Resource Summary Report

Generated by NIF on Aug 20, 2026

B6.Cg-Tg(tetO-APPSwInd)102Dbo/Mmjax

RRID:MMRRC_034845-JAX
Type: Organism

Proper Citation

RRID:MMRRC_034845-JAX

Organism Information

URL: https://www.mmrrc.org/catalog/sds.php?mmrrc_id=34845

Proper Citation: RRID:MMRRC_034845-JAX

Description: Mus musculus with name B6.Cg-Tg(tetO-APPSwInd)102Dbo/Mmjax from MMRRC.

Species: Mus musculus

Notes: Research areas: ; Mutation Type: Transgenic ; Collection:

Affected Gene: tetOp

Catalog Number: 034845-JAX

Background: Transgenic

Database: Mutant Mouse Resource and Research Center (MMRRC)

Database Abbreviation: MMRRC

Source References: PMID:16279840

Alternate IDs: MMRRC_34845-JAX, MMRRC_034845, MMRRC_34845

Organism Name: B6.Cg-Tg(tetO-APPSwInd)102Dbo/Mmjax

Record Creation Time: 20230308T055135+0000

Record Last Update: 20260815T124716+0000

Ratings and Alerts

No rating or validation information has been found for B6.Cg-Tg(tetO-APPSwInd)102Dbo/Mmjax.

No alerts have been found for B6.Cg-Tg(tetO-APPSwInd)102Dbo/Mmjax.

Data and Source Information

Source: Integrated Animals

Source Database: Mutant Mouse Resource and Research Center (MMRRC)

Usage and Citation Metrics

We found 15 mentions in open access literature.

Listed below are recent publications. The full list is available at NIF .

Perez GA, et al. (2026) Neuronal subtype governs amyloid structure, cellular response, and cognitive outcome in genetically targeted APP mouse models. Molecular neurodegeneration, 21(1), 2.

Viana da Silva S, et al. (2024) Localized APP expression results in progressive network dysfunction by disorganizing spike timing. Neuron, 112(1), 124.

Koller EJ, et al. (2024) Doxycycline for transgene control disrupts gut microbiome diversity without compromising acute neuroinflammatory response. Journal of neuroinflammation, 21(1), 11.

Liu P, et al. (2024) A??56 is a stable oligomer that impairs memory function in mice. iScience, 27(3), 109239.

Lusk S, et al. (2023) An automated respiratory data pipeline for waveform characteristic analysis. The Journal of physiology, 601(21), 4767.

Koller EJ, et al. (2022) Temporal and spatially controlled APP transgene expression using Cre-dependent alleles. Disease models & mechanisms, 15(5).

Huichalaf CH, et al. (2019) Cross-species genetic screens to identify kinase targets for APP reduction in Alzheimer's disease. Human molecular genetics, 28(12), 2014.

Chiang ACA, et al. (2018) Discrete Pools of Oligomeric Amyloid-? Track with Spatial Learning Deficits in a Mouse Model of Alzheimer Amyloidosis. The American journal of pathology, 188(3), 739.

Fowler SW, et al. (2014) Genetic modulation of soluble A? rescues cognitive and synaptic impairment in a mouse model of Alzheimer's disease. The Journal of neuroscience : the official journal of the Society for Neuroscience, 34(23), 7871.

Tanifum EA, et al. (2014) Cerebral vascular leak in a mouse model of amyloid neuropathology. Journal of cerebral blood flow and metabolism : official journal of the

International Society of Cerebral Blood Flow and Metabolism, 34(10), 1646.

Zhao R, et al. (2014) Impairments in experience-dependent scaling and stability of hippocampal place fields limit spatial learning in a mouse model of Alzheimer's disease. Hippocampus, 24(8), 963.

Born HA, et al. (2014) Genetic suppression of transgenic APP rescues Hypersynchronous network activity in a mouse model of Alzeimer's disease. The Journal of neuroscience : the official journal of the Society for Neuroscience, 34(11), 3826.

Yetman MJ, et al. (2013) Wild-type neural progenitors divide and differentiate normally in an amyloid-rich environment. The Journal of neuroscience : the official journal of the Society for Neuroscience, 33(44), 17335.

Han HJ, et al. (2012) Strain background influences neurotoxicity and behavioral abnormalities in mice expressing the tetracycline transactivator. The Journal of neuroscience : the official journal of the Society for Neuroscience, 32(31), 10574.

Rodgers SP, et al. (2012) Transgenic APP expression during postnatal development causes persistent locomotor hyperactivity in the adult. Molecular neurodegeneration, 7, 28.