AB003. Local group 2 innate lymphoid cells promote corneal regeneration after epithelial abrasion

Zhijie Li, Jun Liu

Institute of Ophthalmology, Jinan University Medical School, Guangzhou, China

Abstract: Corneal injuries and infections are the leading cause of blindness worldwide. Thus, understanding the mechanisms that control healing of the damaged cornea is critical for the development of new therapies to promptly restore vision. Innate lymphoid cells (ILCs) are a recently identified heterogeneous cell population that has been reported to orchestrate immunity and promote tissue repair in the lungs and skin after injury. However, whether ILCs can modulate the repair process in the cornea remains poorly understood. We identified a population of cornea-resident group 2 ILCs (ILC2s) in mice that express CD127, T1/ST2, CD90, and cKit. This cell population was relatively rare in corneas at a steady state but increased after corneal epithelial abrasion. Moreover, ILC2s were maintained and expanded locally at a steady state and after wounding. Depletion of this cell population caused a delay in corneal wound healing, whereas supplementation of ILC2s through adoptive transfer partially restored the healing process. Further investigation revealed that IL-25, IL-33, and thymic stromal lymphopoietin had critical roles in corneal ILC2 responses and that CCR2- corneal macrophages were an important producer of IL-33 in the cornea. Together, these results reveal the critical role of cornea-resident ILC2s in the restoration of corneal epithelial integrity after acute injury and suggest that ILC2 responses depend on local induction of IL-25, IL-33, and thymic stromal lymphopoietin.

Keywords: Cornea; wound healing; inflammation; innate lymphoid cells (ILCs)

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