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Vascular homeostasis in diabetic retinopathy

Global increase in the diabetic population forecasts a significant rise in the number of people with diabetes-related retinal diseases. An overwhelming cause of vision loss in diabetic individuals is the breakdown of retinal vascular homeostasis, contributing to excess permeability and the development of macular edema, a prominent clinical manifestation of diabetic retinopathy. Despite the use of laser photocoagulation, and available therapeutics, majority of the patients do not fully recover functional vision. Research into areas involving cell-cell communication and blood retinal barrier characteristics has uncovered a significant underlying factor that contributes to both these functional changes. Our studies indicate that abnormal thickening of the vascular basement membrane (BM) can contribute to excess vascular permeability, breakdown in cell-cell communication, and retinal vascular cell loss. It has long been established that vascular BM thickening is a characteristic hallmark of diabetic microangiopathy, however, it is unclear how vascular BM thickening promotes the characteristic lesions seen in diabetic retinopathy. Recent studies have begun to shed light on this subject suggesting vascular BM thickening as a key player that not only compromises the BRB characteristics but also affects vascular homeostasis and promotes cell loss associated with the development and progression of diabetic retinopathy. Importantly, our research has identified several BM genes, fibronectin, collagen IV, and laminin that are abnormally expressed under hyperglycemic condition and contribute to abnormal cell-cell communication and retinal vascular leakage. A strategy for decreasing BM thickening and how this could prevent vascular leakage and contribute to the maintenance of vascular homeostasis in the diabetic retina will be the topic of this presentation.

Biography

Sayon Roy received his Ph.D. from Boston University and completed his postdoctoral training at Schepens Eye Research Institute, Harvard Medical School, Harvard University; Roy is currently a Professor of Medicine, Section of Diabetes, Endocrinology and Nutrition, and a Professor of Ophthalmology at Boston University School of Medicine. Recognized as an expert in retinal vascular biology, Dr. Roy's seminal work has identified several genes in the retina that are abnormally expressed in diabetic retinopathy. His pioneering work has led to novel gene modulatory techniques in retinal vascular cells using antisense oligonucleotides via intravitreal injection. Dr. Roy has received numerous awards including the American Diabetes Association Research Award for the commitment and dedication towards the fight against diabetes, the 2006 Mentor of the Year Award from Boston University, and the 2008 Innovative Award from the Juvenile Diabetes Research Foundation. Research in Roy's laboratory has been funded by several organizations including the National Eye Institute, NIH, National Medical Technology Testbed, American Diabetes Association, Juvenile Diabetes Research Foundation International, Fight for Sight, Research to Prevent Blindness, and the Lions Organization. Dr. Roy currently serves as a chartered member of the NEI Study Section of the National Institutes of Health.

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