School of Optometry

Research Seminar

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PhD

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Gene Therapy for Retinal Degeneration: Generic Strategies to Prolong Neuron Survival through Combatting Oxidation

Date	**:**	**Monday 25 April 2016**
Time	**:**	**11:30 – 12:30 p.m.**
Venue	**:**	**Room HJ203, The Hong Kong Polytechnic University**

Abstract

Why neurons degenerate in diseases as well as during normal aging is an important question to be addressed. We use Retinitis pigmentosa (RP), an inherited retinal degeneration, as a disease model to study the common mechanisms underlying neuron degeneration and to develop novel therapies. Work from our lab and others suggest that the death of cone photoreceptors, the light-sensing neurons that provide our color and high acuity vision, is likely due to a common set of cellular stresses, including oxidation and metabolic dysregulation. We have been aiming to develop generic gene therapies to preserve cone function and survival in RP patients. To address the oxidation problem of cones, we designed two separate therapeutic strategies, one with two essential antioxidant enzymes, SOD2 and catalase, and the other with two antioxidant master transcription regulators, Nrf2 and PGC1a, and compared their efficacy. Via neonatal subretinal injection of adeno-associated virus (AAV) vectors, we can deliver these genes to the majority of cones. We found that both sets of genes can preserve cone survival and function in three different mouse models of RP, with the antioxidant transcription factor Nrf2 working the most effectively. The neuroprotective effects of Nrf2 extended to another neuronal type, retinal ganglion cells, in a model of acute axon injury, suggesting that targeted delivery of antioxidant genes has potential to ameliorate other diseases characterized by oxidative stress-induced cell degradation.

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