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The APP+PS1 Transgenic Mouse: A Model for Testing Drugs to Treat Alzheimer's Disease

Researchers at the University of South Florida have developed mutations in two genes, APP and PS1 that can each cause the disease in some families. Alzheimer's Disease is a devastating illness of late-life resulting from degeneration of the brain. USF's APP+PS1 mouse model combines the expression of two mutant human genes to produce characteristics of the disease.

These "doubly" transgenic mice develop amyloid deposits in their brains that have the same features as those found in Alzheimer's disease. They accumulate in the same areas as in Alzheimer's brain and the first deposits appear at 4 months of age in the mice.

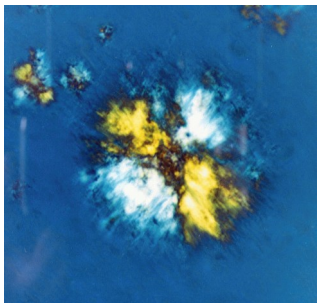
As the mice grow older, they develop learning and memory deficits which can be easily detected in the "radial arm water maze". The extent of the memory deficit corresponds to the amount of amyloid found in each animal's brain, and can be reversed by treatments that remove the amyloid. Duff et al., Nature (1996) 383:710-713.

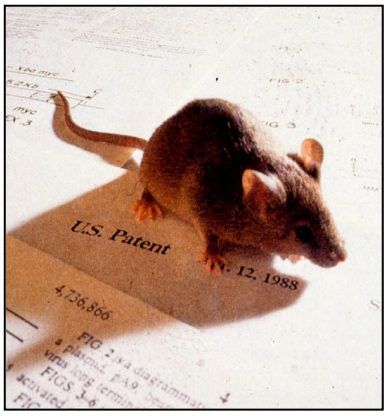
Overexpression of mutant, but not wild-type, PS1 selectively increases brain A beta42(43). The presenilin mutations probably cause Alzheimer's disease through a gain of deleterious function that increases the amount of A beta42(43) in the brain.

ADVANTAGES:

APP/PS doubly transgenic mice show:

- **a selective 41% increase in beta-amyloid (Abeta) in their brains**
- **Abeta neuropathology in mice is low at 5-7 months and very extensive at 15-17 months**
- **Age-related cognitive impairments**





Tech ID # 96B024 **Patent #: 5,898,094**

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