Overview

Also Known As: CB2R KO

CB2 is normally up-regulated following pro-inflammatory and nociceptive stimuli, and in human brain tissues affected by Alzheimer's disease (AD), multiple sclerosis (MS) and amyotrophic lateral sclerosis (ALS). These CB2 knockout mice (CB2-, CB2R- or Cnr2-) may be useful for studying these diseases, pain management and drug/antibody therapeutics.
Cannabinoid receptor 2 (CB₂ or CB₂R) is normally expressed in cells of the haematopoietic lineage, central nervous system and bone. In immune cells, CB₂ is expressed on leukocytes and localized in intracellular membranous structures. Endogenous expression is up-regulated by pro-inflammatory and nociceptive stimuli. CB₂ activation reduces expression of adhesion molecules in brain endothelium, decreasing leukocyte engagement and recruitment.

In these CB₂ knockout mice (CB₂⁻/⁻, CB₂R⁻/⁻, Cnr2⁻/⁻ or Cnr2tm1Dgen⁻/⁻), CB₂ agonists have no effect on minimizing leukocyte-endothelial cell interaction. After treatment with lipopolysaccharide (LPS), CB₂ null mice fail to show complete resolution of inflammation compared to wild type controls. An increase in leukocyte adhesion is seen when Cnr2⁻/⁻ leukocytes are transferred into wildtype recipient mice.

This CB₂ knockout mutant was created and characterized by Deltagen, Inc. View phenotypic data developed by Deltagen.

J20 transgenic amyloid mice express the K670N/M671L/V717F mutant form of amyloid precursor protein under control of the human platelet–derived growth factor β polypeptide promoter (Stock No. 006293). Hemizygous J20 mice are a Familial Alzheimer’s Disease model with progressive amyloid deposition as they age (significant reactive microgliosis by 8 months of age). C57BL/6 mice homozygous for Cnr2tm1Dgen and also harboring the J20 transgene (J20 CNR2⁻/⁻) exhibit increased amyloid pathology (increased soluble Aβ42, plaque deposition and plaque associated microglia), but altered tau processing (suppressed total tau), when compared to J20 mice alone.

Of note, another published CB₂ knockout allele (Cnr2tm1Zim) has been used to show CB₂-deficient macrophages are resistant to the
inhibitory effects of δ-9-tetrahydrocannabinol (THC) and CB2 knockout mice have increased severity in experimental autoimmune encephalitis. Homozygous Cnr2<sup>tm1Zim</sup> mice also harboring the R6/2 transgene (encoding human mutant Huntington exon 1) have increased microglial activation and exacerbated Huntington disease progression/excitotoxicity compared to R6/2 mice alone.

### Development

### Control Suggestions

### Genetics

**Cnr2<sup>tm1Dgen</sup>**

### Disease/Phenotype

**Disease Terms**

**Research Areas By Phenotype**

**Mammalian Phenotype Terms by Genotype**

### References

### Technical Support

**Genotyping Protocols**

Standard PCR: Cnr2

*Genotyping resources and troubleshooting*

**Dietary Information**

LabDiet® 5K52 formulation (6% fat)

**Breeding Considerations**

When maintaining a live colony, heterozygous or homozygous mice may be bred together.

**Additional Breeding and Husbandry Support**

**Mating System**

Homozygote x Homozygote

**Citation**

When using the CB2KO mouse strain in a publication, please cite the originating article(s) and include JAX stock #005786 in your Materials and Methods section.

**Animal Health Reports**

**Facility Barrier Level Descriptions**

**AX11 (Maximum)**

### Pricing & Availability
Live mice available in varying quantities. Ask Customer Service for details.

**Domestic**

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All Related Strains

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