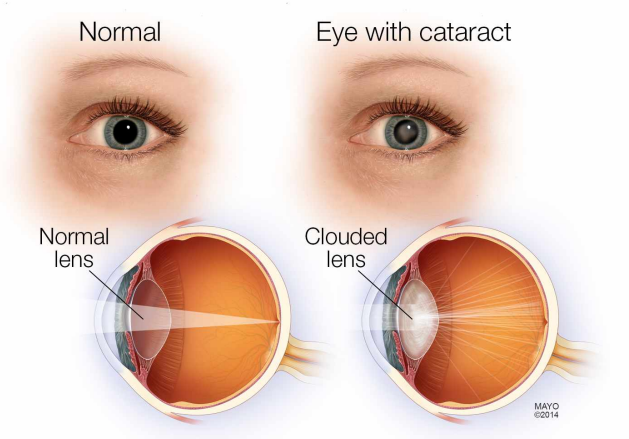
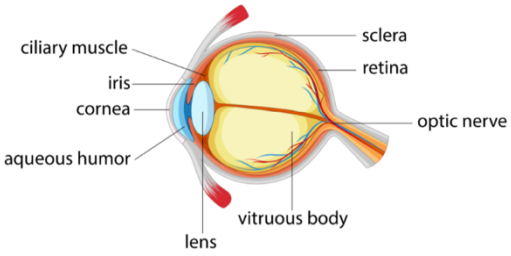
Cataracts Protocol

A cataract is a clouding of the normally clear lens of the eye. The eye lens is positioned behind the iris and focuses light that passes into the eye, producing a clear, sharp image on the retina. The retina is the light-sensitive membrane that functions like the film in a camera. For people who have cataracts, seeing through cloudy lenses is a bit like looking through a frosty or fogged-up window. Clouded vision caused by cataracts can make it more difficult to read and drive a car, especially at night. Most cataracts develop slowly and don't disturb eyesight early on. But with time, cataracts will eventually interfere with an individual's vision.

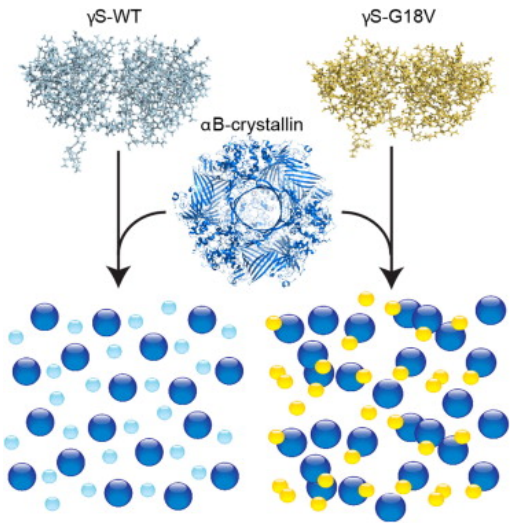


Cataract is the leading cause of reversible vision loss in the world. There are several risk factors for cataract formation with age being the most impactful. Age-related cataracts are responsible for nearly half of all blindness worldwide and half of all visual impairment in the United States. Other factors such as diabetes, glaucoma, and heritability are also known to be significant risks for cataract development. In addition, ultraviolet B radiation (UV-B), body mass index (BMI), smoking, diet, exposure to hyperbaric oxygen, and corticosteroids have been identified as extrinsic or environmental risk factors for cataract.



Cataracts affecting the central zone of the lens, also known as the nucleus, is called nuclear cataracts, that causes the lens to become a yellow color which eventually turns the lens brown. Nuclear cataract is the most common type of age-related cataract. Cataracts that affect the edges of the lens, called cortical cataracts, causes a whitish streak on the outer edges of the lens cortex. As the cortical cataracts progresses, the streaks extend to the center and interferes with light passing through the center of the lens. Diabetes is the main cause of cortical cataracts. There is also a form of cataracts that affects the back of the lens called posterior subcapsular cataracts. A small opaque area forms near the back of the lens, in the path of light. This form of cataracts typically progresses faster than the other two and causes halos around lights. Cortical cataracts and nuclear cataracts are the most common types.

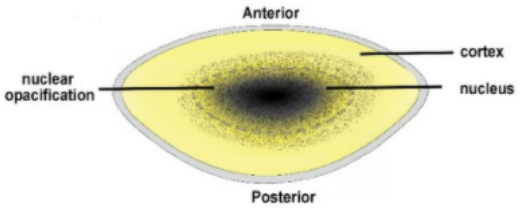
Oxidative stress is a significant factor in the pathogenesis of senile cataract. The oxidative processes increase with age in the human lens that leads to an aggregation of the crystallin inside the fiber cell causing opaque lenses. A crystallin is a water-soluble structural protein in the lens and the cornea and it accounts for the transparency of the structure. The main function of crystallin in the lens is to increase the refractive index while not obstructing light. Crystallin from a vertebrate eye lens are classified into three main types: alpha, beta and gamma crystallin. Transparency in the eye lens is maintained via specific, functional interactions among the structural βγ- and chaperone α-crystallin's. Crosslink of these proteins cause diminished transparency.[1]



Some inherited genetic disorders can also increase the risk of cataracts. For example, G18V variant of human γS-crystallin (γS-G18V) has found to be linked to hereditary childhood-onset cortical cataract because the mutant protein increases its aggregation with αB-crystallins.[1]

Age-related nuclear cataract

Age-related nuclear cataract is initiated in the central core of the lens, which contains primary fiber cells. Oxidative stress is a key factor in nuclear cataract formation. An increase of reactive oxygen species such as through UV exposure or increased oxygen partial pressure and reduction of endogenous antioxidants such as glutathione (GSH) or decrease in antioxidant enzymes change the redox state. It causes lens crystallin proteins to change sulfhydryl groups to thiolated or become cross-linked. The resultant high molecular weight aggregates become insoluble which affects lens transparency, increases yellowing of the lens nucleus, and eventually cause nuclear cataract formation.

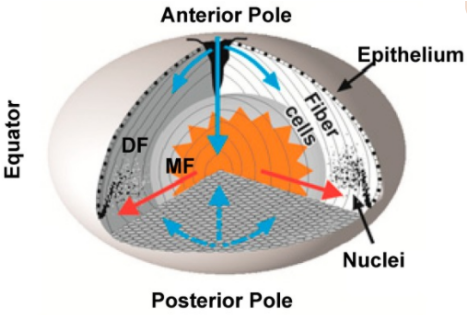


Increased oxidative stress occurs in the fiber cells when glutathione (GSH), the antioxidant in the nucleus, is depleted and the normal robust oxygen radical scavenger systems in the lens fail with advancing age. The eye lens is an avascular organ built from thousands of concentric layers of fiber cells. Fiber cells lose their intracellular organelles including the nucleus and mitochondria soon after they are formed.

At younger ages, the lens is protected against oxidative damage by a robust oxygen radical scavenger system, which utilizes GSH as its principal antioxidant to detoxify reactive oxygen species (ROS). The high levels of GSH in the outer cortex are maintained by a combination of de novo synthesis; direct uptake of GSH from the aqueous; and the recycling of GSSG. In contrast, mature fiber cells in the lens nucleus have lost their capacity to synthesis GSH, and cells can only maintain their GSH levels by delivery of GSH to the nucleus and/or local regeneration of GSH by GR. In age-related nucleus cataract, GSH levels in the nucleus are significantly depleted due to an inability to maintain GSH levels in the lens nucleus caused by the failure to deliver GSH to the lens center.

The lens is not a purely passive optical element. It requires nutrients supply to maintain its deeper lying fiber cell architecture and control the volume of these cells. The avascular lens cannot rely on passive diffusion alone to transport nutrients and antioxidants from the surrounding humors (aqueous humor and vitreous humor) to deeper lying cells, or to transport waste products back to the surface. It has been proposed that the lens operates an internal microcirculation system, which delivers nutrients and antioxidants to the lens core and removes waste faster than would occur by passive diffusion alone.

The nutrient delivery and waste removal are actively operated via an extracellular route driven by the lens microcirculation system. Such microcirculation system employs a circulating current of Na^+ ions that enters at the poles and travels into the lens via the extracellular clefts between fiber cells and returns towards the surface at the equator by crossing fiber cell membranes, where it is actively removed by Na^+ pumps at the lens equator using ATP as the energy source.[2] This circulating ionic current creates a fluid flow that powers a nutrient/antioxidant uptake and removal of metabolic waste from the center core. The peripheral differentiating fiber cells contain mitochondria, while mature fiber cells are devoid of mitochondria. Reduced ATP production by the differentiating fiber cells due to aging can compromise the lens's microcirculation system resulting in undersupply of the GSH to the lens center and leads to the development of nuclear cataract.[2]

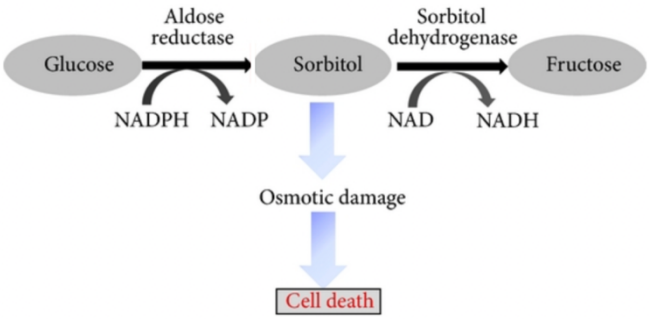


Cortical Cataract

Cortical cataract presents as wedge-shaped or radial spoke opacifications in the lens cortex. It is particularly prevalent in diabetic patients. Cortical cataracts tend to be associated with significant astigmatic shifts, caused by asymmetrical refractive index changes within the lens cortex. This change in refractive index is most likely due to accumulation of fluid in the lens cortex since, at the cellular level, this light scattering is due to a discrete localized zone of cell liquefaction.

An increase of glucose triggers the aldose reductase to catalyze the conversion of glucose to sorbitol, the first step of polyol pathway. This reaction is NADPH dependent and the conversion of NADP to NADPH consumes GSH.[3]

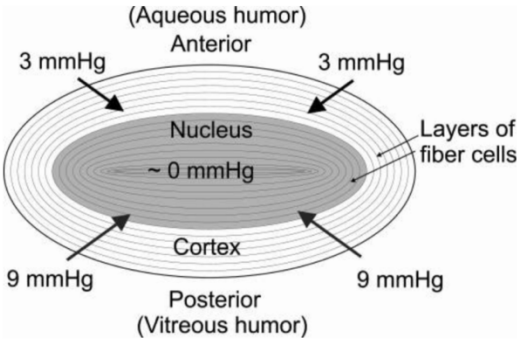
Depletion of GSH causes an increase in ROS. In addition, sorbitol is an osmolyte which increase osmotic stress and attracts fluid to the fiber cell causing fiber cell swelling and liquefaction in the lens cortex. Cell swelling can also cause depolarization with an influx of sodium and calcium ions which triggers the activation of calcium-dependent proteases that damage the cell protein structure. The high glucose level can also cause protein glycation of crystallins resulting in decreased solubility and formation of high molecular weight aggregates and cataract formation.[3]



Effects of Oxygen Partial Pressure and Nutritional Support

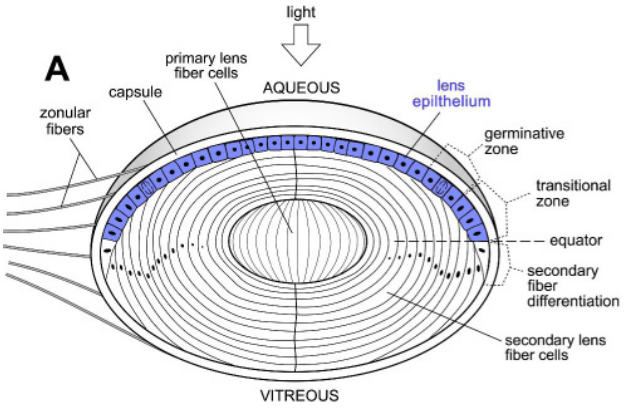
To protect the lens against any type of oxidative damage, an important mechanism that was "developed" during evolution is the maintenance of a very low oxygen partial pressure within the lens through the entire human life. The very low oxygen partial pressure, close to zero in the lens center has to be maintained. Any increase of oxygen partial pressure within the lens interior, caused by age, hyperbaric oxygen treatment, and vitrectomy, are capable of inducing cataract development through the formation of reactive oxygen species.

The mechanisms to maintain low oxygen partial pressure are barriers to oxygen permeation across layers of fiber-cell membranes and oxygen consumption within the lens interior. The oxygen consumption mechanism is through the mitochondrial respiration in the lens epithelium. The lens epithelium, located in the anterior portion of the lens between the lens capsule and the lens fibers, is a simple cuboidal epithelium. The cells of the lens epithelium regulate most of the homeostatic functions of the lens. In most tissues, oxygen partial pressures below 3% (~22 mmHg) are considered hypoxic. The oxygen level near the anterior lens epithelium can be as low as 0.4% (~3 mmHg) due to the oxygen consumption by the mitochondrial in the epithelium.[4] Mitochondria are abundant in the epithelium and differentiating fibers, but mature fibers in the core of the lens lack mitochondria. Mitochondria account for approximately 90% of the total oxygen consumption by the lens.[4]



Decreased nutritional supply due to the reduction of microcirculation of the eye microcapillaries from aging and other factors such as, radiation accumulation, can cause mitochondria decay in number and function which leads to low oxygen consumption in older people. Low oxygen consumption not only causes increased oxygen partial pressure at the lens but also contributes to decreased metabolism in the epithelium to support the lens structure and function leading to the cataract development. In a study involving patients treated with long-term hyperbaric oxygen therapy, it was demonstrated that the older human lens is more susceptible to oxygen-induced damage. In addition, lower oxygen consumption rate in lens equilibrium cells is also found in patients with diabetes or glaucoma which could contribute to cataract development.

The second mechanisms to maintain low oxygen partial pressure are barriers to oxygen permeation across layers of fiber-cell membranes. The lipid bilayer portion of lens membranes, with its unique lipid composition and structure, forms significant barriers to oxygen transport. As the lens need last many years any small amount of oxygen is harmful. During aging process additional mechanism have developed to ensure the lipid bilayer is even less permeable to oxygen.



The fiber-cell membrane phospholipid (PL) composite changes drastically during aging with the increase of sphingolipid content and depletion of phosphatidylcholine. The saturation levels of PL acyl chains also increase with age. The most characteristic change with age is the increase of cholesterol (chol) content up to the chol/PL molar ratio of 4. For the elderly population, the high chol content is often induced formation of cholesterol

crystals. These fiber cell membrane compositions change significantly with increased lens stiffness through the increased membrane mechanical rigidity. The result of increased lens stiffness is that fiber cell plasma membranes become less permeable to oxygen, which help to maintain lens transparency and protect against oxidative damage and cataract development. Such protection against cataract development is at the cost of losing lens flexibility which compromises the focusing property of the lens. The high cholesterol content in fiber-cell plasma membranes is critical to ensure that the lipid bilayer portion of the membranes become even more hydrophobic for permeation of polar molecules. The need for high chol in the eye lens is suggested by the observations that inherited defects in chol metabolism enzymes and the use of cholesterol-biosynthesis-inhibiting drugs contribute to cataract formation. Retrospective cohort studies comparing the risk of development of cataracts between statin users and non-users showed that the risk of cataract development was increased among statin users.

Wellness Recommendation

The wellness recommendation for cataracts includes OptiClear, Eye Brighter, LC Balancer, and Brown. OptiClear helps nurture the eye Yin to improve the fiber cell structure of the eye lens and improve the metabolism of the lens to supply sufficient GSH to the lens nuclear to reverse the protein crosslink. Herbal ingredients in OptiClear address eye health through effectively protecting the retina from neuronal death, apoptosis, glial cell activation, and oxidative stress.[5] Eye-Brighter nurtures eye Qi to enhance blood circulation to the eye and LC Balancer improves systemic microcirculation to enhance nutrient absorption and support the eye with necessary nutrient and improved mitochondrial function. Brown strengthens the liver and further supports eye health. Patients can experience symptom improvements in 3 weeks with clearer vision and up to 3 months of the protocol is recommended for significant improvement.

Selected Case Studies

Case 1: Improved Vision in Cataracts Patient
Michael Biamonte, ND, Nutritionist, CCN, FL

A 92-year-old male patient suffered from cataracts. He was experiencing poor vision, floaters, and double vision. He had to use a magnifying glass to enlarge the size of print and had to have light shining behind his shoulder in order to see anything.

Doctor Biamonte recommended the EyeBrighter formula from Wei Laboratories to improve his vision, and LC Balancer, Brown, and Xcel to improve kidney and liver function. The patient noticed an improvement in his symptoms right away after following the treatment protocol. In two days, his general vision improved. He no longer had double vision and was able to read better. The patient was very surprised. It was a fast and miraculous improvement!

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