Also Known As: D2J, D2, DBA2, DBA, DBA/2

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.
RESEARCH APPLICATIONS
Cardiovascular Research
Neurobiology Research
Sensorineural Research
Research Tools
Dermatology Research
Mouse/Human Gene Homologs
Hematological Research
Immunology, Inflammation and Autoimmunity Research

BASE PRICE
Starting at:
$30.01 Domestic price for male 3-week

Important Note
This strain is homozygous for Cdh23<sup>ahl</sup>, the age related hearing loss 1 mutation, which on this background results in progressive hearing loss that is already severe by three months of age.

Detailed Description
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 um<sup>2</sup> 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 (IOP). 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, GpnmbR150X 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

a

Myo5ad

Asp2

Hbbd

GpnmbR150X

Tyrp1isa

Ahrd

Hc0

Cdhd23ahl

P2rx7s48804829-T
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.

Dietary Information
LabDiet® 5K52 formulation (6% fat)

Breeding Considerations
This strain is a good breeder.

Additional Breeding and Husbandry Support
Mating System
Sibling x Sibling

Appearance
dilute brown

Related Genotype: a/a Tyrp1b/Tyrp1b Myo5a+/Myo5a-

Citation
When using the DBA2 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)

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Domestic
Internation

Pricing effective for USA, Canada and Mexico shipping destinations

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Email: TechTran@jax.org

Related Strains

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