Alzheimer's Disease Genetic Mutation Marked as Therapeutic Target

"Lead Inventors: Tae-Wan Kim, PhD, Gilbert DiPaolo, Diego Berman, Natalie Landman

Alzheimer’s Disease Linked to Familial Inheritance and Amyloid Build-Up in Brain Neurodegenerative disease is an umbrella term for several similar diseases that produce the progressive loss of neuron structure and function that lead to mental and physical disabilities and eventual death. Alzheimer’s is a neurodegenerative disease that is the most common cause of dementia in people over the age of 65. In 2006, 26.6 million people were Alzheimer’s disease patients; it is estimated that 1 in 85 people across the world will suffer with Alzheimer’s disease in 2050. The disease is currently incurable and has been linked to familial inheritance and amyloid build up in the brain. Alzheimer’s patients typically suffer a long and slow progression toward dementia and eventual death, leading to great emotional and financial burden to the families of these patients.

Therapeutic Target for Treating Alzheimer’s Disease Based on Altering Amyloid Beta This technology proposes a novel therapeutic target for treating Alzheimer’s disease. Based upon the amyloid hypothesis, amyloid beta build-up is toxic and precedes disease onset. Research leading to the development of this technology determined that by increasing the levels of phosphoinositol 4-phosphate (PIP) and or phosphotidylinositol 4,5-biphosphate (PIP2), the toxic effect of amyloid beta is mitigated. This technology proposes methods of preventing and/or treating Alzheimer’s, and possibly other neurodegenerative diseases, by methods of identifying and administering compounds that will alter lipid metabolism of amyloid beta.

Applications:
- Treatment of Alzheimer’s by administering agents that boost neural PIP2 levels
- Methods to select appropriate targets to boost neural PIP2 levels
- Possible method of treating other neurodegenerative diseases by reversing synaptic dysfunction

Advantages:
- Currently widespread medications that boost the synthesis, and stop the decomposition of, acetylcholine have proven ineffective at stopping or reversing Alzheimer’s progression
- Metamine therapy combined with donepezil has proven to be on marginally effective.
- Vaccine treatments to reduce amyloid beta build up do not counter or reverse the toxic effect of amyloid beta on neural receptors.


Licensing Status: Available for Licensing and Sponsored Research Support

Publications: Landman et al. "Presenilin Mutations Linked to Familial Alzheimer’s Disease Cause an Imbalance"

Inventors

Tae-Wan Kim Ph.D.