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Therapeutic Reviews

Reactive Oxygen Species and the Aging Eye

Specific Role of Metabolically Active Mitochondria in Maintaining Lens Function and in the Initiation of the Oxidation-Induced Maturity Onset Cataract—A Novel Platform of Mitochondria-Targeted Antioxidants With Broad Therapeutic Potential for Redox Regulation and Detoxification of Oxidants in Eye Diseases

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The authors report the interest in the Intellectual Property of the described modalities protected with the patents. The authors bear primary responsibility for accuracy of made statements and employment of the described products and for the content and writing of the paper.

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Abstract

The aging eye appears to be at considerable risk from oxidative stress. A great deal of research indicates that dysfunctional mitochondria are the primary site of reactive oxygen species (ROS). More than 95% of $O_2^{-\cdot}$ produced during normal metabolism is generated by the electron transport chain in the inner mitochondrial membrane. Mitochondria are also the major target of ROS. Cataract formation, the opacification of the eye lens, is one of the leading causes of human blindness worldwide, accounting for 47.8% of all causes of blindness. Cataracts result from the

deposition of aggregated proteins in the eye lens and lens fiber cell plasma membrane damage, which causes clouding of the lens, light scattering, and obstruction of vision. ROS-induced damage in the lens cell may consist of oxidation of proteins, DNA damage, and/or lipid peroxidation, all of which have been implicated in cataractogenesis. This article is an attempt to integrate how mitochondrial ROS are altered in the aging eye along with those protective and repair therapeutic systems believed to regulate ROS levels in ocular tissues and how damage to these systems contributes to age-onset eye disease and cataract formation. Mitochondria-targeted antioxidants might be used to effectively prevent ROS-induced oxidation of lipids and proteins in the inner mitochondrial membrane in vivo. As a result of the combination of weak metal chelating, $OH^{\cdot}$ and lipid peroxyl radicals scavenging, reducing activities to liberated fatty acid, and phospholipid hydroperoxides, carnosine and carcinine appear to be physiological antioxidants able to efficiently protect the lipid phase of biologic membranes and aqueous environments and act as the antiapoptotic natural drug compounds The authors developed and patented the new ophthalmic compositions, including N-acetylcarnosine, acting as a prodrug of naturally targeted to mitochondria L-carnosine endowed with pluripotent antioxidant activities combined with mitochondria-targeted rechargeable antioxidant (either MitoVit E, Mito Q, or SkQs) as a potent medicine to treat ocular diseases. Such specificity is explained by the fact that developed compositions might be used to effectively prevent ROS-induced oxidation of lipids and proteins in the inner mitochondrial membrane in vivo and outside mitochondria in the cellular and tissue structures of the lens and eye compartments. Mitochondrial targeting of compounds with universal types of antioxidant activity represents a promising approach for treating a number of ROS-related ocular diseases of the aging eye and can be implicated in the management of cataracts.

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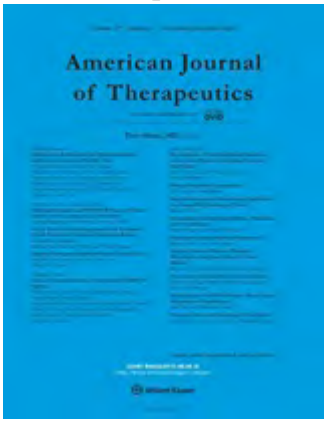

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