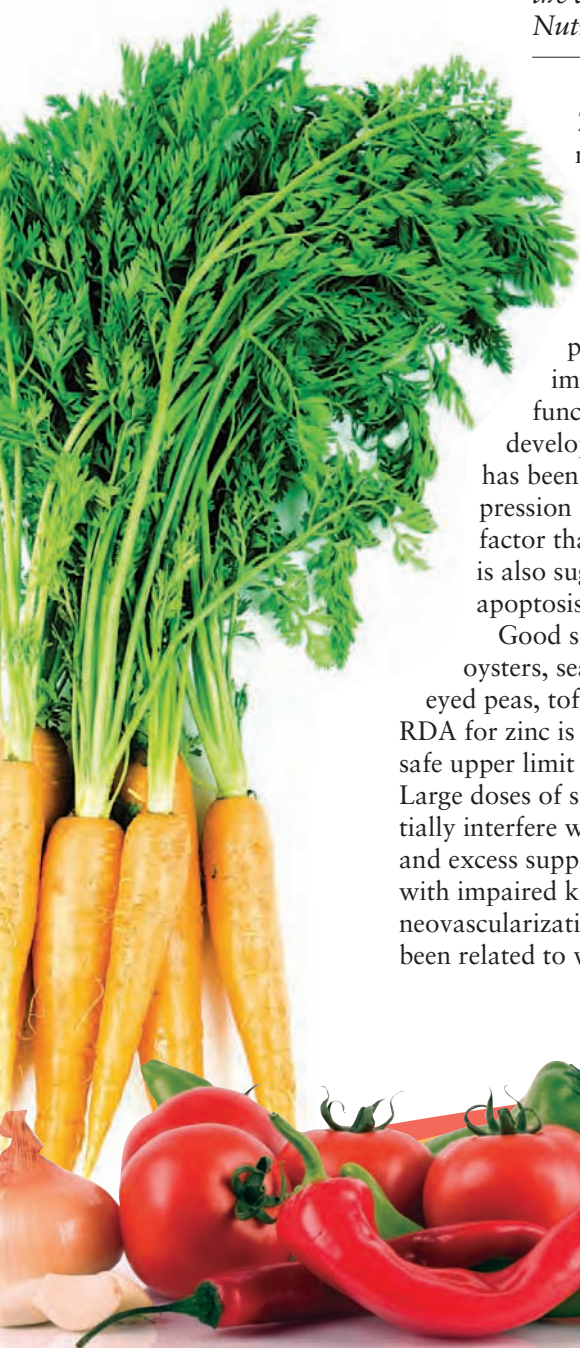


Zinc: A Macular Degeneration Stabilizer

Combinations of Zinc and Antioxidant Vitamins Can Slow AMD Progression.

By Stuart Richer, O.D., Ph.D., F.A.A.O.

Editor's Note: This article is part of a year-long series focusing on vitamins and ocular health and is supported by Bausch & Lomb. To view all the articles in this series, go to www.revoptom.com and click on "Ocular Nutrition from A to Z."



Zinc is an essential trace mineral cofactor used in more than 300 enzyme-governed reactions.

Important in all forms of life, it plays a key role in ATP cellular energy production, cell signaling, immunity, brain and retinal function, as well as growth, development and fertility. Zinc has been found to regulate gene expression by acting as transcription factor that binds to DNA. Zinc is also suggested to play a role in apoptosis (gene directed cell death).

Good sources of zinc include oysters, seafood, meat, eggs, black eyed peas, tofu and wheat germ. The RDA for zinc is 11 mg per day, with the safe upper limit set at 40 mg per day. Large doses of supplemental zinc potentially interfere with copper bioavailability and excess supplementation is associated with impaired kidney function and even neovascularization. Zinc deficiency has been related to visual disturbances, poor

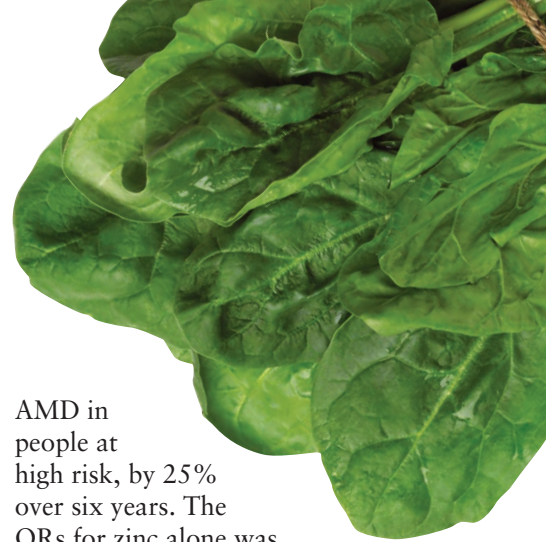
wound healing, mental sluggishness and increased susceptibility to infections. Zinc is synergistic with the AREDS nutrients, antioxidant vitamins C and E, in stabilizing macular degeneration.¹

Zinc Deficiency

The average diet frequently provides less than the Recommended Dietary Allowance for zinc, particularly in vegetarians. Even in developed countries, such as the U.S., low-income pregnant women and pregnant teenagers are at risk for marginal zinc deficiencies. Zinc deficiencies are also more common in alcoholics and people with liver cirrhosis. Zinc deficiencies occur in sickle cell anemia, malabsorption problems, chronic kidney disease and Down's Syndrome patients.²

Zinc helps prevent nyctalopia (night blindness). While night blindness is common in vitamin A deficiency, it's also related to zinc deficiency. This is because zinc enhances the activity of retinol dehydrogenase, a liver enzyme needed to help Vitamin A work properly. Because long-term zinc supplementation may reduce copper levels, 1 mg to 2 mg of copper per day (depending on the amount of

* The views expressed by this article are those of the authors and not Bausch & Lomb.



Zinc (Essential Trace Element)

RDI *	15 mg per day
consequences of deficiency	Night blindness, swelling and clouding of the cornea (severe cases)
dietary source(s)	Mollusks, whole grain cereals
supplement form(s)	Alone or in multivitamin formations

*Recommended Daily Intake

zinc used) is usually recommended for people who are supplementing with zinc for more than a few weeks.

Zinc Excess

Higher levels (up to 50 mg taken three times per day) are reserved for people with certain health conditions, such as AMD, under the supervision of a doctor. Zinc intake in excess of 300mg per day has been reported to impair immune function and blood formation by inducing copper deficiency anemia.³ Some people report that zinc lozenges (providing 13–25 mg of zinc in the form zinc gluconate, zinc gluconate-glycine or zinc acetate) lead to stomach ache, nausea, mouth irritation and a bad taste. Preliminary research suggested that people with Alzheimer's disease should avoid zinc supplements.⁴ However, preliminary evidence in four patients actually showed improved mental function with zinc supplementation.⁵ In a convincing review of zinc/Alzheimer's disease research, one of the most respected zinc researchers in the world concluded that zinc does not cause or exacerbate Alzheimer's disease symptoms.⁶

Some practitioners worry about excess zinc used in AREDS-type supplements. According to AREDS

report number 7, at the five-year exam, the median increase in serum zinc levels for participants assigned to zinc formulations was 17% compared with a 2% increase for participants not assigned to zinc ($P < 0.001$). The differential effect on serum zinc was observed at one year and remained fairly constant over the five-year period. After five years, no significant differences in changes in serum hematocrit, copper or lipids were found between participants assigned to formulations containing zinc and copper, and those assigned to formulations without zinc and copper.⁷

Zinc, AMD and the AREDS Studies

Zinc is an essential cofactor for many of the antioxidant enzymes in the body, and the normal concentration of zinc in the retina is higher than in other parts of the body. The Age-Related-Eye-Disease Study (AREDS) of the National Eye Institute has shown that combinations of zinc and antioxidant vitamins can slow AMD progression. In AREDS, 80 mg of zinc (as zinc oxide & balance 2 mg cupric oxide) was employed. It was part of the four component mixture (along with beta carotene and vitamins C and E), which reduced the relative risk of advanced

AMD in people at high risk, by 25% over six years. The ORs for zinc alone was 0.75 (99% CI, 0.55-1.03) although some 7.5% of zinc-treated patients vs. 5% without zinc, had urinary tract problems requiring hospitalization.¹ In the Rotterdam Study, there was a dose dependent relationship by quartile from food sources of zinc in the Dutch diet. The Beaver Dam results are comparable.⁸ Recently, an ancillary study to the original AREDS trial demonstrated that long-term zinc supplementation had a salutary effect on plasma thiol metabolites and redox status in patients with age-related macular degeneration.⁹ Lower dose zinc is an inclusive part of the new AREDS II clinical trial.¹⁰

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