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Causes of Neuroinflammation

Issac Parker*

Department of Public Health, University of Cape Coast, Ghana, West Africa

Corresponding author: Issac Parker, Department of Public Health, University of Cape Coast, Ghana, West Africa; E-mail: issacpark@yahoo.com

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Commentary

Neuroinflammation is defined as an seditious response within the brain or spinal cord. This inflammation is regulated by the product of cytokines, chemokines, active oxygen species, and secondary couriers. These intercessors are produced by the abiding CNS glia (microglia and astrocyte), endothelial cells, and vulnerable cells located peripherally. There are vulnerable, physical, biochemical, and cerebral goods of these neuroinflammatory responses.

Neuroinflammation is involved in contributing to a variety of neurologic and somatic disorders including Alzheimer's disease (AD), Parkinson's disease (PD), and depression. In this chapter, we focus on the role of neuroinflammation in the management of these three diseases and demonstrate the interaction between the immune response and the central nervous system in the context of gender diversity in disease progression. Much of this chapter is based on clinical findings; however, from time to time we use pre-clinical models where human studies are currently in short supply. We begin by elaborating on the pathology of neuroinflammation, distinguishing between acute inflammation and chronium, and examining donations from innate and flexible immune systems. Next, we summarize the potential mechanisms of immune cells including interleukin-1 beta (IL-1β), tumor necrosis factor α, and IL-6 in AD, PD, and the development of depression. In view of the strong sexual bias seen in these diseases, we also examine the role of sex hormones, e.g., estrogen and testosterone in mediating neuroinflammation at the cellular level. Specifically, we specify how sex hormones can contribute to different behavioral and clinical symptoms as well as speculation between men and women with AD, PD, or depression. Finally, we highlight the potential role of exercise in alleviating neuroinflammation, as well as evidence that anti-inflammatory drugs improve cognitive symptoms seen in brain-related diseases.

Neuroinflammation (NI) is an important component of many CNS diseases, and reducing NI is believed to reduce disease severity and improve patient outcome in most cases. There are a variety of targeted NI target targets such as enzymes, receptors, and ion channels. In this review, the targets are highlighted where advances in medical chemistry have been achieved in the past and include purinergic receptors P2X4 and P2X7, the kynurenine metabolizing enzymes indole 2,3-dioxygenase and kynurenine aminotransferase, toll-like receptors. and TLR9, and TLR9. fractalkine receptor CX3CR1. These goals are also linked to the biological mechanisms underlying NI and disease. Although increased risk of infection is a potential problem for NI targets due to the immune effects, there is a significant chance of finding new molecules to reduce NI in CNS infections.

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Declaration of Conflicting Interests

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