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The neuroinflammatory microenvironment: implications for IL-6- and other cytokine-related therapies

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Abstract

Neuroinflammation, defined as the induction of immune-related processes within the central nervous system (CNS), is recognized as a component of many neurodegenerative disorders, including those of the retina. Generally considered a negative factor in disease progression, neuroinflammation is characterized by a variety of cellular events, including production of inflammatory cytokines and changes in the reactivity state of glial cells. Despite much evidence that neuroinflammatory processes can be detrimental to neuronal survival, we observed that interleukin-6 (IL-6) produced by retinal microglia promotes RGC survival in the presence of elevated pressure, suggesting that microglial reactivity may both promote and inhibit degeneration of RGCs. We recently described that expression and localization of IL-6 and the IL-6-binding subunit of its receptor IL-6R□ are spatially regulated in response to both aging and elevated IOP stressors. In conditions of normal aging (C57 mice), a genetic predisposition to neurodegenerative disease (young DBA/2 mice) and the existence of active neurodegeneration (aged DBA/2 mice), expression of IL-6 and IL-6Ra is not homogeneous across the ganglion cell and nerve fiber layers of the retina, but instead, varies dramatically within discreet spatial parameters. The extent and magnitude of these variations differs dramatically between stressors, suggesting that the formation of these microenvironments of IL-6 signaling are relevant to both aging and neurodegenerative processes in retina. Recent evidence from our laboratory suggests that these spatial variations in IL-6 signaling are part of a larger scheme of neuroinflammatory microenvironments, which are highly regulated and defined by cytokine signaling, glial cell reactivity and neuronal function

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 postdoctoral fellowship from Fight for Sight. Her work is currently supported by a RO1 from the National Eye Institute and a Career Development Award from Research to Prevent Blindness, Inc.