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Alzheimer's Future Drug Therapeutics

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About the Study

Around 60-70 per cent of dementia cases are due to Alzheimer's disease (AD). The development of efficient therapies to treat AD has become urgent and continues to increase patient numbers. All is known about Alzheimer's disease, but an obscure root cause still exists. This makes it harder to find drugs that are capable of halting its effect. Drugs currently available for AD treatment, including cholinesterase inhibitors and an N-methyl-D-aspartate receptor antagonist, may inhibit dementia symptoms for a limited period of time, but may not stop or reverse disease progression. But researcher came off with new generation drug therapeutics which are under process and will slow down the cortical destruction mechanism.

In on-going clinical trials, researchers have developed and are testing a number of possible interventions aimed at different objectives. That Include anti-amyloid and anti-tau interventions, neurotransmitter modification, anti-neuroinflammation and neuroprotection and cognitive enhancement, and behavioural psychological symptoms relief interventions.

It is still unclear about the real causes of AD. There are two pathological characteristics of AD, in terms of senile plaques-amyloid fibrils, brain atrophy-the targeted component for the drug therapeutics. They start clinical trial for inhibitions of targeted molecule.

Beta-amyloid-a precursor protein and a component of plaques. Its aim is to disrupt communication between brain cells and eventually kill them. It is form by clumping of protein and cause toxicity. This beta amyloid is made of Beta-secretase and gamma-secretase enzymes. The objective of researchers is to inhibit their activity. So they targeted two anti-amyloid compounds. They are CAD106 and CNP520.

They are being examined to determine whether symptoms can be avoided or delayed in higher risk cognitively healthy older adults who have two copies of the e4 type of APOE gene. It will determine whether the drugs can fight the accumulation of protein fragment of beta-amyloid in the amyloid plaques. If it is successful then it will allows the brain to store new information.

Beta-secretase (BACE): BACE1 is an essential aspartic acid protease in the production of myelin sheaths in peripheral nerve cells. BACE enables the formation of beta-amyloid. JNJ-54861911 is drug used to minimize the effect and under experimentation. As per researcher view if it will work efficiently. This can slows cognitive decline in people who do not have symptoms of Alzheimer's.

Tau protein: It is a group of six highly soluble protein isoforms generated by alternative splicing of the tau protein associated with the gene microtubule. The leading element of tangles. The other sign of brain abnormality in Alzheimer's disease.it maintain the structure of a neuron. AADvac1 medicine used inhibits the tangling of neuron as well as breakage of neuron. The AADvac1 vaccine is a synthetic peptide derived from the tau sequence of amino acids 294 to 305. It is an active vaccine intended to elicit an immune response to pathologically altered forms of tau protein.

5-HT2A receptor: The family of serotonin receptors and G protein-coupled receptors. The 5-HT2A receptor is a receptor on the cell surface. It found brain cells have been found to be able to lock in chemical neurotransmitters. So pimavanserin drug work opposite to 5-HT2A. It reduces communication among neurons which can reduce the effect.

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