Semaphorin 7a in Herpetic Neurotrophic Keratitis

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Recurrent infection of the ocular surface by herpes simplex virus type I (HSV-1), a neurotropic herpesvirus, is a leading infectious cause of corneal blindness in developed countries.¹ The origin of recurrent infections lies in the fact that the virus establishes latency in sensory neurons of the trigeminal ganglion (TG). While the latent infection of TG is marked by the loss of viral protein synthesis and, hence, very little virus-induced damage to the host neurons, its reactivation and anterograde axonal transport to the cornea via the innervating nerves can contribute to the loss of nerve sensitivity commonly reported by HSV-1–induced neurotrophic keratitis (NTK) patients. Very little is known about the mechanism of this corneal dysfunction during the NTK. In the current issue of IOVS, Chucair-Elliott et al.² provide some novel insights into the molecular mechanism and validate an interesting animal model for HSV-1–induced NTK. The authors provide compelling evidence that sensory nerves and corneal sensitivity are lost within 8 days of HSV-1 infection of the murine corneas. Quite interestingly, the nerves in the cornea regenerated around 30 days after the infection; however, there was no detectable recovery of the corneal sensitivity. The authors successfully used Cochet-Bonnet esthesiometer to quantitatively evaluate corneal sensitivity, which adds credence to use of their murine model in studies of NTK disease symptoms. As part of a possible nerve damage and regeneration mechanism, the authors implicate Semaphorin 7a (SEMA 7A), which is in agreement with a pioneering study on corneal nerve damage performed by Jain and colleagues.³ The latter shows that SEMA7A is central to nerve regeneration and common inflammatory processes reported in the cornea. Overall, Chucair-Elliott et al.² present an excellent starting point for future studies that hopefully will include HSV-1 reactivation models to verify the contribution of SEMA7A, and identify other key molecules and new therapies to control NTK.

References


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