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Lessons learned from the failure of Alzheimer's drug development over the past 20 years

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There are more than 5.5 million people in the US and 43 million people worldwide that have Alzheimer's disease in 2017. Growth in the prevalence of Alzheimer's Disease (AD) over the next few decades is anticipated to result in great pressure on the social and health-care systems of developed and developing economies alike. The current treatment options are considered to be symptomatic. There is an unmet need for therapies that halt or substantially slow disease progression. There were no new drug approvals for treatment of AD; none have been approved in the USA since 2003. Over the past 20 years a majority of failed AD drugs were based on the guiding principle of the amyloid hypothesis, which postulates that accumulation of amyloid plaques trigger a cascade that harms neurons and synapses. Most immunotherapy drugs based on β-secretase, γ-secretase and stimulators of α-secretase have been used to target Aβ pathology include decreasing of Aβ production, preventing aggregation of or stimulating clearance of Aβ. A new non-amyloid approach in the development of a new drug in Phase 2 known as NA-831, a novel neuroprotective and neurogenesis agent for treatment of Alzheimer's disease. The past AD drug development failure and a new clinical strategy for the development of NA-831 is described

Biography

Dr. Brian Tran has completed his MD degree from St. George Medical School. He is board certified\r\nand serves at the Medical Director of NeuroActiva, Inc, which is a clinical-stage biopharmaceutical company dedicated to the discovery, development and commercialization of new drugs to treat Alzheimer's disease.

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