Overview

Also Known As: D2-mdx
The D2.B10 (DBA/2-congenic) Dmd<sup>mdx</sup> mouse (also referred to as DBA/2J-mdx or D2-mdx mice) may be a superior Duchenne muscular dystrophy model as it better recapitulates several of the human characteristics of DMD myopathy (lower hind limb muscle weight, fewer myofibers, increased fibrosis and fat accumulation, and muscle weakness) relative to strains with this mutant allele on other genetic backgrounds.

Our preclinical efficacy testing services offer scientific expertise and an array of target-based and phenotype-based outcome measures, both in vivo and at endpoint, for flexible study designs and assay development in mouse models of Muscular Dystrophy. See our full service platform.

Donating Investigator
IMR Colony, The Jackson Laboratory
**GENETIC OVERVIEW**

<table>
<thead>
<tr>
<th>Genetic Background</th>
<th>Generation</th>
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<tbody>
<tr>
<td>Spontaneous Dmd</td>
<td>?+F17</td>
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<td>(2019-06-12 00:00:00)</td>
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<table>
<thead>
<tr>
<th>Allele Type</th>
<th>Gene Symbol</th>
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<tbody>
<tr>
<td>Spontaneous</td>
<td>Dmd</td>
<td>dystrophin, muscular dystrophy</td>
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</tbody>
</table>

**RESEARCH APPLICATIONS**

- Sensorineural Research
- Mouse/Human Gene Homologs
- Cell Biology Research
- Neurobiology Research

**BASE PRICE**

Starting at:

$0.00 Domestic price for male 5-week

**Details**

Duchenne muscular dystrophy (DMD) is a progressive muscular disorder caused by an imbalance between muscle degeneration and regeneration resulting in muscle degeneration, necrosis, accumulation of fat and fibrosis, and insufficient regeneration/loss of myofibers. The genetic cause of DMD are mutations of the dystrophin muscular dystrophy gene (DMD) on the X chromosome. The Dmd<sup>mdx</sup> mutation in mice has a termination codon in exon 23 that is predicted to result in a truncated protein. Heterozygous females are viable and fertile with no gross phenotypic abnormalities. Homozygous females and hemizygous males are viable and fertile with myopathic features of DMD; although the myopathology is both less severe than the human disease course and variable by mouse strain genetic background.

The muscle pathology observed for C57BL/10ScSn-Dmd<sup>mdx</sup> mice (also referred to as C57BL/10-mdx; Stock No. 001801) includes active fiber necrosis, cellular infiltration, a wide range of fiber sizes, and numerous centrally nucleated regenerating fibers. However, adult C57BL/10-mdx mice fail to exhibit several of the skeletal muscle characteristics of DMD (such as smaller number of myofibers, accumulation of fat and fibrosis, insufficient myofiber regeneration, and loss of muscle weight). In addition, the C57BL/10 inbred strain is shown to possess greater muscle satellite cell self-renewal ability/efficiency than that of the DBA/2 inbred strain which may
increase muscle regeneration/attenuate muscular dystrophy phenotype for MD mutations maintained on the C57BL/10 genetic background. As such, The Jackson Laboratory Repository created this DBA/2J-congenic $Dmd^{mdx}$ mouse model, D2.B10-$Dmd^{mdx}$/J (also referred to as DBA/2J-mdx or D2-mdx; Stock No. 013141).

These DBA/2J-mdx mice may be expected to have a phenotype similar to other $Dmd^{mdx}$ mouse models. That is, when compared to C57BL/10-mdx animals, DBA/2-congenic $Dmd^{mdx}$ mice exhibit additional DMD characteristics such as lower hind limb muscle weight, fewer myofibers, increased fibrosis and fat accumulation, and remarkable muscle weakness. The DBA/2-congenic $Dmd^{mdx}$ phenotype is attributed to diminished myofiber regeneration (rather than accelerated myofiber degeneration). Unlike C57BL/10-mdx mice, no increased incidence of spontaneous rhabdomyosarcoma-like tumors are reported for the DBA/2-congenic $Dmd^{mdx}$ model.

In an attempt to offer alleles on well-characterized or multiple genetic backgrounds, alleles are frequently moved to a genetic background different from that on which an allele was first characterized. This is the case for these DBA/2J-mdx mice (Stock No. 013141). It should be noted that the phenotype could vary from that originally described for C57BL/10-mdx (Stock No. 001801). We will modify the strain description if necessary as published results become available.
**Breeding Considerations**

The \( Dmd^{mdx} \) mutant allele is on the X chromosome. Mutant mice were bred to DBA/2J inbred mice (Stock No. 000671) for many generations using a marker-assisted, speed congenic approach to establish this congenic strain. When maintaining the live congenic colony, heterozygous or homozygous females may be bred with hemizygous males.

**Additional Breeding and Husbandry Support**

**Mating System**

Homozygote x Hemizygote

**Citation**

When using the D2.B10-\( Dmd^{mdx} \)/J mouse strain in a publication, please cite the originating article(s) and include JAX stock #013141 in your Materials and Methods section.

**Animal Health Reports**

**Facility Barrier Level Descriptions**

AX12 (Maximum)

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**Domestic**

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<th>GENOTYPE</th>
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<td>$139.90</td>
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<td>6 weeks</td>
<td>Female</td>
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**Related Products and Services**

| Frozen Mouse Embryo | D2.B10-\( Dmd^{mdx} \)/J | $2595.00 |
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### Related Strains

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All Related Strains