

Ischemia and the Ganglion Cell

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Retinal ganglion cell axons within the globe are functionally specialised being richly provided with many mitochondria. Mitochondria produce the high energy requirement for nerve conduction in the unmyelinated part of the ganglion cell axons and for the maintenance of optimum neuronal function. We have propose that in the initiation of glaucoma an alteration in the quality of blood flow dynamics in the optic nerve head results in sustained or intermittent ischemia. This results in normal mitochondrial function being negatively affected and as a consequence retinal ganglion cell function is compromised. Ganglion cells in this state are now susceptible to secondary insults which they would normally tolerate. One secondary insult to ganglion cells in such a state might be light entering the eye because of its known action on mitochondria. Another insult to the ganglion cells might be substances such as glutamate and nitric oxide, released from astrocytes in the optic nerve head region because of the effects of ischemia. Such cascades of events initiated by ischemia to the optic nerve head region ultimately cause ganglion cells to die at different rates. Thus agents targeted specifically at enhancing ganglion cell mitochondrial energy production, like creatine monohydrate, coenzyme Q₁₀, lipoic acid, nicotinamide or riboflavin should be beneficial in a disease like glaucoma.