B6.129P2-Il10null/J

Stock No: 002251 | Il-10 KO

Congenic, Targeted Mutation

AVAILABLE NOW

PLACE ORDER

Sized to accommodate orders of up to 10 or more with age range. Ask Customer Service for details.
Also Known As: IL-10 KO

B6.129P2-Il10<sup>tm1Cgn</sup>/J mutant mice spontaneously develop a chronic inflammatory bowel disease (IBD). IL10-deficiency is associated with altered lymphocyte and myeloid profiles, elevated serum amyloid A levels, altered responses to inflammatory or autoimmune stimuli, increased prevalence of colorectal adenocarcinoma, and spontaneous development of chronic enterocolitis. IL10-deficient mice exhibit a significant increase in peripheral blood granulocyte populations upon lesion development.

Donating Investigator

Herbert C. Morse III, Laboratory of Immunopathology, NIAID, NIH

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**GENETIC OVERVIEW**

<table>
<thead>
<tr>
<th>Genetic Background</th>
<th>Generation</th>
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Contact Technical Support
(2018-07-27 00:00:00)

**Il10<sup>tm1Cgn</sup>**

<table>
<thead>
<tr>
<th>Allele Type</th>
<th>Gene Symbol</th>
<th>Gene Name</th>
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<tr>
<td>Targeted (Null/Knockout)</td>
<td>il10</td>
<td>interleukin 10</td>
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**RESEARCH APPLICATIONS**

Immunology, Inflammation and Autoimmunity Research
Cancer Research
Hematological Research
Mice homozygous for the II10^m1Cgn targeted mutation are viable and fertile when housed under specific pathogen free (SPF) conditions. Under conventional housing conditions, II10-deficiency is associated with altered lymphocyte and myeloid profiles, elevated serum amyloid A levels, altered responses to inflammatory or autoimmune stimuli (both endogenous and induced), increased prevalence of colorectal adenocarcinoma (especially on 129/Sv and, to a lesser extent, BALB/c genetic background), and spontaneous development of chronic enterocolitis (see below). As The Jackson Laboratory Repository maintains these mice at high health status conditions (high SPF), the observed or experimentally-induced II10-deficient phenotype may vary from that previously published using mice from conventional mouse rooms. These IL-10 mutant mice may be useful studying inflammatory bowel disease (IBD) (Crohn’s disease (CD) and/or colitis), cancer, innate and adaptive immunity, and many other areas of inflammatory or autoimmunity research.

The onset and severity of both spontaneous and experimentally-induced inflammatory phenotype of II10-deficient mice is strongly influenced by the genetic background and the husbandry conditions (specific health status/commensal flora) of the vivaria in which mice are maintained.

For example, inflammatory bowel disease (IBD; colitis and Crohn’s disease) severity in mouse models is dependent upon interactions between specific genetic background and environmental factors (an as yet undefined component of the enteric flora of which Helicobacter spp. appear to be associated, but not specifically the environmental trigger). Both spontaneous and induced models of IBD demonstrate that susceptibility to intestinal inflammation varies markedly among inbred strains of mice. Generally, for II10-deficient models on defined genetic backgrounds, the severity of colitis-related characteristics is most severe on C3H/HeJ/Bir (Stock No. 004326 and Stock No. 003968) or 129/Sv (Stock No. 004368), intermediate on BALB/cJ (Stock No. 004333) or NOD/Lt (Stock No. 004266), and least severe on C57BL/10 (Stock No. 002250) or C57BL/6J (Stock No. 002251). Furthermore, the husbandry conditions (specific health status/commensal flora) of the vivaria in which mice are maintained significantly alter the onset and severity of spontaneous IBD; higher SPF conditions are associated with attenuated colitis. II10-deficient mice on both the C3H/HeJ/Bir and C57BL/6J genetic backgrounds exhibit a significant increase in peripheral blood granulocyte populations upon lesion development and this metric may be used as a robust non-lethal assessment of II10-deficiency induced colitis onset and severity. Other indications of II10-deficiency induced colitic lesion onset may include perianal ulceration (C3H/HeJ/Bir background) or rectal prolapse (C57BL/6J background).
Genetics

II10\textsuperscript{1m1C9n}

Disease/Phenotype

Disease Terms

Research Areas By Genotype

Mammalian Phenotype Terms by Genotype

References

Technical Support

CONTACT TECHNICAL SUPPORT

Genotyping Protocols
High Resolution Melting: II10\textsuperscript{1m1C9n}
Standard PCR: II10\textsuperscript{1m1C9n}
Genotyping resources and troubleshooting

Dietary Information
LabDiet\textsuperset{®} 5K5Z formulation (6% fat)

Breeding Considerations

This strain is a good breeder.
When maintaining a live colony, homozygous mice may be bred together. As homozygous mice are more susceptible to pathogenic bacteria, high specific pathogen-free (SPF) conditions are recommended for optimal breeding. However, the onset and severity of both the spontaneous and experimentally-induced inflammatory phenotype of II10-deficient mice is strongly influenced by the genetic background and the husbandry conditions (specific health status/commensal flora) of the vivaria in which mice are maintained and such high SPF conditions may attenuate the desired II10-deficient phenotype.
Additional Breeding and Husbandry Support

Mating System
Homozygote x Homozygote

Appearance
black
Related Genotype: a/a

Citation
When using the II10 KO mouse strain in a publication, please cite the originating article(s) and include JAX stock #002251 in your Materials and Methods section.
Facility Barrier Level Descriptions

RB16 (Maximum)
## Pricing & Availability

Sized to accommodate orders of up to 10 or more with age range. Ask Customer Service for details.

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All Related Strains

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Related By:  
Autoimmune and Inflammation

NOD/ShiLtJ  
Stock No: 001976 | NOD  
Related By:  
Autoimmune and Inflammation

NZBWF1/J  
Stock No: 100008 | NZB/W  
Related By:  
Autoimmune and Inflammation

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