



## 5. IMPACT OF OXIDATIVE STRESS ON LENS PROTEINS

**Author:** Marciuc Vlada

**Scientific advisor:** Pavlovschi Ecaterina, Assistant Professor, Department of Biochemistry and Clinical Biochemistry, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova

**Introduction.** The impact of oxidative stress (OS) on lens proteins is a crucial aspect of ocular health, as the lens plays an essential role in visual function. The intricate interplay between oxidative stressors and lens proteins, shed a light on its potential implications for eye health and contribute to our understanding of age-related ocular conditions (ex. cataract).

**Aim of study.** To illustrate and convey the effects of oxidative stress on lens proteins, as well as its role in the formation of cataracts.

**Methods and materials.** 20 articles, selected from PubMed databases and published in the last two decades, were analyzed. The keywords employed in this analysis included “oxidative stress”, “cataract”, “lens proteins”.

**Results.** The eye is particularly susceptible to OS due to its continuous exposure to light and the high metabolic activity of various structures. The lens of the eye seems to be a unique organ that is shielded from OS through the regulation of oxygen tension. Lens transparency is maintained by lens proteins, primarily composed of  $\alpha$ A and  $\alpha$ B-crystallin, accounting for approximately 40-50% of the total. While all these proteins contribute to the lens's structure and refraction,  $\alpha$ -crystallin specifically prevents protein aggregation. Oxidation of this molecule leads to the loss of its chaperone functions, playing a crucial role in the development of cataracts. Mitochondrial respiration plays a key role in preserving diminished oxygen partial pressure in the lens, with cortical fiber cells's outermost layers, containing mitochondria that consume nearly 90% of incoming oxygen. The fundamental mechanism, evolved over evolution, maintains low oxygen concentrations within the lens, supported by the resistance to oxidation in both lipid components and cytoplasmic proteins. Disruption of this mechanism can lead to elevated oxygen levels and the onset of cataracts. The structural damage to the crystalline lens and its role in cataract formation result from free radicals, including reactive oxygen species like superoxide anion radical ( $\cdot O_2^-$ ),  $H_2O_2$ , and hydroxyl free radical (OH). OS occurs when pro-oxidants surpass antioxidant levels. A research demonstrate that a treatment of human lens epithelial cells with 17  $\beta$ -estradiol has been shown to protect against oxidative stress by preserving mitochondria and increasing Manganese Superoxide Dismutase (MnSOD) activity - an enzyme that plays a crucial role in antioxidant defense within cells. MnSOD regulates OS in lens epithelial cells through both up- and down-regulation of the enzyme.

**Conclusion.** The primary step in cataract development and lens protection is the maintenance of a low oxygen partial pressure at the lens surface. Recent global research emphasizes the significant role of oxidative stress in the increasing prevalence of cataracts worldwide, highlighting the importance of the lens's antioxidant defenses in preserving transparency.