MRL/MpJ-Fas lpr/J

Stock No: 000485 | MRL-lpr

Spontaneous Mutation

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Overview
Also Known As: lymphoproliferation, MRL-lpr

This strain is commonly known as MRL-lpr or lpr mutant. Mice are homozygous for the lymphoproliferation spontaneous mutation (Fas^{lpr}), and show systemic autoimmunity, massive lymphadenopathy associated with proliferation of aberrant T cells, arthritis, and immune complex glomerulonephrosis. Mice are useful as a model to determine the etiology of systemic lupus erythematosus (SLE) and Sjögren (Sicca) syndrome and to evaluate therapies.

Information about lupus disease phenotypes in MRL-lpr is available here.

GENETIC OVERVIEW

<table>
<thead>
<tr>
<th>Genetic Background</th>
<th>Generation</th>
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<tr>
<td></td>
<td>F5pF13</td>
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Fas^{lpr}

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<tr>
<th>Allele Type</th>
<th>Gene Symbol</th>
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<tr>
<td>Spontaneous (Hypomorph)</td>
<td>Fas</td>
<td>Fas (TNF receptor superfamily member 6)</td>
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RESEARCH APPLICATIONS

Immunology, Inflammation and Autoimmunity Research
Internal/Organ Research
Apoptosis Research
Cancer Research
Mouse/Human Gene Homologs

VIEW ALL RESEARCH APPLICATIONS
Important Note
July 2007: This strain has been recovered from cryopreservation and the original phenotype was observed: The sixteen-week old mice have lymph nodes that were 4.5 (females) to 10.1 times (male) larger than age and sex matched individuals from the former colony. Splenomegaly is 3 to 6 times greater and their life spans were also greatly reduced. The former version of this line, which displayed a loss of lymphoproliferative phenotype, has been renamed MRL/MpJ-Fas²pr/²J and is available as Stock No. 006825.

Detailed Description
Mice homozygous for the lymphoproliferation spontaneous mutation (Fas²pr) show systemic autoimmunity, massive lymphadenopathy associated with proliferation of aberrant T cells, arthritis, and immune complex glomerulonephritis. Starting at about three months of age, levels of circulating immune complexes rise greatly in the MRL-Ipr/Ipr mouse but not the MRL normal (Hewicker 1990). Onset and severity of symptoms associated with the Fas²pr gene is strain-dependent. For example, lymphoproliferation varies greatly with congenic strain C57BL/6J-Fas²pr/Fas²pr at a 24 fold increase over control lymph node weight, MRL/Mp-Fas²pr/Fas²pr at 75 fold and congenic strain C3H/HeJ-Fas²pr/Fas²pr highest at 116 fold increase over control lymph node weight (Morse et al 1985). Variance in renal pathology ranks from extensive in MRL/Mp-Fas²pr/Fas²pr at 4 to 7 months to negligible at 14 to 16 months in mice with C57BL/6J and C3H/HeJ backgrounds and homozygous for the Fas²pr (Kelley and Roths 1985). Spontaneous production of anti-dsDNA autoantibodies is likewise affected with percentage binding of radiolabeled dsDNA in Fas²pr/Fas²pr mice varying from 5 percent on C57BL/6J to 26 percent on C3H/HeJ to as high as 49 percent on MRL/Mp (izui et al 1984). Female MRL/Mp-Fas²pr mice die at an average age of 17 weeks of age and males at 22 weeks. This compares to between 42 and 52 weeks in females on the C57BL/6J or C3H/HeJ background (Roths 1987). Embryonic stem cell lines have been established with MRL/Mp-Fas²pr/Fas²pr mouse strains (Kawase et al 1994). This mouse is a model for systemic lupus erythematosus-like autoimmune syndromes.

MRL/MpJ and one of its ancestral strains LG/J display heightened wound healing relative to a panel of other inbred strains. At 4 weeks post-injury, 2mm ear punch wounds healed to 0-0.4mm in MRL/MpJ mice but were still 1.2-1.6mm in C57BL/6 mice. At 15 days post-injury C57BL/6 showed a maximal closure of 30% reduction in ear hole size while MRL showed 85% reduction. The process of healing in MRL/MpJ mice was faster, more complete, showed increased swelling, angiogenesis, fibroblast migration, extracellular matrix deposition, and decreased scarring and fibrosis. Additionally, hair follicles and accompanying sebaceous glands were regenerated to a much greater degree. The other ancestral strains of MRL/MpJ (C3H, C57BL/6, and AKR) do not display this enhanced healing. Bone marrow transplantation showed that the MRL/MpJ healing phenotype did not readily transfer with bone marrow and did remain in the irradiated host tissues. Enhanced healing of cardiac wounds has also been reported in MRL/MpJ mice. In this model a very high mitotic index (10-20%) was found, similar to that seen in non-mammalian tissue regeneration. Using F2 and backcross mapping of MRL/MpJ-Fas²pr x B6 progeny McBrearty et al. identified wound healing QTLs: the heal2 and heal3 loci were identified on MRL/MpJ Chromosome 13 in the region of D13Mit115 and D13Mit129 respectively; the heal5 locus was identified on MRL/MpJ chromosome 12 in the region of D12Mit233; the heal1 locus was identified on chromosome 8 of C57BL/6 in the region of DBMit211; and a highly suggestive locus was found on MRL/MpJ Chromosome 7 in the region of D7Mit220. (Clark et al., 1998; Leferovich et al., 2001; Kench et al., 1999; McBrearty et al., 1998.)

Microarray and SELDI ProteinChip analysis have identified multiple genes and proteins that have varied expression in the ear punch wounds of MRL/MpJ-Fas²pr versus C57BL/6. The changes in expression patterns suggest that in MRL/MpJ mice there is less of an inflammatory response and an earlier transition into tissue repair than is seen in C57BL/6. (Li et al., 2000 and 2001.) Blankenhorn et al. found that MRL/MpJ females heal faster and more completely than males. Some heal QTL are sexually dimorphic with heal 2, 3, 7, 8, 10, and 11 having greater effect in males and heal 4, 5, and 9 having greater effect in females. Castration improves
wound healing in MRL/MpJ males to nearly the degree seen in females, but ovariectomy does not improve the degree of healing seen in MRL/MpJ females. (Blankenhorn et al., 2003) Relative to B10.D2SnJ mice, MRL/MpJ mice have decreased Neutrophil accumulation in the bronchiolar lavage in response to LPS infusion and tests using bone marrow chimeras revealed that the pulmonary inflammatory response transfers with bone marrow. Transforming growth factor beta 1 autologous induction is reduced in MRL/MpJ splenocytes while macrophages show a reduction in the transforming growth factor beta 1 induction of interleukin 1 beta and tumor necrosis factor alpha production but no significant reduction in transforming growth factor beta 1 production. (Kench et al., 1999) MRL-Fas<sup>lpr</sup> are also highly susceptible to Mycobacterium leprae (Yogi et al., 1989).

Genotyping Protocols
Melt Curve Analysis: Fas<sup>lpr</sup> MCA
Standard PCR: Fas<sup>lpr</sup>
Genotyping resources and troubleshooting
Dietary Information
LabDiet® 5K52 formulation (6% fat)
Breeding Considerations

**This strain is a challenging breeder.**

Due to the heightened healing which occurs in mice with the MRL genetic background, ear punch is not a good method for individual mouse identification in this strain. Mice may have only 2 litters before developing phenotype.

**Additional Breeding and Husbandry Support**

**Mating System**

Homozygote x Homozygote

**Appearance**

albino

Related Genotype: a/a Tyr<sup>C</sup>/Tyr<sup>C</sup>

**Citation**

When using the MRL dd mouse strain in a publication, please cite the originating article(s) and include JAX stock #000485 in your

Animal Health Reports

<table>
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<th>Facility Barrier Level Descriptions</th>
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<td>RB03 (Maximum)</td>
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**Domestic**

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