

Apobec2 Cas9-CKO Strategy

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Reviewer: Ruirui Zhang

Design Date: 2020/2/12

Project Overview



Project Name

Apobec2

Project type

Cas9-CKO

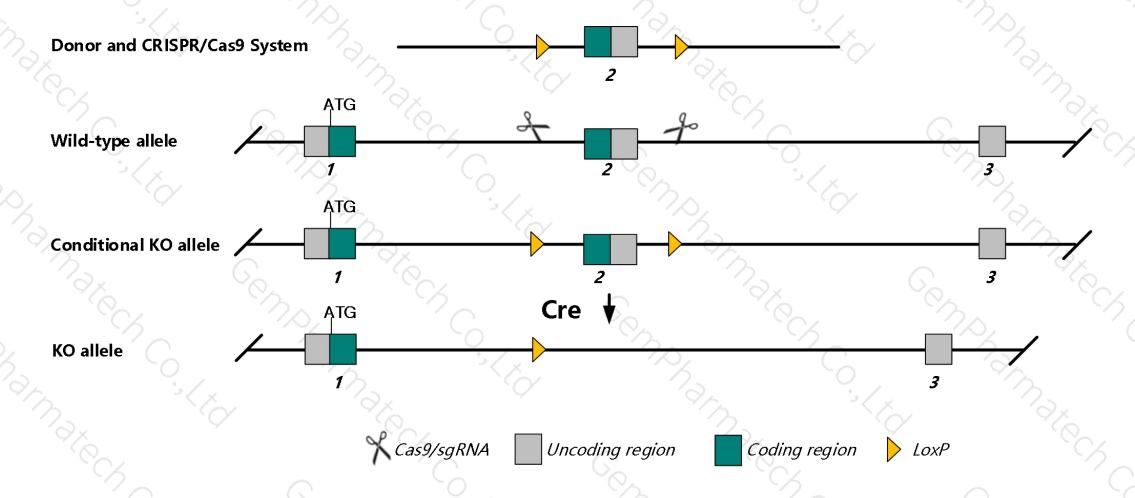
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Apobec2 gene. The schematic diagram is as follows:



Technical routes



- ➤ The *Apobec2* gene has 2 transcripts. According to the structure of *Apobec2* gene, exon2 of *Apobec2-201* (ENSMUST00000046549.4) transcript is recommended as the knockout region. The region contains most of coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Apobec2* 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, Mice homozygous for a gene trapped allele display growth retardation and decreased bone mineralization and density. Mice homozygous for a knockout allele exhibit reduced body mass and a shift in muscle fiber type and develop a mild myopathy as as they age.
- ➤ The *Apobec2* gene is located on the Chr17. 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)



Apobec2 apolipoprotein B mRNA editing enzyme, catalytic polypeptide 2 [Mus musculus (house mouse)]

Gene ID: 11811, updated on 12-Aug-2019

Summary

△ ?

Official Symbol Apobec2 provided by MGI

Official Full Name apolipoprotein B mRNA editing enzyme, catalytic polypeptide 2 provided by MGI

Primary source MGI:MGI:1343178

See related Ensembl: ENSMUSG00000040694

Gene type protein coding
RefSeq status VALIDATED
Organism Mus musculus

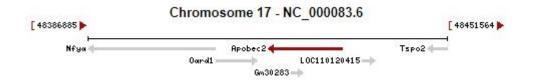
Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia;

Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus

Also known as Arp1

Expression Biased expression in heart adult (RPKM 62.8), mammary gland adult (RPKM 42.7) and 3 other tissues See more

Orthologs human all



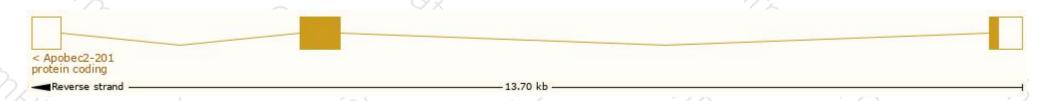
Transcript information (Ensembl)



The gene has 2 transcripts, all transcripts are shown below:

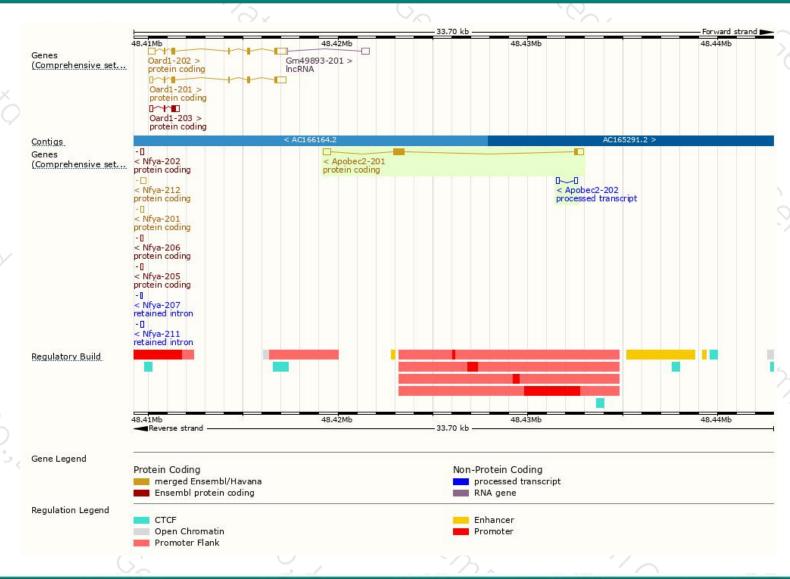
| Name 🌲 | Transcript ID | bp 🌲 | Protein | Biotype | CCDS . | UniProt 4 | Flags | | |
|-------------|----------------------|------|------------|----------------------|-------------|-----------|-------|---------------|-----------|
| Apobec2-201 | ENSMUST00000046549.4 | 1420 | 224aa | Protein coding | CCDS28868 ₽ | Q9WV35₽ | TSL:1 | GENCODE basic | APPRIS P1 |
| Apobec2-202 | ENSMUST00000233707.1 | 317 | No protein | Processed transcript | 120 | 5 | | (2) | |

The strategy is based on the design of Apobec2-201 transcript, The transcription is shown below



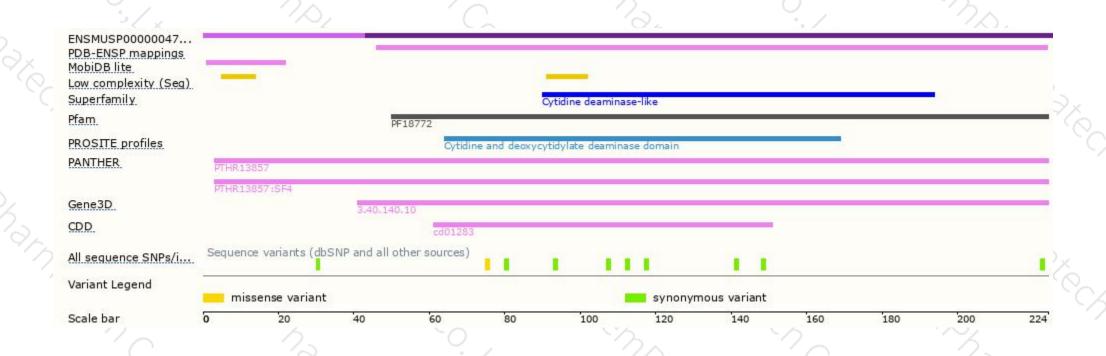
Genomic location distribution





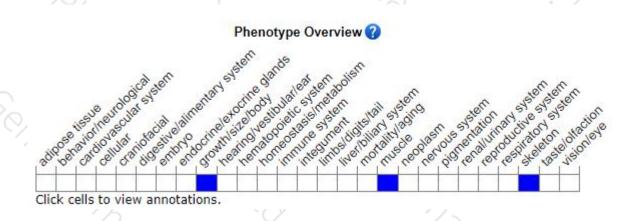
Protein domain





Mouse phenotype description(MGI)





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

Mice homozygous for a gene trapped allele display growth retardation and decreased bone mineralization and density. Mice homozygous for a knockout allele exhibit reduced body mass and a shift in muscle fiber type and develop a mild myopathy as as they age.



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





