

Mc1r Cas9-KO Strategy

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Design Date: 2023-12-26

Overview

Target Gene Name

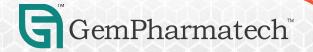
• Mc1r

Project Type

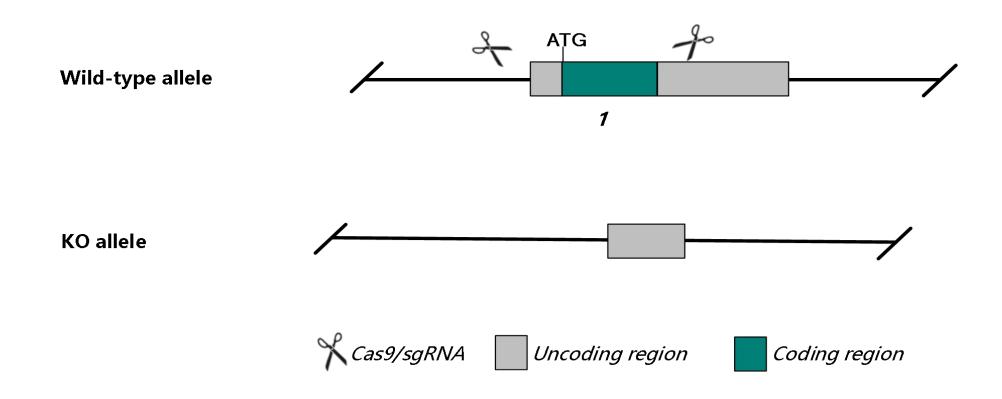
• Cas9-KO

Genetic Background

• C57BL/6JGpt



Strain Strategy



Schematic representation of CRISPR-Cas9 engineering used to edit the Mc1r gene.



Technical Information

- The *Mc1r* gene has 1 transcript. According to the structure of *Mc1r* gene, exon1 of *Mc1r*-201 (ENSMUST00000098324.4) transcript is recommended as the knockout region. The region contains all of the coding sequence. Knocking out the region will result in disruption of protein function.
- In this project we use CRISPR-Cas9 technology to modify *Mc1r* gene. The brief process is as follows: gRNAs were transcribed in vitro. Cas9 and gRNAs were microinjected into the fertilized eggs of C57BL/6JGpt mice. Fertilized eggs were transplanted to obtain positive F0 mice which were confirmed by PCR and ontarget amplicon sequencing. A stable F1-generation mouse strain was obtained by mating positive F0-generation mice with C57BL/6JGpt mice and confirmation of the desired mutant allele was carried out by PCR and on-target amplicon sequencing.



Gene Information

Mc1r melanocortin 1 receptor [Mus musculus (house mouse)]

▲ Download Datasets Gene ID: 17199, updated on 23-Nov-2023

Summary

Official Symbol Mc1r provided by MGI

Official Full Name melanocortin 1 receptor provided by MGI

Primary source MGI:MGI:99456

See related Ensembl:ENSMUSG00000074037 AllianceGenome:MGI:99456

Gene type protein coding RefSeg status VALIDATED Organism Mus musculus

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha;

Muroidea: Muridae: Murinae: Mus: Mus

Also known as e; Tob; Mcr1; Mshra

Summary Enables melanocyte-stimulating hormone receptor activity. Involved in intracellular signal transduction; positive regulation of intracellular signal

transduction; and positive regulation of transcription by RNA polymerase II. Acts upstream of or within melanin biosynthetic process; pigmentation; and sensory perception of pain. Predicted to be located in membrane. Predicted to be integral component of membrane. Predicted to be active in cytoplasm and plasma membrane. Is expressed in ductus deferens; epididymis; esophagus; and skin. Human ortholog(s) of this gene implicated in familial melanoma; major depressive disorder; melanoma; oculocutaneous albinism type II; and pigmentation disease. Orthologous to human MC1R (melanocortin 1 receptor). [provided by Alliance of Genome Resources, Apr 2022]

Orthologs human all

Try the new Gene table

Try the new Transcript table

Source: https://www.ncbi.nlm.nih.gov/

△ ?

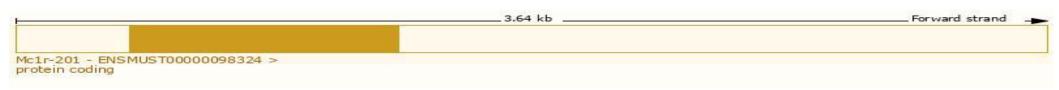


Transcript Information

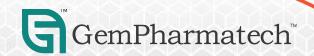
The gene has 1 transcript, and the transcript is shown below:



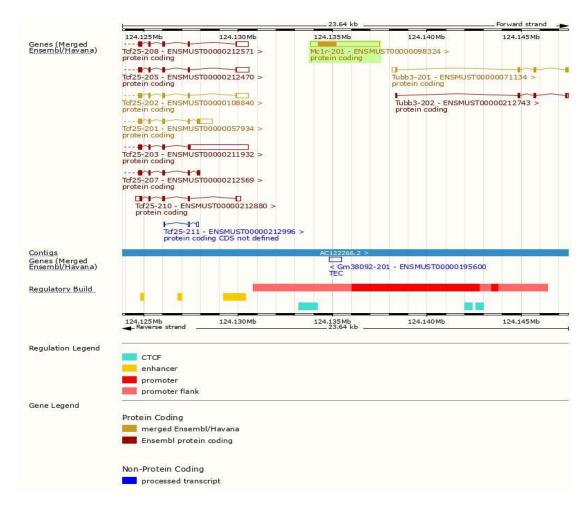
The strategy is based on the design of Mc1r-201 transcript, the transcription is shown below:



Source: https://www.ensembl.org



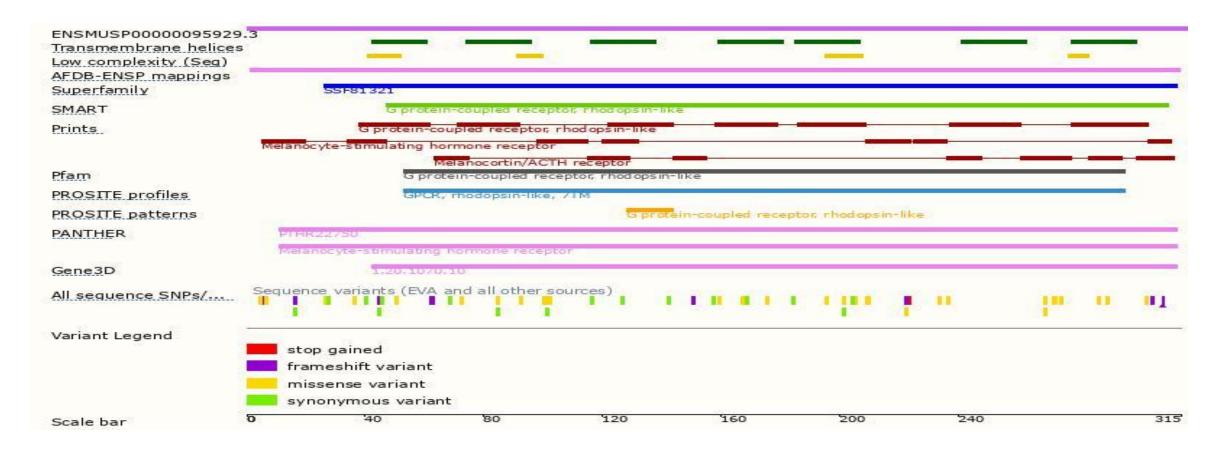
Genomic Information





Source: : https://www.ensembl.org

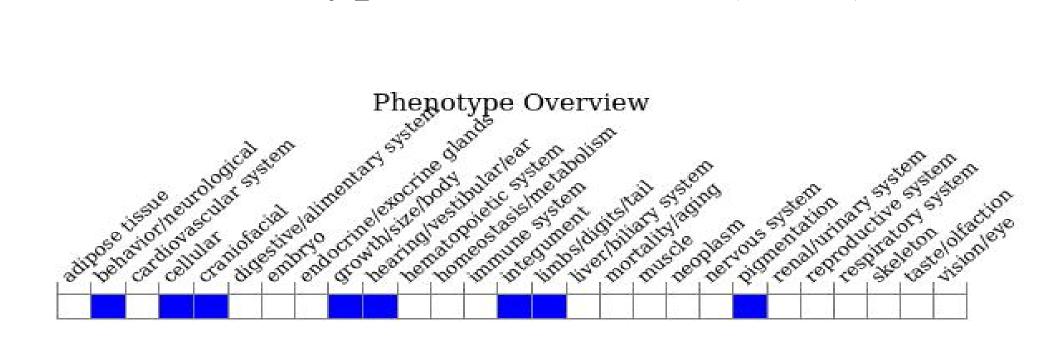
Protein Information





Source: : https://www.ensembl.org

Mouse Phenotype Information (MGI)



• Mutant alleles at this locus extend or restrict the amount of black pigment (eumelanin) in hair with the opposite effect on yellow pigment (phaeomelanin). Some variants affect pain sensitivity.



Source: https://www.informatics.jax.org

Important Information

- According to MGI information, mutant alleles at this locus extend or restrict the amount of black pigment (eumelanin) in hair with the opposite effect on yellow pigment (phaeomelanin). Some variants affect pain sensitivity.
- The KO region is about ~2.5kb away from the N-terminal of *Tubb3* gene, this strategy may influence the regulatory function of the N-terminal of *Tubb3* gene.
- *Gm38092* gene may be deleted.
- *Mc1r* is located on Chr8. If the knockout mice are crossed with other mouse strains to obtain double homozygous mutant offspring, please avoid the situation that the second gene is on the same chromosome.
- This Strategy is designed based on genetic information in existing databases. Due to the complexity of biological processes, all risks of the mutation on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

