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Altered glutamate metabolism in diabetic retina: Potential mechanism of neurodegeneration in diabetic retinopathy

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Excess extracellular glutamate is known to cause excitotoxicity and neurodegeneration in the brain and retina under various pathological conditions. Diabetes-induced altered level of glutamate is believed to be a major factor to cause neurodegeneration in diabetic retina, leading to diabetic retinopathy. The purpose of this study was to understand the regulation of glutamate metabolism in non-diabetic and diabetic rat retinas. We performed glutamate metabolic studies in the rat retinas and in cultured Muller cells. We analyzed both rate of anaplerosis (de novo synthesis of glutamate and glutamine) and cataplerosis (catabolism of glutamate) in the control and diabetic rat retinas. We measured metabolites in those rat retinas using spectrophotometric, high performance liquid chromatography and radioisotopic techniques. In addition, we also analyzed glutamate uptake in the cultured retinal Muller cells under glutamate depletion conditions. Results of anaplerosis indicated that the level of glutamate and glutamine synthesis significantly decreased in diabetic retina compared to control ($p<0.05$). Cataplerosis experiments showed that glutamate oxidized to CO2 and lactate, and their levels were significantly decreased in diabetic retina compared to control. Glutamate uptake experiments in Muller cells suggested that the rate of glutamate transport increased when cells were depleted of glutamate. Thus, glutamate metabolism experiments suggest that the rate of anaplerosis of glutamate did not increase in the diabetic rat retina, however cataplerosis decreased, which gives the basis of an increased intracellular level of glutamate within Muller cells. Glutamate uptake experiments in cultured Muller cells suggest that either high or low intracellular levels of glutamate may hinder the rate of glutamate uptake. Thus, these results suggest the basis of glutamate excitotoxicity and neurodegeneration due to increased intracellular level of glutamate in the diabetic retina.

Biography

Mohammad Shamsul Ola is an Associate Professor in the Department of Biochemistry at King Saud University, Riyadh, Saudi Arabia. He completed his Postdoctoral Training at Medical College of Georgia and Pennsylvania State University, USA and subsequently joined as a Faculty Member at King Saud University in 2008. He is an established Scientist working in the research area of cellular and molecular mechanism of diabetic retinopathy. He has made fundamental discoveries that have greatly added to our understanding of vision impairment caused by diabetes. His research on altered metabolism in diabetes has contributed a thorough understanding of the molecular causes of impairment in the early stages of diabetic retinopathy. His major area of research interest includes molecular mechanism of neurodegeneration, and oxidative stress and neuroprotection in diabetic retinopathy.

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