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Poster Presentation

The Role of Amyloid Beta-Peptides and Tau Protein in Alzheimer's Disease

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Abstract

Alzheimer's desease is the most common age-related neurodegenerative disorder, and cognitive problems such as defects in learning and memory are of its symptoms. Among the factors involved in the pathogenesis of the disease are biochemical disorders in protein production, oxidative stress, decreased acetylcholine secretion and inflammation of the brain tissue. Extra-neuronal accumulation of Beta-amyloid and hyperphosphorylated Tau neurofibrils degredate the dendrites and destroy the synapses, which ultimately results in memory loss in Alzheimer's patients. Amyloid Beta is an important molecule in the pathogenesis of Alzheimer's disease, which progressively accumulates in the mitochondrial matrix and directly associated with the mitochondrial toxicity, which leads to the production of ROS and the oxidative stress that result in the neural dysfunction and eventualy the death of neurons. Beta-amyloid fibrils as plaque activate the microgelia, which results in the release of inflammatory cytokines and destruction of neurons. One of the most important effects of beta-amyloid is the damage to synaptic activity and inhibition of stimulant synapses, which in fact causes disruption in the the learning and memory system. It seems that the production of abnormal forms of Beta-amyloid peptides and Tau proteins is one of the main causes of Alzheimer's disease and observing the amyloid plaques in the cortex and the hippocampus in the early stages of the disease and their spread to other areas of the brain at the higher stages are the warnings for this disease. Therefore, it is possible to delay its progress to the debilitating s tages by modulating the Beta-amyloid level and preventing the formation of abnormal forms of peptide chains. This review study aimed to investigate the role of Beta-amyloid peptides in the pathogenesis of Alzheimer's disease.

Keywords: Alzheimer Disease, Amyloid Beta-Peptide, Tau Proteins

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