Primary Care Section Symposium in Collaboration with The Anterior Segment Special Interest Group

AAO Phoenix 2012

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Cornea and Ocular Surface Disease: Application of Cutting Edge Optometric Research

The focus of the symposium is to bring leading optometric researchers in cornea and ocular surface disease together who will present their cutting edge research and explicitly point to applications that will change eye care.

Moderator:

Anthony J. Adams, OD, PhD, FAAO
Editor-in Chief, Optometry and Vision Science; Professor and Dean Emeritus, School of Optometry, University of California Berkeley

Speakers:

Joseph Bonanno, OD, PhD, FAAO
Professor and Dean of Optometry, Indiana University, Bloomington

Why are topical CAIs contraindicated in corneas with low endothelial cell count?

The corneal endothelium is responsible for maintaining the hydration and transparency of the cornea. Trauma, inflammation, or dystrophies (e.g. Fuchs dystrophy) can reduce endothelial function leading to corneal edema and loss of vision. The endothelial "pump" has been described as a bicarbonate and carbonic anhydrase dependent ion secretory mechanism that creates osmotic gradients leading to water efflux that exactly counterbalances water influx driven by stromal glycosaminoglycans. We now present evidence indicating that bicarbonate and carbonic anhydrase activity act to buffer corneal lactic acid transport across the endothelium and that this facilitation of lactate transport is a significant contributor to the endothelial pump. These new insights also provide a context for the role of carbonic anhydrases and an explanation for reports of corneal decompensation in patients using topical carbonic anhydrase inhibitors.

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Suzanne M.J. Fleiszig, OD, PhD, FAAO
Professor of Optometry and Vision Science, Infectious Diseases & Immunity, and Microbiology, University of California Berkeley

Eradicating infection

Despite a plethora of new products, the incidence of contact lens related infection has not changed since soft lenses were first introduced onto the market about 40 years ago. It is becoming increasingly clear that basic research to understand the mechanisms cannot be circumvented if the problem is ever to be solved. In recent years, our laboratory has capitalized on new technologies and an explosion of information in the field of molecular and cellular biology to develop various new models and methods. These are now providing answers to long standing questions about to how the healthy cornea resists infection, how contact lens wear compromises that resistance, and how microbes take advantage of that to cause disease. Data arising from these experiments is informing us about how lens wear could be made safer, and suggest exciting and novel strategies for managing infection whatever the cause.

Nancy A. McNamara, OD, PhD
Associate Professor, Departments of Anatomy and Ophthalmology
University of California San Francisco
Sjögren's Syndrome International Collaborative Clinical Alliance (SICCA)

Tackling the clinically recalcitrant dry eye in autoimmune disease

One of the most debilitating forms of keratoconjunctivitissicca (KCS) results from autoimmune-mediated destruction of the lacrimal gland. Despite powerful immunosuppressive and immunomodulatory therapy, KCS in autoimmune diseases like Sjögren's syndrome can progress to complete corneal opacification and blindness through a process known as squamous metaplasia. Little is known about the pathogenesis of squamous metaplasia and there is no cure. *The goal of our research is to decipher how autoimmune-mediated inflammation provokes KCS and vision-threatening squamous metaplasia.* Using three model systems, (i) human patients with Sjögren's syndrome; (ii) a validated mouse model that mimics the clinical characteristics of Sjögren's syndrome; and (iii) *in vitro* studies of cultured corneal epithelial cells, my lab has demonstrated an essential role for autoreactive CD4+ T cells and their interplay with the proinflammatory cytokine interleukin (IL)-1 in the pathogenesis of autoimmune KCS. Accordingly, in clinical studies we have shown IL-1β protein is elevated in the tears of human patients with Sjögren's syndrome and its ocular surface expression is a significant predictor of ocular disease. The development of immunoassays to identify and validate novel biomarkers fromsmall volume tear samples is underway, as well as the identification of novel drug targets to treat autoimmune dry eye and prevent the devastating consequences of squamous metaplasia.

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Danielle Robertson, OD, PhD, FAAO
Assistant Professor of Ophthalmology, UT Southwestern Medical Center

Mechanisms of corneal epithelial maintenance: understanding the effects of systemic disease and contact lens wear

The corneal epithelium is a self-renewing stratified epithelial sheet that provides a barrier against invading pathogens and a smooth refracting surface essential for vision. A coordinated balance between proliferation, differentiation and apoptotic desquamation coupled with dynamic regulation of intercellular junction formation contributes to homeostatic tissue maintenance as cells divide, migrate and shed throughout the course of normal cellular turnover. Using contemporary imaging, molecular and biochemical techniques, we are investigating fundamental gene regulatory mechanisms and protein-protein interactions that regulate normal corneal epithelial homeostatic renewal and are disrupted by injury or disease. To accomplish this, we have implemented two clinically relevant models that perturb normal epithelial homeostatic renewal: hyperglycemic-induced corneal epithelial changes as seen in diabetic disease; and contact lens wear, where mechanical and chemical effects from lenses and/or solutions alter both renewal and barrier function properties, predisposing the cornea to infection.

Anthony J. Adams, OD, PhD, FAAO
Moderator-Panel Discussion