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Potential contribution of IL-6 trans-signaling to the pathophysiology of glaucoma

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Interleukin-6 (IL-6) is a potential neuroprotective factor for retinal ganglion cells (RGCs) exposed to elevated pressure. In glaucomatous retina, IL-6 is produced primarily by microglia and RGCs are the primary targets of IL-6 signaling via expression of IL-6 receptor (IL-6R). We recently examine the impact of IL-6 depletion on disease progression and outcomes in the microbead model of murine glaucoma. Contrary to findings from in vitro studies, our data suggests that global depletion of IL-6 improves RGC and visual outcomes in vivo. Like many cytokines, IL-6 signaling can occur through one of two pathways, the classical pathway and the trans-signaling pathway. The classical pathway is mediated by membrane-bound IL-6R, while trans-signaling is mediated by the soluble form of the IL-6 binding subunit (sIL-6Rα) of the receptor. The trans-signaling pathway is often associated with neurodestructive outcomes of IL-6 signaling. As such, we examined the potential for the trans-signaling to contribute to IL-6-mediated outcomes in glaucoma. Using protein fractionation and immunohistochemical analysis, we determined that expression of sIL-6Rα is elevated in both aged and glaucomatous retina. Interestingly, sIL-6Rα appears to be produced by RGCs and is primarily associated with RGC axons in the nerve fiber layer. Given the axogenic nature of RGC degeneration in glaucoma, these findings have potential implications for identifying novel mechanisms of neuronal-glial interactions that impact axon degeneration in glaucoma.

Biography

Rebecca Sappington earned her M.S. and Ph.D. in Neuroscience from the University of Rochester School of Medicine and Dentistry, where she studied mechanisms of ganglion cell death in glaucoma. Rebecca continued her work on neuronal-glial interactions in glaucoma as a postdoctoral fellow at the Vanderbilt Eye Institute, Vanderbilt University School of Medicine (2004-2009). In 2009, Rebecca joined the faculty at the Vanderbilt Eye Institute, where her laboratory focuses on neuroinflammation in retinal neurodegeneration. Rebecca's training was supported by institutional training grants from the National Institute for Neurological Disease and Stroke and the National Eye Institute as well as a predoctoral fellowship from the American Foundation for Aging Research and a postdoctoral fellowship from Fight for Sight, Inc. Her work is currently supported by a RO1 from the National Eye Institute and a Career Development Award from Research to Prevent Blindness, Inc.

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