

Transgenic Mouse with ACT Gene

Researchers at the University of South Florida have created a transgenic mouse that harbors foreign DNA encoding an alpha1-antichymotrypsin (ACT) gene, a key inflammatory protein associated with Alzheimer's Disease (AD). When crossed with a mouse expressing a human mutant Amyloid Precursor Protein (APP) gene, the progeny exhibit a key inflammatory profile associated with AD.

There is great need for animal models exhibiting the pathology and cognitive deficits associated with Alzheimer's Disease and other related neurological disorders. Such an animal model, it is believed, would greatly facilitate the development of therapeutic treatments. One of the key aspects of AD is inflammation in the brain, which is essential for both the amyloid formation and the cognitive decline.

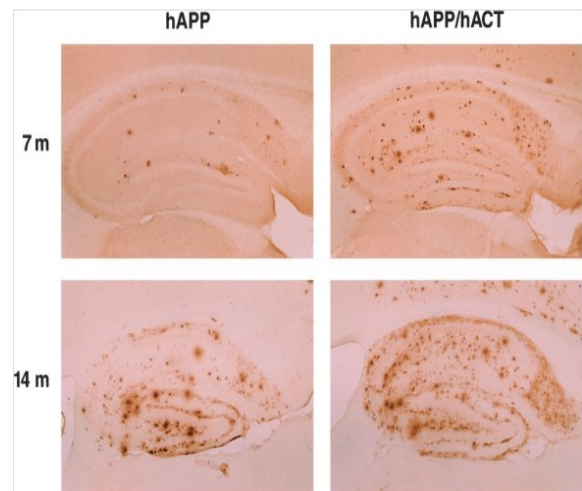
The normal function of ACT is to reduce potential damage inflicted by inflammation enzymes that break down normal serum protein and healthy tissue outside of the immediate area of inflammation. The ACT gene encodes for an inhibitor of these inflammation enzymes and is normally produced in the liver in response to inflammation. However, in AD patients, ACT is overproduced in brain cells where it combines with other proteins (Ab peptide and apoE4) and promotes the formation of neurotoxic deposits in the brain. Polymorphisms in the ACT gene have been associated with increased risk of AD.

This animal model can be used for studying the role of the ACT gene in Alzheimer's disease and potentially other neurological disorders. The model also displays a full range of AD symptoms including cognitive memory loss, behavioral disturbances, inflammation, and amyloid-plaque formation.

ADVANTAGES:

- Excellent animal model for Alzheimer's disease
- Full range of symptoms displayed
- Screening tool for promising pharmaceuticals

Animal Model for Alzheimer's Disease Research



Astroglial expression of hACT increases amyloid deposition in hAPP/hACT mice. Brain sections from hAPP mice (left) and hAPP/hACT mice (right) were labeled with the anti-Aβ antibody 3D6 at 7 or 14 months (m) of age. Aβ-immunoreactive deposits in the hippocampus were visualized by immunoperoxidase reaction and light microscopy.

Tech ID # 98B046

Patent #: [6,781,029](#)