What Is New in *Ophthalmic Research* – Focus on the Lens

Lens Calcium and Cataract

Borchman et al., Louisville, USA

Ophthalmic Res 2008;40:86–93

In virtually all cells, calcium regulates many physiological and cellular events by acting as a second messenger; calcium also triggers pathological events in cell injury and death. The lens is no exception, and every cataractous lens contains 3–1,000 times more calcium than clear lenses. It has been hypothesized that the dearrangement of calcium balance is a significant factor in the cataractogenic cascade. Calcium homeostasis is accomplished by a delicate balance between cell membrane permeability and the active transport of calcium by Ca-ATPases at the level of the cell membrane (PMCA) and the endoplasmic reticulum. In this study, the authors found that the lack of expression of PMCA cannot account for the elevated levels of calcium with increasing age and cataract. Other factors such as an increase in membrane permeability or the direct inhibition of PMCA or its activators must account for the high amount of total calcium in cataractous lenses.

Antioxidative Actions by NSAIDs/ASA in the Lens

Petersen et al., Göteborg, Sweden

Ophthalmic Res 2008;40:77–85

This study examined the mechanisms behind the anticataractogenic effects of the nonsteroidal anti-inflammatory substances indomethacin, diclofenac, celecoxib and acetylsalicylic acid (NSAIDs/ASA). The authors found a reduction of superoxide and peroxide production as well as a reduction of glutathione depletion in oxidatively stressed human lens epithelial cells incubated with low concentrations of NSAIDs/ASA. These results indicate

antioxidative properties of NSAIDs/ASA, which is separate from their ability to reduce inflammation, pain and fever by inhibition of the enzyme cyclooxygenase. This study provides an explanation for previous reports on anticataractogenic effects of these substances. The understanding of reactive-oxygen-species-scavenging actions by NSAIDs/ASA may lead to further therapeutic/prophylactic applications in the future.

Involvement of PI3K/Akt Pathways in TGF- β_2 -Induced Epithelial Mesenchymal Transition

Yao et al., Hangzhou, China

Ophthalmic Res 2008;40:69–76

The major goals of this study were to determine the effect of TGF- β_2 on epithelial mesenchymal transition (EMT) and to identify the effect of signaling pathways in lens epithelial cells (LECs) activated by TGF- β_2 . EMT of postoperative remnants of LECs can lead to posterior capsule opacification (PCO). In this study, the authors demonstrated that TGF- β_2 is a potent growth factor for LECs and may contribute to the development of PCO. Stimulation of HLEB-3 cells with TGF- β_2 suppressed connexin 43 protein expression, and increased fibronectin, desmin and integrin β_1 protein expression. In addition, TGF- β_2 activated PI3K/Akt in a time-dependent manner, but not ERK and p38 MAPK. The activation of PI3K/Akt was necessary for the TGF- β_2 -stimulated downregulation of connexin 43, which in turn was necessary for TGF- β_2 -induced EMT in HLEB-3 cells. Therefore, TGF- β_2 -induced EMT in LECs is mediated by the downregulation of connexin 43, which is regulated through the PI3K/Akt pathway.

Uwe Pleyer, Editor-in-Chief