C3H/HeJ

Stock No: 000659 | C3H

Inbred Strain

PLACE ORDER

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C3H/HeJ mice, commonly called C3H, are used as a general purpose strain in a wide variety of research areas including cancer, infectious disease, sensorineural, and cardiovascular biology research. A spontaneous mutation in Tlr4 occurred in C3H/HeJ at the lipopolysaccharide response locus (mutation in toll-like receptor 4 gene, Tlr4Lps-d) making C3H/HeJ mice more resistant to endotoxin. C3H/HeJ mice are highly susceptible to infection by Gram-negative bacteria such as Salmonella enterica. The C3H substrains at The Jackson Laboratory are homozygous for the retinal degeneration 1 mutation (Pde6brd1), causing blindness by weaning age.

READ MORE

GENETIC OVERVIEW

<table>
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<tr>
<th>Genetic Background</th>
<th>Generation</th>
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RESEARCH APPLICATIONS
Cardiovascular Research
Cancer Research
Research Tools
Sensorineural Research
Immunology, Inflammation and Autoimmunity Research
Neurobiology Research
Mouse/Human Gene Homologs

BASE PRICE
Starting at:
$25.33 Domestic price for male 3-week

Details

Important Note
This strain does not carry mouse mammary tumor virus (MMTV). This strain is homozygous for retinal degeneration allele Pde6b<sup>rd1</sup>, the defective lipopolysaccharide response allele Tlr<sup>4<sub>Lps<sup>-</sup>d</sub></sup>, and for a chromosomal inversion on Chromosome 6. A sighted alternative is Stock No. 003648, C3Sn:BliA-Pde6b<sup>-</sup>/DnJ.

Detailed Description
C3H/HeJ mice are used as a general purpose strain in a wide variety of research areas including cancer, immunology and inflammation, sensorineural, and cardiovascular biology. C3H/HeJ mice and all other Jackson substrains are homozygous for the retinal degeneration 1 mutation (Pde6b<sup>rd1</sup>), which causes blindness by weaning age, but lack the nob5 allele of Gpr179 (Chang, 2015). White belly spots, ranging in phenotype from a few white hairs to a defined spot are common in C3H/HeJ mice. There is also a high incidence of hepatomas in C3H mice (reportedly 72-91% in males at 14 months, 59% in virgin females, 30-38% in breeding females). Despite the lack of exogenous mouse mammary tumor virus (MMTV), virgin and breeding females may still develop some mammary tumors later in life. C3H/HeJ mice, fed an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat), fail to develop atherosclerotic aortic lesions in contrast to several highly susceptible strains of mice (e.g. C57BL/6J, Stock No. 000664; C57L/J, Stock No. 000668, C57BR/rdJ, Stock No. 000667, and SM/J, Stock No. 000687). C3H/HeJ mice spontaneously develop alopecia areata (AA) at a reported incidence of approximately 0.25% by 5 months of age. In older mice (12-18 months old), incidences as high as approximately 20% are reported. Females as young as 3-5 months can develop AA, but onset typically is delayed until after 6 months in males. Alopecia areata can be surgically-induced by grafting a small piece of skin from an older, donor animal with AA onto a younger, isogenic C3H/HeJ recipient.

A spontaneous mutation occurred in C3H/HeJ at the lipopolysaccharide response locus (later identified as a mutation in the toll-like receptor 4 gene, Tlr<sup>4<sub>Lps<sup>-</sup>d</sub></sup>) making C3H/HeJ mice endotoxin resistant. C3H/HeJ (Tlr<sup>4<sub>Lps<sup>-</sup>d</sub></sup>) mice are highly susceptible to infection by Gram-negative bacteria such as Salmonella enterica. Mice infected with Salmonella exhibit delayed chemokine production, impaired
nitric oxide generation and attenuated cellular immune responses. Mortality in infected mice appears to result from enhanced bacterial growth within the liver Kupffer cell network (Vazquez-Torres et al., 2004).

The C3H/HeJ substrain is homozygous for an inversion on Chromosome 6 (symbol: In(6)1J). The inversion covers 20% of Chromosome 6 between D6Mit124 (~30.3 cM) and D6Mit150 (~51.0 cM), but results in no reported phenotype. Results from screening other C3H substrains and cryopreserved stock from C3H/HeJ suggest that the mutation arose after 1952. The spontaneous mutation, spike wave discharge 1 (Gria4spkwt), is present in C3H/HeJ, but not C3HeB/FeJ. Mice homozygous for this mutation exhibit a modest incidence of absence seizures. This strain is also homozygous for a hypomorphic allele in Pcnx2, which is caused by an IAP insertion and which dampens the severity of the absence seizure phenotype caused by Gria4spkwt (Frankel et al., 2014).

Development

Selected References

Genetics

Hrh1Bphs-r

Tlr4Lps-d

ln(6)1J

Pcnx2C3HHeJ

Ahrb-2

Cd51

Gria4spkwt

Pde6bDl

Disease/Phenotype

Disease Terms

Research Areas By Genotype

Mammalian Phenotype Terms by Genotype

Phenotype Information

References

Technical Support
Genotyping Protocols
End Point Analysis: Tlr4<sup>Lps<sup>-</sup></sup>-Alternate 2
Genotyping resources and troubleshooting
Inbred mouse strains are maintained through sibling (sister x brother) matings: no genotyping required.

Dietary Information
LabDiet<sup>®</sup> 5K52 formulation (6% fat)

Breeding Considerations
This strain is a good breeder.
Additional Breeding and Husbandry Support

Mating System
Sibling x Sibling

Appearance
agouti
Related Genotype: A/A

Citation
When using the C3H mouse strain in a publication, please include JAX stock #000659 in your Materials and Methods section.

Animal Health Reports

- MP14 (Maximum)
- RB09 (Maximum)
- MP24 (High)

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