

Bmper Cas9-KO Strategy

Designer: Huimin Su

Reviewer: Ruiuri Zhang

Design Date: 2020-6-28

Project Overview



Project Name

Bmper

Project type

Cas9-KO

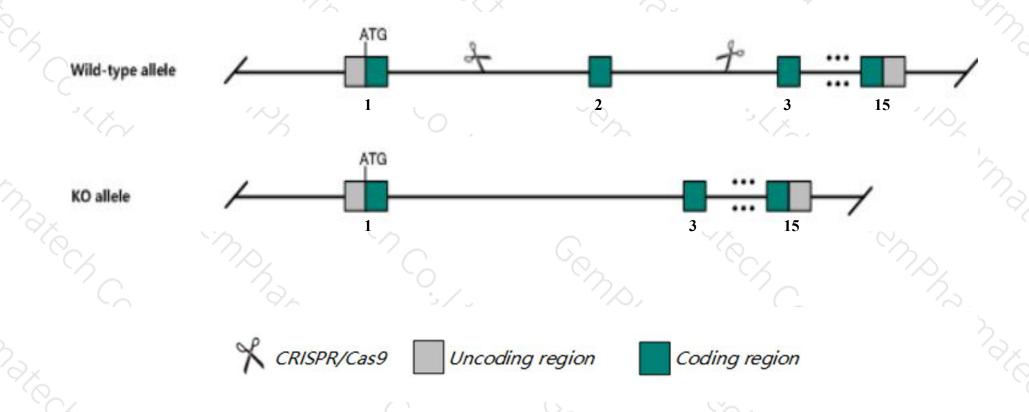
Strain background

C57BL/6JGpt

Knockout strategy



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



Technical routes



- > The *Bmper* gene has 3 transcripts. According to the structure of *Bmper* gene, exon2 of *Bmper-*201(ENSMUST00000071982.6) transcript is recommended as the knockout region. The region contains 86bp coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Bmper* gene. The brief process is as follows: CRISPR/Cas9 system 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.

Notice



- > According to the existing MGI data, mice homozygous for a knock-out mutation exhibit neonatal lethality associated with abnormal lung and skeleton development. Mice heterozygous for a null allele exhibit abnromal lung development.
- ightharpoonup Transcript *Bmper-202* is incomplete, so the effect on it is unknown.
- > The *Bmper* 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 the gene knockout on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

Gene information (NCBI)



Bmper BMP-binding endothelial regulator [Mus musculus (house mouse)]

Gene ID: 73230, updated on 26-Jun-2020

Summary

↑ ?

Official Symbol Bmper provided by MGI

Official Full Name BMP-binding endothelial regulator provided by MGI

Primary source MGI:MGI:1920480

See related Ensembl: ENSMUSG00000031963

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 Cv2; CV-2; Crim3; 3110056H04Rik

Summary This gene encodes a secreted protein that contains five Von Willebrand factor type C domains and a Von Willebrand factor type D domain and a trypsin inhibitory-like

domain. The encoded protein binds to bone morphogenetic proteins (BMP) and regulates their activity. Mutation of the related gene in humans causes

diaphanospondylodysostosis. [provided by RefSeq, Mar 2013]

Expression Broad expression in lung adult (RPKM 4.5), limb E14.5 (RPKM 4.1) and 20 other tissues See more

Orthologs human all

Transcript information (Ensembl)



The gene has 3 transcripts, all transcripts are shown below:

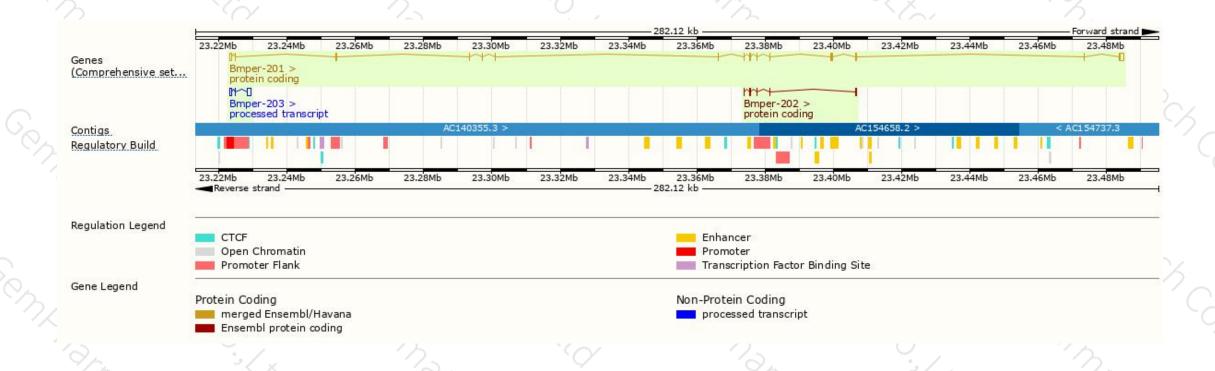
Name 🍦	Transcript ID	bp 🛊	Protein A	Biotype	CCDS	UniProt	Flags
Bmper-202	ENSMUST00000214050.1	640	213aa	Protein coding	-	A0A1L1SS01₽	CDS 5' and 3' incomplete TSL:5
Bmper-201	ENSMUST00000071982.6	3791	685aa	Protein coding	CCDS22929 ₽	Q8CJ69@	TSL:1 GENCODE basic APPRIS P1
Bmper-203	ENSMUST00000217648.1	1616	No protein	Processed transcript	1-0	-	TSL:1

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



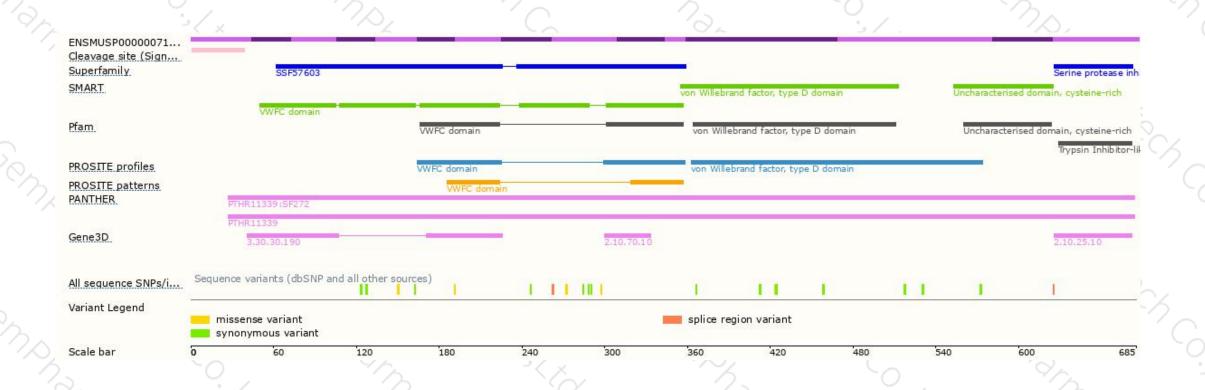
Genomic location distribution





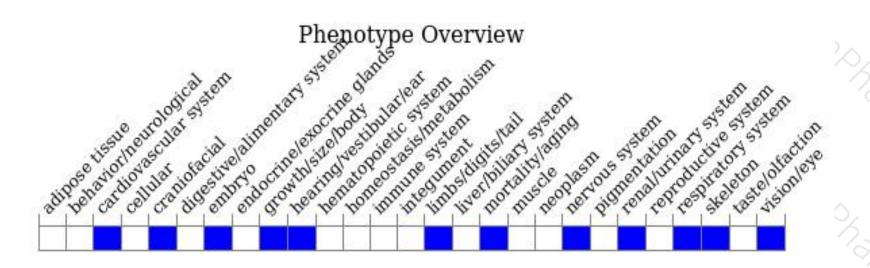
Protein domain





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,mice homozygous for a knock-out mutation exhibit neonatal lethality associated with abnormal lung and skeleton development. Mice heterozygous for a null allele exhibit abnromal lung development.



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





