Intracellular Toll-Like Receptors Help Retinal Microglia Sense Corneal Infections

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Microglia reactivity is a well-known phenomenon that occurs in response to several retinal pathologies, including infection, injury, degeneration, and autoimmunity.1 On the one hand, this resident macrophage population of the retina controls local immune homeostasis, but on the other hand, microglia can trigger chronic inflammatory events and neuronal cell death. Not so long ago, it was common sense that the eye is an immune-privileged organ and that local immune processes in the anterior and posterior segments do not mutually influence each other.

In this edition of IOVS, Chinnery and colleagues2 follow up on their important earlier findings that inflammatory stimuli at the corneal surface can influence retinal microglia behavior.3 Here, they used topical application of different toll-like receptor (TLR) ligands onto the injured corneal surface of Cx3cr1gfp/+ microglia reporter mice to mimic corneal infections.2 The authors thereby identified that specific agonists binding to intracellular TLRs (TLR7 and TLR9), but not those targeting cell surface TLR4, can rapidly induce the production of proinflammatory cytokines and vitritis. This is then followed by a widespread activation of retinal microglia that transform to amoeboid phagocytes migrating to the outer retina and even subretinal space. Chinnery et al.2 speculate that DNA and single-stranded RNA released during bacterial and viral corneal infections may thereby also disturb immune balance of the nearby retina. This raises the possibility that corneal microbial infections may have detrimental side effects on the progression of chronic inflammatory processes that often occur in retinal pathologies, including AMD, diabetic retinopathy, and glaucoma.

References