# Preservation of vision after CaMKII-mediated protection of retinal ganglion cells

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Retinal ganglion cells (RGCs) are the sole output neurons that transmit visual information from the retina to the brain. Diverse insults and pathological states cause degeneration of RGC somas and axons leading to irreversible vision loss. A fundamental question is whether manipulation of a key regulator of RGC survival can protect RGCs from diverse insults and pathological states, and ultimately preserve vision. Here, we report that CaMKII-CREB signaling is compromised after excitotoxic injury to RGC somas or optic nerve injury to RGC axons, and reactivation of this pathway robustly protects RGCs from both injuries.

CaMKII activity also promotes RGC survival in the normal retina. Further, reactivation of CaMKII protects RGCs in two glaucoma models where RGCs degenerate from elevated intraocular pressure or genetic deficiency. Last, CaMKII reactivation protects long-distance RGC axon projections in vivo and preserves visual function, from the retina to the visual cortex, and visually guided behavior.



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