

B6(Cg)-Mrp13^{dcr}/Dcr

Stock No: **009408**

 Spontaneous Mutation

Typically mice are recovered in 10-14 weeks. Contact Customer Service to place an order or for more information.

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Donating Investigator

Dr. Derry Roopenian, The Jackson Laboratory

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GENETIC OVERVIEW

Genetic Background

Generation

Mrp13^{dcr}

Alele Type

Spontaneous (Hypomorph)

Gene Symbol

Mrp13

Gene Name

mitochondrial ribosomal protein L3

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RESEARCH APPLICATIONS

Neurobiology Research

VIEW ALL RESEARCH APPLICATIONS

BASE PRICE

Starting at:

Details

Detailed Description

MRPL3 is one of the proteins essential in the mitochondrial ribosome and the spontaneous mutation decrepit (*Mrpl3^{dcr}*) is an approximately 133 bp insertion containing an extensive T repeat inserted into the intron between exons 6 and 7. While *Mrpl3* homozygous null mutations cause embryonic lethality by E9.5, the decrepit mutation is a hypomorph that causes adult onset neurodegeneration. Increased citrate synthase activity was found in mitochondria extracted from brains of adult decrepit homozygotes and assessment via the Barnes maze revealed impaired learning and progressive loss of spatial memory. Homozygotes develop progressive bilateral focal ischemia in the brain beginning by 12 weeks of age, when an eosin-pale, sharply bounded region is found in the cortex of most homozygotes. This expands by 14 weeks with all homozygotes showing the phenotype, by 16 weeks the inferior hippocampus and other areas are also affected, and by 18 weeks those homozygotes that survive have large regions of vacuolated tissue in the inferior cortex and hippocampus, and the dorsal cortex is also affected. T2-weighted MRI revealed hyperintensities indicative of cell death in the piriform and entorhinal cortices at 70 days, the limbic area, cingulate cortex, striatum, and nucleus accumbens at 86 days, the motor cortex and hippocampus at 96 days, and the substantia nigra at 125 days, which is consistent with the initial histological findings. The volume of several brain structures differs significantly from normal, either larger or smaller depending on the structure, between 50 and 150 days of age. As a result of this underlying pathology, homozygotes develop an unkempt scruffy coat and hunched posture by approximately 10 weeks of age and are over-excited by stimulation. Wasting begins around 12 weeks of age, lethargy by approximately 15 weeks, and death generally occurs between 18 and 22 weeks, but can be earlier or later. Histological assessment found pathology restricted to the brain. The brain vacuoles show bilateral symmetry, and no hemorrhage or emboli are found, suggesting these vacuoles do not result from strokes. X-ray micro-CT found an increase in the number of primarily small vessel segments in the cerebrovasculature after 100 days of age when neurodegeneration has already occurred. (Sproule et al., 2010; Cahill et al., 2020.)

Development

Control Suggestions

Selected References

Genetics

Mrpl3^{dcr}

Disease/Phenotype

+ Disease Terms

+ Research Areas By Phenotype

+ Mammalian Phenotype Terms by Genotype

+ References

- Technical Support

C O N T A C T T E C H N I C A L S U P P O R T

Genotyping Protocols

[Genotyping resources and troubleshooting](#)

Citation

When using the B6(Cg)-*Mrp13^{dcr}*/Dcr mouse strain in a publication, please [cite the originating article\(s\)](#) and include JAX stock #009408 in your Materials and Methods section.

Animal Health Reports

[Facility Barrier Level Descriptions](#)

Production of mice from cryopreserved embryos or sperm occurs in a maximum barrier room, [G200](#)

- Pricing & Availability



Cryo
Recovery

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SERVICE/PRODUCT	DESCRIPTION	PRICE
Cryo Recovery	Heterozygous for dcr, 1 pair minimum	\$2,854.50

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B6(Cg)-dcr/Dcr

\$2595.00

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