

## CATARACTS

### Overview

Cataract is clouding of the eye lens that reduces the amount of incoming light and results in deteriorating vision. Cataract is often described as being similar to looking through a waterfall or waxed paper. Daily functions such as reading or driving a car may become difficult or impossible. Eyeglass prescriptions may require frequent changes. An estimated 200 million people worldwide have cataracts.

Minor lens opacities at birth may never progress to cataract in adulthood, while others progress to a degree requiring surgery or causing blindness. Many factors influence vision and cataract development including age, nutrition, heredity, medications, toxins, health habits, sunlight exposure, and head trauma. Hypertension, kidney disease, diabetes, or direct trauma to the eye can also cause cataract.

Today, cataract surgery is a common surgery in the United States, with over 1.5 million surgeries done yearly. Annual costs associated with cataract treatment are estimated to be over \$3.4 billion.<sup>1,2</sup> Cataract surgery costs Medicare more than any other medical procedure: 60% who initially qualify for Medicare already have cataracts.<sup>3</sup>

There are three main types of cataracts. The most common is nuclear cataract. Nuclear cataract occurs when proteins of the nucleus (center) degenerate and darken, causing light to scatter. The second most common type, cortical cataract, occurs in the cortex (or periphery) of the lens. Cortical cataract forms when the order of fibers in the cortex is disturbed and the gaps fill with water and debris, thus altering the pathophysiology of light by scattering and/or absorbing it. The least common type affects the back of the lens and is called posterior subcapsular cataract.

### Summary

This protocol provides information about cataracts; its nature, etiology, physiology, pathophysiology, and current treatments. Nutritional approaches to protecting the eye and preventing or slowing cataract progression of cataracts is provided. This information on cataracts and nutritional supplements should enable the reader to understand the beneficial effects of nutrition on cataract prevention.

### *Scientific Summary*

The most widely accepted conventional surgical treatment is removal of the lens and implantation of an artificial lens (IOL). Surgical treatment is recommended when a cataract progresses to the point that it impairs visual function. Before this point is reached, conventional medicine often takes a “watchful waiting” approach, considering cataracts to be an age-related, unfortunate, but inevitable, fact of life. In contrast are a growing contingent of physicians, researchers, and nutritional scientists who have a more proactive view of cataract prevention and treatment. This holistic approach to maintain healthy lens function and eye health includes awareness of risk factors (e.g., smoking, alcohol, and sunlight), compliance with a sensible diet (e.g., low-fat, high-fiber), exercising, and nutritional therapy specifically for the eye.

### *Lifestyle Changes*

Wear protective eyewear and avoid the following risk factors:

- Avoid smoking, excessive alcohol consumption, exposure to sunlight (particularly UV radiation), and excessive exposure to X-ray and gamma irradiation.
- Life Extension’s Solarshield sunglasses to protect from:
  - blue and UV radiation
  - preservation of essential macular pigments

### *Nutritional Supplements*

- Glutathione: 500 mg daily
- Vitamin C: 500 mg daily

- Vitamin B2: 50–150 mg daily
- Selenium: 200–400 mcg daily
- Vitamin E: 400 IU daily
- Gamma E Tocopherol w/Sesame Lignans: 1 softgel daily
- R-lipoic acid: 210–420 mg daily
- N-acetyl-cysteine: 600 mg daily
- Melatonin: 300 mcg–3 mg at bedtime
- Vitamin B6: 50-250 mg daily
- Acetyl-L-carnitine arginate: three-four capsules daily
- Aminoguanidine: 150–300 mg daily
- Carnosine: 500–1000 mg daily
- Life Extension Mix: 3 tablets, 3 times daily, provide N-acetyl-cysteine, selenium, inositol, vitamins B2, B6, C, and E, bioflavonoids, and many other antioxidants and anti-glycating nutrients.
- Brite Eyes II: One-two drops in each eye daily
- Lutein Plus: One tablespoon daily taken with a fatty meal.
- Super Zeaxanthin with Lutein: One-two capsules daily.
- Coenzyme Q10: 100–200 mg daily
- Potassium: 400 mg daily, but consult your physician
- Magnesium: 800 mg daily
- Gingko biloba: 120 mg daily
- Bilberry: 100 mg daily
- Kyolic Reserve Garlic: One-three capsules daily

**Note:** Many of the nutrients suggested may be contained in the Life Extension Mix

### **For More Information**

Contact the National Eye Health Education Program of the National Institutes of Health, (301) 496-5248, or the American Society of Cataract Surgery, (703) 591-2220.

### **Product Availability**

Life Extension Mix, Brite Eyes II, glutathione, Super Carnosine, Super Absorbable CoQ10, Solarshield sunglasses, Super Zeaxanthin with Lutein, vitamin C, vitamin B2, vitamin B6, vitamin E, Gamma E Tocopherol with Sesame Lignans, ginkgo biloba extract, bilberry extract, R-lipoic acid, potassium, magnesium, melatonin, Kyolic Reserve Garlic, N-acetyl-cysteine, acetyl-L-carnitine arginate, and selenium can be ordered by calling (800) 544-4440 or by ordering online at [www.lef.org](http://www.lef.org).

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## OVERVIEW

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## EPIDEMIOLOGY AND GENETICS

- Prevalence
- Risk Factors
- Genetics
- Symptoms and Disease Progression

### ***Prevalence***

Estimates are that 20.5 million Americans older than 40 years, representing 17.2% of that population, have a cataract in at least one eye and that 6.1 million (or 5.1%) have had cataract surgery to remove a lens (aphakia) or to replace a lens with an artificial lens (pseudophakia).<sup>4</sup> There is evidence that genetics plays a role in the formation of cataract, especially congenital cataract.<sup>5</sup> Cataract is seen primarily in adults, and the incidence grows rapidly after age 50, affecting 50% of individuals between 65 to 74 and 70% of individuals age 75 and older.<sup>6</sup> Because of the growing elderly population, by the year 2020, the number of individuals with a cataract could climb to 30.1 million; 9.5 million would be expected to have aphakia or pseudophakia.<sup>4</sup>

### ***Risk Factors***

Identification and awareness of risk factors for cataract could have an important benefit. Estimates are that if cataract onset could be delayed by 10 years, the number of cataract surgeries could be reduced annually by 45%.<sup>2</sup>

### **Gender**

In the United States, women have a significantly higher age-adjusted prevalence of cataract, with 58% of cataract cases.<sup>4</sup> Women have a higher risk for most types of cataracts,<sup>7</sup> though evidence suggests estrogen may protect against cataract formation.<sup>8</sup> The anti-estrogen drug tamoxifen (used to block estrogen receptors) increases risk of cataract when taken long-term.<sup>9</sup>

### **Education Status and Socioeconomic Factors**

Risk for cataract is greater among individuals with lower socioeconomic status or educational level. This is attributed to nutritional deficiencies from poor diet, increased exposure to disease, poor general health, and greater exposure to conditions inducing cataract development.<sup>2</sup>

### **Exposure to Excessive Sunlight**

Geographical areas with more hours of sunshine have a greater prevalence of cataract, showing an association between ultraviolet B irradiation and cataract formation.<sup>10</sup>

### **Exposure to Radiation**

Exposure to X-rays or gamma radiation is a risk factor for cortical and posterior subcapsular cataracts in humans. Radiologists routinely minimize exposing the lens to ionizing radiation. When this is not possible, cataracts frequently develop and require surgical treatment.<sup>11</sup>

### **Nutrition**

A diet lacking a high intake of antioxidants, particularly vitamins A, C, and E, fails to protect the lens from cataract formation.<sup>12,13</sup>

### **Smoking and Alcohol**

There is an increased risk of nuclear cataract smokers.<sup>14,15</sup> Risks for all cataract types increase with heavy alcohol consumption.<sup>7</sup>

### **Diabetes**

Diabetics are more likely to develop cortical opacities or require cataract surgery.<sup>16</sup>

### **Corticosteroids**

Corticosteroid use is associated with posterior subcapsular cataracts.<sup>17</sup>

### **Genetics**

Lens-specific genes include genes encoding proteins for growth and transformation of lens fiber cells (cystallins) and mediation of cellular respiration and metabolism, such as major intrinsic polypeptide (MIP) and certain connexins.<sup>18,19</sup> Mutations in lens-specific genes are associated with hereditary cataracts possibly through a mechanism which produces a protein interfering with normal proteins, thus disrupting normal function and cataract formation.<sup>20</sup>

Numerous hereditary syndromes manifest cataracts as a characteristic feature. The gene mutations are identified for some syndromes: Lowe's syndrome, neurofibromatosis type 2, galactosemia, and Werner syndrome.<sup>21,22</sup> In most diseases identifying the genes responsible for the development of the cataract offers no explanation as to why cataracts manifest.<sup>20</sup> A better understanding of biochemical and molecular mechanisms underlying cataract formation may provide more information about cataract formation.<sup>5,23</sup>

## **Symptoms and Disease Progression**

### **Symptoms**

*Decreased visual acuity.* Decrease in visual acuity, a measure of eyesight sharpness and focus, is one of the first signs of cataract. Measurement of visual acuity is most commonly used to detect changes in visual function caused by cataracts (and other causes) over time.<sup>24</sup> Often an individual with a cataract will notice worsening vision that requires frequent changes to a stronger lens correction. Visual acuity testing uses the Snellen Visual Acuity Chart.

A Snellen test is not always the best measure of cataract or indicator for surgery. Clinically, visual acuity can remain high despite age-related lens opacities. The Snellen test may not always reflect visual disabilities occurring under less than ideal (clinical) circumstances, as with contrast sensitivity.<sup>24</sup> The Preferred Practice Pattern of the American Academy of Ophthalmology recommends Snellen acuity tests as the best guide for appropriateness of surgery with respect to the patient's functional and visual needs, environment, and risk factors.<sup>25</sup>

*Reduced contrast sensitivity and glare.* Common complaints are loss of ability to see objects in bright sunlight and being blinded by strong lights at night, such as oncoming headlights.<sup>26</sup> All cataracts lower contrast sensitivity, but do so most severely in posterior subcapsular cataract. Cataracts that reduce contrast sensitivity normally occur within the pupil diameter.

Complaints of glare are another symptom of cataracts. Even minor degrees of lens opacity produce glare because of the scatter of light toward the front of the lens. All cataract forms can cause glare, especially cortical and posterior subcapsular types. Patients with glare symptoms frequently have poorer vision in daylight conditions and when driving. Unlike contrast sensitivity reduction, some glare can be produced by opacities not within the pupil diameter.<sup>24</sup>

*Myopic shift.* The natural aging process in human lens produces a progressive shift toward hyperopia (i.e., farsightedness). When a cataract is forming in the nucleus of the lens, clouding of the lens changes the way light bends (or refracts). This produces greater nearsightedness (a myopic shift). Myopic shift enables an aging person who previously needed reading glasses to read without corrective lens. This phenomenon called "second sight" is indicative of hardening of the lens nucleus, a predictor of a

developing nuclear cataract.<sup>27</sup>

**Double vision and color shift.** Other common signs of cataract are double vision in one eye and change in color vision. Monocular diplopia (double vision in one eye) occurs with lens opacities, particularly cortical spoke cataracts. In cortical spoke cataracts, water clefts form radial wedge shapes that contain a fluid with a lower refractive index than the surrounding lens. All light entering the lens is not bent to the same extent; producing double or multiple images. There may be a perception of haloes around light.<sup>28</sup>

Color shift is produced by a lens that is more absorbent at the blue end of the spectrum, causing color perception to fade. Color shift is common with nuclear cataracts. Usually patients are not aware of a defect in color perception, although it becomes apparent after cataract surgery when they readjust to normal color perception.<sup>28</sup>

### **Disease Progression**

**Observation and assessment.** Cataracts are usually observed and assessed with a slit lamp biomicroscope; a microscope with two eyepieces. Different magnifications combined with a strong light are focused into a slit to examine the eye. A slit lamp examination measures visual acuity and the amount of light scattered in the eye.<sup>27</sup> Cataracts can be detected with a funduscope, an optical instrument that inspects the retina. Retinal blood vessels are blurred by light scattering caused by opacity in the lens.

**Lens clouding.** Disease progression in all types of cataract is indicated by increased lens opacity, though opacity manifests differently in each type.

**Nuclear cataract.** In nuclear cataract, lens density initially increases in the central lens nucleus. Opacity follows producing color changes beginning clear, changing to yellow, and to brown at more advanced states.<sup>28</sup>

**Cortical cataract.** Changes in transparency involve the periphery (or cortex) of the lens. Water gets into the lens cortex and creates pockets under the lens capsule called vacuoles. The vacuoles gradually lengthen into ray-like spaces and fill with fluid which is first transparent and later opaque. Vacuoles begin at the periphery and gradually spread toward the center, taking on an appearance of wedges or spokes. Because a cortical cataract begins at the periphery, vision may not be affected at first, but eventually visual acuity decreases.<sup>28</sup>

**Posterior subcapsular cataract.** Cataracts form in front of the posterior capsule as a cluster of swollen cells. The posterior capsule is the lens casing at the back of the lens. These cataracts develop as independent, isolated entities, but are associated with cataract formation in nuclear or cortical regions. Granules and vacuoles in front of the posterior capsule are signs of posterior subcapsular cataract. Although they are not common, progression and severity can be more extreme than other types.<sup>28</sup>

**Cataract classification.** Cataracts are immature, mature, and hypermature. A lens with remaining clear areas is an immature cataract. A mature cataract is completely opaque. A hypermature cataract has a liquefied surface that leaks through the capsule. The leaking material can cause inflammation in other eye structures.<sup>6</sup>

## **ETIOLOGY AND MECHANISMS OF ACTION**

- Cataract: Underlying Causal Mechanisms
- Age-Related Cataracts: Specific Causal Mechanisms

### ***Cataract: Underlying Causal Mechanisms***

Cataract is any type of opacification of the lens. Cataract is considered clinically significant when opacification interferes with visual function. Decreased lens transparency results in increased light scattering as light passes through the lens and then to the retina where the diminished focus of light impairs vision. Cataract adversely impacts vision by light absorption in a less transparent lens.

The underlying mechanism for cataracts involves: disruption of the structure of the lens fiber cells, increases in protein aggregation, or cytoplasm dysfunction in the lens cell.<sup>29</sup>

### ***Age-Related Cataracts: Specific Causal Mechanisms***

Each type of age-related cataract has a specific mechanism that leads to their development. These include: oxidative damage, protein aggregation, breakdown of the glutathione, damage to fiber cell membranes, protein breakdown, elevated calcium, abnormal lens epithelial cell migration, or aberrant changes in lens fiber cells.

## **Nuclear Cataract**

Nuclear cataracts show increased oxidative damage to lens proteins and lipids<sup>30</sup> causing protein-to-protein interactions that cause aggregation and increase light scattering. A lens with a cataract has increased interaction between crystallins and lens fiber cell membranes.<sup>31</sup>

Evidence suggests a strong connection between aging and increased amounts of oxidized glutathione in the lens nucleus indicative of an imbalance between protein and lipid oxidation, and glutathione-dependent reduction.<sup>32,33</sup> Nuclear cataract formation may be caused by separation of lens cell cytoplasm (a jelly-like substance) into protein-rich and protein-poor liquid phases,<sup>34</sup> accounting for the opacity.<sup>35</sup>

## **Cortical Cataract**

Cortical opacities start in small regions of the lens periphery. Opacity may spread around the circumference of the lens. Several mechanisms may initiate the cortical cataract: damage to the fiber cell plasma membrane, loss of protective molecules such as glutathione, excessive breakdown of proteins (proteolysis), and damage to systems responsible for calcium homeostasis. These factors are interrelated because any one of them leads to the others in the initial formation of cortical cataracts.<sup>20</sup>

Loss of calcium homeostasis spreads opacification around the lens periphery and towards the nucleus. Calcium levels are elevated in damaged cells in cortical cataracts.<sup>36</sup> Elevated calcium leads to proteolysis, protein aggregation, and light scattering.<sup>37</sup>

## **Posterior Subcapsular Cataract**

These cataracts are caused by environmental stresses such as ultraviolet light, diabetes, and drug ingestion.<sup>2,38</sup> Light scattering occurs in a cluster of swollen cells at the back of the lens, beneath the lens capsule. Because opacity produced by the cell cluster is within the optical axis (or the line of sight), these cataracts can be particularly debilitating. These cataracts are associated with abnormal migration of lens epithelial cells or aberrant changes in lens fiber cells at the back of the lens.<sup>39</sup>

## **ANATOMY AND PHYSIOLOGY (STRUCTURE AND FUNCTION)**

- The Lens
- Zonules
- Refractive Properties of the Lens

### ***The Lens***

A lens is formed from specialized epithelial cells during embryonic development. The epithelium is a sheet of cube-shaped cells covering the anterior surface of the lens near the cornea. The major part of the lens consists of concentric layers of elongated fiber cells. The outermost shells of fiber cells extend from beneath the epithelium to the posterior lens surface near the vitreous body. The lens is one centimeter from front to back, surrounded by the capsule--an elastic matrix of cells produced during embryonic development by secretions from epithelial and fiber cells on the lens surface.<sup>40</sup>

In an adult lens, only a few epithelial cells replicate, proliferating slowly, producing new fiber cells that elongate and accumulate crystallins (lens proteins). Crystallins give the lens its refractive power to focus light on the retina.<sup>41</sup> During maturation layers of fiber cells build up.<sup>42</sup>

After the elongation process, a differentiation begins that degrades all intracellular, membrane-bound organelles.<sup>43</sup> Mature fiber cells are buried deeper within the lens as generations of fiber cells go through this process. The lens increases in size and cell numbers throughout life.<sup>44</sup> Because protein synthesis stops with organelle degradation, mature fiber cells are more stable than cells having other functions in the body.<sup>45</sup>

### ***Zonules***

The lens is suspended by inelastic microfibrils called zonules located above and below the lens in the anterior part of the lens and extending into the lens capsule. Zonules are inelastic compared to other fibrils in the body (e.g., in the skin and arterial walls), but stretch enough to create the tension responsible for altering lens curvature. This is required for focusing on objects at different distances, a process known as accommodation.<sup>46</sup>

### ***Refractive Properties of the Lens***

The refractive properties of the lens result from the high concentration of crystallins in the cytoplasm of lens fiber cells and the curvature of the lens. Lens crystallins are water-soluble proteins in lens fibers that provide a high refractive index. The lens is able to focus light on photoreceptors in the retina.<sup>47</sup> In a healthy lens, refractive error is caused by abnormalities in corneal curvature or length of the ocular globe, but rarely from defects in the curvature or refractive index of the lens itself.<sup>20</sup>

An essential component of lens transparency is a high concentration of lens crystallins and minimization of light scattering and absorption. Light passes through the lens because of the regular structure of lens fibers, an absence of membrane-bound organelles, and small, uniform spaces between the cells. This reduced light scattering is due to short-range interactions among densely packed crystalline molecules.<sup>48</sup>

# CATARACTS

## PATHOPHYSIOLOGY

- Nuclear Cataract Formation
- Cortical Cataract Formation
- Posterior Subcapsular Cataract Formation

### ***Nuclear Cataract Formation***

Cataract formation, especially in nuclear cataracts, is caused by oxidative stress that occurs in all biological systems and particularly the lens. Oxidative stress and generation of free radicals results from normal activity of mitochondria and other metabolic processes.<sup>49</sup> Oxidation is controlled by an environment of reducing agents. Reducing agents produced in the mitochondria neutralize free radicals.

Production of reducing agents requires energy output, a challenge for the deeper lens fiber cells that lack mitochondria. The enzyme systems in deeper cells are less active because they were synthesized decades earlier.<sup>20</sup> These central lens fiber cells are delicate balanced between being damaged by oxidation of membrane lipids and cytoplasmic protein, and being protected by reducing agents transported from epithelial cells and immature lens fiber cells near the surface. Transport of reducing agents is difficult because there is little space between lens fiber cells. Movement is by diffusion.<sup>50</sup>

Another challenge is maintenance of protein stability for many decades. Once a lens is formed, proteins are synthesized in outer fiber cells close to the surface. Proteins deeper in the lens generated during embryogenesis have to last a hundred years or more. Accumulated damage to these proteins reduces enzymatic activity and increases protein aggregation, a component of cataract formation.<sup>29</sup>

### ***Cortical Cataract Formation***

Unlike nuclear cataracts, cortical cataracts show disorganization of fiber cell structure. Causes of cortical cataracts include loss of calcium balance, protein breakdown and aggregation, and diminished antioxidant protection (from glutathione). There is evidence for a genetic cause of cataract formation.<sup>51</sup> There is no overall explanation why initial damage is restricted to the center of affected cells or why the preferred location of cortical cataracts is the lower half of the lens.<sup>52</sup>

### ***Posterior Subcapsular Cataract Formation***

Posterior subcapsular cataracts are less common and occur with the other two types. A “pure” posterior subcapsular cataract is uncommon, occurring in only 10% of cases.<sup>16,53</sup>

An important risk factor in posterior subcapsular cataract development (and cortical cataracts) is exposure to excessive X-ray or gamma-radiation.<sup>54</sup> Mechanisms that initiate cellular or molecular dysfunction are poorly understood.<sup>20</sup>

## ENDOCRINOLOGY AND BIOCHEMISTRY (REGULATION AND METABOLISM)

- Energy Sources
- Oxidative Damage: Protective Biomechanisms

### ***Energy Sources***

The lens' oxygen concentration is lower than most parts of the body because it has no direct blood supply.<sup>55</sup> The lens depends on glycolytic metabolism to produce much of the adenosine triphosphate (ATP) and reducing agents for metabolism.<sup>56</sup>

Glycolysis is the process by which sugars (like glucose) are metabolized to produce the energy currency of the body, adenosine triphosphate (ATP). When glycolysis occurs in differentiated lens fiber cells deep within the lens, the absence of oxygen

(anaerobic glycolysis) only allows 10% of the energy available to be conserved. The glucose comes from the aqueous humor, the fluid sac between the lens and cornea. Energy from glucose is derived from (aerobic) oxidative pathways in superficial lens fiber cells and epithelial cells containing mitochondria. In animal studies, 50% of the ATP produced by epithelial cells came from oxidative metabolism and glycolysis accounted for almost all ATP produced in most lens fiber cells.<sup>56</sup>

## ***Oxidative Damage: Protective Biomechanisms***

### **Glutathione**

Although the oxygen level within the lens is very low, the lens still derives a substantial proportion of ATP from mitochondrial (aerobic) oxidative phosphorylation, which creates free radicals as an unwanted by-product. Glutathione provides the most important protection against damage from free radical and other oxidants.<sup>57</sup> Glutathione is a very small specialized protein (a tripeptide) consisting of three amino acids: glutamic acid, cysteine, and glycine. Glutathione is concentrated within the lens and is readily oxidized by damaging oxidants. Those oxidants are chemically reduced (neutralized) as glutathione is chemically oxidized in cytoplasm of cells within the lens. When glutathione levels decline in the epithelial cells (or the entire lens), cell damage and cataract formation can occur unabated.<sup>58</sup>

Lens epithelial cells and superficial lens fiber cells synthesize glutathione. Additional glutathione is transported into the lens from the aqueous humor.<sup>59</sup> Oxidized glutathione can be regenerated (i.e., reduced) by the enzyme glutathione reductase that uses the coenzyme called reduced nicotinamide adenine dinucleotide phosphate (NADPH), which is the cofactor derived from the dietary or supplemental B vitamin: niacin or niacinamide, also known as vitamin B3.<sup>57</sup> Regeneration of reduced glutathione from oxidized glutathione is especially important because it is the chemically reduced form of glutathione that is effective in neutralizing (chemically reducing) free radicals. Glutathione is unique in its ability to regenerate its chemically reduced state by simply finding an electron donor. This cycle allows one molecule of glutathione to continually act as a free radical scavenger.

Reduced glutathione diffuses into the lens fiber cells, moving toward the lens center, while oxidized glutathione moves toward the lens surface.<sup>33</sup> Impediment of diffusion in an older lens is a possible cause of nuclear cataract.<sup>33</sup> The rate of diffusion between superficial and deeper layers of the lens decreases with age. Consequently, proteins and lipids in nuclei of older lens are more affected by oxidative stress.

### **Vitamin C**

Ascorbic acid (vitamin C) protects the lens from oxidative damage. In the aqueous humor, ascorbic acid reaches concentrations that are 30 to 50 times the levels in blood. Ascorbic acid is in the lens and surrounding ocular tissues in substantial quantities.<sup>60</sup> Dehydroascorbate (the oxidized form of ascorbic acid) can enter the lens through a glucose transporter. It is then reduced by glutathione-dependent processes.<sup>61</sup> Ascorbic acid reacts readily with free radicals and other oxidants in the aqueous humor and lens, preventing damage to lens proteins, lipids, and nucleic acids.

## **PHARMACOLOGY**

### ■ Conventional Therapy

No successful anti-cataract drug is available. Research continues on possible anti-cataract agents, including nonsteroidal anti-inflammatory drugs (NSAIDs) such as salicylic acid and ibuprofen. Animal trials have tested the effects of aldose reductase inhibitors. High levels of aldose reductase (an enzyme) are associated with diabetic cataracts. No clinical trials have demonstrated that these substances have any convincing anti-cataract effect.<sup>62</sup>

### ***Conventional Therapy***

#### **Surgical Removal and Intraocular Lens Implantation**

The most common treatment is surgical removal of the cataract and replacement with an artificial lens. Widely used surgical procedures are phacoemulsification and extracapsular extraction. In phacoemulsification, a small incision is made in the cornea. A probe vibrating with ultrasound waves is then used to emulsify the cataract and the fragments are removed by suction. The lens capsule is left in place to provide support for a lens implant.<sup>63</sup>

If a cataract has advanced to an extent that phacoemulsification cannot effectively break up the lens, the preferred alternative is extracapsular extraction, requiring a larger incision so the lens nucleus is removed in one piece through the open lens capsule. The softer lens cortex is vacuumed out, leaving the shell in one piece.<sup>63</sup>

After the cataract is removed, an artificial lens is implanted into the empty lens capsule. This implant, an intraocular lens (IOL), is made of plastic, acrylic, or silicone. An IOL requires no care and becomes a permanent part of the eye. Early IOLs were rigid

plastic and the incision required several sutures. IOLs currently used are flexible, allowing a smaller incision requiring no sutures. Flexible IOLs are folded by a surgeon and inserted into the capsule. Reading glasses will be required after surgery.<sup>63</sup>

## **Secondary Cataracts**

A common complication of extracapsular cataract extraction is formation of secondary cataracts. Secondary cataracts occur because lens epithelial cells migrate under the IOL to the posterior capsule that has been denuded of cells by surgery. These cells are then abnormally transformed into a mass of fiber-like cells (globular clusters) or fibrotic plaques, which scatter light, degrade visual images, and cause secondary cataract formation.<sup>20</sup> Secondary cataracts develop postoperatively in one out of two cases.<sup>63</sup>

A common, effective method for secondary cataract removal uses a laser procedure called YAG capsulotomy. A YAG (yttrium aluminum garnet) laser delivers tiny, rapid bursts of energy that pass through the front of the eye and the IOL. When the laser beam reaches the posterior capsule, it makes a tiny opening. Light can then pass into the vitreous body and reach the retina. Enough of the posterior capsule is left to hold the IOL in place.<sup>63</sup>

## **NUTRITIONAL THERAPY**

- Protection from Free Radical Damage
- Lens Protein Protection
- Lens Metabolism Support
- Ocular Environment Support

### ***Protection from Free Radical Damage***

The benefits of dietary supplements for cataracts are widely documented. Free-radical action is directly linked to cataracts and is a major cause of damage to eyes and cataract formation.<sup>64</sup> Numerous studies have documented the effects of supplements, including their ability to reduce free-radical damage and reverse the damage in some cases.<sup>65,66</sup>

### **Maintaining Glutathione Levels**

A healthy eye contains glutathione in very high concentrations, whereas low levels adversely effect the eye.<sup>67</sup> Glutathione maintains the water balance in the lens. It is synthesized in the lens (and elsewhere) and is essential to normal metabolism. Glutathione can benefit lens function by:<sup>40,57</sup>

- Preserving the physicochemical integrity of proteins in the lens<sup>33</sup>
- Maintaining action of the sodium-potassium transport pump and molecular integrity of lens fibers (protein)<sup>33</sup>
- Maintaining molecular integrity of lens fiber membranes and acting as a free radical scavenger to protect membranes and enzymes from oxidation<sup>66</sup>
- Preventing free-radical-induced photochemical generation of harmful by-products<sup>61</sup>
- Reactivating oxidized vitamin C, which improves antioxidant capability in the lens<sup>68</sup>

A suggested glutathione dose is 500 mg daily.

### **Vitamin C**

Vitamin C (ascorbic acid) is essential for normal ocular metabolism and occurs in the lens at a concentration 30-50 times higher than blood. This concentration is second only to the central nervous system and adrenal cortex. Vitamin C is found in high concentrations in eyes of animals active during daylight hours; low concentrations are found in nocturnal animals.<sup>69</sup> Prior to cataract formation, vitamin C concentrations significantly drop. Vitamin C provides protective benefits for the lens by:<sup>70,71</sup>

- Protecting the lens from photochemical oxidation<sup>72</sup>
- Helping increase levels of glutathione<sup>57</sup>
- Supporting delicate membranes regulating transport of nutrients and ions (minerals and electrolytes) into the lens<sup>60</sup>
- Protecting against damaging UV radiation and visible light<sup>73</sup>
- Protecting against superoxide radical, O<sub>2</sub><sup>-</sup> (known to be extremely destructive in every cell)<sup>74</sup>

A suggested dose of vitamin C is 500 mg daily.

### **Vitamin B2**

Vitamin B2 (riboflavin) is a required precursor to the cofactor, reduced flavin adenine dinucleotide (FADH) used by glutathione

reductase, which in the lens enzymatically reduces, and thereby, activates glutathione; and makes that glutathione available for the enzyme glutathione-selenium peroxidase, which chemically reduces peroxide free radicals to harmless water. Deficiency of glutathione creates a faulty antioxidant defense system in the lens.<sup>75</sup>

Light, especially ultraviolet (UV) light, destroys riboflavin and FADH. Most B vitamins are not stored so they must be replaced daily. Riboflavin deficiency is a prime cause of photosensitivity making the eye more sensitive to UV damage. A daily dose of 50 to 150 mg of riboflavin reduces this photosensitivity.<sup>76</sup>

### **Selenium and Vitamin E**

Low plasma levels of vitamin E increase the risk of lens opacities.<sup>77</sup> Selenium works with alpha-lipoic acid to increase cellular concentrations of glutathione, which protects the eye lens from free radical damage.<sup>74</sup> Taking 400–800 IU daily of vitamin E and 200–400 mcg daily of selenium is prudent to protect the lens from cataract formation and maintain overall good health.

*Note: See Appendix for Cautions and Contraindications*

### **Alpha-Lipoic Acid**

Supplementation of animals with alpha-lipoic acid prevents cataract formation resulting from inhibition of glutathione synthesis. Alpha-lipoic acid reduced cataract formation by 40% and protected the lens from losing vitamins C, E, and glutathione. Unsupplemented animals lose these nutrients.<sup>78</sup> A suggested dose of lipoic acid is 150-300 mg daily.

### **N-Acetyl-Cysteine and Garlic**

A combination of diallyl disulfide (a major organosulfide in garlic oil) and N-acetyl-cysteine (NAC) completely prevented cataract development in animals.<sup>79</sup> NAC assists in glutathione production because it is a source of cysteine, one of the three amino acids in this tripeptide.<sup>80</sup> A suggested dose of NAC is 600 mg daily.

*Note: See Appendix for Cautions and Contraindications*

### **Melatonin**

Melatonin is an antioxidant that could impede cataract development. In animals,<sup>81</sup> melatonin potently inhibited cataract formation, due to free-radical scavenging or through stimulation of glutathione production. Melatonin production slows after age 40, but by age 60 virtually no melatonin is produced at a time when most cataracts develop. A suggested dose of melatonin is 500 mcg to 3 mg at bedtime.

*Note: See Appendix for Cautions and Contraindications*

## **Lens Protein Protection**

### **Vitamin B6**

Vitamin B6 (pyridoxine) is essential for amino acid and protein metabolism, absorption of vitamin B12, and proper synthesis of nucleic acids. Its coenzyme is required for many reactions of amino acids and related metabolic functions. Vitamin B6 is suggested for nutritional support for cataract patients.<sup>82</sup> A suggested dose of vitamin B6 is 50-250 mg daily.

### **Acetyl-L-Carnitine**

Acetyl-L-carnitine is an amino acid that maintains cellular metabolism of fatty acids. During aging, mitochondria (energy-producing organelles within the cell) begin to deteriorate, resulting in accumulation of cellular debris and eventual cell death. Acetyl-L-carnitine can diminish advanced glycation end product (AGE) damage that leads to cataract formation.<sup>83</sup> Acetyl-L-carnitine can acetylate (deactivate) potential glycation sites on crystallins and protect them from glycation-mediated protein damage.<sup>84</sup> A suggested dose of acetyl-L-carnitine arginate is 3-4 capsules daily.

*Note: See Appendix for Cautions and Contraindications*

### **Aminoguanidine**

Aminoguanidine inhibits advanced glycation end products (AGEs) and may treat diabetic cataracts. In moderately and severely diabetic rats, aminoguanidine inhibited cataracts only in moderately diabetic rats.<sup>85</sup> It is important to maintain control over blood sugar levels, so that antiglycating agents such as aminoguanidine can protect against cataract. A suggested dose of aminoguanidine is 300 mg daily.

*Note: Although aminoguanidine has been safely used throughout the world for decades, clinical experience is limited in the United States. Aminoguanidine has not been approved by the U.S. Food and Drug Administration. Aminoguanidine should be taken under the supervision of a physician. It can inhibit vitamin B6 uptake so co-administration of B6 is suggested.*

**Note:** The Alteon Corporation (USA) has aminoguanidine (Pimagine®) in stage III trials for diabetes.

# CATARACTS

## *Lens Metabolism Support*

### **Bioflavonoids**

Bioflavonoids are powerful inhibitors of the enzyme aldose reductase.<sup>68</sup> If aldose reductase activity falls, sorbitol is not synthesized. This reduces the accumulation of water in the lens.<sup>86</sup> The bioflavonoids quercetin, myricetin, and kaempferol (from limes) specifically inhibit diabetic cataracts.<sup>71</sup> Ginkgo is a widely used flavonoid that maintains microcirculation to the eye and inhibits free radicals.<sup>87</sup> A suggested dose of ginkgo biloba is 120 mg daily.

### **Inositol**

Inositol nicotinate is a B vitamin that occurs in high concentrations in the lens. Inositol exhibits antioxidant property resulting in the quenching of reactive oxygen and scavenging of glucose.<sup>88</sup> Inositol works best taken with B complex vitamins. A suggested dose of inositol is 250 mg daily.<sup>89</sup>

### **Carnosine**

Carnosine inhibits formation of advanced glycation end products (AGEs) and protects normal proteins from the toxic effects of existing AGEs.<sup>90,91</sup> Eye drops containing N-acetyl-L-carnosine can delay vision senescence in humans: effective in 100% of primary senile cataract cases and 80% of mature senile cataract cases. N-acetyl-L-carnosine enter the aqueous and lipid parts of the eye and prevent and repair light-induced breaks to DNA strands.<sup>92</sup> N-acetyl-L-carnosine eye drops are approved for human use in Russia for the treatment of many eye diseases. Brite Eyes II is an advanced eye formula that contains 1% N-acetyl-L-carnosine in a soothing eye drop. A suggested oral dose of carnosine is 500-1000 mg daily.

## *Ocular Environment Support*

### **Carotenoids**

Carotenoids are fat-soluble, yellowish pigments found in some plants, algae, and photosynthetic bacteria. Carotenoids are light-gathering pigments that provide protection from the toxic effects of oxygen free radicals and singlet oxygen which are generated in the presence of light and oxygen.<sup>93</sup> Lutein and zeaxanthin are carotenoids found in high concentrations in the macula of the retina.<sup>94,95</sup> Lutein and zeaxanthin protect the eye from age-related macular degeneration and cataract formation.<sup>65</sup> Lutein is derived from dark green leafy vegetables (spinach, broccoli, kale, and collard greens). Zeaxanthin is found in yellow fruits and vegetables (corn, peaches, and mangoes). Suggested doses are 5 mg of zeaxanthin and 10 mg of lutein.

### **Coenzyme Q10**

Coenzyme Q10 (CoQ10) is an antioxidant that provides protection from free radical damage in the eye.<sup>96</sup> A combination of antioxidants including CoQ10, acetyl-L-carnitine, polyunsaturated fatty acids (PUFAs), and vitamin E improved mitochondrial function (linked to age-related macular degeneration) in retinal pigment epithelium.<sup>83</sup> Mitochondrial dysfunction in lens epithelial cells and superficial fiber cells of the eye may lead to oxidative stress and cataract formation. Mitochondrial dysfunction occurs throughout the body and produces damaging reactive oxygen species thought to cause aging and disease.<sup>97</sup> A suggested dose of CoQ10 is 100-200 mg daily.

### **Potassium and Magnesium**

A lens with cataracts has decreased concentrations of potassium and magnesium.<sup>98</sup> Potassium and magnesium are often deficient in aging humans. Supplementation with 400 mg of elemental potassium and 800 mg of elemental magnesium increases availability of these minerals to the lens and protects the arterial system.<sup>98</sup>

### **Ginkgo and Bilberry**

Ginkgo biloba extract is an antioxidant, increases circulation to the optic nerve<sup>75</sup> and has exhibit potential anti-cataract ability.<sup>87</sup> Bilberry (from *Vaccinium myrtillus fructus*) is a proanthocyanidin historically used for eye conditions, including glaucoma, cataracts, macular degeneration, diabetic retinopathy, and retinitis pigmentosa.<sup>75</sup> Ginkgo biloba and bilberry may restore microcapillary circulation.<sup>83</sup> Suggested doses are Ginkgo biloba, 120 mg daily, and bilberry, 100 mg daily. After taking Ginkgo and bilberry for a month; taking 400 mcg of selenium, 500 mg of glutathione, and 300 mg of alpha-lipoic acid daily has been suggested. **Note:** See *Appendix For Cautions and Contraindications*.

## FUNCTIONAL AND PRACTICAL MEDICINE

- Overview
- Free Radical Reduction

- Protection from Free Radicals: The Glutathione Mechanism
- Lens Protein Protection and Cellular Metabolism Maintenance
- Maintaining a Healthy Ocular Environment

## **Overview**

Treatment for cataracts is reactive: when cataracts adversely impact vision, they are surgically removed and replaced with an implanted lens. Cataract surgery is very effective and has a high success rate. Nutritional therapy can prevent the onset of cataracts, particularly because proven anti-cataract drugs are not available.<sup>62</sup>

Intervention strategies should address the underlying causes of cataracts: oxidative stress, free radical production, the breakdown and aggregation of lens proteins, dysfunction of metabolism in the lens, and inability to maintain a healthy ocular environment. Nutritional therapy is available for each of these underlying causes.

## **Free Radical Reduction**

### **Metabolic Changes and Cataract**

An aging lens undergoes metabolic changes that predispose it to cataracts. Some metabolic changes occur from reduced oxygen and nutrient supply which increases eye vulnerability to free-radical damage. The eye is protected by cellular antioxidants: glutathione and vitamin C. Healthy eyes are protected from free radical damage by a mechanism that produces and recycles antioxidants in the eye that neutralize free radicals. Cataract formation is associated with a breakdown in the mechanism that regulates utilization of glutathione and vitamin C and/or decreases their concentration in the lens and surrounding structures.<sup>58</sup>

### **Hydrogen Peroxide and Cataract**

Cataract formation is initiated by the free radical hydrogen peroxide found in the aqueous humor.<sup>99</sup> Hydrogen peroxide oxidizes glutathione, or conversely, glutathione chemically reduces hydrogen peroxide, ultimately damaging the energy-producing system of the eye and allowing sodium to leak into the lens. Excess sodium attracts water to maintain osmolality, which initiates the edema phase of a cataract. Normal body heat in the lens catalyzes oxidation of the lens' proteins, which become opaque and insoluble (similar to the process by which egg protein changes from clear to opaque upon cooking). Free radicals break down fatty acids in membranes and lens protein fibers, generating more free radicals. This cross-links (or denatures or breaks down) the laminate-like structural proteins inside the lens capsule. The lens capsule can swell or shrink (dehydrate) and these changes in pressure breaks lens fiber membranes, forming microscopic spaces that trap water and debris.<sup>101</sup>

### **Metabolism Support: Key Components**

The key components are glutathione and vitamin C. In particular, glutathione is required to protect mature lens fiber cells from free radical damage. Vitamin C (ascorbic acid) protects the lens from oxidative damage.<sup>99,100</sup>

## **Protection from Free Radicals: The Glutathione Mechanism**

Nutritional supplements reduce the risks of developing cataracts and slow or reverse cataract growth.<sup>101</sup> Blood circulation within the eye is minimal so supplementing with oral nutrients is challenging.<sup>102</sup> The most important nutrients maintain or stimulate antioxidant mechanisms utilizing glutathione and include vitamins C, B2, E, selenium, alpha lipoic acid, melatonin, N-acetylcysteine with garlic, and glutathione. Decreased glutathione and vitamin C are associated with cataracts.<sup>103,104</sup>

## **Lens Protein Protection and Cellular Metabolism Maintenance**

Proteins deep in the lens are generated during embryogenesis and must retain functionality for many decades. The inability to maintain protein stability over time leads to formation of a nuclear cataract (the predominant form). Once the lens forms (embryologically), proteins are only synthesized in the outermost fiber cells close to the lens surface.<sup>79</sup> Accumulated damage to the proteins causes loss of enzymatic activity and increases the likelihood of protein aggregation, a component of cataract formation.<sup>20</sup>

The glycation (glycosylation) process can change (denature) lens proteins and significantly contribute to diabetic cataract formation and retinopathy. Glycation occurs when proteins react with sugars and form advanced glycation end products (AGEs), essentially, proteins strongly (covalently) bind to sugars, compromising the function of that protein. AGEs are biochemically altered proteins, DNA, and lipids with altered physiological properties.<sup>105</sup>

Nutritional supplements that may decrease breakdown of lens proteins and help maintain cellular metabolism include vitamin B6, acetyl-L-carnitine, aminoguanidine, bioflavonoids, inositol, and carnosine.

### ***Maintaining a Healthy Ocular Environment***

Cataract formation is connected to the aging process, associated with increased oxidative stress, and a consequence of free radical attacks, and reduced efficiency of metabolic processes. The lens provides an environment where these processes proliferate at a rate faster than that in other parts of the body. The lens consists of multiple layers of cells without the usual cellular organelles for energy production and other regenerative mechanisms for cellular biostability.<sup>40</sup> Lens fiber cells dependent upon a small number of lens surface cells and surrounding cells for support. Over time these support mechanisms require increased nourishment and more antioxidants.<sup>50</sup>

Decrease efficiency in these supportive mechanisms is inevitable in aging, but it is possible to counteract these age-related processes by maintaining a healthy ocular environment with optimally maintained levels of antioxidant and cellular metabolism to ensure optimal lens function.<sup>64</sup> Aging and oxidative stress, in particular, affects the entire body. Free radical proliferation can be minimized in the eye through proper diet and lifestyle, positively affecting overall health.

Maintaining a healthy ocular environment begins by avoiding common lifestyle and environmental risks that promote eye disease, cataract development in particular, and by following dietary and nutritional recommendations that support overall eye health.

### **Risk Factor Avoidance**

Environmental risk factors to avoid include smoking, excessive alcohol consumption, excessive exposure to sunlight, especially UV radiation, and exposure to X-rays and gamma radiation.

### **Dietary Recommendations**

Recommendations include increased consumption of vegetables and fruits, “good fats” found in oily fish (e.g., salmon and tuna), whole grains, and legumes, and minimal consumption of saturated fats and cholesterol.<sup>106</sup> Consuming foods rich in the carotenoids lutein and zeaxanthin is especially important.

### **Nutritional Supplement Support**

Important nutritional supplements for maintaining healthy eyes include coenzyme Q10 (CoQ10), potassium, magnesium, Ginkgo, bilberry, and taurine.

*CoQ10.* CoQ10 is an antioxidant that improves mitochondrial function in the retina. This function is linked to age-related macular degeneration.<sup>96,83</sup>

*Potassium and magnesium.* Decreased potassium and magnesium concentrations are found in a lens with cataract.

*Ginkgo biloba and bilberry.* Ginkgo biloba<sup>107</sup> and bilberry support restoration of microcapillary circulation to the eye by protecting and strengthening ocular blood vessels.<sup>75</sup> Ginkgo and bilberry can be effectively combined with daily doses of 400 mcg of selenium, 500 mg glutathione, and 300 mg of lipoic acid.<sup>33,75</sup> Molecular and cellular assessment of ginkgo biloba extract as an ophthalmic drug determined it was an excellent antioxidant that readily scavenged free radicals, inhibited oxidative damage to proteins, and protected cells from light-mediated stress and DNA breakage, but did not alter endogenous antioxidant enzyme activity or protect against phototoxicity. It significantly retarded lens opacification in cataracts induced in rats.<sup>87</sup>

## **SUMMARY**

- Scientific Summary
- Lifestyle Changes

This protocol provides information about cataracts; its nature, etiology, physiology, pathophysiology, and current treatments. Nutritional approaches to protecting the eye and preventing or slowing cataract progression of cataracts is provided. This information on cataracts and nutritional supplements should enable the reader to understand the beneficial effects of nutrition on cataract prevention.

### ***Scientific Summary***

The most widely accepted conventional surgical treatment is removal of the lens and implantation of an artificial lens (IOL).

Surgical treatment is recommended when a cataract progresses to the point that it impairs visual function. Before this point is reached, conventional medicine often takes a “watchful waiting” approach, considering cataracts to be an age-related, unfortunate, but inevitable, fact of life. In contrast are a growing contingent of physicians, researchers, and nutritional scientists who have a more proactive view of cataract prevention and treatment. This holistic approach to maintain healthy lens function and eye health includes awareness of risk factors (e.g., smoking, alcohol, and sunlight), compliance with a sensible diet (e.g., low-fat, high-fiber), exercising, and nutritional therapy specifically for the eye.

## ***Lifestyle Changes***

Wear protective eyewear and avoid the following risk factors:

- Avoid smoking, excessive alcohol consumption, exposure to sunlight (particularly UV radiation), and excessive exposure to X-ray and gamma irradiation.
- Life Extension’s Solarshield sunglasses to protect from:
  - blue and UV radiation
  - preservation of essential macular pigments

## **LIFE EXTENSION’S INTEGRATED PROTOCOL**

- Supplement Recommendations

## ***Supplement Recommendations***

- Glutathione: 500 mg daily
- Vitamin C: 500 mg daily
- Vitamin B2: 50–150 mg daily
- Selenium: 200–400 mcg daily
- Vitamin E: 400 IU daily
- Gamma E Tocopherol w/Sesame Lignans: 1 softgel daily
- R-lipoic acid: 210 –420 mg daily
- N-acetyl-cysteine: 600 mg daily
- Melatonin: 300 mcg–3 mg at bedtime
- Vitamin B6: 50-250 mg daily
- Acetyl-L-carnitine arginate: three-four capsules daily
- Aminoguanidine: 150–300 mg daily
- Carnosine: 500–1000 mg daily
- Life Extension Mix: 3 tablets, 3 times daily, provide N-acetyl-cysteine, selenium, inositol, vitamins B2, B6, C, and E, bioflavonoids, and many other antioxidants and anti-glycating nutrients.
- Brite Eyes II: One-two drops in each eye daily
- Lutein Plus: One tablespoon daily taken with a fatty meal.
- Super Zeaxanthin with Lutein: One-two capsules daily.
- Coenzyme Q10: 100–200 mg daily
- Potassium: 400 mg daily, but consult your physician
- Magnesium: 800 mg daily
- Gingko biloba: 120 mg daily
- Bilberry: 100 mg daily
- Kyolic Reserve Garlic: One-three capsules daily

**Note:** *Many of the nutrients suggested may be contained in the Life Extension Mix*

## ***For More Information***

Contact the National Eye Health Education Program of the National Institutes of Health, (301) 496-5248, or the American Society of Cataract Surgery, (703) 591-2220.

## **Product Availability**

Life Extension Mix, Brite Eyes II, glutathione, Super Carnosine, Super Absorbable CoQ10, Solarshield sunglasses, Super Zeaxanthin with Lutein, vitamin C, vitamin B2, vitamin B6, vitamin E, Gamma E Tocopherol with Sesame Lignans, ginkgo biloba extract, bilberry extract, R-lipoic acid, potassium, magnesium, melatonin, Kyolic Reserve Garlic, N-acetyl-cysteine, acetyl-L-carnitine arginate, and selenium can be ordered by calling (800) 544-4440 or by ordering online at [www.lef.org](http://www.lef.org).

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