Gap junctions serve to distribute health-signals among neurons of the diseased retina (Rethealthsi)

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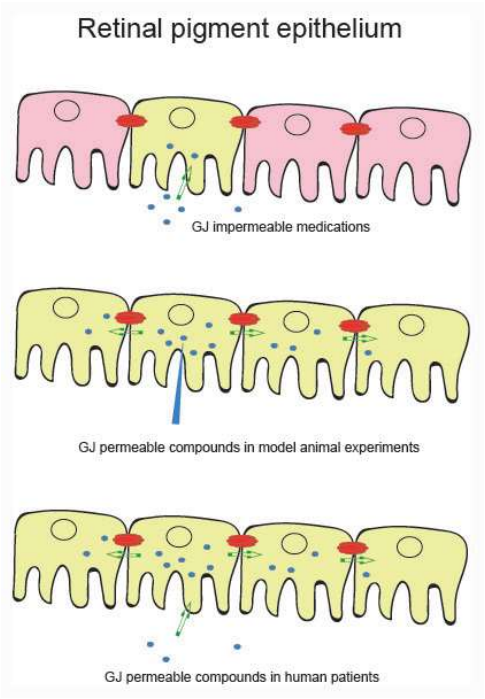

In retinal diseases, the loss of directly insulted cells is accompanied by a secondary en mass cellular death due to the so called bystander effect. Although bystander effect blocking interventions may have therapeutic value, the underlying mechanisms are unknown. Hypothetically, gap junctions (GJs - communication routes between cells) pass 'death-signals' from dying cells to neighbours, thus, it has been put forward that a GJ blockade could rescue cells in progressive diseases, such as diabetic retinopathy, Retinitis Pigmentosa (RP) or glaucoma. However, a GJ blockade for a chronic treatment would deteriorate vision as well. We propose a treatment modality, in which GJs are utilized as avenues for cell-to-cell diffusion of health-signal molecules. We will characterize the physical and chemical barriers of this GJ mediated diffusion of potential health-signals (endogenous molecules, epigenetic factors and medications) in both healthy and RP retinas. RP mutations cause the degeneration of rods (night-vision) and a bystander effect mediated secondary death of cones (daylight vision). Our goal is to characterize health-signals that by crossing GJs, reach many cells in the RP retina and rescue vision. Our long term aim is to develop a new therapeutic modality to arrest vision loss by ocular delivery of health-signal molecules.

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