Septa Therapeutics believes that it has identified the “trigger” that initiates AD. The data have been filed in US patent application serial no. 62/613,621.

We have shown that a septapeptide (“septa”) contained in the amyloid beta protein that accumulates in the brains of AD patients has hMCP-1 chemokine activity. This chemokine transforms stem cells into microglia in the CNS. A vast overabundance of microglia is thought to result in neuron death, culminating in AD.

Our second patent application, a CIP, will describe the structure of a compound that blocks the trigger, and will serve as our new drug to treat AD. Our strategy, therefore, is to block the activity, rather than to remove plaque or microglia from the CNS.

Early diagnosis of AD in the future remains essential. Once diagnosed, AD patients would be obliged to remain on their medication to prevent further deterioration in brain function.

It must be emphasized that homologous septas are also located in proteins found in prions and viruses, like HIV, that establish chronic infections in the CNS. We expect that effective blocker(s) will have to inhibit the activity in all of its sites, both plaque and viral.

Septa Therapeutics is currently discussing pre-clinical trials in mice with BiOasis Inc.

Background

This work represents a culmination of 25 years of research by D. Van Alstyne in the delineation of chronic viral infection in the CNS (see www.septatherapeutics.com). Specifically, earlier research on bacterial and viral meningitis provided the drug discovery platform for the development of the AD work.

Septa Therapeutics Inc. was incorporated December 14, 2015 to capitalize on the meningitis platform technology. As such, it represents a late-stage, pre-clinical trial company. It is privately held by several people with 6 million shares outstanding.
Development of a new therapeutic for the treatment of Alzheimer's Disease
May, 2018