Improving Neuroplasticity Through Neuroinflammation Pathways as a Therapeutic Goal in the Treatment of Autism

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Abstract
Neuroplasticity is the brain’s ability to reorganize itself by forming new neural connections throughout life. Neuroplasticity allows the neurons in the brain to compensate injury and disease and to adjust their activities in response to new situations or to changes in their environment. At the other side, it is now well established that neuronal function is strongly influenced by both central and peripheral inflammation and it is able to modulate the efficacy of synaptic transmission and the induction of the main forms of synaptic plasticity. Astrocytes, Glial and microglial cells are recognized as active elements of synapses, playing a central role in neuro-inflammatory processes. This feature can be used as therapeutic goal in many brain diseases such as Multiple sclerosis, Alzheimer, Epilepsy, Parkinson’s disease, Autism spectrum disorder, etc. Autism, or autism spectrum disorder (ASD), refers to a range of conditions characterized by challenges with social skills, repetitive behaviors, speech and nonverbal communication, as well as by unique strengths and differences. Newly, some studies suggested that an abnormal function of glia and astrocytes may be involved in the development of autism. Recent studies reported some markers that are effect ASD mechanism including glial fibrillary acidic protein (GFAP), aquaporin4, connexin43, methyl-CpG-binding protein 2 (MeCP2) and etc. The present review summarizes the latest understanding of novel ASD treatment that effect neuron plasticity, especially proteins and cytokines that are involved in neuro-inflammation pathways and the process that damages neuroplasticity. However, there is yet no direct evidence showing how neuro-inflammation pathways can improve neuroplasticity in the brain of autistic patient and improve the life of these patients. This review indicates more research is essential to study on these markers as a therapeutic goal in the treatment of autism.

Keywords: Autism spectrum disorders, Brain disease, Neuroinflammation, Synaptic plasticity

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