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IDENTIFICATION AND QUANTIFICATION OF COLLAGEN TYPES, LAMININ, AND FIBRONECTIN IN THE TRABECULAR MESHWORK OF GLAUCOMATOUS AND NORMAL HUMAN EYES

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ABSTRACT

The trabecular meshwork, located at the corneoscleral angle, is the site of aqueous outflow resistance which results in elevated intraocular pressure in primary open-angle glaucoma. Vision loss associated with glaucoma is due to optic nerve atrophy at the site of the lamina cribrosa, a specialized fenestrated continuation of the sclera through which the axons of the optic nerve pass. The progression of glaucoma is unpredictable in that some people with normal intraocular pressures show progressive optic atrophy and visual field loss, some have consistently high intraocular pressures without having optic nerve degeneration or abnormalities, and many will present with both high pressure and optic atrophy. An anatomical link between the trabecular meshwork and lamina cribrosa may exist. Immunofluorescence microscopy and immunoelectron microscopy using the protein A-gold labelling technique were used to identify and quantify the collagen types, fibronectin, and laminin in the trabecular beams and juxtacanalicular zone respectively of the trabecular meshwork in normal and glaucomatous human eyes. Specimens from 12 glaucomatous and 12 age-matched normal or nonglaucomatous eyes were reacted with monoclonal antibodies specific for collagen types I, II, III, IV, V, and attachment proteins fibronectin and laminin. The results indicated the presence of collagen types I, III, IV, V, fibronectin and laminin in both the glaucomatous and normal eyes. Quantification revealed an overall decrease in five of these extracellular matrix components in

the glaucomatous eyes to varying degrees. Collagen type V did not change. The trabecular meshwork findings, when compared with previous studies of the lamina cribrosa, suggest that a collagen disorder may explain the characteristic abnormalities of glaucoma which are noted in these two anatomically and functionally separate tissues.