

Atp5a1 Cas9-CKO Strategy

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Project Overview

Project Name

Atp5a1

Project type

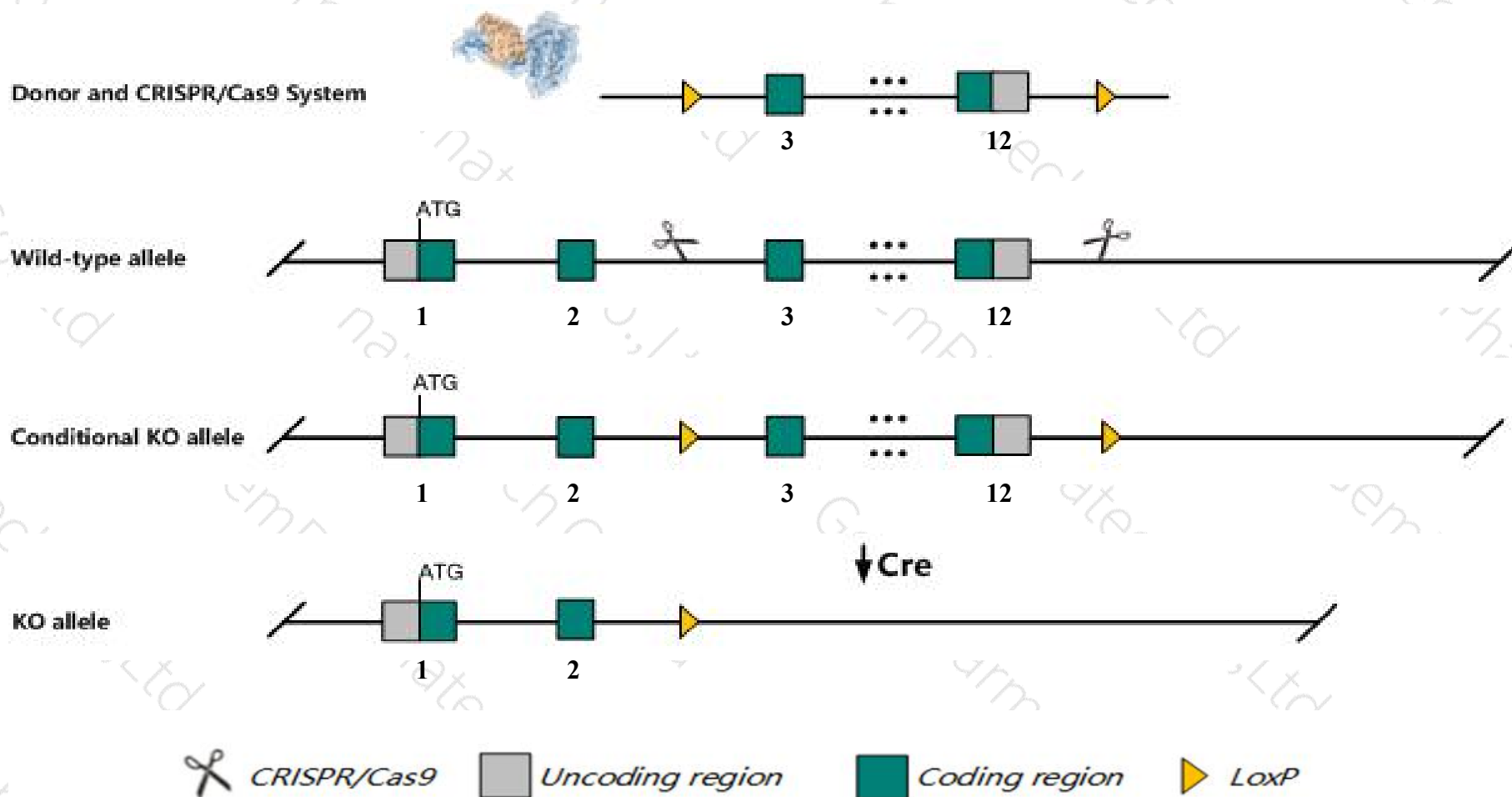
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Atp5a1* gene. The schematic diagram is as follows:



Technical routes

- The *Atp5a1* gene has 5 transcripts. According to the structure of *Atp5a1* gene, exon3-exon12 of *Atp5a1-201*(ENSMUST00000026495.14) transcript is recommended as the knockout region. The region contains 1523bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Atp5a1* 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 carrying a targeted mutation of this gene display preweaning and embryonic lethality. Heterozygous mutants exhibit decreased body weight and lean body mass and reduced circulating insulin, serum albumin, and total protein levels.
- The *Atp5a1* gene is located on the Chr18. 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)

Atp5a1 ATP synthase, H⁺ transporting, mitochondrial F1 complex, alpha subunit 1 [Mus musculus (house mouse)]

Gene ID: 11946, updated on 13-Mar-2020

Summary

Official Symbol Atp5a1 provided by [MGI](#)

Official Full Name ATP synthase, H⁺ transporting, mitochondrial F1 complex, alpha subunit 1 provided by [MGI](#)

Primary source [MGI:MGI:88115](#)

See related [Ensembl:ENSMUSG00000025428](#)

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 AI035633, AL022851, AL023067, Atp5f1a, Atpm, D18Ertd206e, Mom2

Expression Ubiquitous expression in heart adult (RPKM 1425.7), kidney adult (RPKM 741.8) and 28 other tissues [See more](#)

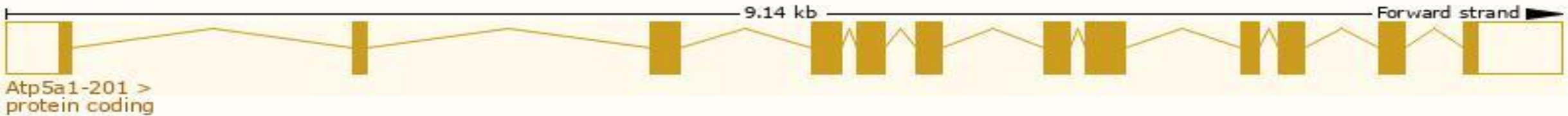
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

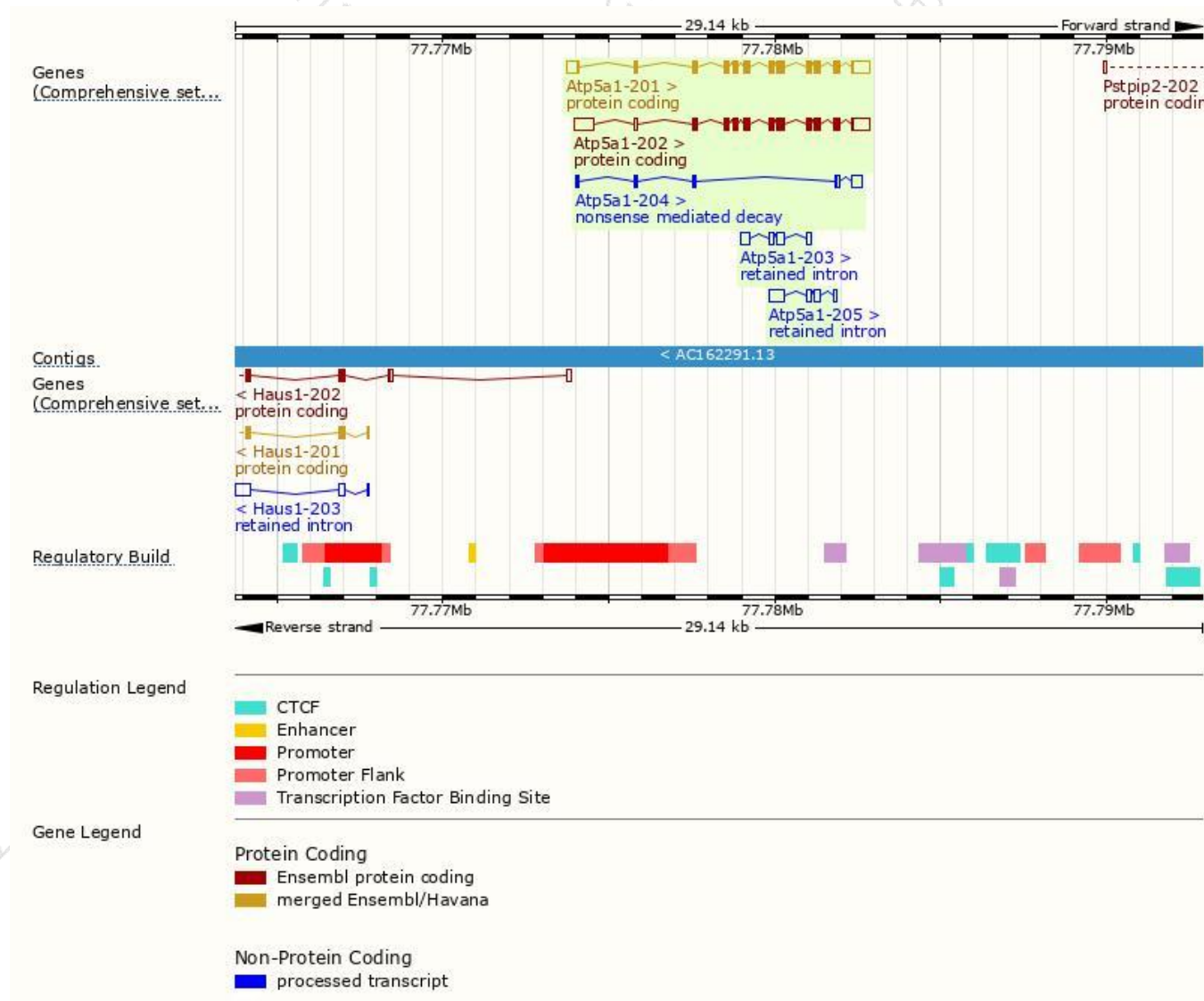
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Atp5a1-201	ENSMUST00000026495.14	2471	553aa	Protein coding	CCDS29358	Q03265	TSL:1 GENCODE basic APPRIS P1
Atp5a1-202	ENSMUST00000114748.1	2674	503aa	Protein coding	-	D3Z6F5	TSL:1 GENCODE basic
Atp5a1-204	ENSMUST00000135678.1	774	89aa	Nonsense mediated decay	-	D6RJ16	TSL:5
Atp5a1-205	ENSMUST00000146869.1	816	No protein	Retained intron	-	-	TSL:2
Atp5a1-203	ENSMUST00000128073.1	765	No protein	Retained intron	-	-	TSL:2

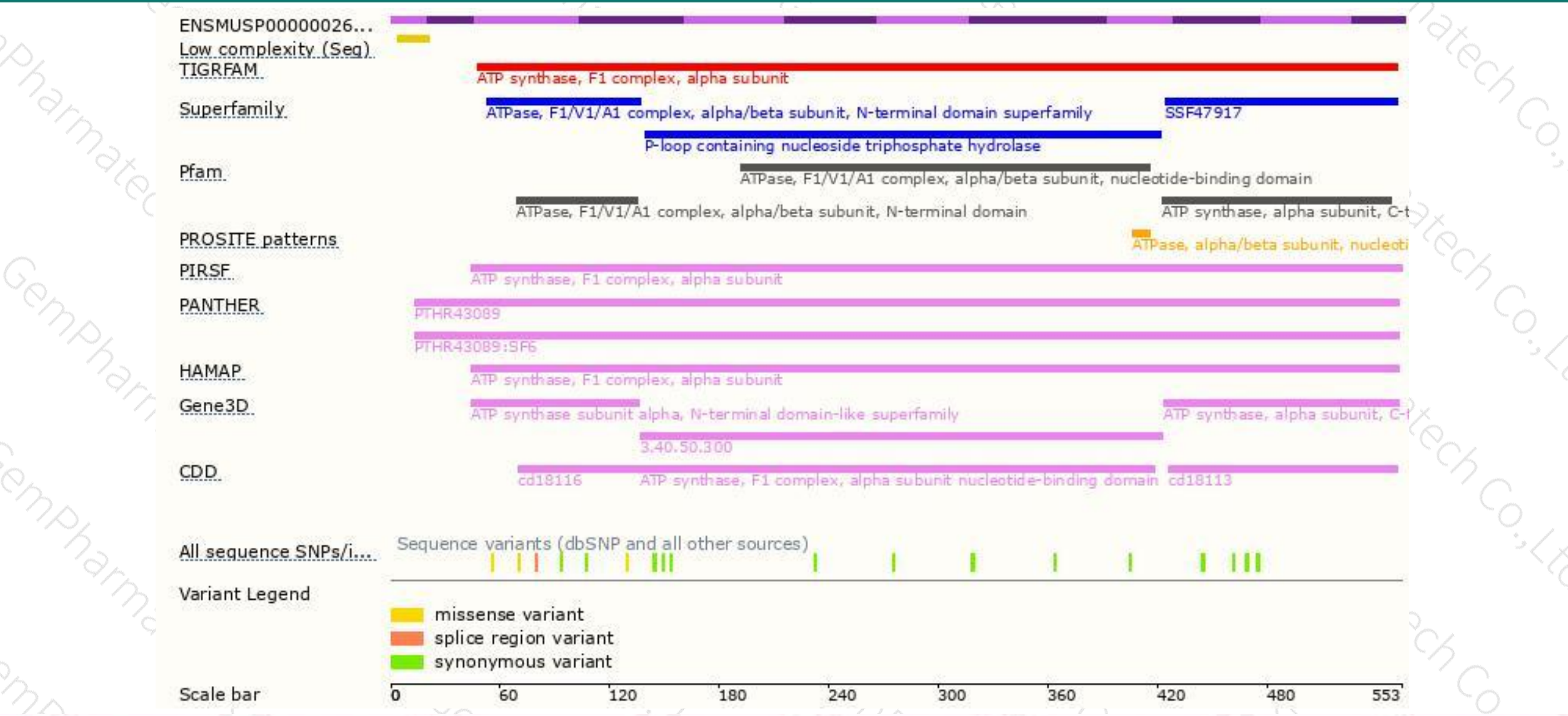
The strategy is based on the design of *Atp5a1-201* transcript,the transcription is shown below:



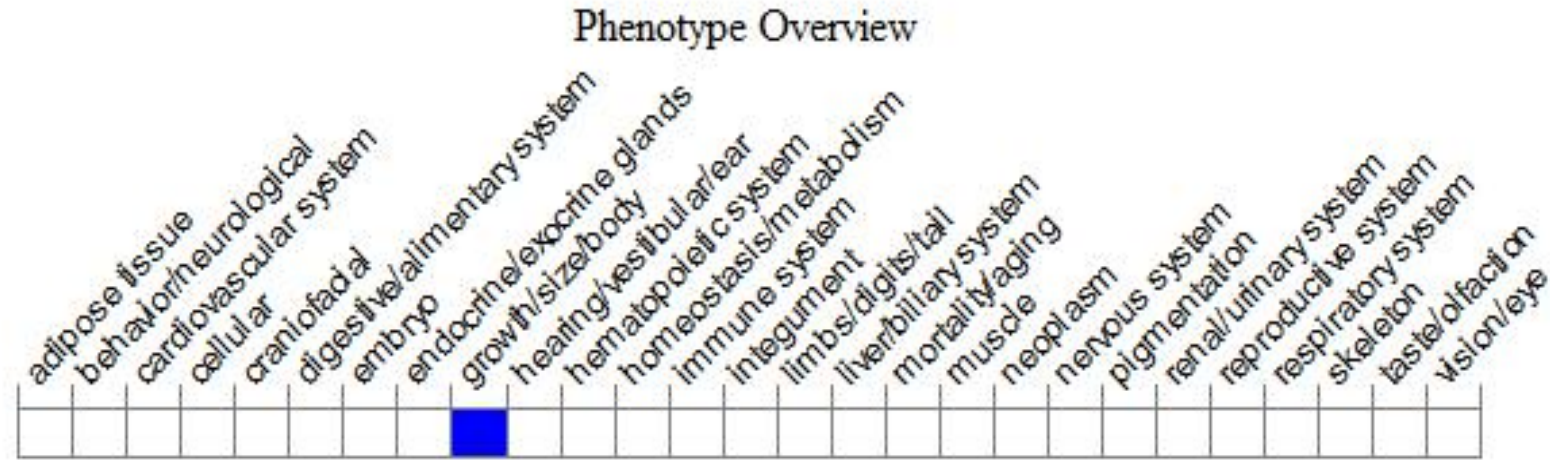
Genomic location distribution



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 carrying a targeted mutation of this gene display preweaning and embryonic lethality. Heterozygous mutants exhibit decreased body weight and lean body mass and reduced circulating insulin, serum albumin, and total protein levels.

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

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