B6.129P2-Il10tm1Cpr/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
The onset and severity of both spontaneous and experimentally-induced inflammatory phenotype of IL10-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 IL10-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. IL10-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 IL10-deficiency induced colitis onset and severity. Other indications of IL10-deficiency induced colitic lesion onset may include perianal ulceration (C3H/HeJ/Bir background) or rectal prolapse (C57BL/6J background).
Genotyping Protocols
High Resolution Melting: Il10^Im1Cgn
Standard PCR: Il10^Im1Cgn
Genotyping resources and troubleshooting

Dietary Information
LabDiet® 5K52 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 Il10-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 Il10-deficient phenotype.
Additional Breeding and Husbandry Support

Mating System
Homozygote x Homozygote

Appearance
black
Related Genotype: a/a

Citation
When using the Il10 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

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## Domestic

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