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Vascular basement membrane abnormalities in diabetic retinopathy

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Abstract

The changing landscape of the global diabetic population points to significant increase in the prevalence of diabetes worldwide. Based on this projected increase, the number of people with diabetes-related retinal diseases is estimated to increase significantly. The overwhelming cause of vision loss in diabetic individuals is typically due to macular edema and the altered blood-retinal barrier. Despite the use of laser photocoagulation, and molecular therapeutics targeting vascular endothelial growth factor, majority of the patients do not fully recover functional vision. It is well recognized that a significant clinical manifestation of diabetic retinopathy is the breakdown of the blood retinal barrier leading to excess vascular leakage. Importantly accumulating evidence suggests that the abnormal thickening of the vascular basement membrane (BM) can contribute to excess vascular permeability. 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 in the development of retinal vascular lesions in 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 hyperglycemia-driven vascular lesions including retinal vascular leakage. To combat the pathophysiological event, we have developed a strategy for decreasing BM thickening and thereby reduce vascular leakage in the diabetic retina. These findings provide novel insights into the development and progression of diabetic retinopathy.

Biography

Sayon Roy received his PhD from Boston University and completed his postdoctoral training at Schepens Eye Research Institute, Harvard Medical School, Harvard University. He 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, 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. 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. Roy currently serves as a chartered member of the NEI Study Section of the National Institutes of Health.