ANGIOTENSIN CONVERTING ENZYME MODULATES CORNEAL ANGIOGENSIS *IN VIVO*

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Purpose: We tested (i) whether a renin-angiotensin system exists within the cornea, and (ii) its role in modulating corneal neovascularization (CNV) *in vivo.*

Methods: Rabbit corneal fibroblast and corneal epithelial cell cultures were used *in vitro* and New Zealand White rabbits were used *in vivo*. Total RNA was extracted from cells/tissues and reverse-transcribed to cDNA using standard molecular biological techniques. PCR detected angiotensin converting enzyme (ACE), angiotensin II (AT1) receptor and angiotensin II (AT2) receptor expression. CNV was induced in rabbit eyes by vascular endothelial growth factor (VEGF) using micro-pocket assay. Animals either received Enalapril, an ACE inhibitor, (3 mg/kg) or water IM each day for 12 days. Stereomicroscopy and immunohistochemical techniques were used to measure the length and number of corneal blood vessels. NIH Image Java software was used for quantification.

Results: Rabbit corneal fibroblasts showed AT1 receptors but no ACE expression, whereas rabbit corneal epithelium demonstrated ACE but no AT1 receptor expression. Neither of these cells demonstrated AT2 receptors. Enalapril-treated animals showed a roughly 23 percent decrease in the number of blood vessels entering the cornea compared to controls at days 7 and 12. However, due to small sample size (n=4) this data was not found statistically significant. Surprisingly, no appreciable change in corneal blood vessel length was noted between the two groups.

Conclusions: This study suggests a renin-angiotensin system exists within the cornea and plays an important role in corneal angiogenesis. More studies are needed to explore the therapeutic potential of Enalapril to control CNV.

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