Overview

Also Known As: reeler

Mice homozygous for the reeler (Reln<sup>r</sup>) mutation exhibit an ataxic gait, dystonic posture and. Neuronal layer formation fails in laminated brain regions during development, and there are reduced numbers of granule and Purkinje cells. T-cell and macrophage function are suppressed in homozygous mutants. Mutant reeler mice may serve as a murine model for general lissencephalic disorders that affect humans. Additionally, mice heterozygous for the ReIn<sup>r</sup> mutation may be useful in studies of dopamine-related pathophysiologic disorders such as schizophrenia.
Mice homozygous for the reeler (Reln<sup>fl</sup>) mutation exhibit an ataxic gait, dystonic posture and tremors starting around 2 weeks of age. These mutants are incapable of maintaining their hindquarters upright and often fall over during locomotor activity. Moreover, viability and fertility are greatly reduced, especially when the gene is carried on an inbred genetic background. Heterozygotes are visually indistinguishable from wildtype controls. Neuropathies characteristic of Reln<sup>fl</sup>/Reln<sup>fl</sup> mutants include a failure of neuronal layer formation in laminated brain regions during development. Neuronal positioning is abnormal within cerebellar, cerebral and hippocampal cortices. The behavioral phenotype is primarily attributed to the severe hypoplasia of the cerebellum, which lacks foliation. Here, there are reduced numbers of granule and Purkinje cells and these cells are aberrantly dispersed among the layers. In the Reln-deficient neocortex, neurons normally destined to migrate past the subplate remain confined to deeper nuclei, thus ablatting normal cortical layer formation. Similarly, pyramidal and granule cells of the developing hippocampus are scattered throughout the hippocampal tracts causing gross disorganization. RELN is required for normal spinal cord formation since migration of sympathetic preganglionic neurons in the intermediolateral column becomes disrupted in developing Reln<sup>fl</sup>/Reln<sup>fl</sup> mice. While somatic motor neurons and cholinergic interneurons are positioned normally in the Reln<sup>fl</sup>/Reln<sup>fl</sup> spinal cord, parasympathetic and sympathetic preganglionic neurons migrate medially past their normal destinations, indicating that RELN may act in a cell-specific manner.
Neurons are also found abnormally positioned in the facial nucleus, inferior olivary complex, and mesencephalic trigeminal nucleus of affected reeler mutants. A RELN deficiency additionally results in an alteration in the structure and function of retinal synaptic circuitry. There is a reduction in the number of rod bipolar cells and physiologic responsiveness is compromised. Specifically, electoretinography analysis demonstrated a reduction in rod b-wave amplitudes. RELN may also play a role in the development of immune function since T-cell and macrophage function are suppressed in Reln^r/Reln^r mutants. Taken together, the data suggest that RELN functions in the extracellular matrix as a patterning signal for postmitotic neuronal migration along radial glial cell pathways. It may alternatively function to modulate neuron-neuron adhesivity and/or stability. Severe defects in neuronal cell migration underlie general lissencephalic disorders that affect humans. Therefore, the reeler mice may serve as a murine model for such neuronal ectopia disorders. Additionally, mice heterozygous for the Reln^r mutation are currently being pursued as a model for dopamine-related pathophysiological disorders such as schizophrenia. These Reln^r/+ mice exhibit a reduction in 1) the number of tyrosine hydroxylase-immunoreactive cell bodies, 2) tyrosine hydroxylase and dopamine transporter immunoreactivity, 3) tyrosine hydroxylase and D2 dopamine receptor mRNA levels in the mesolimbic dopamine system, and 4) oxytocin receptors in the piriform cortex, neocortex, retrosplenial cortex and certain regions of the hippocampus (reviewed by Rice and Curran, 2001, D’Arcangelo and Curran, 1998; and Hatten, 1999; Falconer, 1951; Soriano et al., 1997; Hunter-Schaadie, 1997; Caviness and Rakic, 1978; Caviness, 1982; Caviness et al., 1972; Rice et al., 2001; Yip et al., 2000; Phelps et al., 2002; Bailmaier et al., 2002; Liu et al., 2005).
Genotyping Protocols
SEPARATED MELT: Reln<sup>rl</sup>
MELT: Generic Pde6b Alternate1

Genotyping resources and troubleshooting
At The Jackson Laboratory, we use the combination of breeding scheme and phenotype to maintain our B6C3Fe a/a Reln<sup>rl</sup>/J colony.

1) Reln<sup>rl</sup>/Reln<sup>rl</sup> x B6C3FeF1/J a/a to generate obligate heterozygotes (Reln<sup>rl</sup>/+).
2) Reln<sup>rl</sup>+/+ x Reln<sup>rl</sup>+/+ to generate homozygotes, which are identified based on their phenotype: ataxic gait, dystonic posture and tremors.

Appearance
black, ataxic
Related Genotype: a/a Reln<sup>rl</sup>/Reln<sup>rl</sup>

black, unaffected
Related Genotype: a/a +/+ or a/a Reln<sup>rl</sup>+/+

Citation
When publishing the results of your use of this strain in a publication, please cite the originating article(s) and include JAX stock #000235 in your Materials and Methods section.

Facility Barrier Level Descriptions
Production of mice from cryopreserved embryos or sperm occurs in a maximum barrier room, G200

Pricing & Availability

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

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<th>SERVICE</th>
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<td>Cryo Recovery</td>
<td>Homozygous for a, Heterozygous Homozygous or wildtype for Reln&lt;sup&gt;rl&lt;/sup&gt;, 1 pair minimum</td>
<td>$2,595.00</td>
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We will fulfill your order by providing at least two carriers for each strain ordered. The total number, sex, and genotypes provided will vary, although typically 8 or more animals are provided. Please check genotypes which will be recovered. While the genotypes of all animals produced will be communicated to you prior to scheduling shipment, the genotypes of animals provided may not reflect the mating scheme and genotypes described in the strain description. Animals are typically ready to ship in 11-14 weeks. If a second recovery is required to produce the minimum number of animals, then delivery time would increase to approximately 25 weeks. If we fail to produce animals of the correct genotype, you will not be charged. We cannot guarantee the reproductive success of mice shipped to your facility. If the mice are lost after the first three days (post-arrival) or do not produce progeny at your facility, a new order and fee will be necessary.

Cryo recovery to establish a Dedicated Supply for greater quantities of mice. Mice recovered can be used to establish a dedicated colony to contractually supply you mice according to your requirements. Price by quotation.
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Terms of Use

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