

# Fabp3 Cas9-CKO Strategy

Designer:Xueting Zhang

Reviewer: Yanhua Shen

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## **Project Overview**



**Project Name** 

Fabp3

**Project type** 

Cas9-CKO

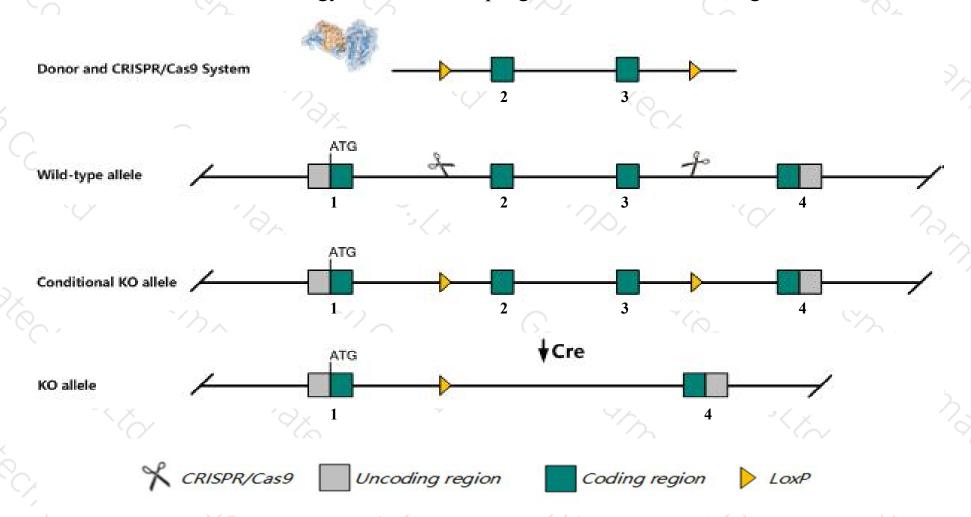
Strain background

C57BL/6JGpt

## Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Fabp3 gene. The schematic diagram is as follows:



### Technical routes



- ➤ The *Fabp3* gene has 1 transcript. According to the structure of *Fabp3* gene, exon2-exon3 of *Fabp3-201*(ENSMUST00000070532.7) transcript is recommended as the knockout region. The region contains 275bp coding sequence.

  Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Fabp3* 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, Inactivation of this locus results in impaired fatty acid utilization. Homozygous null mice show exercise intolerance and cardiac hypertrophy.
- The floxed region is near to the N-terminal of Gm10570 and C-terminal of Zcchc17 gene, this strategy may influence the regulatory function of the N-terminal of Gm10570 and C-terminal of Zcchc17 gene.
- The *Fabp3* gene is located on the Chr4. 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)



#### Fabp3 fatty acid binding protein 3, muscle and heart [ Mus musculus (house mouse) ]

Gene ID: 14077, updated on 12-Oct-2019

#### Summary

☆ ?

Official Symbol Fabp3 provided by MGI

Official Full Name fatty acid binding protein 3, muscle and heart provided by MGI

Primary source MGI:MGI:95476

See related Ensembl: ENSMUSG00000028773

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 Mdgi; Fabph1; Fabph4; H-FABP; Fabph-1; Fabph-4

Expression Biased expression in heart adult (RPKM 2334.3) and placenta adult (RPKM 178.9) See more

Orthologs human all

#### Genomic context



**Location:** 4 D2.2; 4 63.43 cM

See Fabp3 in Genome Data Viewer

Exon count: 4

Annotation release	Status	Assembly	Chr	Location	
108	current	GRCm38.p6 (GCF_000001635.26)	4	NC_000070.6 (130308738130315463)	
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	4	NC_000070.5 (129986022129992707)	

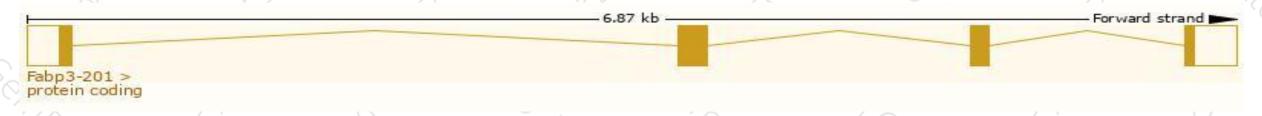
## Transcript information (Ensembl)



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

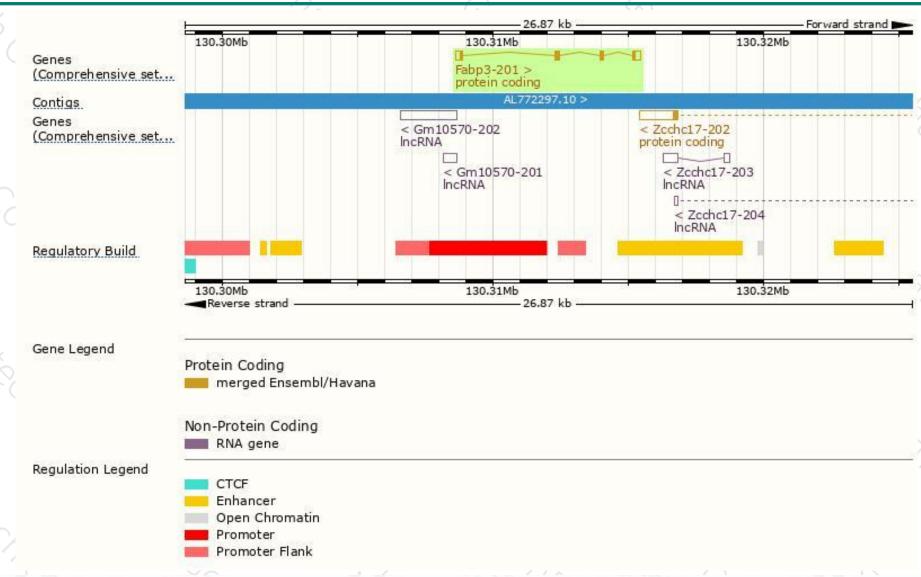
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags	l
Fabp3-201	ENSMUST00000070532.7	823	<u>133aa</u>	Protein coding	CCDS18709	P11404 Q5EBJ0	TSL:1 GENCODE basic APPRIS P1	Ľ

The strategy is based on the design of Fabp3-201 transcript, The transcription is shown below



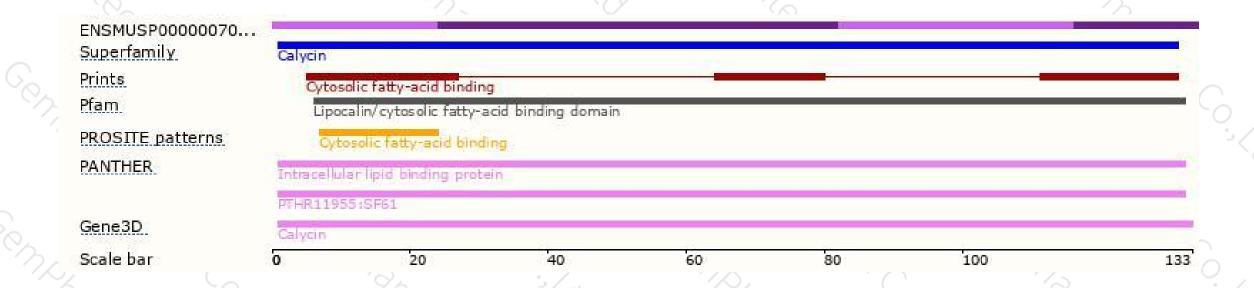
### Genomic location distribution





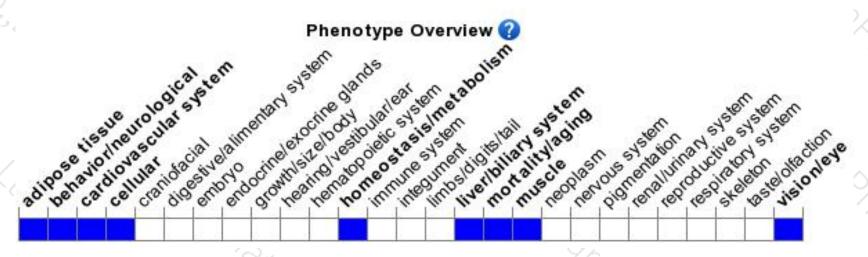
### Protein domain





## 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, Inactivation of this locus results in impaired fatty acid utilization. Homozygous null mice show exercise intolerance and cardiac hypertrophy.



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





