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Poster Presentation Review

Lipopolysaccharide and BDNF in Anxiety and Depression

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Abstract

Brain derived neurotrophic factor (BDNF) is a neurotrophin acting at several levels in the brain. Additionally, BDNF was shown to be responsible for the survival, maintenance and growth of neurons. Also, BDNF plays an important role in synaptic plasticity and memory processes. In activity-dependent synaptic plasticity, BDNF enhances long-term potentiation (LTP) in the hippocampus but blocks the induction of long-term depression (LTD) in the visual cortex. A relation between BDNF expression and anxiety was shown in the elevated plus maze where BDNF overexpression in mouse hippocampal astrocytes promotes local neurogenesis and elicits anxiolytic like activities. Moreover, BDNF is implicated in the pathophysiology of depression. Postmortem studies have shown that hippocampal BDNF levels are decreased in depressed patients and increased in patients receiving antidepressant treatment. On the other hand, it has been suggested that infections and inflammatory processes may act as causative factors in emotional disorders, including anxiety and depression. Lipopolysaccharide (LPS) is a component of the cell wall of gram negative bacteria and is known to induce a profound inflammatory and immunostimulatory response. Substantial evidence exists to suggest that the negative effects of LPS on synaptic function are due to the neurodegenerative effects. It elicits through its activation of proinflammatory cytokines such as interleukin-1β (IL-1β). However, it has been proposed that LPS can affect expression of neurotrophins and their receptors in the CNS and that this action may contribute to the functional impairments observed upon LPS injection; injection of LPS has been reported to significantly decrease BDNF expression in the hippocampus and several cortical regions may create to anxiety and depression.

Keywords: Lipopolysaccharide, BDNF, Anxiety, Depression.

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