B6;129S1-Atp7b<sup>lmt<sup>Ltk</sup>/LtskJ</sup>

Stock No: 032624 | Atp7b-

Targeted Mutation

AVAILABLE FOR PRE-ORDER

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Estimated to begin distribution on Sep 2, 2019

Overview

Also Known As: Atp7b-

Atp7b<sup>−/−</sup> mice contain a neo cassette replacing part of exon 2, of the ATPase, Cu++ transporting, beta polypeptide (Atp7b) gene, abolishing protein expression. These mice may be useful when studying hepatic copper accumulation.

Donating Investigator

Svetlana Lutsenko, Johns Hopkins University

READ MORE »
GENETIC OVERVIEW

<table>
<thead>
<tr>
<th>Genetic Background</th>
<th>Generation</th>
</tr>
</thead>
<tbody>
<tr>
<td>?+pN1</td>
<td>(2019-04-15 00:00:00)</td>
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Atp7b<sup>im1Tcg</sup>

<table>
<thead>
<tr>
<th>Allele Type</th>
<th>Gene Symbol</th>
<th>Gene Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Targeted (Null/Knockout)</td>
<td>Atp7b</td>
<td>ATPase, Cu++ transporting, beta polypeptide</td>
</tr>
</tbody>
</table>

RESEARCH APPLICATIONS

Developmental Biology Research
Internal/Organ Research

BASE PRICE
Starting at:

Details

Detailed Description

Atp7b<sup>−/−</sup> mice contain a neo cassette replacing part of exon 2, of the ATPase, Cu++ transporting, beta polypeptide (Atp7b) gene, abolishing protein expression. Atp7b encodes a copper-transporting enzyme expressed predominantly in the liver, with lower levels in the brain, intestine, mammary gland, placenta and other tissues. Mutations in this gene are associated with the onset of Wilson disease, a copper misbalance disorder characterized by a build-up of intracellular copper in the liver leading to hepatitis and/or cirrhosis. ~40% of cases also manifest neurological and/or psychiatric disorders. Hematological, renal and endocrine forms of the disease are much less common. The insertion of the neo cassette results in alternative splicing which produces a truncated ATP7B mRNA that lacks 1270bp and a frame shift mutation resulting in multiple stop codons. Heterozygous animals are viable and fertile and do not develop disease. Homozygous mice are viable and fertile with shortened life expectancy. They display copper overload with metabolic changes in the liver and altered lipid metabolism by 6 weeks. By 10-12 weeks of age these mice develop liver inflammation and other pathologic changes. The life expectancy of homozygous mice varies from 20 weeks to one year of age.

Homozygous mice display a gradual accumulation of hepatic copper that increases to a level 60-fold greater than normal by 5 months of age. An increase in copper concentration was also observed in the kidney, brain, placenta and lactating mammary glands of homozygous mutants, although milk from the mutant glands was copper deficient. Morphological abnormalities resembling cirrhosis developed in the majority of the livers from homozygous mutants older than 7 months of age. Progeny of the homozygous mutant females demonstrated neurological abnormalities and growth retardation characteristic of copper deficiency.

Development
Genotyping Protocols
MELT: Atp7btm1Tcg
Genotyping resources and troubleshooting

Breeding Considerations
When maintaining a live colony, heterozygous mice are viable and fertile. Homozygous mice are also viable and fertile but have a shortened life expectancy of from 20 weeks to one year of age. An increase in copper concentration was observed in the kidney, brain, placenta and lactating mammary glands of homozygous mutants, although milk from the mutant glands was copper deficient.

Additional Breeding and Husbandry Support

Mating System
Heterozygote x Heterozygote

Citation
When using the Atp7b mouse strain in a publication, please cite the originating article(s) and include JAX stock #032624 in your Materials and Methods section.

Facility Barrier Level Descriptions

FGB29 (Standard)
Pricing & Availability

Estimated to begin distribution on Sep 2, 2019

Available for Pre-order

Domestic  International
Pricing effective for USA, Canada and Mexico shipping destinations

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All

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