MOUSE STRAIN DATASHEET

B6;129-Tg(APPSwe,tauP301L)1Lfa/Mmjax

MMRRC Stock No: 34830-JAX | **3xTg-AD**

Targeted Mutation,Transgenic

AVAILABLE NOW

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Live mice available in varying quantities. Ask Customer Service for details.

Overview

Also Known As: 3xTg-AD

3xTg-AD mice are useful when studying plaque and tangle pathology associated with synaptic dysfunction and Alzheimer's disease.

Donating Investigator

Frank LaFerla, University of California, Irvine

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GENETIC OVERVIEW

Genetic Background	Generation
	F?+F23 (2019-12-10 00:00:00)

Tg(APPSwe,tauP301L)1Lfa

Allele Type

Transgenic (Inserted expressed sequence, Humanized sequence)

Psen1^{tm1Mpm}

Allele Type	Gene Symbol	Gene Name
Targeted	*Psen1*	presenilin 1

VIEW GENETICS

RESEARCH APPLICATIONS

Neurobiology Research
Mouse/Human Gene Homologs
Developmental Biology Research
Research Tools

VIEW ALL RESEARCH APPLICATIONS

Details

Detailed Description

Mice homozygous for all three mutant alleles (3xTg-AD; homozygous for the *Psen1* mutation and homozygous for the co-injected APPSwe and tauP301L transgenes (Tg(APPSwe,tauP301L)1Lfa)) are viable, fertile and display no initial gross physical or behavioral abnormalities. Translation of the overexpressed transgenes appears to be restricted to the central nervous system, notably in Alzheimer's disease-relevant areas including the hippocampus and cerebral cortex. The initial characterization of this mouse line indicated a progressive increase in amyloid beta peptide deposition, with intracellular immunoreactivity being detected in some brain regions as early as 3-4 months. Synaptic transmission and long-term potentiation are demonstrably impaired in mice 6 months of age. Between 12-15 months aggregates of conformationally altered and hyperphosphorylated tau are detected in the hippocampus. This mutant mouse exhibits plaque and tangle pathology associated with synaptic dysfunction, traits similar to those observed in Alzheimer's disease patients.

In February 2014, the donating investigator communicated that, in contrast to the initial observations, male transgenic mice may not exhibit the phenotypic traits originally described. No reports of diminished traits in female carriers have been reported.

Belfiore *et al.* 2019 Aging Cell 18:e12873 [PMID:30488653] characterized C57BL/6;129 genetic background 3xTgAD females for the onset, severity, and incidence of amyloidβ, phosphorylated tau, hippocampal and cortical plaques, neuroinflammation and cognitive decline. Most phenotypes that were evaluated were evident by 6 months of age. However, it was noted that cortical plaques were first detected at 12 months. For more detailed information, please see that publication. If any more detailed characterization is completed by The Jackson Laboratory, we will modify the strain description accordingly.

Development

Expression Data

Control Suggestions

Selected References

Genetics

Tg(APPSwe,tauP301L)1Lfa

Psen1^{tm1Mpm}

Disease/Phenotype

Disease Terms

Research Areas By Genotype

Mammalian Phenotype Terms by Genotype

References

Technical Support

CHAT OFFLINE

CONTACT TECHNICAL SUPPORT

Genotyping Protocols

Restriction Enzyme Digest:Psen1^{tm1Mpm}
Pyrosequencing:Psen1^{tm1Mpm}
End Point Analysis:Psen1^{tm1Mpm}-EP
Standard PCR:Generic APP human genomic or cDNA
Standard PCR:Tg(TAU*P301S)#Elan
Probe:Tg(APPSwe,tauP301L)1Lfa-Chr2
Genotyping resources and troubleshooting

Dietary Information

LabDiet® 5K52 formulation (6% fat)

Breeding Considerations

When maintaining a live congenic colony, mice that are homozygous for the co-injected APPSwe and tauP301L transgenes [Tg(APPSwe,tauP301L)1Lfa on chromosome 2] and homozygous for the PS1M146V knock-in mutation [*Psen1*^{tm1Mpm} on chromosome 12] may be bred together.

Additional Breeding and Husbandry Support

Mating System

See "Breeding Considerations"

Citation

When using the 3xTg-AD mouse strain in a publication, please cite the originating article(s) and include MMRRC stock #34830 in your Materials and Methods section.

Animal Health Reports

Facility Barrier Level Descriptions

AX12 (Maximum)

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