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Poster Presentation

Evaluation of Calcineurin Role in Neuroinflammation: Possible Targets for Early Detection and Treatment

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Abstract

Calcineurin (CaN) is a Ca^{2+} /calmodulin ($\text{Ca}^{2+}/\text{CaM}$)-dependent serine/threonine protein phosphatase expressed in most mammalian tissues but found at higher concentration in brain. In the last decade there have been a steadily increasing number of studies identifying neuronal CaN as a primary suspect in neuronal vulnerability, synapse loss, dendritic atrophy, synaptic dysfunction and neuroinflammation. Subsequently despite the apparently selective association of CaN with neurons and neuronal signaling cascades, many studies found that CaN, can also appear in primary glial cells and glial cells of healthy brain tissue and astrocytes, prominently following inflammatory insult. The clear connection between glial cells / astrocytes and neuroinflammatory signaling, in addition to the well-known the role of CaN in cytokine production in peripheral immune cells, suggested a potent association between glial CaN and the neuroinflammation inherent to most acute and chronic neuroinflammatory autoimmune diseases. In this review we aim to evaluate the Calcineurin role in neuroinflammation as an early event. Recent studies highly confirmed CaN as a major modulator of immune/inflammatory processes in glial cells and astrocytes that higher expression of CaN associated with early stage of neuroinflammatory autoimmune disease that makes CaN as novel target for early detection and treatment of neuroinflammatory autoimmune diseases.

Keywords: Calcineurin, Neuroinflammation, Early detection, Treatment

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