Editorial

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Drug Therapeutics for Alzheimer Disease

Phoebe Stevens*

Editorial office, The Journal of Immunology and Immunotherapy, United Kingdom

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Corresponding author: Phoebe Stevens

jmso@emedicinejournals.org

Editorial office, The Journal of Immunology and Immunotherapy, United Kingdom.

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Editorial Note

Alzheimer's disease is responsible for 60%-70% of dementia cases (AD). The need for effective medicines to treat Alzheimer's disease has grown urgent, as the number of patients with the disease continues to rise. Although much is known about Alzheimer's disease, there is still an unknown underlying cause. This makes it more difficult to identify medicines that can stop it in its tracks. Current Alzheimer's disease treatments, such as cholinesterase inhibitors and an N-methyl-D-aspartate receptor antagonist may temporarily relieve dementia symptoms but do not stop or reverse disease development. However, researchers have developed new generation pharmacological treatments that would slow down the cortical breakdown mechanism.

Researchers have created and are evaluating a variety of proposed treatments aiming at various aims in ongoing clinical studies. Anti-amyloid and anti-tau therapies, neurotransmitter modulation, anti-neuroinflammation and neuroprotection, cognitive improvement, and behavioral psychological symptom alleviation interventions are some of the treatments available.

The true causes of Alzheimer's disease are still unknown. In terms of senile plaques, there are two pathological features of AD: amyloid fibrils and brain atrophy, which are the target components for pharmacological treatments. They have begun a clinical trial for the suppression of a specific chemical.

Beta-amyloid is a precursor protein and a plaque component. Its goal is to impair brain cell communication and finally destroy them. It is caused by protein clumping and causes toxicity. Beta-secretase and gamma-secretase enzymes make up this beta amyloid. The goal of researchers is to stop them from doing what they're doing. As a result, they focused on two anti-amyloid chemicals. They are CAD106 and CNP520, respectively. They're being studied to see if symptoms may be prevented or delayed in older individuals who have two copies of the APOE gene type e4 and are cognitively healthy. It will be determined if the medicines can prevent the accumulation of beta-amyloid protein fragments in amyloid plaques. If it succeeds, the brain will be able to store new knowledge.

Beta-secretase: BACE1 is an aspartic acid protease that is required for the formation of myelin sheaths in peripheral nerve cells. BACE is a protein that aids in the production of beta-amyloid. JNJ-54861911 is a medication that is being tested to see whether it can reduce the impact. It will operate efficiently according to the researcher's opinion. This can help people who don't have Alzheimer's symptoms slow down their cognitive loss.

Tau protein: It's a collection of six highly soluble protein isoforms produced by alternative splicing of the tau protein linked to the microtubule gene. It is the most important component of tangling. The other symptom of Alzheimer's disease is a brain malfunction. It keeps a neuron's structure intact. Neuron tangling and breakage are both inhibited by the AADvac1 medication. The AADvac1 vaccine is a synthetic peptide generated from amino acids 294 to 305 of the tau protein. It's an active vaccination that elicits an immune response against pathologically altered tau protein types.

5-HT2A receptor: Serotonin receptors and G protein-coupled receptors are a family of receptors. The 5-HT2A receptor is a cell surface receptor. It was discovered that brain cells may lock in chemical neurotransmitters. As a result, pimavanserin works in the opposite direction of 5-HT2A. It inhibits neuronal communication, which can diminish the impact.

