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Poster Presentation

The Study of Some Factors Which Effect on Beta-Amyloid Signaling in Neuroinflammation

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Abstract

Neurological inflammatory diseases are developing rapidly. Different factors involved in the pathogenesis of these diseases. In this article, we discuss some of the mechanisms are dealt with. An aberrant procedure of beta-amyloid precursor protein (BAPP) to form neurotoxic beta-amyloid peptides and an accumulated insoluble polymer of beta –amyloid (BA) that forms the senile plaque. The above process shows one of the major pathogenic involving in Alzheimer's disease. Actually, the mutations in the preseniline genes PS1 and PS2 cause irregular beta -amyloid precursor protein processing with consequent overexpression of beta-amyloid42 (BA42) and related neurotoxic peptides. The overexpression of RAGE (receptor for advance glycation end products) causes neuroiflammation (NI). This process caused the aggregation of beta-amyloid which increased inflammation and destruction memory. The RAGE signaling in microglia contributes to inflammatory reaction that impaires neuronal function. For improving this condition; the blockade of microglial RAGE may be effective. Intracerebral streptozotocin (i.c.stz) causes NI by increasing Ptau, ABPP, AB42 and reducing the level of synaptophysin and specially IGF1.Since T3D-959 increased IGF1, AKT and P70S6K, using it may be effective for improving brain signaling and reducing NI. Also, in another study, the usage of Tobacco had the same effects. Tobacco suppressed the expression of proteins required for signaling through AKT, P70S6K and increased ABPP-AB. With above expressions, we can conclude that BA may be the most important factors in neuroinflammatory processes. With these interpretations, we can propose that the control of the production BA and prevent the development of inflammatory diseases is gene therapy. In this way, it will be appropriate if we control the genes mutations to control the production of the factors which ultimately lead to overproduction of BA.

Keywords: Neuroinflammation, Signaling, Beta-amyloid

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