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, \( Tlr4^{Lps-d} \)) 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 (\( Pde6b^{rd1} \)), causing blindness by weaning age.

**GENETIC OVERVIEW**

<table>
<thead>
<tr>
<th>Genetic Background</th>
<th>Generation</th>
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<tbody>
<tr>
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<td>(2019-06-14 00:00:00)</td>
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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:

$26.09 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 \( Pde6b^{rd1} \), the defective lipopolysaccharide response allele \( Tlr4^{Lps-d} \), and for a chromosomal inversion on Chromosome 6. A sighted alternative is Stock No. 003648, C3Sn.BLiA-Pde6b\(^+\)/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 (\( Pde6b^{rd1} \)), which causes blindness by weaning age, but lack the \( nob5 \) allele of \( Gpr179 \) (Chang,
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/cdJ, 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, *Tlr4*). C3H/HeJ mice endotoxin resistant. C3H/HeJ (*Tlr4*) 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 (*Gria4*), 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 *Gria4* (Frankel et al., 2014).
Genotyping Protocols
End Point Analysis: Tlr4<sup>Lps-d</sup>
Genotyping resources and troubleshooting
Inbred mouse strains are maintained through sibling (sister x brother) matings; no genotyping required.

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
Facility Barrier Level Descriptions
  - AX29 (Maximum)
  - EM02 (Maximum)
  - RB09 (Maximum)

Pricing & Availability
Sized to accommodate orders of up to 100 or more. Ask Customer Service for details.
## Live Mouse

<table>
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<tr>
<th>AGE</th>
<th>SEX</th>
<th>GENOTYPE</th>
<th>PRICE</th>
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## Related Products and Services

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General Terms and Conditions

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Phone: 207-288-6470
Email: TechTran@jax.org

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All

By Allele

By Gene

By Collection

All Related Strains

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