

## Neuro-immune modulations in Alzheimer's disease

Interactions between brain and the immune system has received immense interest over decades, and in the recent years the interest is greatly renewed due to the discovery of functional lymphatic vasculatures in the dura. Evidences show that besides resident immune cells of the CNS, the peripheral blood cells, such as dendritic cells and monocytes, constantly survey the CNS in steady state, contributing to a bidirectional communication between the CNS and the periphery.

Cognitive deficit or dementia is the outcome of Alzheimer's disease (AD), and to date is the most common form of dementia in the world. Inflammation is one of the prominent features of AD, the trigger of which is yet uncertain. Inflammation can contribute to the onset/progression of Alzheimer's disease by activation of the innate immune response or by impaired adaptive immunity leading to chronic inflammation. On the other hand, protective anti-inflammatory cytokines, along with growth factors, have been shown to attenuate the disease progress in AD. Interestingly, deficits in peripheral immunity is also observed in subjects with AD. For example, myeloid dendritic cells were shown to be reduced, associated with severity in disease progression and depressive symptoms. In addition, several blood-derived metabolic and protein biomarkers are also emerging as crucial players in the diagnosis and prognosis of the disease. Therefore, a broader picture on the inter-communication between immunity and the CNS in Alzheimer's disease is emerging.

In this special issue, we invite submission of original research articles (in-vitro, animal and human studies), review articles, and perspectives that could stimulate the continuing efforts to better understand the relationship between immunity and Alzheimer's disease. We welcome submissions from a broad range of topic, including (but not limited to): CNS and peripheral immune crosstalk, mechanism of action, immune modulatory factors, pathogenesis, biomarkers, experimental therapeutics, gut-brain axis, and disease models of AD. Articles focusing on other tauopathies are also welcome.

**Submission Deadline:** 31 January 2022

**Submission:** <https://jin.imrpress.com>

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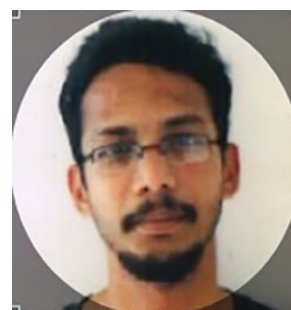
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