The Effect of Stress on Neuroinflammation in Multiple Sclerosis

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Abstract

Multiple sclerosis (MS) is a chronic autoimmune disorder of central nervous system. This demyelinating disease affects more than 2.3 million people worldwide. Most of patients are young adult. There are many possible triggering factors including infections, toxin, immunization, trauma, sunlight exposure and hormonal variable in pathogenesis of MS. One of the important triggers is stress. There are some substantial evidence that indicates stress can precipitate or worsen symptoms and signs of inflammation in general and more specifically in multiple sclerosis. This review has focused on elucidating the effect of stress on neuroinflammation in multiple sclerosis. The mechanism of stress in MS is not completely understood. There are some proposed theories such as secretion of Corticotropin Relapsing Hormone (CRH) and neurotensin (NT). These inflammatory factors activate microglia and mast cells leading to maturation and activation of T17 autoimmune cells. Disruption of Blood Brain Barrier (BBB) cause T17 cells to enter into the CNS. Finally, brain inflammation will be worsened. The pathway of stress neuroinflammation can be considered as a therapeutic target for M.S patients. Some evidences indicate Glatiramer Acetat, stress management therapy, Diazepam, Alprazolam and CRH antagonists can reduce brain inflammation. So the mechanism of stress in pathogenesis of M.S can be novel therapeutic target.

Keywords: Stress, Neuroinflammation, Multiple Sclerosis

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