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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 (A $\beta$ ) 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 A $\beta$  pathogenesis of AD. A $\beta$  induces toxicity lead to increasing ROS. In the other hand, 5-HT<sub>6</sub> and Aloe arborescence recently reported to protect cells from this effect. Additionally, A $\beta$  oligomers interact with neurons through Nrx2a and NL1 receptors by blocking these receptors; one can reduce the A $\beta$ -induced memory impairment. Moreover, A $\beta$  aggregation correlates with high concentration of Fe (III) and Cu (II). And chelators decreased significantly aggregation of A $\beta$  in synaptic cleft. By knowing the mechanism of A $\beta$  toxicity, new therapeutic approaches can be developed to prevent AD or alleviate disability caused by it. JC-124 treatment leads to decrease levels of A $\beta$  deposition. Bosentan, a dual endothelin receptor antagonist, offers protection against A $\beta$ -induced endothelial damage. Anti Nrx2a and NL1 reduces A $\beta$ -induced memory impairment in mice. Clicaquinol inhibits disaggregation of A $\beta$  at low pHs. In this article we review the substances that have a role in the toxicity of A $\beta$  and can be considered as a new target for the management of AD.

**Keyword:** Amyloid beta, Alzheimer disease, Neurodegenerative, Treatment

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