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What is the role of mitochondria in diabetic retinopathy?

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Diabetic retinopathy remains one of the most debilitating complications negatively affecting the social entity of a patient. Hyperglycemia is considered as the main instigator initiating an array of metabolic, physiological and functional abnormalities in the retina and its vasculature. Despite extensive research in the field, the molecular mechanism of its development remains elusive, thus making the rationale for possible therapies limited. Available evidence suggests that diabetes increases oxidative stress, and oxidative stress plays a pivotal role in the development of diabetic retinopathy. It affects mitochondrial integrity causing mitochondrial DNA (mtDNA) damage, and this initiates a vicious cycle leading to decreased transcription and protein synthesis. This is further exacerbated due to subsequent decreased electron transport and increased superoxide radicals. Mitochondria biogenesis is tightly controlled by nuclear-mitochondrial transcriptional factors. Maintenance of the mtDNA is critical for the mitochondria to function properly, and mitochondria biogenesis is tightly controlled by nuclear-mitochondrial transcriptional factors. The enzymes important in mtDNA replication/repair, the DNA polymerase gamma and the base excision repair enzymes, are also encoded by the nuclear genome. We are investigating the role of nucleus-mitochondrial communication and the mtDNA replication machinery in the development of diabetic retinopathy. Our studies have documented that in diabetes, retinal mitochondria copy number is decreased, D-loop of mtDNA is damaged and the replication/repair machinery is impaired, and these abnormalities in mitochondria biogenesis are under the control of superoxide. We believe that understanding the mechanism(s) of retinal mitochondria damage should help identify novel targets for future therapeutic to slow down the progression of this devastating disease that is now the leading cause of blindness in young adults.

Biography

Renu A. Kowluru is currently Professor of Ophthalmology and Anatomy/Cell Biology at Wayne State University in Detroit, MI. She is a very well respected and established authority on diabetic retinopathy, and has published over 110 scientific papers. She serves on the editorial board of many leading journals including Investigative Ophthalmology and Visual Sciences, Expert Opinion on Investigational Drugs (Ashley Publication Ltd, London), and as a scientific reviewer for over 30 reputed journals. Her research interests are focused on understanding the molecular mechanism of diabetic retinopathy, especially elucidating the role of mitochondrial dysfunction and epigenetic modifications. The research is funded by RO1 awards from the National Institutes of Health, and also awards from the Juvenile Diabetes Research Foundation and other private organizations.

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