DBA/2J
Stock No: 000671
Inbred Strain

Sized to accommodate orders of up to 100 or more. Ask Customer Service for details.

Also Known As: D2J, D2, DBA2
DBA/2J is a widely used inbred strain. Some characteristics include low susceptibility to developing atherosclerotic aortic lesions, high-frequency hearing loss, susceptibility to audiogenic seizures, development of progressive eye abnormalities that closely mimic human hereditary glaucoma, and extreme intolerance to alcohol and morphine.

GENETIC OVERVIEW

Genetic Background
Generation
Contact Technical Support
(2018-07-27 00:00:00)
DBA/2J is a widely used inbred strain that is valuable in a large number of research areas, including cardiovascular biology, neurobiology, and sensorineural research. Its characteristics are often contrasted with those of the C57BL/6J inbred strain (Stock No. 000664). DBA/2J mice show a low susceptibility to developing atherosclerotic aortic lesions (20 to 350 um2 atherosclerotic aortic lesions /aortic cross-section) following 14 weeks on an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat). They also exhibit high-frequency hearing loss beginning roughly at the time of weaning/adolescence (between three to four weeks of age) and becoming severe by two to three months of age. The age related hearing loss 8 mutation arose spontaneously in DBA/2J between 1951 and 1975. This strain possesses three recessive alleles that cause progressive cochlear pathology initially affecting the organ of Corti. Decreasing anteroventral cochlear nucleus volume decreases and neuron loss parallel the progression of peripheral hearing loss. Young DBA/2J inbred mice are also susceptible to audiogenic seizures due to the asp2 mutation, however, this susceptibility decreases as animals reach adulthood. There is high incidence of calcareous pericarditis, and calcified lesions of the testes, tongue and skeletal muscle. This strain is among the least responsive to phytohemagglutinin (Heiniger et al., 1975), but highly sensitive to haloperidol (Kanes et al, 1993).

Aging DBA/2J mice develop progressive eye abnormalities that closely mimic human hereditary glaucoma. Defects include iris pigment dispersion, iris atrophy, anterior synechia (adhesion of the iris to the cornea), and elevated intraocular pressure.
The onset of disease symptoms begins between three and four months of age with 56% of females and 15% of males showing signs of iris pigment epithelium loss and transillumination of the peripheral iris. By six to seven months of age, all mice demonstrate significant widespread transillumination and thickening of the iris border. Elevation of IOP is evident in some females by six months of age. By nine months of age, both sexes exhibit elevated IOP, with pressures higher in females (mean: 20.3 +/-79; 1.8 mmHg) compared to males (mean: 16.2 +/-79; 1.4 mmHg). Retinal histopathology reveals retinal ganglion cell, as well as GABAergic and cholinergic amacrine cell, loss. (Moon JI et al. 2005). Two alleles contribute to the eye phenotype, Gpnmbr19s and Tyrp1isa; both are present in DBA/2J mice.

DBA/2J mice also show an extreme intolerance to alcohol and morphine. In 2002, Vance et al. reported that NK cells in DBA/2J exhibit the unique characteristic that they lack surface expression of CD94/NKG2A receptors. CD94/NKG2 receptors are normally expressed on the surface of most fetal NK cells. Expression of CD94/NKG2 is thought to play a role in self tolerance and the ability of NK cells to distinguish between MHC Ilow and MHC Ihigh target cells. CD94 is the product of the mouse Klrd1 locus, on mouse Chromosome 6. A subsequent publication by Wilhelm and coworkers identified a deletion in the 3’ end of the Klrd1 gene of DBA/2J mice. This ~2.4 kb deletion does not prevent transcription of the gene, but prevents translation and cell surface expression of the CD94 protein. Analysis of DNA samples held at The Jackson Laboratory (unpublished results) confirmed the presence of the deletion of Klrd1 in the DBA/2J strain. The deletion, which occurred sometime between 1984 and 1989, is homozygous within our colonies, making DBA/2J mice naturally CD94 deficient.

Development

Selected References

Genetics

Fscn2ahl8
Gpr84del
P2rx7P451L
Tyrp1isa
a
Hc0
Fbrwt2DBA/2J
Fbrwt1DBA/2J
Cdh23ahl
Mx1s1
Ahrd
Asp2
**Disease/Phenotype**

**Disease Terms**

**Research Areas By Phenotype**

**Mammalian Phenotype Terms by Genotype**

**Phenotype Information**

**References**

**Technical Support**

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**Genotyping Protocols**

Sanger sequencing: Taar1 rs33645709-SEQ  
Genotyping resources and troubleshooting

Inbred mouse strains are maintained through sibling (sister x brother) matings; no genotyping required.

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**Dietary Information**

LabDiet® 5K52 formulation (6% fat)

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**Breeding Considerations**

This strain is a good breeder.

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**Additional Breeding and Husbandry Support**
Mating System
Sibling x Sibling
Appearance
dilute brown
Related Genotype: \(a/a\ Tyrp1^b/Tyrp1^b\ Myo5a^d/Myo5a^d\)

Citation
When using the DBA/2J mouse strain in a publication, please include JAX stock #000671 in your Materials and Methods section.

Animal Health Reports
Facility Barrier Level Descriptions

- RB09 (Maximum)
- RB11 (Maximum)
- EM03 (Maximum)
- MP14 (Maximum)
- AX29 (Maximum)

Pricing & Availability

Sized to accommodate orders of up to 100 or more. Ask Customer Service for details.

![Domestic Internat](image)

Pricing effective for USA, Canada and Mexico shipping destinations

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### Volume Pricing Details

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**Volume Pricing Program**

Quantities: Volume pricing is automatically applied when a minimum quantity per strain for a shipment is reached

Sexes: Sexes of the same strain may be combined to reach minimum quantity levels to receive the volume pricing

Shipment: All shipping destinations qualify

This strain is available from some international Charles River (CR) breeding facilities in Japan and/or Europe. For more information, see the [Worldwide Distributor List for JAX® Mice](#).

### Related Products and Services

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Terms Of Use

General Terms and Conditions

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

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