Neural Plasticity

Hindawi

Special Issue on

Impact of Inflammation on Neuroplasticity in the Central Nervous System

CALL FOR PAPERS

Inflammation in the central nervous system (CNS) mediates disease processes leading to neurological dysfunction. It is well recognized that there is extensive crosstalk between the nervous and immune systems. Trauma and infections of the CNS are characterized by high levels of inflammatory mediators such as cytokines and immunomodulatory neuropeptides. Such mediators can sensitize resident immune and neural cells and recruit immune cells from periphery in a positive feedback loop-like manner. Recruited periphery immune cells are involved with removal of dead tissue and debris by phagocytosis and secretion of a wide spectrum of cytokines having trophic, mitogenic, and chemotactic properties. These events affect the behavior of resident cells in the area of injury. Inflammation in the brain as a result of trauma, aging, changes in microbiome, autoimmune, infectious, and neurodegenerative diseases leads to the activation of resident immune cells, increased blood-brain barrier permeability, and infiltration of peripheral immune cells which may lead to reduced neuroplasticity. Understanding and regulating inflammatory responses in a timely manner is therefore critical in preserving normal CNS functions.

Immune responses may be protective as well as harmful to the CNS, and while they may limit infection and trauma associated damage, immune responses may also be detrimental if not controlled and limited. Severity and duration of injury can foster dysfunctional immune reactions that become damaging. Indeed, several neurodegenerative disorders are associated with exacerbated immune responses involving resident and peripheral cells in the brain affecting neuroplasticity. Thus, a fine balance is necessary for limiting the damage in CNS.

Immune responses are likely involved in most CNS disease states since they influence essentially every pathophysiologic area of neurological function, including the neuroendocrine system, neurotransmitter metabolism, regional brain activity, and behavior. Functional decline, including cognitive, affective, and motor function, may follow acute and chronic neuroinflammatory states.

The identification of treatment options capable of limiting CNS inflammation and modulating neuroplasticity represents an important challenge. Corticosteroids represent an obvious means for controlling CNS inflammation. However, they may interfere with the bodies' ability to clear infections and hence could induce adverse effects. Furthermore, increased susceptibility to inflammatory and autoimmune disease could stem from corticosteroid-dependent impairments in the activity of the hypothalamic pituitary axis (HPA). Antioxidant based therapies represent an important new area for study. The administration of effective antioxidants has the potential to limit inflammation, and reactive oxygen species (ROS) are known to play an important role in inflammatory brain diseases.

Neuroplasticity in the form of neurogenesis, remyelination, and synaptic plasticity are key contributors to brain repair and functional recovery during inflammatory processes in the CNS. The interaction between inflammation and subsequent neuroplasticity is not well understood. Understanding such interaction could lead to the development of new therapies to promote brain repair mechanisms.

This special issue will focus on the interplay between CNS inflammation, neuroplasticity, and how these interactions influence CNS repair and functional recovery following infectious and sterile neuroinflammation.

Potential topics include but are not limited to the following:

- Neuroinflammation and the role various diseases have on neuroplasticity, both centrally and peripherally
- The impact of infectious, cerebrovascular, neurodegenerative diseases and so forth on recovery potential of nervous system
- Autoimmunity and demyelinating disease influence on neuroplasticity
- Impact of comorbidities (e.g., malnutrition, diabetes mellitus, hypertension, and psychological stress) on nervous system inflammation and immunity
- Potential and timing of neuroinflammation treatment options

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