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The Role of Inflammasomes for aβ Microglia Phagocytosis in Alzheimer Disease

Authors : Francesca La Rosa , Marina Saresella, Mario Clerici, Michael Heneka

Abstract : Neuroinflammation plays a key role in the modulation of the pathogenesis of neurodegenerative disorder such as Alzheimer's Disease (AD). Microglia, the main immune effector of the brain, are able to migrate to sites of Amyloid-beta (Aβ) deposition to eliminate Aβ phagocytosis upon activation by multiple receptors: Toll like receptors and scavenger receptors. The issue of whether microglia are able to eliminate pathological lesions such as neurofibrillary tangles or senile plaques from AD brain still remains the matter of controversy. Recent data suggest that the Nod Like Receptor 3 (NLRP3), multiprotein inflammasome complexes, plays a role in AD, as its activation in the microglia by Aβ triggers. IL-1β is produced as a biologically inactive pro-form and requires caspase-1 for activation and secretion. Caspase-1 activity is controlled by inflammasomes. We investigate about the importance of inflammasomes complex in the Aβ phagocytosis and its degradation. The preliminary results of phagocytosis assay and immunofluorescent experiment on primary Microglia cells to lipopolysaccharide (LPS) an Aβ exposure show that a previous treatment with LPS reduce Aβ phagocytosis. Different results were obtained in Primary Microglia wild type, NLRP3 and ASC Knockout suggesting a real inflammasomes involvement in Alzheimer's pathology. Inflammasomes inactivation reduces the production of inflammatory cytokines prolonging the protective activity of microglia and Aβ clearance, featuring a typical microglia phenotype of the early stage of AD disease.

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