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Glia: A real culprit of neuronal vulnerability in Alzheimer's disease

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Sporadic Alzheimer's disease (SAD) is a progressive neurodegenerative disorder with dysfunctional insulin signaling and energy metabolism. Growing evidence supports that impairment in brain insulin responsiveness, glucose utilization and energy metabolism may be a major cause of amyloid precursor protein mishandling. A support for this notion comes from the studies where Streptozotocin (STZ) induced brain insulin resistance in murine model, resulting into the SAD like brain pathology with cognitive decline. Intriguingly in vitro models have been used to understand the metabolic basis of SAD. However, mechanistic effects of brain insulin resistance on neurons and glia are not well understood. To understand the status of insulin signaling pathway, glucose uptake, glucose metabolism and energy homeostasis, STZ induced glial-neuronal co-culture model of SAD has been established. Present study suggests that glial cells are more compromised for insulin signaling than neurons. The evidence has been supported by glial activation in co-culture SAD model system. These changes were found to be correlated with amyloid deposition in this cellular model system which indicates that insulin signaling in glia may be a major regulator of amyloidogenesis in SAD brain.

Biography

Smriti Gupta is a PhD Scholar, Department of Biochemistry at Panjab University, India. She has done her bachelor's and master's Degree in Zoology at Banaras Hindu University. Then she has been a Project Assistant at Indian Institute of Technology Kanpur. Her research focuses on glial-neuronal crosstalk to understand the pathophysiology of Alzheimer's disease.

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