

# Map2k1 Cas9-CKO Strategy

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**Design Date:** 2019-11-14

## **Project Overview**



**Project Name** 

Map2k1

**Project type** 

Cas9-CKO

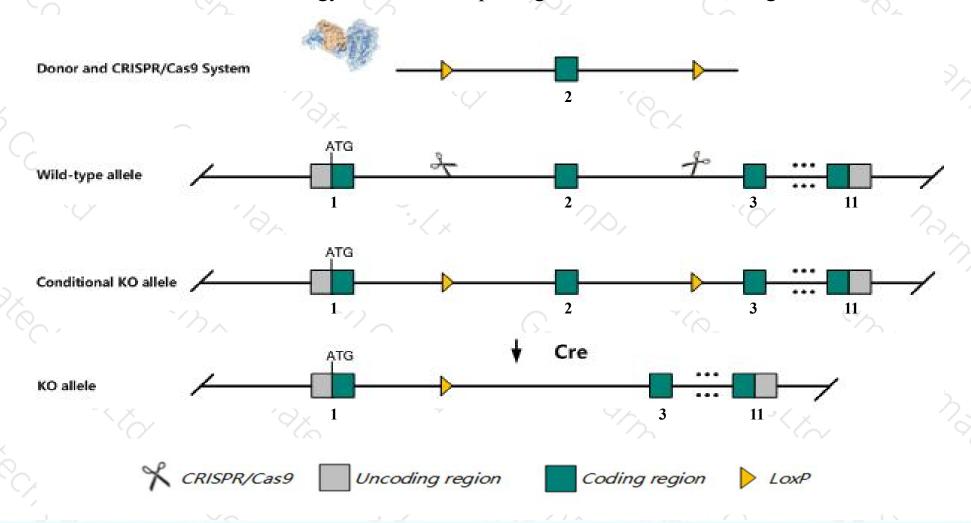
Strain background

C57BL/6JGpt

## Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Map2k1 gene. The schematic diagram is as follows:



### Technical routes



- The *Map2k1* gene has 4 transcripts. According to the structure of *Map2k1* gene, exon2 of *Map2k1-201*(ENSMUST0000005066.8) transcript is recommended as the knockout region. The region contains 211bp coding sequence.

  Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Map2k1* 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 sequencing. A stable F1 generation mouse model was obtained by mating positive F0 generation mice with C57BL/6JGpt mice.
- 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.

### **Notice**



- ➤ According to the existing MGI data, Homozygous inactivation of this gene leads to reduced embryo size and midgestational lethality due to impaired development and hypovascularization of the placenta with decreased labyrinth cell proliferation and enhanced cell apoptosis. Mutant MEFs fail to exhibit fibronectin-induced migration.
- The *Map2k1* gene is located on the Chr9. If the knockout mice are crossed with other mice strains to obtain double gene positive homozygous mouse offspring, please avoid the two genes 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 existing technological level.

## Gene information (NCBI)



#### Map2k1 mitogen-activated protein kinase kinase 1 [Mus musculus (house mouse)]

Gene ID: 26395, updated on 7-Apr-2019

#### Summary

☆ ?

Official Symbol Map2k1 provided by MGI

Official Full Name mitogen-activated protein kinase kinase 1 provided by MGI

Primary source MGI:MGI:1346866

See related Ensembl:ENSMUSG00000004936

Gene type protein coding
RefSeq 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 MAPKK1, MEKK1, Mek1, Prkmk1

Expression Ubiquitous expression in frontal lobe adult (RPKM 50.3), cortex adult (RPKM 32.1) and 27 other tissuesSee more

Orthologs human all

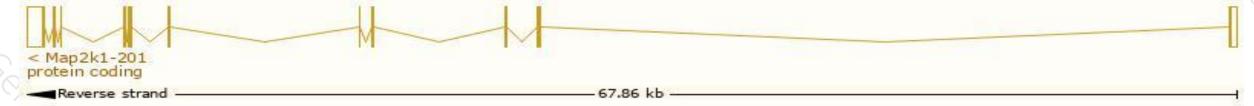
## Transcript information (Ensembl)



The gene has 4 transcripts, all transcripts are shown below:

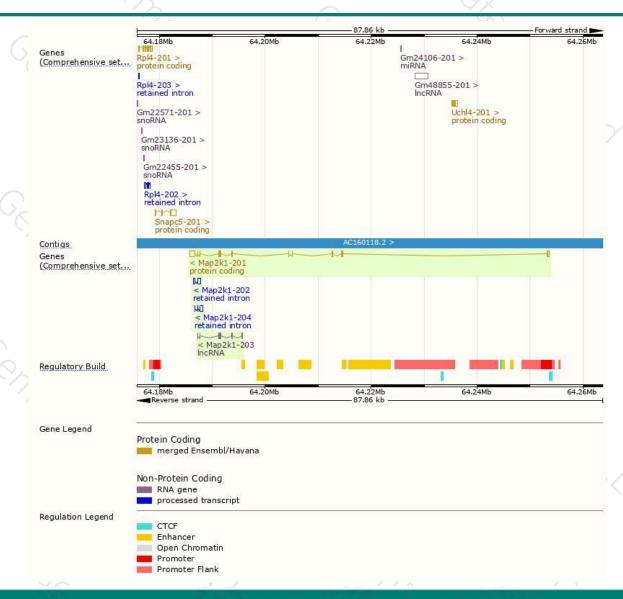
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Map2k1-201	ENSMUST00000005066.8	2436	393aa	Protein coding	CCDS23277	P31938 Q3TMJ8	TSL:1 GENCODE basic APPRIS P1
Map2k1-202	ENSMUST00000147796.1	844	No protein	Retained intron	ē	243	TSL:3
Map2k1-204	ENSMUST00000152675.1	688	No protein	Retained intron		(4)	TSL:2
Map2k1-203	ENSMUST00000152535.1	597	No protein	IncRNA	<u> </u>	-	TSL:3

The strategy is based on the design of Map2k1-201 transcript, The transcription is shown below



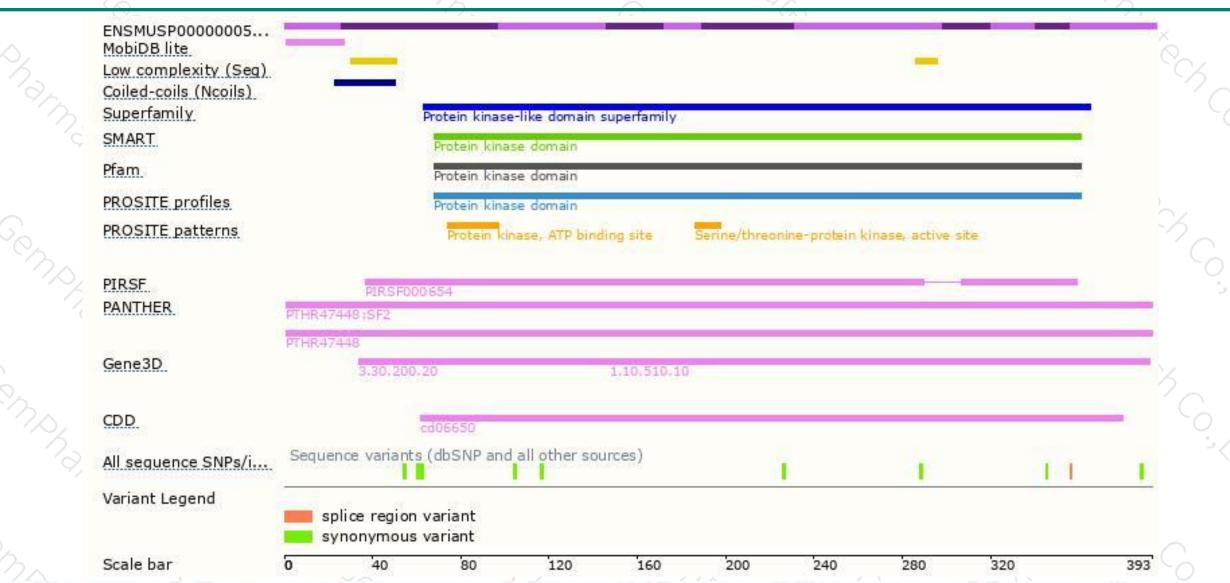
## Genomic location distribution





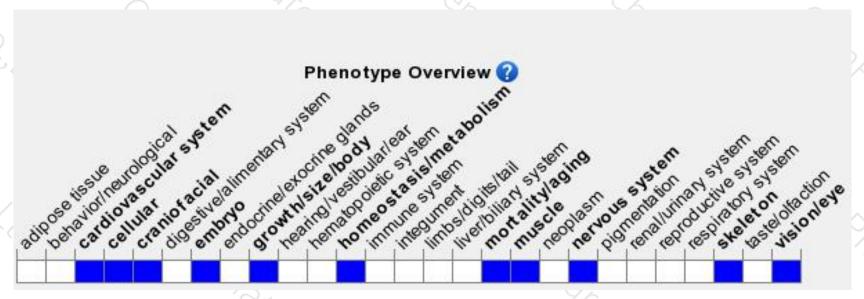
### Protein domain





## Mouse phenotype description(MGI)





Phenotypes affected by the gene are marked in blue.Data quoted from MGI database(http://www.informatics.jax.org/).

According to the existing MGI data, Homozygous inactivation of this gene leads to reduced embryo size and midgestational lethality due to impaired development and hypovascularization of the placenta with decreased labyrinth cell proliferation and enhanced cell apoptosis. Mutant MEFs fail to exhibit fibronectin-induced migration.



If you have any questions, you are welcome to inquire. Tel: 400-9660890





