



Facultad de Óptica y Optometría
Universidad Complutense de Madrid

IV CICLO SEMINARIOS

“Novedades que el Óptico-Optometrista debe conocer sobre...”:
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“Focus on the enhancement of mitochondrial function in the treatment of glaucoma”

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RESUMEN

Explaining why individual retinal ganglion cells (RGCs) die at different times in glaucoma remains a challenge. It is proposed that glaucoma is initiated because the quality of the blood supply in the optic nerve head region is affected to result in a type of ischemia. This causes an alteration in RGC mitochondrial homeostasis and an activation of glial. Thereafter, as the disease progresses substances released from activated glial as well as other factors (e.g. blue light reaching the retina) act synergistically to affect RGC mitochondria further to eventually result in their death at varying times. The repertoire of receptors and number of mitochondria in individual RGCs might ultimately determine when individual neurones die in glaucoma. This implies that substances with a single mode of action is unlikely to be sufficient for effective clinical neuroprotection but that enhancing mitochondrial function might be away forward. This can theoretically be achieved, among other possibilities, by use of spectacles to blunt any insult of blue light and employ none invasive red light therapy to enhance mitochondrial function.

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