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Genetic scenario of Fuchs' endothelial corneal dystrophy in India

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Fuchs' endothelial corneal dystrophy (FECD) is a progressive deteriorative condition of corneal endothelial cells that predominantly affects women and onsets in the fourth to fifth decade of life. As an autosomal dominant and heterogeneous disease, FECD displays a peculiar trait of corneal guttata formation and excrescences from the Descemet membrane (DM) which are clinically detected using specular microscopy. So far, many genes have been attributed to cause this disease, thereby revealing its genetic complexity. With respect to Asian ethnicity, not many studies have been done to elucidate demographic specific genetic contributors for the disease. Transcription factor 4 (TCF4), that encodes for E2-2 protein, a group of E protein transcription factors known for cellular growth and differentiation, is one such gene that has been associated with this disease. Our recent study substantiates the fact that the intronic tri-nucleotide repeat expansion in this gene is a major contributing factor for the disease pathogenesis. In search of other genetic signatures for this disease, our recent findings suggest that intronic polymorphisms in TCF8 and FEN1 genes have strong associations with the disease in Indian population. Ours is the first report to identify a polymorphic marker for TCF8 gene in association with FECD, thereby rendering a stronger genetic load upon TCF8 for being a causative agent for the disease. Association of FEN1 vouches for the role of oxidative stress as one of the contributing aspect for the pathogenesis of FECD.

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A posterior-eye drug delivery system for treatment of age-related macular degeneration

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Age-related macular degeneration (AMD) is a progressive, neurodegenerative, occular disease and a leading cause of irreversible loss of vision in aging adults in developed countries. Its pathogenesis is characterized by uncontrolled proliferation of cells and cell growth in blood vessels, leaking of blood and proteins and aberrant folding, aggregation and accumulation of proteins. Over-expression of the vascular epithelial growth factor (VEGF) causes uncontrolled blood vessel growth resulting in violation of the blood-retina barrier and accumulation of blood and protein debris causes neuro-degeneration of cells in the retinal pigment epithelium (RPE) and tissue dysfunction. The current treatment of AMD is primarily based on anti-VEGF drugs which are administered by intra-vitreal injection. It has been recently proposed to administer exogenous heat shock proteins such as Hsp70 by intra-vitreal injection in order to clear accumulated debris from RPE and inhibit aggregate-based cell neuro-degeneration. An equally effective and less vision-threatening than intra-vitreal injection route of administration of the above macromolecular drugs is trans-scleral delivery from an implant in the posterior eye, made out of a poly (N-isopropyl acrylamide) (NIPAM) thermally sensitive gel. This gel undergoes a phase transition characterized by a lower critical solution temperature (LCST) of 33oC, below which the drug is loaded in the gel and above which the drug is released from the gel.

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