Inflammation in the Pathogenesis of Depression

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Abstract
Depression is a mental disorder that results from changes in the central nervous system (CNS) that may result from immunological abnormalities. According to the World Health Organization, major depression will become the leading cause of disability worldwide. Accumulating evidence has indicated the existence of reciprocal communication pathways between nervous, endocrine and immune systems. The immune system affects the CNS through cytokines, which regulate brain activities and emotions. Cytokines affect the activity of the two biological systems that are most associated with the pathophysiology of depression: The hypothalamic-pituitary-adrenal axis and the catecholamine/sympathetic nervous system. Pro-inflammatory cytokines and stress are important in inflammatory and neurogenesis and neuroprotection. Stress induces pro-inflammatory cytokines over secretion, which result in activation the HPA axis and neurotransmitter turnover, thus leading to depression. The use of cytokine inhibitors and anti-inflammatory drug is effective in the treatment of the depression. Although there are known effective treatment for depression, fewer than half of these affected in the world. In this review calculated recent literature related to the effect of inflammation on the pathogenesis depression.

Keywords: Inflammation, Central nervous system, Cytokine, Endocrine, Stress

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