

10<sup>th</sup> International conference on

## OPHTHALMOLOGY AND OPTOMETRY

August 10-11, 2017 Beijing, China

**Janardan Kumar**

Becker College, USA

**Aneurysm mediated release of thrombin into the aqueous humor may be a causative factor of glaucoma**

Glaucoma has been known to be associated with ocular hypertension due to age-related dysfunction of outflow pathway tissues. Based on the geometry of outflow pathway tissues, only the inner wall of Schlemm's canal (SC) cells in the conventional outflow pathway appeared to be arranged as a monolayer consisting of irregular pores. Several studies suggest the biomechanical properties of the endothelial SC cells to be modulators of endothelial barrier function, outflow resistance and intraocular pressure (IOP). Our studies of thrombin perfusion in enucleated porcine eyes *ex vivo*, procured after thorough investigation of unnoticed impurities, revealed an increased outflow resistance. Of significance, as opposed to the effect of thrombin in other cell types for the endothelial barrier function, treatment of thrombin to the human endothelial SC cells monolayer permeability barrier-function assay exhibited a significant decrease in the permeability of horse-radish peroxidase. Even if there was the di-phosphorylated form of myosin light chain that is located in the peripheral region of the cell, formation of stress fibers and focal adhesions were increased significantly and that resembles the characteristics for the activation of Rho kinase. Such cellular changes of human SC endothelial cells have been correlated with cellular contraction and/or stiffness, causing increased resistance of the outflow. Since the thrombin can cause increased outflow resistance and is known to be involved in increased extracellular matrix (ECM) protein deposition and fibrosis, we speculate that release of thrombin in the aqueous humor followed by an aneurysm may be associated with the etiology of glaucoma. Hence, the clinical diagnosis of an aneurysm in glaucomatous patients and detection of thrombin in glaucomatous aqueous humor would provide a better understanding of glaucoma.

**Biography**

Janardan Kumar has completed his undergraduate studies in Botany with honors in 1981 at Rajendra College in India. In 1992, he has graduated from Kanpur University at the Central Drug Research Institute, India. In 1998, he was a Junior Faculty Member in the Department of Cell Biology at Duke University in North Carolina. He opted to pursue Postdoctoral studies in the field of glaucoma. He led the way in studying Rho kinase activation and inhibition while working in Duke University's laboratory. He is currently a Professor of Microbiology and Former Chair of the Department of Natural Sciences at Becker College in USA. He has lectured at several international conferences, edited books and served as a Reviewer for several peer-reviewed journals. His main research interests are membrane biology, intracellular membrane dynamics, differentiation of stem cells, generation of therapeutic monoclonal antibodies and glaucoma.

Janardan.kumar@becker.edu

**Notes:**