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Update
CORNEA

TEAR DYSFUNCTION
Emerging therapies show promise for reducing pain and improving vision
by Howard Larkin in Orlando

Recent insights into molecular and cellular pathways of tear dysfunction are leading to new therapies for superficial epithelial corneal disease, one of the most common problems treated by ophthalmologists, Stephen C Pflugfelder MD of Baylor College of Medicine, Houston, Texas, US, told the American Academy of Ophthalmology in the 67th Edward Jackson Memorial Lecture.

With symptoms including severe dry eye, pain, photophobia and blurred vision, superficial epithelial corneal disease can be as disabling as unstable angina, making it a significant threat to health and wellbeing. Several recently developed treatments targeting tear dysfunction can often prevent or reverse these symptoms and the related corneal disease, and more treatments are in development, Dr Pflugfelder said.

The cornea accounts for 65 per cent of the optical power of the eye, but is also the most exposed mucosal surface in the body, open 92 per cent of the time during waking hours, Dr Pflugfelder noted. Replenished about 15 times per minute by blinking, tears play several essential roles in both maintaining visual quality and corneal health.

Precorneal tear gel improves vision by masking light scattering due to irregular microplicae on the superficial corneal epithelium, Dr Pflugfelder said. Tears also maintain corneal homeostasis, supplying the avascular cornea with essential factors for defending against microbial attachment, suppressing corneal inflammation and degradation and promoting rapid wound healing. At the same time, tears protect the cornea from environmental, recreational and occupational desiccating stress. But they are not always up to the task, Dr Pflugfelder said. “Unfortunately, dry eye is an unavoidable complication of certain professions.”

Risk factors for tear dysfunction include diabetes mellitus, low dietary intake of omega n-3 and n-6 essential unsaturated fatty acids, prolonged computer or video use, chronic exposure to low humidity, chronic contact lens wear and LASIK. Tear dysfunction has been recognised for more than 100 years as the most common cause of superficial corneal disease, and addressing it is often the key to treating corneal surface conditions.

Lacrimal functional unit Tear secretion is regulated by the lacrimal functional unit, which consists of sensory nerves in the cornea and conjunctiva communicating with the organs that produce tear components. The precorneal tear film that protects and nourishes the cornea is a mixture of growth factors, cytokines, antimicrobial peptides, immunoglobins and protease inhibitors secreted by the lacrimal glands. Mucins that lubricate and smooth the corneal surface are produced by epithelial membranes and conjunctival goblet cells. The tear layer is topped by lipids that reduce evaporation produced mainly by the meibomian glands.

When working properly together, the components of the lacrimal functional unit create a balanced tear film that supports corneal health and normal function. However, disruption of the signalling or disease of one or more of these components result in changes in tear composition that in turn promote a variety of pathological changes in corneal epithelial cells, Dr Pflugfelder said. “Tear dysfunction activates stress signalling in the corneal epithelium.”

Severe dry eye can be treated with the PROSE contact lens device that keeps the eye surface hydrated, Dr Pflugfelder added. “It shields the cornea from environmental stress, and bathes it with body-temperature saline. This silences corneal nociceptors. Over a week, this reduces patient reaction to cool and dry air. When we put the lens in the eye, patients say it’s like getting their life back. How often do you get that response from your dry-eye patients?”

Pharmaceutical interventions that inhibit cytokine and MMP activity also have been found to prevent corneal barrier disruption in response to acute desiccating stress in mice, Dr Pflugfelder said. Dexamethasone, doxcycline and cyclosporine all have been shown to improve optical performance. T helper cytokine inhibitors, selective glucocorticoid receptor agonists, MMP inhibitors and leukocyte trafficking blockers, including LFA-1, VLA-4 and chemokine receptor 2 antagonists, all show promise for tamping down corneal stress signalling and resulting damage.

Contact
Stephen Pflugfelder – steven@bcm.edu