AB086. Influence of the intraocular pressure on the expression of tight junctions in the corneal endothelium

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Background: Cells are influenced by their environment. *In vivo*, the corneal endothelium is subjected to intraocular pressure (IOP). The purpose of this project was to evaluate *in vitro*, the effect of the IOP on the formation of tight junctions in the corneal endothelium.

Methods: Cultivated corneal endothelial cells (P2-P3; n=6 populations) were seeded on devitalized on corneas (n=10 pairs). Native corneas and devitalized corneas were respectively used as positive (n=2 pairs) and negative controls (n=3 pairs). Corneas were placed in artificial anterior chambers and subjected to a hydrostatic pressure between 0.3 and 0.4 psi during 4–5 days. Unpressured control corneas were maintained in cell culture dishes. Pictures of the corneas were taken following the experiment to assess stromal transparency. Morphology, corneal thickness and distribution of ZO-1, n-cadherin, b-catenin, NaK ATPase pump and HCO_3-cotransporter were evaluated by electron microscopy, histological staining and immunofluorescences.

Results: Pressure treated corneas were more transparent than the controls. Thickness was accordingly reduced by 38.4%±4.9% for cultivated endothelium and 32.2%±2.7% for native endothelium. Negative controls change in transparency and thickness were marginal. Pressure treated cells showed none or at most marginal difference in morphology and expression of ZO-1, n-cadherin, b-catenin, NaK ATPase pump and HCO_3-cotransporters and failed to recreate a phenotype similar to native corneas. Pressure however increased cortical localisation of the protein ZO-1 in both cultivated and native endothelium.

Conclusions: These results suggest that anterior chamber hydrostatic pressure may enhance endothelial functionality by modulating the distribution of tight junction's proteins.

Keywords: Corneal endothelium; intraocular pressure (IOP); tissue engineering; tight junctions

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