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Mathematical models of retinal degeneration

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The retina is a tissue layer at the back of the eye that uses photoreceptor cells to detect light. Photoreceptors can be characterised as either rods or cones. Rods provide achromatic vision under low light conditions, while cones provide high-acuity colour vision under well-lit conditions. The term Retinitis Pigmentosa (RP) refers to a range of genetically mediated retinal diseases that cause the loss of photoreceptors and hence visual function. RP leads to a patchy degeneration of photoreceptors and typically directly affects either rods or cones, but not both. During the course of the disease, degenerate patches spread and the photoreceptor type unaffected by the mutation also begins to degenerate. The cause underlying these phenomena is currently unknown. The oxygen toxicity hypothesis suggests that secondary photoreceptor loss is due to hyperoxia (toxically high oxygen levels), which results from a decrease in oxygen uptake following the initial loss of photoreceptors. We have constructed mathematical models, formulated as systems of PDEs in 1D/2D, to investigate this hypothesis. Using a combination of numerical simulations and mathematical analysis, we find that degeneration may spread due to hyperoxia, replicating some, but not all, of the spatio-temporal patterns of degeneration seen in vivo. We determine the conditions under which a degenerate patch will spread or remain stable and show that the wave speed of degeneration is a decreasing function of the local photoreceptor density. Lastly, the effects of treatment with antioxidants and trophic factors upon the dynamics of photoreceptor loss and recovery are also considered. In time, these models could be used to inform personalised treatment strategies.

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