GemPharmatech™

Mmp13 Cas9-CKO Strategy

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Reviewer: Xueting Zhang

Design Date: 2024-3-5

Overview

Target Gene Name

- Mmp13

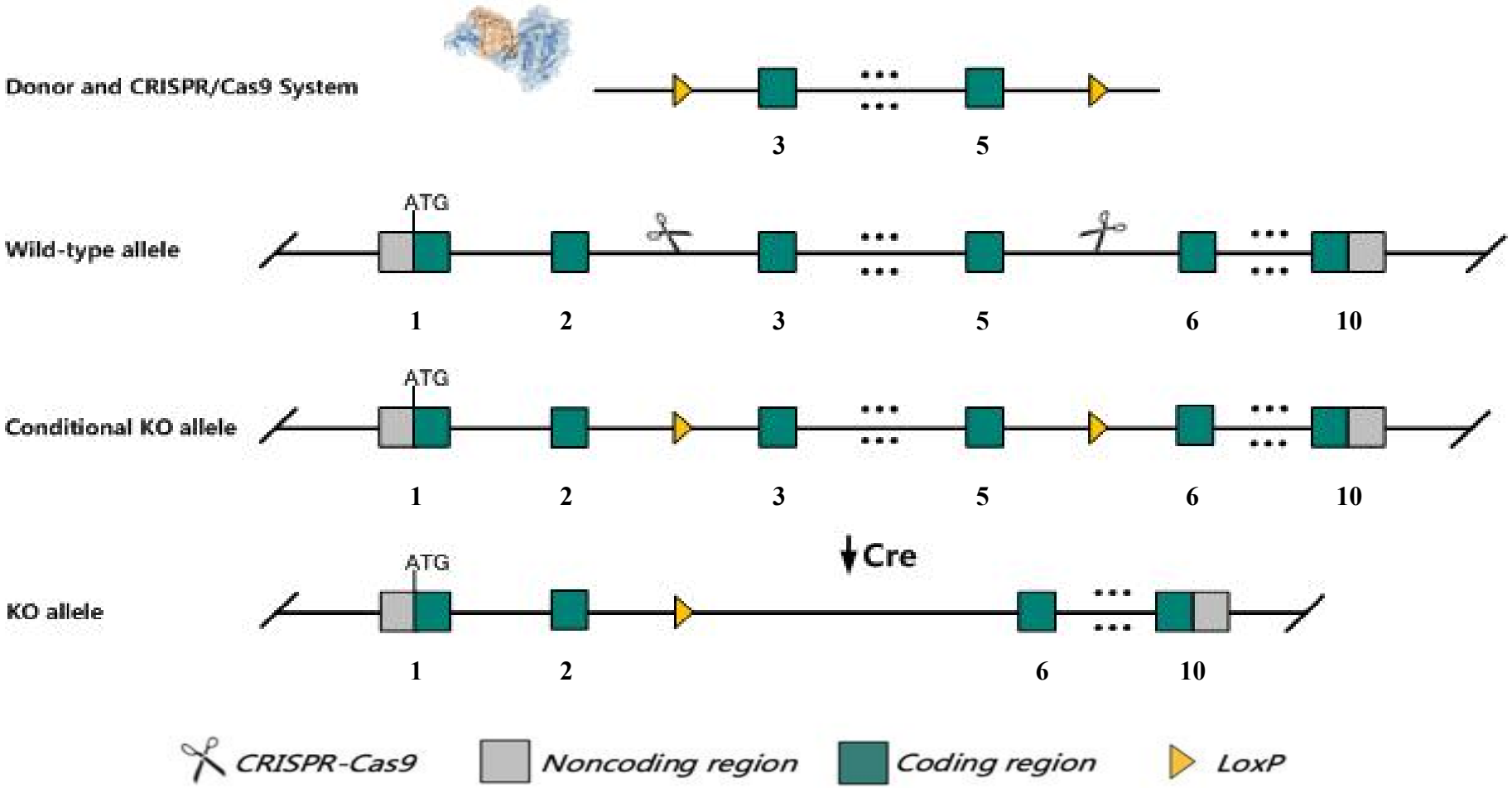
Project Type

- Cas9-CKO

Genetic Background

- C57BL/6JGpt

Strain Strategy



Schematic representation of CRISPR-Cas9 engineering used to edit the *Mmp13* gene.

Technical Information

- The *Mmp13* gene has 1 transcript. According to the structure of *Mmp13* gene, exon3-exon5 of *Mmp13*-201 (ENSMUST00000015394.10) transcript is recommended as the knockout region. The region contains 437bp coding sequence. Knocking out the region will result in disruption of protein function.
- In this project we use CRISPR-Cas9 technology to modify *Mmp13* gene. The brief process is as follows: CRISPR-Cas9 system and Donor 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 on-target 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.
- The flox mice will be knocked out after mating with mice expressing Cre recombinase, resulting in the loss of function of the target gene in specific tissues and cell types.

Gene Information

Mmp13 matrix metallopeptidase 13 [*Mus musculus* (house mouse)]

Download Datasets

Gene ID: 17386, updated on 1-Mar-2024

Summary

Official Symbol	Mmp13 provided by MGI
Official Full Name	matrix metallopeptidase 13 provided by MGI
Primary source	MGI:MGI:1340026
See related	Ensembl:ENSMUSG00000050578 AllianceGenome:MGI:1340026
Gene type	protein coding
RefSeq status	REVIEWED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Clg; Mmp1; MMP-13
Summary	This gene encodes a member of the matrix metalloproteinase family that plays a role in wound healing, skeletal development and bone remodeling. The encoded protein is activated by the removal of an N-terminal activation peptide to generate a zinc-dependent endopeptidase enzyme that can cleave various native collagens, including types I - IV, X and XIV. Mice lacking the encoded protein display profound defects in growth plate cartilage as well as a delay in the endochondral bone development. Lack of the encoded protein also impairs the wound healing process due to reduced keratinocyte migration and vascular density at the wound site. This gene is located in a cluster of other matrix metalloproteinase genes on chromosome 9. [provided by RefSeq, Jun 2015]
Expression	Biased expression in CNS E18 (RPKM 1.3), limb E14.5 (RPKM 0.9) and 13 other tissues See more
Orthologs	human all

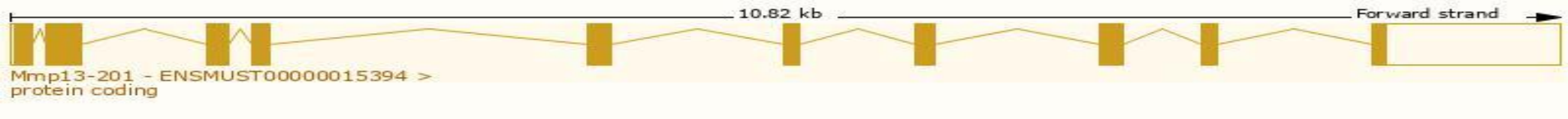
Source: https://www.ncbi.nlm.nih.gov/

Transcript Information

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

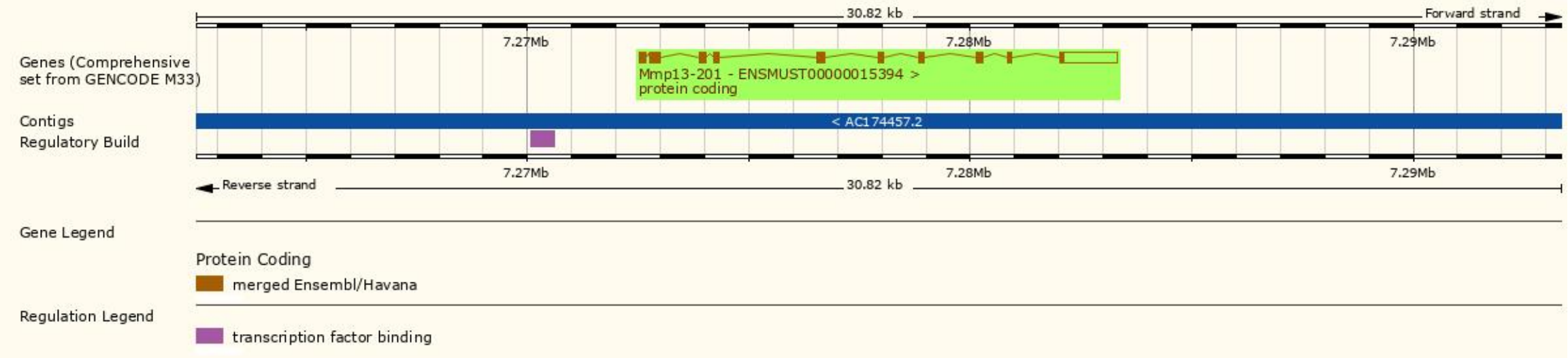
Transcript ID	Name	bp	Protein	Biotype	CCDS	UniProt Match	Flags
ENSMUST00000015394.10	Mmp13-201	2673	472aa	Protein coding	CCDS22803	P33435 Q3U9V5	Ensembl Canonical GENCODE basic APPRIS P1 TSL:1

The strategy is based on the design of *Mmp13*-201 transcript, the transcription is shown below:



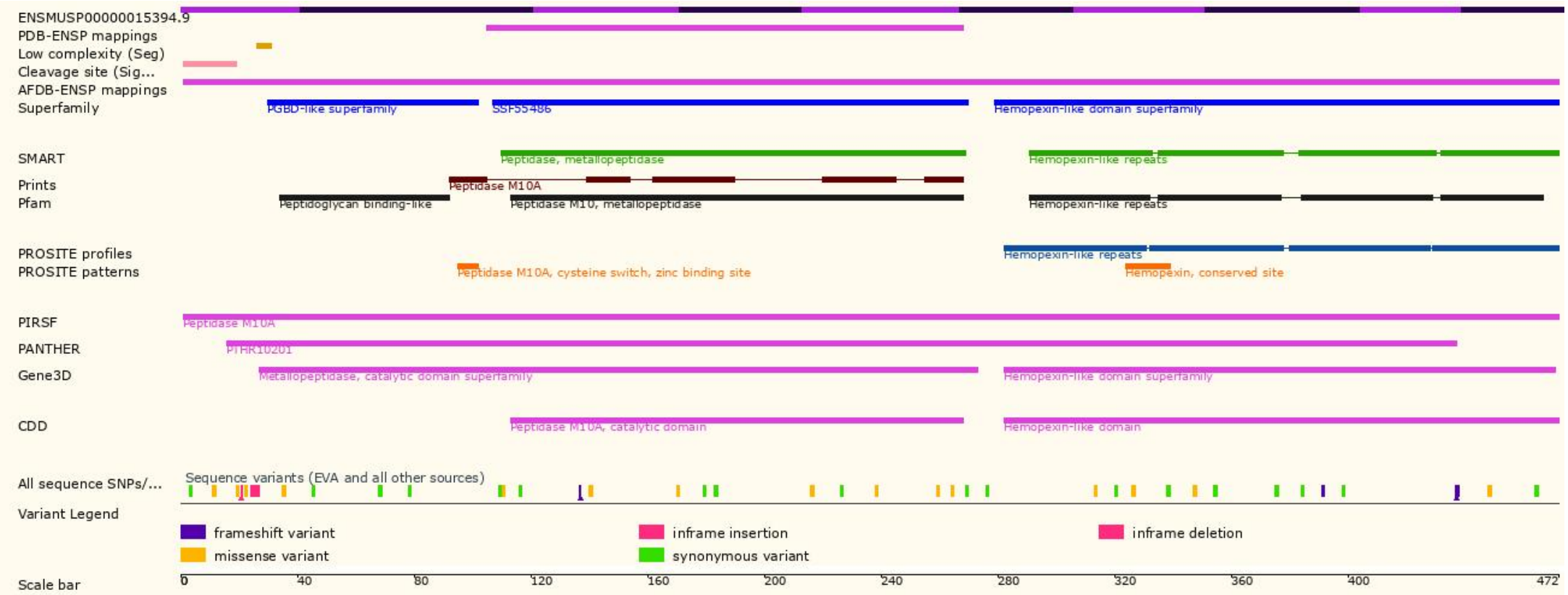
Source: https://www.ensembl.org

Genomic Information



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

Protein Information



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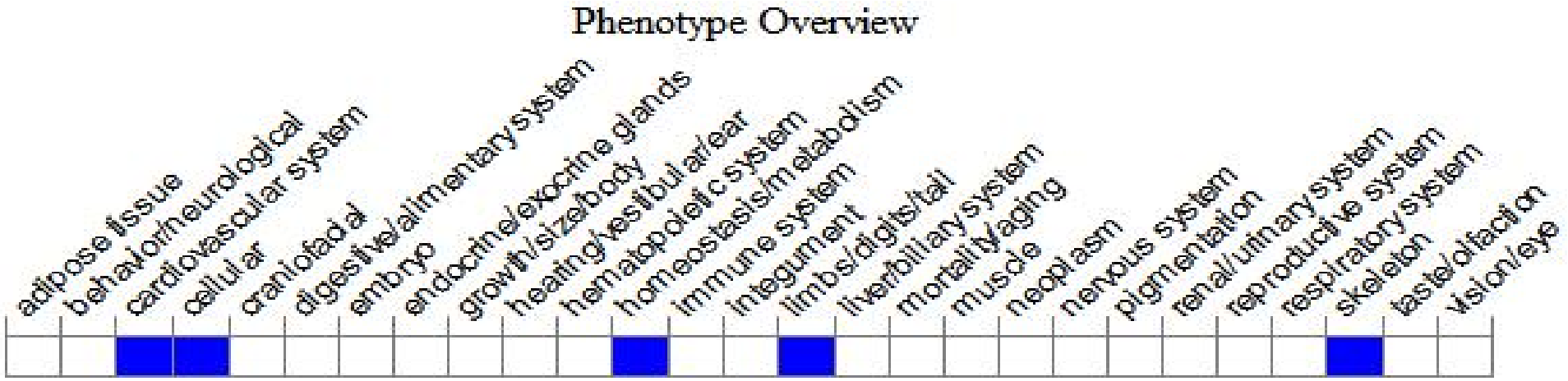
Source: : https://www.ensembl.org

Mouse Phenotype Information (MGI)



- Homozygous null mice display increased width of hypertrophic chondrocyte zone and increased trabecular bone.

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

Important Information

- *Mmp13* is located on Chr9. 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 risk of loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.