

Neuroinflammation and its role in the development of neurological diseases (Multiple Sclerosis, Neurodegeneration)

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Abstract

Neuroinflammation refers to immune-related inflammatory processes within the central nervous system (CNS). It involves activation of microglia and astrocytes, altered blood–brain barrier (BBB) function, recruitment of peripheral immune cells, and release of inflammatory mediators (cytokines, chemokines, reactive oxygen/nitrogen species). In acute or controlled form, neuroinflammation can be protective by clearing debris and supporting repair. However, when inflammation becomes chronic or dysregulated, it contributes to neuronal dysfunction, synaptic damage, demyelination, and progressive neurodegeneration.

Keywords:

Neuroinflammation; microglia; astrocytes; cytokines; chemokines; blood–brain barrier; immune dysregulation; oxidative stress; synaptic dysfunction;

In Multiple Sclerosis (MS), neuroinflammation is a core driver of pathology. Immune cells infiltrate the CNS across a disrupted BBB and trigger inflammatory cascades that damage myelin and oligodendrocytes, forming demyelinating lesions. Over time, sustained inflammatory activity also promotes axonal injury and neurodegeneration, which are strongly associated with irreversible disability. This explains why controlling inflammatory activity is crucial in relapsing MS, while progressive forms may involve additional compartmentalized CNS inflammation and degeneration.

Misfolded or aggregated proteins (β -amyloid and tau in Alzheimer's; α -synuclein in Parkinson's) can chronically activate microglia and astrocytes. Persistent activation

may shift these cells toward a pro-inflammatory state, increasing oxidative stress, impairing synaptic function, and reducing clearance of toxic proteins—creating a vicious cycle where inflammation and neurodegeneration reinforce each other. Understanding neuroinflammation therefore supports improved diagnostics (biomarkers, imaging) and new therapies aimed at modulating glial activation, BBB integrity, and inflammatory signaling pathways. In neurodegenerative disorders, neuroinflammation is often not the original trigger, but it strongly shapes disease progression and symptom severity. In Alzheimer's disease (AD), accumulation of β -amyloid plaques and tau tangles is associated with chronic microglial and astrocytic activation. Microglia recognize aggregated proteins via pattern-recognition receptors and may initially help clear amyloid. But persistent exposure can push microglia toward a sustained inflammatory state that damages synapses and reduces effective clearance. Inflammatory mediators can also promote tau phosphorylation and spread, linking immune activation to hallmark pathology. Synaptic dysfunction and loss—closely correlated with cognitive decline—can be worsened by inflammatory cytokines and complement-mediated synaptic pruning mechanisms that become overactive.

Conclusion

Neuroinflammation plays a dual role in CNS disease: it can support repair in the short term, but chronic or uncontrolled activation becomes a major pathological mechanism. In MS, neuroinflammation drives demyelination and contributes to axonal loss and long-term disability. In neurodegenerative disorders, persistent glial activation, oxidative stress, and impaired protein clearance accelerate neuronal and synaptic damage. Targeting neuroinflammatory pathways—while preserving protective immune functions—represents a promising strategy for slowing progression and improving outcomes across a wide range of neurological diseases.