Journal of Traumatic Stress Disorders & Treatment

Commentary

A SCITECHNOL JOURNAL

Impact of Depression on Body Immune System

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Introduction

The World Health Organization has identified major depression as a main cause of disability globally. It is a prevalent and often fatal condition. While antidepressants are unquestionably helpful in roughly 70% of cases, a significant number of individuals remain partially or completely unresponsive to treatment. Treatment resistance has no easy explanation; however it is possible that modern antidepressants do not adequately address all of the pathogenic mechanisms that cause the primary symptoms of depression. As a result, there is a pressing need to widen the range of targets on which antidepressants are thought to work [1].

All currently available antidepressants are based on the monoamine theory of depression, which claims that a malfunction of biogenic amines in the limbic and cortical circuits is the source of depression's principal symptoms. As a result, antidepressants are thought to work by rectifying these anomalies. In recent years, however, more emphasis has been paid to the interplay between the brain and peripheral organs (the "body-mind" connection), in which changes in the endocrine and immunological systems play a key part in the pathological changes that occur in depression [2]. As a result, inflammation is beginning to emerge as a major contributor factor not just to depression and other serious psychiatric illnesses, but also to the link between mental disease and medical disorders. For example, it is now clear that there is a link between the severity and duration of serious depression and an increased risk of heart disease, type 2 diabetes, different autoimmune illnesses, arthritis, and cancer.

The idea of a malfunctioning immune system playing a vital influence in mental health dates back to antiquity. However, clinical and experimental evidence suggesting that parts of both cellular and humoral immunity are defective in serious depression has only been gained in the last 30 years. The brain was once thought to be an immunologically privileged organ, shielded from the peripheral immune system by the blood-brain barrier. It is now clear that this viewpoint is wrong, and that peripherally produced cytokines, chemokines, prostenoids, and glucocorticoids, as well as some immune cells, have direct influence on the brain. The impact of big molecules from the periphery on the brain is surprising, given the lack of specialised transporters for peptides like interleukins at the blood-brain barrier.

However, there is now experimental evidence that such molecules could enter the brain a) through a leaky blood-brain barrier that occurs in major depression, b) through activation of endothelial cells that line the cerebral vasculature and produce inflammatory mediators inside the barrier, and c) through binding to cytokine receptors associated with the vagus nerve and thus signaling inflammatory mediators inside the barrier. The proinflammatory cytokines activated both neuronal and non-neuronal (for example, microglia, astrocytes, and oligodendroglia) cells in the brain via the nuclear factor-kappa-beta (NF-kB) cascade, like the peripheral inflammatory response. The focus of this review is on the negative effects of proinflammatory cytokines, which are expected to cause cellular harm in pathological amounts in the brain and periphery. However, these same cytokines offer trophic support for neurons, increase neurogenesis, and contribute to proper cognitive function at physiological concentrations [3]. Inflammation is also a defense mechanism against bacteria, viruses, and oncogenes, as well as a key component of the stress response. Inflammation begins as a time- and site-specific defence mechanism aimed at not only defending the body from harmful germs, but also eliminating injured neurons and, under physiological conditions, mending damaged neural networks. Inflammatory mediators only perform a pathogenic role in instances where their concentrations exceed the physiologically appropriate range. While it is clear that this circumstance applies to people with serious depression, it is important to note that inflammation is an important part of infection prevention. Immunomodulators must be developed with caution since, in order to become clinically effective antidepressants, they will need to be given to patients for months, if not years, in order to maintain remission from depression.

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Citation: *Huang Z (2021) Impact of Depression on Body Immune System. J Trauma Stress Disor Treat 10:7.*

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Received: July 01, 2021 **Accepted:** July 15, 2021 **Published:** July 22, 2021

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