclear density was inversely associated with riboflavin intake. Moreover, in an open trial of 18 patients with lens opacities and 6 with fully developed cataracts, there was dramatic improvement after only 1 to 2 days of supplementation with 15 mg of riboflavin daily; and, after 9 months, all lens opacities had disappeared. While these preliminary findings are impressive, they may well be limited to patients with riboflavin deficiency and need confirmation with controlled trials.

Selenium

Glutathione peroxidase requires selenium for its activity. Serum selenium levels, which are positively correlated with aqueous humor levels of erythrocyte glutathione peroxidase, appear to be reduced in patients with senile cataracts; and selenium deprivation induces cataract in rats. Moreover, patients with senile cataracts may have decreased selenium levels in the aqueous humor, and findings from a random sample of 1,274 older women have suggested that a higher intake of selenium may be associated with decreased risk of progression to severe cataract.

However, while one study found the selenium concentration of cataractous lenses to be only 15% of normal, another found a significant increase in lens selenium content with increasing opacification and coloration, with the highest selenium concentration in lenses with a mature cataract.

Since excessive selenium intake is known to promote cataract formation in experimental animals, increasing selenium intake is not recommended until there is adequate experimental evidence of its efficacy.

Superoxide Dismutase

The enzyme superoxide dismutase (SOD) also appears to act as a barrier against photooxidation. As with glutathione, levels of SOD decrease in human lenses with senile cataracts as the cataract advances; moreover, glutathione levels are positively correlated with SOD activity in the human cataractous lens.

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