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Role of epigenetics in regulation of astroglial excitatory amino acid transporter 2 (EAAT2) expression

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In the CNS, extracellular glutamate is predominantly cleared by astroglial cells through the high-affinity excitatory amino acid transporter 2 (EAAT2). The expression of the pro-inflammatory cytokine IL-1□ and EAAT2 are dysregulated in various neurodegenerative diseases including HIV-1 induced NeuroAIDS. Dysregulation of astroglial EAAT2 expression leads to accumulation of extracellular glutamate which results in excitotoxic neuronal cell death. The notion that glutamate-induced brain damage can be attenuated by restoring glutamate homeostasis in the injured brain has drawn our attention in elucidating the role of epigenetics in EAAT2 expression. Our studies demonstrate that epigenetic modifications of EAAT2 gene at the chromatin level are involved in the transcriptional inactivation of the gene in response to IL-1□. Furthermore, our studies also demonstrate the role of microRNAs (miRNAs) in the post-transcriptional regulation of EAAT2 gene expression at the protein level. These observations demonstrate that targeting chromatin modifiers and miRNAs may have potential as novel therapeutic strategies for modulating EAAT2 expression not only in NeuroAIDS but also in other neurodegenerative diseases.

Biography

Dr. Datta is Assistant Professor in the department of Neuroscience, Temple University School of Medicine, Philadelphia, USA. He received his Ph.D. from University of Calcutta, India and did his postdoctoral training at Chicago Medical School, N. Chicago, Illinois. His research focuses on the identification of molecular mechanisms involved in the pathogenesis of HIV-1 induced NeuroAIDS, with particular interest in the role of neuroinflammation in regulation of Complement and EAAT2 genes, and the identification of putative targets for effective treatment and prevention of NeuroAIDS.

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