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EDITORIAL

# Editorial

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Far from the original concept that the central nervous system (CNS) is immunologically privileged, growing evidences suggests that the brain and immune system are intricately connected and engaged in significant crosstalk to maintain homeostasis. It is therefore not surprising that activation of various component of the immune system is invariably observed during CNS injury and is now recognized as a prominent and common feature of many neurological disorders. Yet, the outcome of altered or dysfunctional immune system on pathogenesis is hardly predictable as both neuroprotective and neurodestructive outcomes have been attributed to immune activation.

In this special issue, the authors provide overviews of the pathophysiological involvement of neuroinflammation in various neurological disorders. In particular, they discuss the mechanisms by which neuroinflammatory processes may, in some cases, prevent disease progression but they also show that in other circumstances neuroinflammation can be instrumental in neuronal and glial degeneration. This double-edged feature of neuro-immune interactions is undoubtedly linked to disease context and phase and immune component involved. In this special issue, conceptually different pathological situations associated with neuroinflammatory processes are covered.

Thus, in the first article, the author outlines the dual role of neuroinflammation during viral infection of the CNS showing that while immune responses against the infectious agent control its spread, collateral neuronal damages can ensued and are therefore essential in determining disease outcome. In a following article, the authors discuss the role of neuro-immune interactions in multiple sclerosis, a degenerative disorder in which a primary dysfunction of the immune system is believed to be critically involved in the destruction of myelinating cells. They outline how non-invasive imaging methods can help our understanding of the development of inflammatory lesions and define the molecular and subcellular alterations caused by neuroinflammation. Then, a series of review articles go on to highlight the role of neuro-immune interactions in neurodegenerative disorders for which immune responses secondary to neuronal injury are though to contribute to the aggravation of neuronal damages and progression of the disease. This concept is first illustrated in Alzheimer's disease with two articles covering emerging aspects of amyloid plaque clearance by local (microglia) as well as blood-born (macrophages) innate immune cells and how these unexpected mechanisms could be stimulated for therapeutic purposes. In additional articles, the role of neuroinflammation is then discussed in the context of Parkinson's disease, amyotrophic lateral sclerosis and Huntington's disease pathogenesis with chapters highlighting ways in which detrimental immune system responses could be harness and provide therapeutic benefits for these devastating neurological disorders. Lastly, the role of inflammation in the disease pathomechanism of developmental disorders such as perinatal brain damage is

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further discussed with as a special emphasis placed onto the importance of the blood brain barrier which differs during development and in adulthood.

Finally, in a last review article, the authors explore future directions for immune modulation in neurodegenerative disorders. Focusing on Parkinson's disease, they identify new strategies aimed at manipulating the immune response by acting on the orphan nuclear receptor Nurr-1 or by producing the neuropeptide vasoactive intestinal peptide (VIP). These new ways of manipulating the neuroinflammatory processes might open new avenues for reducing neuronal and glial alteration associated to the neuroinflammatory processes in the central nervous system.