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Immunohistochemistry of adheren junctions in the optic nerve head highlights potential fatal sites for aqueous humor-induced ionic stress and points to a new paradigm in glaucoma diagnosis and treatment

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Is aqueous humor toxicity the missing link in glaucoma? Glaucoma is a prevalent disease characterized by axonal loss related to difficulties in aqueous humor outflow that may manifest as increased intraocular pressure. We have shown in perfusion experiments that the optic nerve with a fluid-conducting paravascular glial sheat is easily permeable. There is increasing evidence that in some regions of the CNS, axons rely on lactate rather that glucose for their nourishment. We have communicated that based on the presence of transport molecules; lactate is the main energy source at the astrocyte-rich pre-laminar level of the optic nerve. Along with them, adherens junctions (AJ) rich in N-cadherin depend of calcium ion to maintain the binding. We study the response of the cultured pre-laminar tissue of the pig to calcium ion withdrawal by transmission electron (TEM) and confocal (CLSM) microscopes to detect changes in the exposed N-Cadherin attachments. There is an increase both in apoptosis and increase in the cleft separation in AJ in the cultured pre-laminar region with aqueous humor-levels of calcium reduction. Phosphorylation of Beta-Catenin shown by immune-labeling and incremented apoptosis as measured by Western Blot of caspases is both induced by calcium reduction. Altogether, this suggests that activation of the signaling system related to AJ triggers a wave of anoikis in astrocytes. Axon loss in glaucoma can be linked to astrocyte detachment provoked by aqueous misdirection through the optic nerve. This mechanism opens new avenues for pathogenetically-oriented treatment of axon loss and corresponding visual field defects in glaucoma.

Biography

Francisco Javier Carreras is a Tenured Professor of Ophthalmology, University of Granada. His research has pivoted around the pathogenesis of the glaucomas. He has contributed the description and characterization of the Cameral Mucous Gel in humans. He has described a fluid conducting role for the perivascular glial sheat as well as the fenestrations of the basal lamina in the inner limiting membrane. His description of a pathogenetic role of the misdirection of aqueous humor flow is been incrementally delineated by his late contributions. He has developed a computational model of the optic pathways that helps to understand the structural/functional relationship in glaucoma.

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