

Bmp10 Cas9-CKO Strategy

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Project Overview



Project Name

Bmp10

Project type

Cas9-CKO

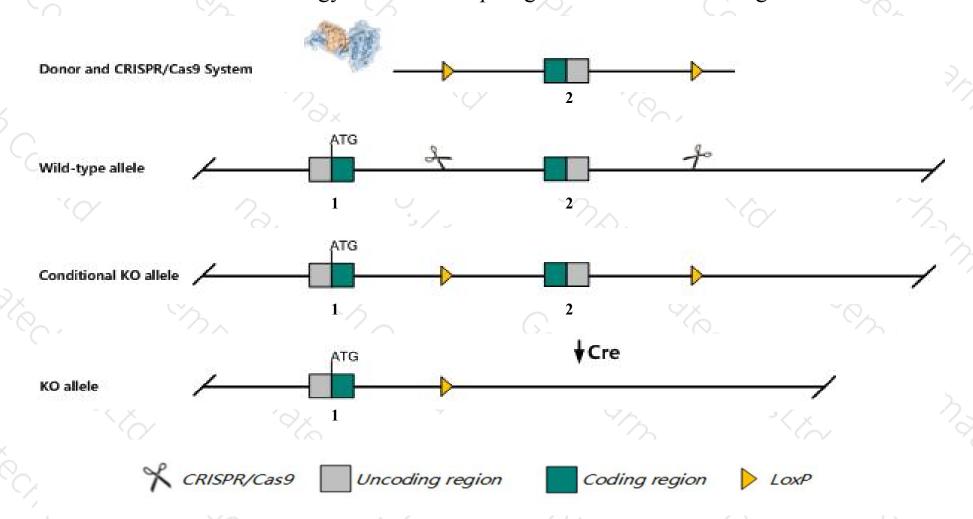
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Bmp10* gene. The schematic diagram is as follows:



Technical routes



- ➤ The *Bmp10* gene has 1 transcript. According to the structure of *Bmp10* gene, exon2 of *Bmp10-201* (ENSMUST00000032125.6) transcript is recommended as the knockout region. The region contains 932bp coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Bmp10* 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 null mice display decreased embryo size, cardiac dysgenesis, defects in early embryonic vascular development, enlarged pericardium, arteriovenous malformations, and embryonic lethality.
- The *Bmp10* gene is located on the Chr6. 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)



Bmp10 bone morphogenetic protein 10 [Mus musculus (house mouse)]

Gene ID: 12154, updated on 13-Mar-2020

Summary

☆ ?

Official Symbol Bmp10 provided by MGI

Official Full Name bone morphogenetic protein 10 provided by MGI

Primary source MGI:MGI:1338820

See related Ensembl: ENSMUSG00000030046

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 b2b2711Clo

Summary This gene encodes a secreted ligand of the TGF-beta (transforming growth factor-beta) superfamily of proteins. Ligands of this family bind

various TGF-beta receptors leading to recruitment and activation of SMAD family transcription factors that regulate gene expression. The encoded preproprotein is proteolytically processed to generate the mature protein, which binds to the activin receptor-like kinase 1 (ALK1) and plays important roles in cardiovascular development including cardiomyocyte proliferation and regulation of heart size, closure of the ductus arteriosus, angiogenesis and ventricular trabeculation. Homozygous knockout mice for this gene exhibit impaired heart development and

embryonic lethality. [provided by RefSeq, Aug 2016]

Expression Biased expression in heart adult (RPKM 23.3) and liver E18 (RPKM 1.3)See more

Orthologs human all

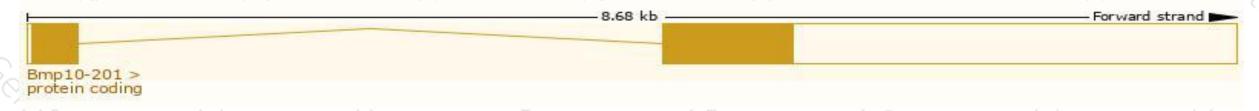
Transcript information (Ensembl)



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

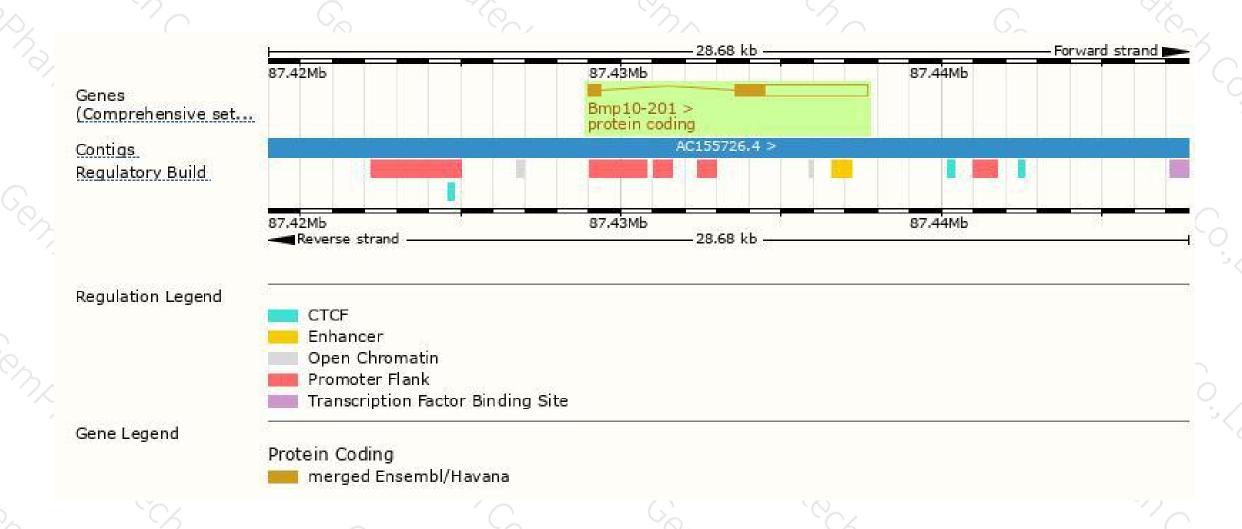
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags	1
Bmp10-201	ENSMUST00000032125.6	4490	421aa	Protein coding	CCDS51838	Q9R229	TSL:1 GENCODE basic APPRIS P1	

The strategy is based on the design of *Bmp10-201* transcript, the transcription is shown below



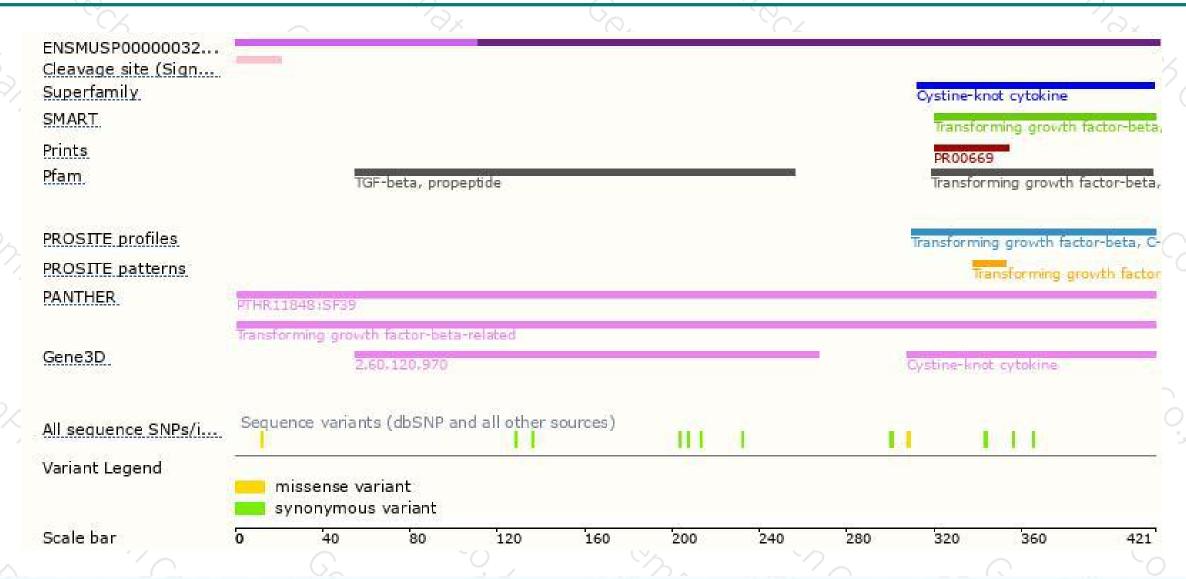
Genomic location distribution





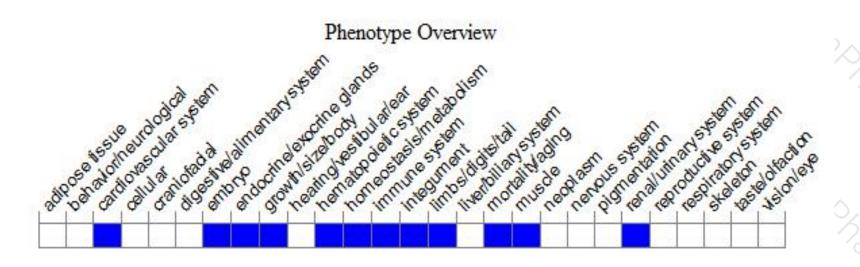
Protein domain





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 null mice display decreased embryo size, cardiac dysgenesis, defects in early embryonic vascular development, enlarged pericardium, arteriovenous malformations, and embryonic lethality.



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





