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Neuroinflammation: The Silent Culprit behind a Range of Neurological Disorders

Sadeghdoust Mikhailova*

Department of Biological Science, Chosun University, Gwangju, Republic of Korea

Introduction

Neuroinflammation, a term that is gaining increasing attention in both research and clinical practice, refers to the inflammatory response that occurs within the brain and spinal cord. It is a complex and multifaceted process, involving various cells, molecules and signaling pathways. While inflammation is a protective mechanism in response to injury or infection in peripheral tissues, neuroinflammation is often subtle, persistent and can become maladaptive, contributing to a variety of neurological disorders. The brain, in particular, is highly susceptible to neuroinflammatory responses, as it is a vital organ that plays a central role in regulating the body's overall function. Recent research has begun to elucidate the significant role of neuroinflammation in the pathogenesis of numerous neurological conditions, including Alzheimer's disease, Parkinson's disease, multiple sclerosis, epilepsy and even psychiatric disorders such as depression and schizophrenia. Despite its importance, neuroinflammation often operates silently, without overt symptoms, which makes it difficult to detect in its early stages and a challenge to treat effectively [1].

Description

Neuroinflammation is the brain's response to injury, infection, or disease. The inflammatory process in the brain is orchestrated by microglia, which are the resident immune cells of the Central Nervous System (CNS), as well as astrocytes, oligodendrocytes and neurons. Microglia are constantly surveying the environment for signs of distress and are the first responders to any damage or infection. Upon activation, they undergo morphological and functional changes, releasing pro-inflammatory cytokines, Reactive Oxygen Species (ROS) and other immune mediators that aim to repair the injury or eliminate the cause of inflammation. Astrocytes, which are supportive cells in the brain, can also become activated during neuroinflammation. In their reactive form, astrocytes release inflammatory molecules that further exacerbate the inflammatory environment. This chronic inflammatory state can create a cycle that perpetuates neuronal damage, impairs neuroplasticity and ultimately disrupts normal brain function. Inflammation in the brain can be caused by a variety of factors, including infections, Traumatic Brain Injury (TBI), ischemic stroke, toxins, or chronic neurodegenerative diseases. In some cases, neuroinflammation arises without an obvious trigger, as is the case in many neurodegenerative disorders. The brain, which is protected by the Blood-Brain Barrier (BBB), can still become vulnerable to inflammatory insults under certain conditions. When the BBB is compromised, neuroinflammatory responses can be exaggerated and lead to further complications [2].

Neuroinflammation has been implicated in a wide array of neurological disorders, ranging from neurodegenerative diseases to psychiatric conditions. Understanding the role of inflammation in these disorders is critical for the development of novel therapies. Below, we explore some of the key neurological conditions where neuroinflammation plays a central role. Alzheimer's disease is the most common form of dementia, characterized by progressive cognitive decline and memory loss. Inflammation is considered a key feature of AD pathology and recent studies suggest that neuroinflammation contributes significantly to disease progression. In AD, the accumulation of amyloid-beta plaques and tau tangles triggers the activation of microglia, which release pro-inflammatory cytokines and other toxic molecules. While microglial activation initially tries to clear amyloid plaques, sustained inflammation leads to neuronal damage and synaptic loss, accelerating cognitive decline. The relationship between amyloid-beta, tau and neuroinflammation in AD is complex, with each factor exacerbating the others. It has been proposed that reducing neuroinflammation through anti-inflammatory therapies could slow or even halt the progression of AD, but clinical trials targeting inflammation have so far yielded mixed results [3].

Parkinson's disease is a neurodegenerative disorder characterized by the progressive loss of dopaminergic neurons in the substantia nigra. Neuroinflammation is a hallmark of PD, with activated microglia and astrocytes present in the brains of affected individuals. The presence of alpha-synuclein, a protein that aggregates in the brains of PD patients, triggers an inflammatory response. This chronic inflammation contributes to the death of dopaminergic neurons and the worsening of motor symptoms, including tremors, rigidity and bradykinesia. Recent studies have suggested that targeting neuroinflammation could offer a therapeutic strategy for PD. Anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), have shown promise in preclinical models, although human studies are still inconclusive. Furthermore, there is increasing interest in using immune-modulating therapies to reduce neuroinflammation and protect dopaminergic neurons [4].

Neuroinflammation has also been linked to psychiatric disorders such as depression and schizophrenia. In depression, there is growing evidence that pro-inflammatory cytokines, such as Interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), are elevated in the blood and cerebrospinal fluid of patients. These inflammatory markers are thought to contribute to the disruption of neurotransmitter systems, particularly serotonin and dopamine, which are involved in mood regulation. Moreover, neuroinflammation may impair neurogenesis and synaptic plasticity, further exacerbating depressive symptoms. In schizophrenia, neuroinflammation may play a role in the pathophysiology of the disorder by influencing brain regions involved in cognition, emotion and perception. Microglial activation has been observed in individuals with schizophrenia and studies suggest that targeting neuroinflammation may offer a novel approach to treating treatment-resistant symptoms of the disorder. The key players in neuroinflammation are microglia, astrocytes and neurons. The intricate interactions between these cells and the molecules they produce contribute to the inflammatory response in the brain [5].

Conclusion

Neuroinflammation is a critical but often overlooked factor in the development and progression of many neurological disorders. From Alzheimer's and Parkinson's disease to multiple sclerosis, epilepsy and even psychiatric conditions like depression, neuroinflammation plays a central role in causing neuronal damage, cognitive decline and functional impairment. While the mechanisms of neuroinflammation are complex, recent advances in our understanding of its molecular and cellular underpinnings offer hope for

***Address for Correspondence:** Sadeghdoust Mikhailova, Department of Biological Science, Chosun University, Gwangju, Republic of Korea; E-mail: sadeghdoustikha.milova@ous.kr*

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the development of targeted therapies. As research continues to uncover the intricacies of neuroinflammation, it is likely that novel approaches to prevent, manage, or even reverse its damaging effects will emerge. The challenge, however, lies in the fact that neuroinflammation often operates silently, without obvious symptoms, until significant damage has already occurred. Early detection, prevention and intervention will be key to combating the silent culprit of neuroinflammation and protecting brain health for future generations.

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Conflict of Interest

None.

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