Role of Neuroinflammation in Depression

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Abstract

The prevalence of mental health disorders continues to rise worldwide, such that it is estimated that 1 in 4 individuals will be affected by a mental health disorder at some point in their lifetime. Research has found that the most prevalent mental health disorders are depressive disorder. Depression is a common condition, particularly within the aging population. The development of depression, multifactorial disorder with the signing of neuroinflammatory, appears to be associated with the body allostatic disorder. Research has linked neuroinflammation as a major contributing factor to depression diseases. Recent findings link between inflammation and depression and hypothalamic-pituitary-adrenal particular role (HPA) axis in depression have created. This article reviews the clinical and experimental studies investigating the role of axis HPA, HPA hyperactivity (resulting in increased levels of cortisol), as well as pro-inflammatory cytokine tumor necrosis factor, C-reactive protein and interleukins, in depressed patients. The main reason neuroinflammation effects on depression show lie within the dysregulation of the control and release of pro- and anti-inflammatory cytokines. This can come from an internal or external insult to the system, or from changes in the individual due to aging which peaking in immune dysregulation. The need to reduce neuroinflammation has led to extensive research into neuroprotectants. We discuss the efficacy found with nicotine, alcohol, resveratrol, curcumin, and ketamine. Our main focus will be on what research tells us about the connections between neuroinflammation and depression, and the hope that neuroprotectants research gives people suffering from depression stemming from neuroinflammation.

Keywords: Hypothalamic pituitary adrenal axis, Neuroinflammation, Depression

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