MICROGLIA- A THERAPEUTIC TARGET IN NEUROLOGICAL DISEASES AND DISORDERS

Aims & Scope:

Microglia, the resident cells of the central nervous system (CNS) respond swiftly to a variety of acute and chronic stimuli. In response to various stimuli, both exogenous and endogenous, activated microglia showed increased production of trophic factors (e.g. insulin-like growth factors) and proinflammatory cytokines (e.g. TNF-α and IL-1β) or mediators (e.g. nitric oxide, reactive oxygen species) that are either protective or detrimental to neighboring neurons and oligodendrocytes. Such a phenomenon is not only observed in the adult but is also evident in the developing CNS including the brain and the retina. Associated with the above, various signaling pathways linked to the production or regulation of the various factors e.g. NF-κB and prostanoid pathways in the activated microglia are up-regulated. Recent focus on microglial research has been aimed at activated microglia which play a crucial role in the pathogenesis of many diseases such as multiple sclerosis, Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis by producing a vast number of neurotoxic mediators such as cytokines, free radicals, prostaglandins and glutamate. In search of potential therapeutic interventions for neurodegenerative diseases or injuries, several therapeutic agents for modulating microglia activation such as minocycline, inhibitors of prostaglandin synthesis, herbal medicines {e.g. extract of *Panax notoginseng* (NotoG™)}, inhibitors of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase (apocynin, diphenyliodonium and others) are under investigation. This special issue will deal with the trophic and toxic roles of microglia and will highlight the pharmacological approaches attempted at suppression of microglial activation in neuroinflammatory/neurodegenerative diseases of the CNS.

Key words:

Microglia, Central Nervous System, insulin-like growth factors, cytokines, oligodendrocytes.

Subtopics:

Screening for inhibitors of microglia to reduce Neuroinflammation

Towards a novel glioma treatment: the potential of genetically enhanced microglia

Microglial senescence and degeneration

Microglia as a therapeutic target in neuropathic pain

Potential Drugs Targeting Microglia: Current Knowledge and Future Prospects

When friends become foes- immunosuppression in brain disease

Glutamate Receptors in Microglia

Effects Of Iron Accumulation In Microglia on the Brain
Role of microglia in the pathogenesis of septic encephalopathy

Gastrodin inhibits LPS-induced MAPK activation and inflammatory responses via activation of the phosphatidylinositol 3-kinase Pathway in murine BV-2 microglial cells

Bone marrow-directed downregulation of CCR5 protects the brain from microglial inflammation in acute excitotoxic seizures

A friend in need may not be a friend indeed: the role of microglia in neurodegenerative diseases

Schedule:

Manuscript Submission Deadline: August 2012

Peer Review Due: September 2012

Revision Due: October 2012

Notification of Acceptance by the Guest Editor: November 2012

Final Manuscripts Due: December 2012