The Role of Nutrition in the Prevention of Age-Related Macular Degeneration

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The leading cause of blindness in the elderly is age-related macular degeneration (AMD). The number of cases is expected to reach 17.8 million in 2050 in the world which is almost double that reported in 2010 (9.1 million) [1]. This not only seriously affects the quality of life for those suffering from AMD but places a huge financial burden on society. For example, the costs associated with caring for AMD patients in the USA is already in order of $340 billion [2]. While its treatment is limited, any nutritional interventions that can prevent or slow down the development of AMD would accrue significant social and economic benefits to an already severely stressed world economy.

Research conducted over the past two decades points to a number of potentially beneficial nutritional strategies. These include treatment with polyunsaturated fatty acids, carotenoids, some vitamins and zinc.

The retina is very rich in lipids which account for 20% of its dry weight. These lipids are composed of around 60% polyunsaturated fatty acids with a high concentration of docosahexaenoic acid (DHA) in the outer segments of the photoreceptors [3]. Prolonged exposure of the retina to radiant energy and high oxygen consumption suggested the possible role of oxidative stress in the pathology of AMD [4-7]. Extensive research has since identified the protective effects exerted by dietary intake of omega-3 fatty acids, obtained from fish or nuts [3,8].

In addition to omega-3 fatty acid sources, antioxidants may also provide some additional protection against the development AMD. A randomized clinical trial by the Age-Related Eye Disease Study Group (AREDS) conducted in 2001 reported that antioxidants (vitamin C, E and beta-carotene) and zinc significantly reduced the chances of developing advanced AMD and loss of vision by 25% compared to the placebo control [9]. Huang and co-workers [10] recently reported the beneficial effects of supplementing the carotenoids lutein and zeaxanthin in Chinese subjects exhibiting early signs of AMD. In addition to increasing the serum levels of both of lutein and zeaxanthin they also enhanced macular pigment optical density (MPOD) compared to the placebo group. Earlier work by Bian et al. [11] showed that both lutein and zeaxanthin modulated inflammatory responses following phototoxidation of cultured retinal pigment epithelial cells. One of the mechanisms was their ability to protect the proteosome from oxidative inactivation which could explain their role in reducing the risk of AMD. Abdel-Aal and co-workers [12] recently reviewed the importance of dietary sources of lutein and zeaxanthin to eye health.

Of the vitamins examined, there appeared to be an inverse association between vitamin E and the risk for AMD [13]. Nevertheless after a series of extensive studies vitamin E did not appear to be a strong preventative of AMD [14]. A recent study by Pahl and co-workers [15], found 1,25-dihydroxyvitamin D decreased HTRA1 promoter activity in the rhesus monkey. The AMD disease-associated region in humans is located in 10q25 which includes two genes ARMS2 and HTRA1. Pahl et al. [15] reported down regulation of HTRA1 in the rhesus monkey which could also be relevant to the expression of HTRA1 in humans. This discovery has important implications for the treating the development of dry AMD, the least aggressive form of AMD.

Research conducted so far, suggests the potential of dietary intervention for the prevention of the least aggressive dry form of AMD. While genetic factors, such as genes associated with immune system modulation and the complement system, may provide future screening methods for assessing those susceptible to AMD development. The current dietary intervention strategy appears the most effective way of preventing or delaying the onset of AMD.

REFERENCES


