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Molecular Mechanism and Regulation in Neuroinflammation

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Message from the Guest Editor

Neuroinflammation is defined as an inflammatory response within the brain or spinal cord, including infection, traumatic brain injury, toxic metabolites, or autoimmunity. This inflammation is mediated by the production of cytokines, chemokines, reactive oxygen species and second messengers. These mediators are produced by resident CNS glial cells (microglia and astrocytes), endothelial cells, and immune cells of peripheral origin. These neuroinflammatory responses have immunological, physiological, biochemical, and psychological consequences. From a pathological point of view, in the central nervous system, neuroinflammation is associated with damage from direct penetrating physical injury: e.g., traumatic brain injury (TBI), spinal cord injury (SCI), or neurodegenerative multiple sclerosis (MS) and other biochemical diseases), Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD) and amyotrophic lateral sclerosis (ALS), tumors (gliomas) or senescence. The research topics of this special issue aim to cover promising, recent and novel molecular regulation and research mechanisms for the diagnosis and treatment of neuroinflammation.