

Abhd5 Cas9-CKO Strategy

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Project Overview

Project Name

Abhd5

Project type

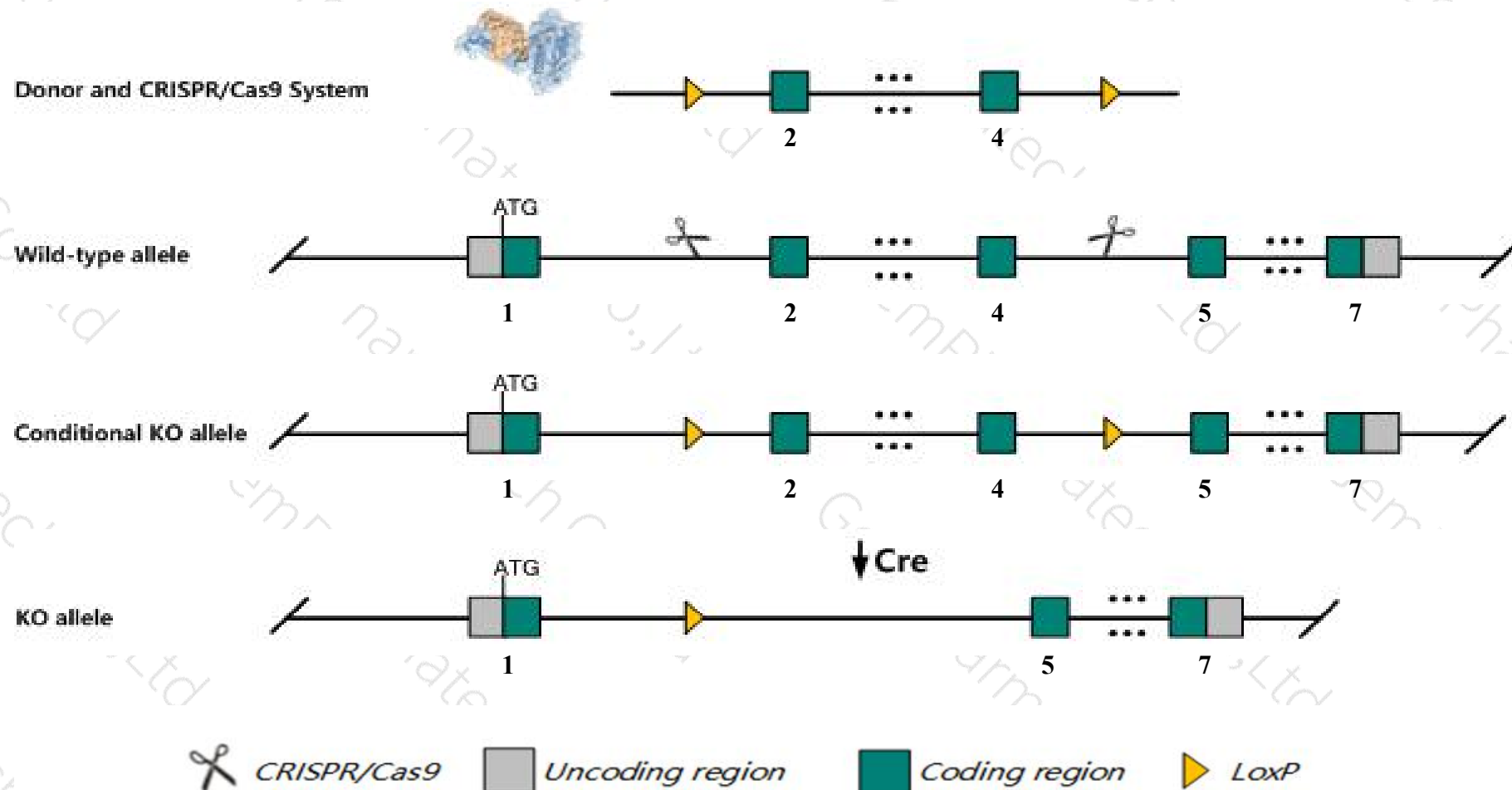
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Abhd5* gene. The schematic diagram is as follows:



- The *Abhd5* gene has 8 transcripts. According to the structure of *Abhd5* gene, exon2-exon4 of *Abhd5*-204 (ENSMUST00000156520.7) transcript is recommended as the knockout region. The region contains 614bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Abhd5* 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.

- According to the existing MGI data, mice homozygous for a knock-out allele exhibit growth retardation, impaired triacylglycerol catabolism, hepatic steatosis, and lethal skin barrier defect.
- The *Abhd5* 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.
- Transcripts 206,207 may not be affected.

Gene information (NCBI)

Abhd5 abhydrolase domain containing 5 [Mus musculus (house mouse)]

Gene ID: 67469, updated on 13-Mar-2020

Summary



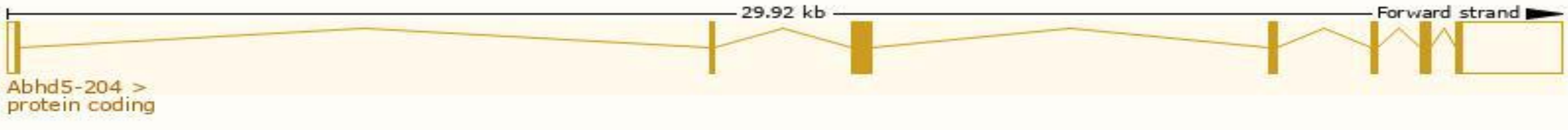
Official Symbol	Abhd5 provided by MGI
Official Full Name	abhydrolase domain containing 5 provided by MGI
Primary source	MGI:MGI:1914719
See related	Ensembl:ENSMUSG00000032540
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	1300003D03Rik, 2010002J10Rik, CGI-58, IECN5, NCIE2
Expression	Broad expression in testis adult (RPKM 60.2), liver E18 (RPKM 34.9) and 23 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

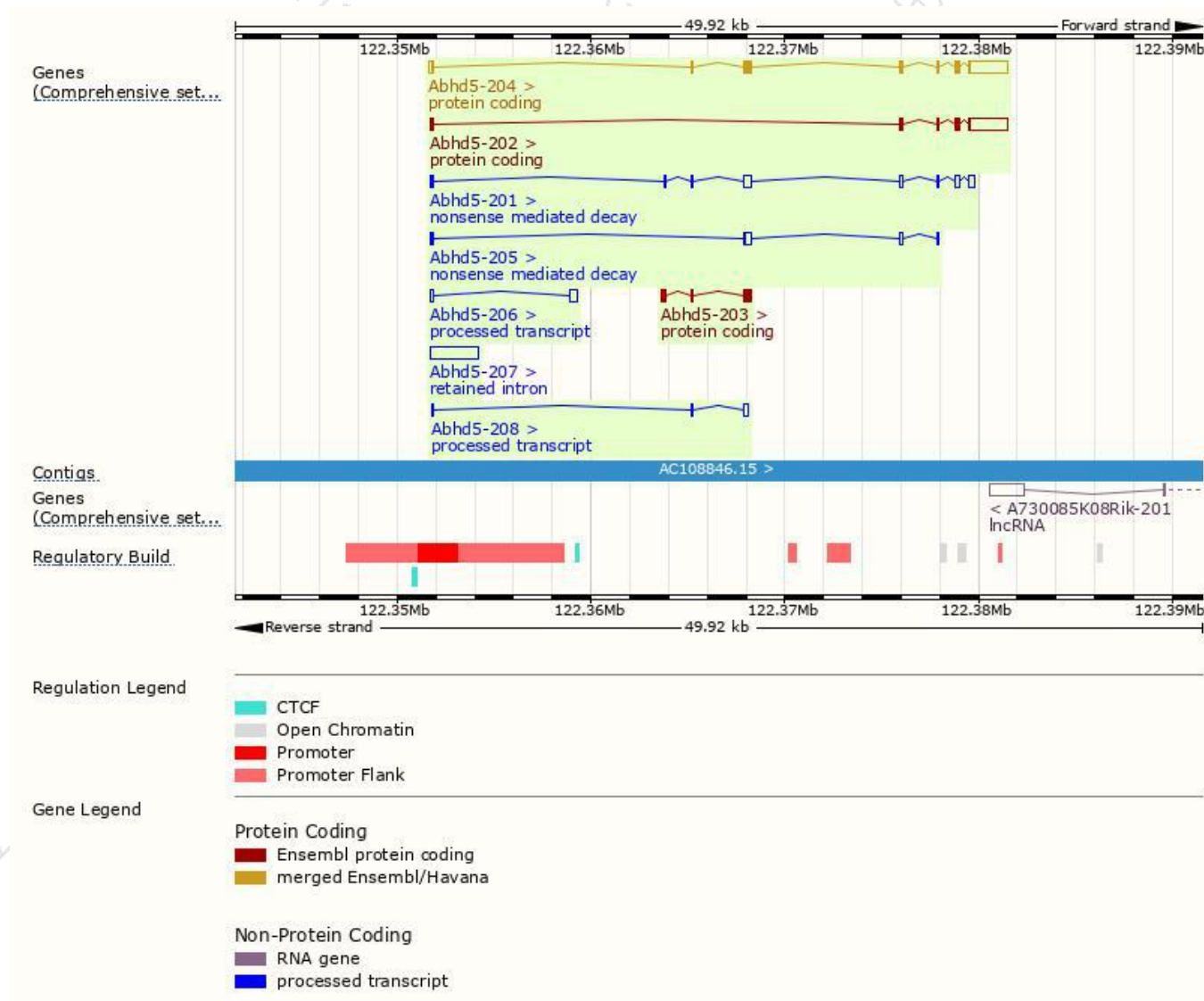
The gene has 8 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Abhd5-204	ENSMUST00000156520.7	3157	351aa	Protein coding	CCDS23646	Q9DBL9	TSL:1 GENCODE basic APPRIS P1
Abhd5-202	ENSMUST00000111497.4	2576	198aa	Protein coding	-	Q9DBL9	TSL:1 GENCODE basic
Abhd5-203	ENSMUST00000154161.2	561	127aa	Protein coding	-	D3Z7K3	CDS 3' incomplete TSL:3
Abhd5-201	ENSMUST00000035128.10	1386	38aa	Nonsense mediated decay	-	H3BKV1	TSL:1
Abhd5-205	ENSMUST00000175973.1	710	46aa	Nonsense mediated decay	-	H3BLJ0	TSL:2
Abhd5-206	ENSMUST00000176005.1	467	No protein	Processed transcript	-	-	TSL:1
Abhd5-208	ENSMUST00000216775.1	387	No protein	Processed transcript	-	-	TSL:3
Abhd5-207	ENSMUST00000214061.1	2417	No protein	Retained intron	-	-	TSL:NA

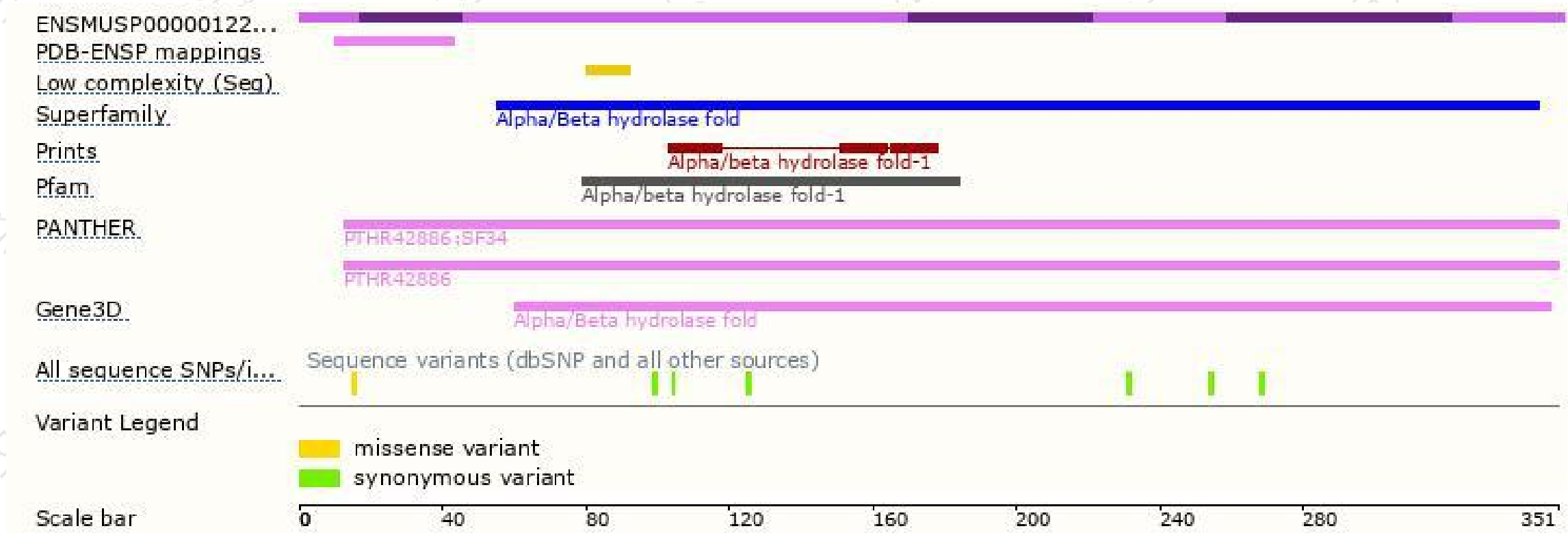
The strategy is based on the design of *Abhd5-204* transcript,the transcription is shown below:



Genomic location distribution

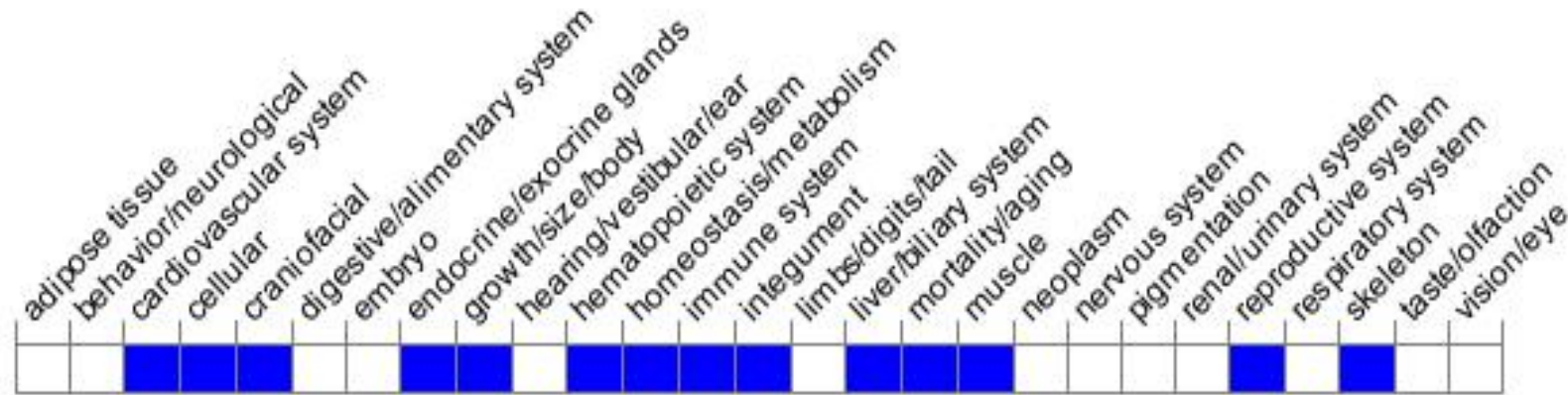


Protein domain



Mouse phenotype description(MGI)

Phenotype Overview



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 allele exhibit growth retardation, impaired triacylglycerol catabolism, hepatic steatosis, and lethal skin barrier defect.

If you have any questions, you are welcome to inquire.

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