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## Poster Presentation

### Emerging Perspectives on Mtor-Associated Inflammation in Neurodegenerative Diseases

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#### Abstract

Inflammatory processes have been shown to be involved in development and progression of neurodegenerative diseases. Mammalian target of rapamycin (mTOR) involves in various cellular processes including autophagy, apoptosis and energy metabolism. Recently, studies have been shown an association between mTOR pathway and inflammation, supporting the role of the pathway in the pathogenesis of inflammatory disorders including neurodegenerative diseases. There are several studies have been shown that rapamycin, an antagonist of mTOR pathway, or PF-4708671, a mTOR substrate inhibitor, exhibits high neuroprotective effects through reducing inflammation. For example, rapamycin attenuates proinflammatory responses by increasing anti-inflammatory activity of regulatory T cells to restrain post-stroke neuro-inflammation. Moreover, pharmacological inhibition of mTOR decreases neuronal inflammation in cerebral palsy mice model subjected to hypoxia-ischemia and lipopolysaccharide-induced inflammation. Similarly, Liu et al indicated that inhibition of mTOR inhibits amyloid- $\beta$  or LPS-induced neuro-inflammation in mice models. Consistent with the anti-inflammatory effects of mTOR inhibitors, Ding et al, reported that melatonin negatively regulates the release of proinflammatory cytokines by inhibition of the mTOR in traumatic brain injury in animal models. Taken together, these results clearly suggest that mTOR inhibitors can be considered as a promising therapeutic target to suppress neuronal inflammation in neurodegenerative diseases. Understanding of the exact molecular mechanism of mTOR signaling could be helpful to design a novel mTOR inhibitor to regulate the inflammatory responses in neurodegenerative diseases.

**Keywords:** MTOR Signaling, Neuro-Inflammation, Lipopolysaccharide

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