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Poster Presentation

The Amyloid Beta as a Therapeutic Target in Alzheimer Disease

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Abstract

Alzheimer disease (AD) is a neurodegenerative disorder marked by cognitive and behavioral impairment. Amyloid beta (Aβ) peptides are involved in AD as the main component of amyloid plaques found in the brain. Recent in vivo and in vitro studies have shown that there is a lot of substances that alter Aβ pathogenesis of AD. Aβ induces toxicity lead to increasing ROS. In the other hand, 5-HT6 and Aloe arborescence recently reported to protect cells from this effect. Additionally, Aβ oligomers interact with neurons through Nrx2a and NL1 receptors by blocking these receptors; one can reduce the Aβ-induced memory impairment. Moreover, Aβ aggregation correlates with high concentration of Fe (III) and Cu (II). And chelators decreased significantly aggregation of Aβ in synaptic cleft. By knowing the mechanism of Aβ toxicity, new therapeutic approaches can be developed to prevent AD or alleviate disability caused by it. JC-124 treatment leads to decrease levels of Aβ deposition. Bosentan, a dual endothelin receptor antagonist, offers protection against Aβ-induced endothelial damage. Anti Nrx2a andante NL1 reduces Aβ-induced memory impairment in mice. Clicaquinol inhibits disaggregation of Aβ at low pHs. In this article we review the substances that have a role in the toxicity of Aβ and can be considered as a new target for the management of AD.

Keyword: Amyloid beta, Alzheimer disease, Neurodegenerative, Treatment

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