

## **Current State of Understanding Regarding the Immunopathogenesis of Dry Eyes and Implications for Therapy**

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There is considerable evidence to link dry eye disease (DED) with immune-mediated inflammation: There is strong evidence for (i) early and sustained overexpression of proinflammatory cytokines in the ocular surface (cornea, conjunctiva, tear film) of patients and animals with DED; (ii) activation and expansion of T cell subsets in the lymphoid compartment in DED; (iii) homing of CD4<sup>+</sup> T cells to the ocular surface of patients and animals with DED; (iv) over-expression of chemokines by the ocular surface that mediate leukocyte recruitment in all clinical forms and experimental models of DED. Still, the precise immunopathogenesis of DED, and the potential for immune modulation via ‘biologic’ treatments that target specific ligands/receptors involved in the disease pathogenesis remain incompletely understood. This brief presentation will review some of the current knowledge about novel strategies targeting immunogenic inflammation in DED.