

Resource Summary Report

Generated by [PRECISE-TBI](#) on Aug 22, 2026

[Gars^{C201R}/Gars⁺](#)

RRID:MGI:3849420

Type: Organism

Proper Citation

RRID:MGI:3849420

Organism Information

URL:

Proper Citation: RRID:MGI:3849420

Description: Allele Detail: Chemically induced (ENU) This is a legacy resource.

Species: Mus musculus

Notes: Allele Detail: Chemically induced (ENU) This is a legacy resource.

Phenotype: abnormal sensory neuron morphology, decreased nerve conduction velocity, abnormal innervation pattern to muscle, abnormal neuromuscular synapse morphology, decreased grip strength, abnormal neuromuscular synapse morphology, abnormal tibialis anterior morphology, decreased nerve conduction velocity, decreased body weight, decreased skeletal muscle fiber number, decreased grip strength, nervous system phenotype, muscle weakness, decreased tibialis anterior weight

Affected Gene: Gars

Genomic Alteration: C201R

Catalog Number: 3849420

Background: involves: BALB/cAnN * C3H/HeH * C57BL/6J

Database: MGI, Mouse Genome Informatics MGI

Database Abbreviation: MGI

Availability: Availability unknown check source stock center

Source References: [PMID:22144914](#), [PMID:19470612](#)

Organism Name: Gars^{C201R}/Gars⁺

Record Creation Time: 20240120T190335+0000

Record Last Update: 20240130T201842+0000

Ratings and Alerts

No rating or validation information has been found for Gars^{C201R}/Gars⁺.

No alerts have been found for Gars^{C201R}/Gars⁺.

Data and Source Information

Source: [Integrated Animals](#)

Source Database: MGI, Mouse Genome Informatics MGI

Usage and Citation Metrics

We found 2 mentions in open access literature.

Listed below are recent publications. The full list is available at [PRECISE-TBI](#) .

Sleigh JN, et al. (2020) Altered Sensory Neuron Development in CMT2D Mice Is Site-Specific and Linked to Increased GlyRS Levels. *Frontiers in cellular neuroscience*, 14, 232.

Sleigh JN, et al. (2020) Developmental demands contribute to early neuromuscular degeneration in CMT2D mice. *Cell death & disease*, 11(7), 564.