The Jackson Laboratory

B6;C3-Tg(APPswe,PSEN1dE9)85Dbo/Mmjax

MMRRC Stock No: **34829-JAX** | APP/PS1

Transgenic

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Also Known As:APP/PS1

APP/PS1 are double transgenic mice expressing a chimeric mouse/human amyloid precursor protein (Mo/HuAPP695swe) and a mutant human presenilin 1 (PS1-dE9), both directed to CNS neurons. Both mutations are associated with early-onset Alzheimer's disease. These mice may be useful in studying neurological disorders of the brain, specifically Alzheimer's disease, amyloid plaque formation and aging.

Donating Investigator

Dr. David R Borchelt, University of Florida

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GENETIC OVERVIEW

Genetic Background

Generation

N4F4pF4
(2021-01-12 00:00:00)

Tg(APPswe,PSEN1dE9)85Dbo

Allele Type

Transgenic (Inserted expressed sequence, Humanized sequence)

VIEW GENETICS

RESEARCH APPLICATIONS

Neurobiology Research
Mouse/Human Gene Homologs

VIEW ALL RESEARCH APPLICATION

Details

Detailed Description

APP/PS1 are double transgenic mice expressing a chimeric mouse/human amyloid precursor protein (Mo/HuAPP695swe) and a mutant human presenilin 1 (PS1-dE9), both directed to CNS neurons. Both mutations are associated with early-onset Alzheimer's disease. The "humanized" Mo/HuAPP695swe transgene allows the mice to secrete a human A-beta peptide. Both the transgenic peptide and holoprotein can be detected by antibodies specific for human sequence within this region (Signet Laboratories' monoclonal 6E10 antibody). The included Swedish mutations (K595N/M596L) elevate the amount of A-beta produced from the transgene by favoring processing through the beta-secretase pathway. This "humanized" Mo/HuAPP695swe protein is immunodetected in whole brain protein homogenates. The transgenic mutant human presenilin protein (PS1-dE9), which in high levels displaces detectable endogenous mouse protein, is also immunodetected in whole brain protein homogenates. The donating investigator reports that transgenic mice develop beta-amyloid deposits in brain by 6 to 7 months of age. Between 6 and 15 months of age, mice exhibit a gender-based disparity in beta-amyloid burden. Females develop a 5-fold ($A\beta_{42}$) and 10-fold ($A\beta_{40}$) increase in beta-amyloid deposits in the cerebellum by 15 months as compared to males. Accumulation of plaques is more abundant in the molecular layer than in the granular layer. In the cortex, the beta-amyloid burden is increased in both sexes in parallel (Ordonez-Guiterrez *et al.* Jnl Alz Dis 2016).

APP/PS1 hemizygotes on a C57BL/6;C3H genetic background (Stock No. 004462) do not exhibit any seizure phenotype. These animals also display a slight alteration in their tail phenotype (*e.g.*, kinked tail) that is believed to be due to the mixed genetic background of the strain and is not related to transgene expression. This strain does not carry the retinal degeneration allele *Pde6b*^{rd1}.

In contrast, APP/PS1 hemizygotes on a C57BL/6J-congenic background (see Stock No. 005864) exhibit seizure activity. Specifically, hemizygous mice on the C57BL/6 background (N9B6) exhibit a high incidence of seizures, as detected by video-EEG. 25% of transgenic mice, 3 to 3.5 months in age, exhibit at least 1 seizure. By 4.5 months of age, seizure incidence increases to 55%. 10-15% mortality is reported for transgenic mice on the congenic (N9) C57BL/6 background (Minkeviciene *et al.* J Neurosci. 2009). At 17-18 weeks of age, hemizygous mice on the congenic C57BL/6J background (N13) exhibit epileptiform discharges as detected by video-EEG. Mortality was 38% (6/16) and some mutant mice experienced spontaneous seizures during the experiments. Antiepileptic drugs (carbamazepine, phenytoin, valproate) reduce the frequency of spontaneous electrographic epileptiform discharges (Ziyatdinova *et al.* Epilepsy Res 2011).

Development

Expression Data

Control Suggestions

Selected References

Genetics

Tg(APPswe,PSEN1dE9)85Dbo

Disease/Phenotype

Disease Terms

Research Areas By Phenotype

Mammalian Phenotype Terms by Genotype

References

Technical Support

CONTACT TECHNICAL SUPPORT

Genotyping Protocols

Standard PCR:Tg(PSEN1)
Standard PCR:Generic Tg(APP)
Probe:Generic Human PSEN1 cDNA Probe
Probe:Pde6b Probe
Standard PCR:Tg(APPswe,PSEN1dE9)85Dbo-Chr9
Genotyping resources and troubleshooting

Breeding Considerations

When maintaining a live colony, hemizygotes may be bred with wildtype (noncarrier) siblings. Coat color expected from breeding is black or agouti. Please note: male aggression is sometimes observed in this strain and may require separate housing.

Additional Breeding and Husbandry Support

Mating System

+/+ sibling x Hemizygote

Citation

When using the APP/PS1 mouse strain in a publication, please cite the originating article(s) and include MMRRC stock #34829 in your Materials and Methods section.

Animal Health Reports

Facility Barrier Level Descriptions

AX12 (Maximum)

THE JACKSON LABORATORY'S GENOTYPE PROMISE

The Jackson Laboratory has rigorous genetic quality control and mutant gene genotyping programs to ensure the genetic background of JAX® Mice strains as well as the genotypes of strains with identified molecular mutations. JAX® Mice strains are only made available to researchers after meeting our standards. However, the phenotype of each strain may not be fully characterized and/or captured in the strain data sheets. **Therefore, we cannot guarantee a strain's phenotype will meet all expectations.** To ensure that JAX® Mice will meet the needs of individual research projects or when requesting a strain that is new to your research, we suggest ordering and performing tests on a small number of mice to determine suitability for your particular project. We do not guarantee breeding performance and therefore suggest that investigators order more than one breeding pair to avoid delays in their research.

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