

Eye and Central Nervous System Protection by Astaxanthin

Current theory on diseases and injuries of the eye and central nervous system is that they are caused by the increased generation and presence of singlet oxygen and other free radicals (superoxide, hydroxyl, hydrogen peroxide, etc.) or by decreased removal ability. This includes but is not limited to age-related macular degeneration, the leading cause of blindness in the United States, retinal arterial and venous occlusion, glaucoma and diabetic retinopathy and injuries resulting from trauma and inflammation.

These free radicals are generated by continuous or excessive exposure to light and the highly oxygenated environment of the normal eye, ischemia (some form of blockage that deprives the eye of nutrition and oxygen) and reperfusion (the reoxygenation of tissue after blockage removal), and enzymatic processes. Free radicals oxidize the polyunsaturated fatty acids in the retina which leads to functional impairment of the retinal cell membranes, causing temporary and permanent damage to the retinal cells. Once the retina is damaged, it cannot be replaced. An antioxidant that can reach the inner eye by crossing the blood-brain and blood-retinal barriers would certainly afford the eye protection from these damaging conditions.

Astaxanthin is a carotenoid not found in the eye. Dr. Mark Tso first of all proved that astaxanthin could cross the blood-brain and blood-retinal barriers by feeding astaxanthin to rats and finding it in their eyes. He then proved it protected the eye from light-induced damage, photoreceptor cell damage, ganglion cell damage, neuronal damage and inflammatory damage. Dr. Tso fed rats either astaxanthin or placebo, exposed them to damaging light and then compared the thickness of their retinas to a normal eye. The astaxanthin retina was 42 micrometers thick, the placebo retina 32 micrometers thick and the normal retina 45 micrometers thick. Astaxanthin provided significant protection.

In the ischemia-reperfusion experiment, rats were fed either astaxanthin or placebo, exposed to highly elevated intraocular pressure (ischemia) for an hour and returned to normal pressure (reperfusion). After a week, the retinas of the astaxanthin-treated rats were about 70 micrometers while the placebo retinas were 62 micrometers (normal being 120 micrometers). Once again, this is statistically significant protection.

Not only did astaxanthin protect the photoreceptor cells but rhodopsin levels also and performed better than beta-carotene used in the same type of experiment. Astaxanthin also exerts a protective effect on the central nervous system in general.

Tso, Mark O. M., Lam, Tim-Tak, "Method of Retarding and Ameliorating Central Nervous System and Eye Damage," Patent No. 5,527,533. Washington, D.C., U.S. Patent and Trademark Office, June 18, 1996.