

Title:

Galectin-1-Mediated Suppression of *Pseudomonas aeruginosa*-Induced Corneal Immunopathology

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Abstract:

Corneal infection with *Pseudomonas aeruginosa* leads to a severe immunoinflammatory lesion often causing vision impairment and blindness. Although past studies have indicated a critical role for CD4⁺ T cells, particularly Th1 cells, in corneal immunopathology, the relative contribution of recently discovered Th17 and Treg cell is undefined. In this study, we demonstrate that after corneal *P. aeruginosa* infection, both Th1 and Th17 cells infiltrate the cornea with increased representation of Th17 cells. In addition to Th1 and Th17 cells, Treg also migrate into the cornea during early as well as late stages of corneal pathology. Moreover, using galectin-1 (Gal-1), an immunomodulatory carbohydrate-binding molecule, we investigated whether shifting the balance among various CD4⁺ T cell subsets can modulate *P. aeruginosa*-induced corneal immunopathology. We demonstrate here that local recombinant Gal-1 (rGal-1) treatment by subconjunctival injections significantly diminishes *P. aeruginosa*-mediated corneal inflammation through multiple mechanisms. Specifically, in our study, rGal-1 treatment significantly diminished corneal infiltration of total CD45⁺ T cells, neutrophils and CD4⁺ T cells. Furthermore, rGal-1 treatment significantly reduced proinflammatory Th17 cell response in the cornea as well as local draining lymph nodes (DLN). Also, rGal-1 therapy promoted anti-inflammatory Th2 and IL-10 response in secondary lymphoid organs. Collectively, our results indicate that corneal *P. aeruginosa* infection induces a strong Th17-mediated corneal pathology and treatment with endogenously derived protein such as Gal-1 may be of therapeutic value for the management of bacterial keratitis, a prevalent cause of vision loss and blindness in humans worldwide.