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Inducing alzheimer's-like plaques in neuronal stem cells in order to trace the pathway.

Alzheimer's disease is a neurological disorder that diminishes the function of nerve cells. Amyloid-beta plaques and tangles have been seen as the possible sources of Alzheimer's. In the case of amyloid-beta plaques, they are caused by an enzyme, beta-secretase, improperly cutting amyloid precursor proteins (APP) into beta-amyloid fragments. These fragments come into contact with other beta-amyloid fragments, forming plaques. Both the plaques and tangles are being heavily investigated by scientific researchers and so far, it is unclear which dominates in Alzheimer's disease or if both play an equal role. Researching Alzheimer's disease is important because the source of the amyloid beta plaques and tangles are not truly understood due to the thousands of pathways and factors involved with this disease. As life expectancy increases, more people are being diagnosed with this disease. However, there are many anti-dementia drugs used on Alzheimer's patients to control behavioral, cognitive and psychological symptoms but to date no cure. Though the anti-dementia drugs help slow down the progression, they do not reverse the damages caused by Alzheimer's.

The ultimate goal of this set of experiments was to establish a model to better examine the mechanism's behind increased levels of alpha, beta and gamma secretase's.