Journal of Ophthalmology

Hindawi

Special Issue on

Molecular Mechanisms of Corneal Inflammation and Therapeutic Interventions to Inhibit Corneal Damage

CALL FOR PAPERS

Cornea is a complex sensory tissue that must maintain its transparency for optimal vision. The normal cornea is devoid of lymphatic and blood vessels that contribute to its immune privilege via suppressed immune responses. Inflammation; however, breaks this unique feature of the cornea and leads to development of blood vessels in inflamed ocular surface providing a route for the entry of inflammatory cells. The lymphatic vessels facilitate the exit of antigen-presenting cells and antigenic material from the cornea to draining lymph nodes and as a consequence promote the induction of adaptive immune response. Accumulation of inflammatory cells and angiogenesis in cornea can damage its structural integrity that could block or diffract the light ultimately leading to keratitis and scarring of cornea. Keratitis can be classified into different types based on its location, severity, and cause. Corneal inflammation may arise because of infectious or noninfectious cause. Bacteria, viruses, fungi, and other parasitic organisms infecting the cornea could potentially cause infectious or microbial keratitis. In addition, contact-lens, physical, or chemical trauma are also among the common causes of other forms of keratitis. Exploring the mechanisms of the immune privilege, molecular events that occur during inflammation/angiogenesis in the cornea and how the eye strives to maintain its transparency by inhibiting various inflammatory events could potentially be useful to design new therapeutics to prevent or reduce corneal damage.

We invite researchers to contribute original research articles as well as review articles that will stimulate the continuing efforts to understand the immune privilege mechanisms, the molecular pathways of various forms of keratitis, and angiogenesis that will help in the development of therapies for these diseases.

Potential topics include, but are not limited to:

- Mechanisms how cornea maintains vascular, lymphatic, and immune privilege
- Various aspects of ulcerative and nonulcerative keratitis
- Contribution of neutrophils, T cells, and other cell types such as macrophages in keratitis and neovascularization
- Role of inflammasomes in ocular inflammation
- Role of microRNAs and other inflammatory mediators in corneal neovascularization and lymphangiogenesis
- Endogenous mechanisms of corneal wound healing
- Potential of proresolution drugs to treat corneal inflammation
- Role of gut and local microbiota in the pathogenesis of ocular inflammation and keratitis

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/joph/mmc/.

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