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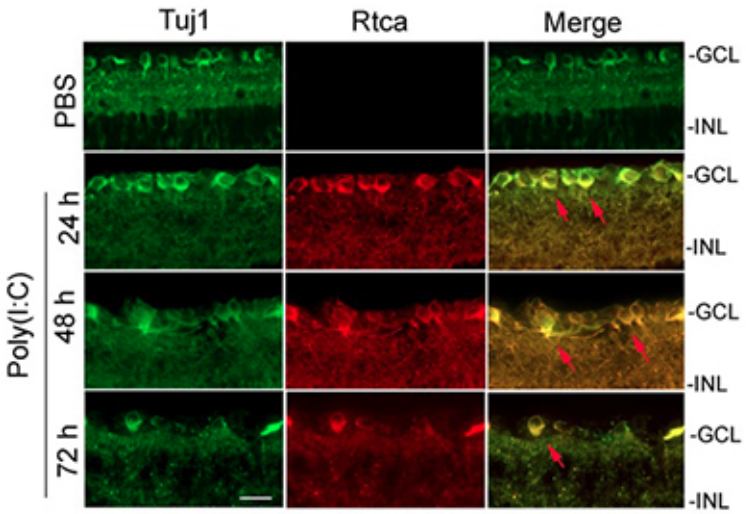
Undergraduate Naveena Daram is coauthor on an article in Investigative Ophthalmology and Visu Science

Undergraduate biomedical sciences major Naveena Daram is coauthor on an article published in the October 2016 issue of the journal Investigative Ophthalmology and Visual Science (Volume 57, Pages 5338-5347). Daram is a member of the Honor's College, participated in the ERI's Summer Undergraduate Program in Eye Research (SUPER), and is a holder of a Michael P. and Elizabeth A. Kenny Scholarship for the Sciences, awarded by the College of Arts and Sciences. She worked with Dr. Shravan Chintala, formerly of Oakland University's Eye Research Institute and now with the University of Southern California. They studied the degeneration of retinal ganglion cells, which leads to vision loss in several diseases that affect 60 million people worldwide. The article is open access, meaning you can **read it for free**. The abstract is reproduced below:



Localization of Rtca protein in RGCs of C57BL/6J mice. From Investigative Ophthalmology & Visual Science, 57:5338-5347, 2016.

Down-Regulation of RNA 30-Terminal Phosphate Cyclase Attenuates Toll-Like Receptor 3-Mediated Axonal Loss in the Retina and Optic Nerve

Purpose: To investigate the role of RNA 3′-terminal phosphate cyclase (Rtca) in Toll-like receptor 3 (TLR3)-mediated loss of retinal ganglion cells (RGCs) and their axons.

Methods: Polyinosinic-polycytidylic acid (Poly[I:C]) or PBS was injected into the vitreous humor of C57BL/6J and Tlr3 knockout mice. C57BL/6J mouse eyes were treated with Rtca silencing RNA or control RNA, with or without PBS or Poly(I:C). At 24, 48, and 72 hours after treatments, RGC loss was determined with the brain-specific homeobox/POU domain protein 3a antibody, and axonal loss was assessed by using the neuronal class III beta-tubulin (Tuj1) antibody. Axonal loss in the optic nerves was determined by anterograde-labeling of Cholera Toxin B. Western blot assays were performed to determine TLR3, Rtca, c-jun N-terminal kinase 3 (JNK3), and phospho-JNK3 (pJNK3) levels, and immunohistochemistry assays were performed to determine the cells that synthesize Rtca.

Results: Poly(I:C) significantly up-regulated the protein levels of TLR3, Rtca, JNK3, and pJNK3 in the retina. Rtca levels were increased in RGCs, and an increase in Rtca levels promoted significant loss of RGCs and their axons. In Tlr3 knockout mouse retinas, Poly(I:C) failed to elevate Rtca, JNK3, and pJNK3 protein levels and did not promote significant axonal loss. Also, Rtca silencing RNA down-regulated Rtca, JNK3, and pJNK3 in C57BL/6J mouse retinas, and down-regulation of Rtca attenuated Poly(I:C)-mediated loss of RGCs and their axons.

Conclusions: The results presented in this study show that the activation of TLR3 promotes the loss of RGCs and their axons by elevating Rtca levels in the retina. Also, the results presented in this study show that Rtca regulates JNK3 expression in the retina.

*Naveena Daram is also the president of a student organization that travels abroad to conduct service missions. The **Foundation for International Medical Relief of Children** took their first trip in August to Per and they are planning their next trip for spring 2017.*