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Editorial

Molecular Mechanisms Underlying Cataract Development

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EDITORIAL NOTE

Cataracts are the most shared cause of blindness worldwide and a very significant reason of visual damage in infants and children. Congenital cataracts are seen in 10 to 60/100,000 births in the United Kingdom and in 50 to 150/100,000 births in emerging countries.

Classifying the genetic variants causing congenital cataracts has not only better our understanding of the pathogenesis of infantile cataracts, the most frequent treatable reason of childhood blindness, but also their more common counterpart, adult-onset cataracts.

The ocular lens provides a unique model for sympathetic the inductive interaction of various embryonic tissues, as well as cell difference, signaling, proliferation, physiology, biochemistry, longevity, and organelle degradation. The structure is surrounded by a thick capsule and full with elongated lens epithelial cells (LECs) that have differentiated into lens fiber cells. The lens is the only organ that continually changes and increases in size without substituting any cell in its system. It performs its role to bend light onto the retina until mutation or the effects of aging, the environment, or intrauterine infections cooperation its transparency and optical function. Such insults cause changes to the biomolecules and trigger homeostatic imbalance in the lens, leading to protein combination and cataracts, thus cumulative light scatter to affect refraction and cause loss of vision. Lens development is a result of a series of inductive events during eye morphogenesis. The eye begins to develop during gastrulation at the beginning of week 4 (day 22, Carnegie stage 9).

A single eye field (eye primordium) arises in the middle of the anterior neural plate (diencephalon region of the developing brain), which separates into 2 optic vesicles and induces the overlying surface ectoderm to form the lens placode (lens primordium) by day 28 (Carnegie stages 12-13). Throughout this stage, a series of inductive interactions begin to shape the eye, driven by signaling molecules such as bone morphogenetic proteins and fibroblastic growth factor 2, and by eye field transcription factors including PAX6, RAX, SIX3, and LHX2. The lens placode invaginates to form the cup-shaped lens pit, which brands a complete circle of cells and splits from the surface ectoderm to develop into the lens vesicle. The portion of the optic vesicle that faced the lens placode gives rise to the retina. The retina, in turn, provides oxygen and inductive signals that regulate the growth and apical-posterior axis of the lens. This tissue addition lasts to enable the functional optimization of eye function with the establishment of emmetropia. In the early optic cup stage, the lens vesicle releases signals that induce the overlying surface ectoderm to differentiate into the corneal epithelium. After the lens vesicle has closed (weeks 4-5; Carnegie stage 15), secondary fiber cells add to the growing lens as the fetal nucleus starts to form in weeks 6 to 7 (Carnegie stages 16-19), derived from the epithelial cells located at the equator of the developing lens. Around week 8 (Carnegie stage 20), the Y-shaped suture appears at the anterior and posterior poles of the embryonic nucleus of the lens, as the terminal ends of the secondary lens fibers about each other. Yet, it is important to remember that phenotypic variability is seen within families with the same mutation.Different variants in different genes can present with the same phenotype.

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