BioMed Research International

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Special Issue on

Molecular and Cellular Mechanisms of Neuroinflammation

CALL FOR PAPERS

In the central nervous system (CNS), the innate immune response plays a significant role in both physiological and pathological conditions. CNS diseases including traumatic brain injury, ischemic stroke, brain tumor, and cerebrovascular and neurodegenerative diseases trigger a cascade of events broadly defined as neuroinflammation, which is characterized by the activation of the microglia and astrocyte population. Reactive microglia and astrocytes potentially cause injury to the blood-brain barrier and account for neuronal impairment.

Microglia/macrophages are major cells in an organism's immune system, and they play important roles in CNS repair and regeneration. While microglia are derived from primitive macrophages during brain development, monocyte-derived macrophages are recruited from the blood following injury or disease. Functionally differentiated microglia and macrophages are commonly subdivided into two categories: the classically activated phenotype (M1), which is characterized by the secretion of proinflammatory and cytotoxic mediators, and the alternatively activated phenotype (M2), involved in tissue repair and remodeling. Similarly, the potential roles of reactive astrocytes, NG2-positive cells, oligodendrocytes, and endothelial cells and neurons in the neuroinflammatory process have been recently established.

The aim of this special issue is to publish high-quality research papers as well as review articles addressing recent advances on the cellular and molecular mechanisms of neuroinflammation. We encourage submission of manuscript exploring the cross-talk microglia/macrophage-astrocyte/neuron and its role in different CNS diseases.

Potential topics include but are not limited to the following:

- All potential causes of neuroinflammation (infection, neurodegenerative disorders, vascular dementia, teratogenesis, brain tumors, obesity, hypertension, autoimmunity, and toxic exposure)
- Mechanisms driving physiological outcomes of neuroinflammation (i.e., tissue-repair response and adaption to stress)
- Mechanisms driving pathological outcomes of inflammation (i.e., tissue damage and tumor growth)

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/bmri/neuroscience/mcmn/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

Lead Guest Editor
Anna Di Vito, Magna Graecia University, Calabria, Italy
divito@unicz.it

Guest Editors
Giuseppe Donato, Magna Graecia University, Calabria, Italy
gdonato@unicz.it

Daniele Tomassoni, University of Camerino, Marche, Italy
daniele.tomassoni@unicam.it

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