

Atp6v1h Cas9-CKO Strategy

Designer:

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Project Overview

Project Name

Atp6v1h

Project type

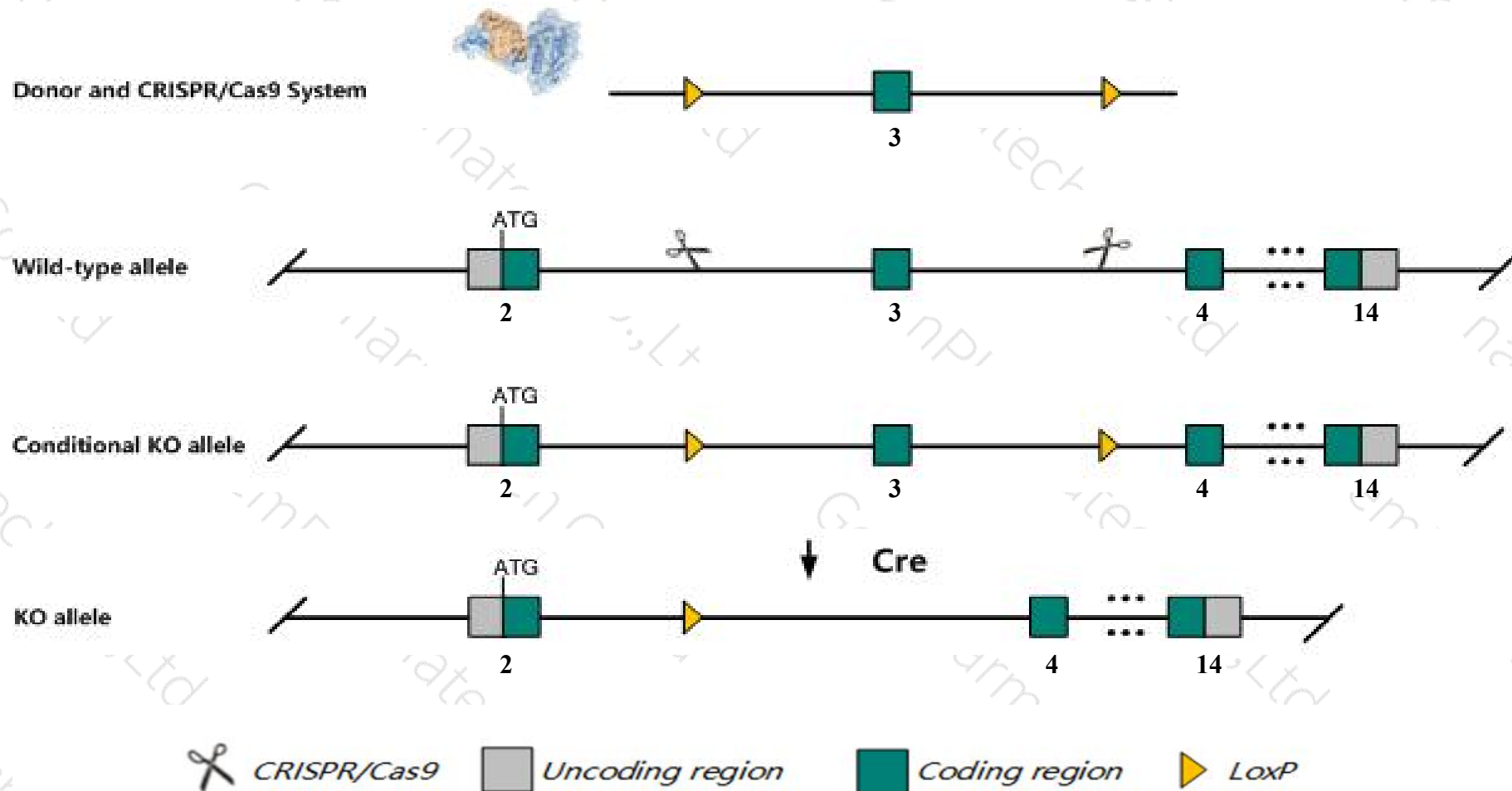
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Atp6v1h* gene. The schematic diagram is as follows:



- The *Atp6v1h* gene has 8 transcripts. According to the structure of *Atp6v1h* gene, exon3 of *Atp6v1h-201* (ENSMUST00000044369.12) transcript is recommended as the knockout region. The region contains 103bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Atp6v1h* 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 embryonic lethality. Mice heterozygous for the allele exhibit bone loss with altered bone absorption and decreased bone formation.
- The *Atp6v1h* gene is located on the Chr1. 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)

Atp6v1h ATPase, H⁺ transporting, lysosomal V1 subunit H [Mus musculus (house mouse)]

Gene ID: 108664, updated on 7-Apr-2019

Summary



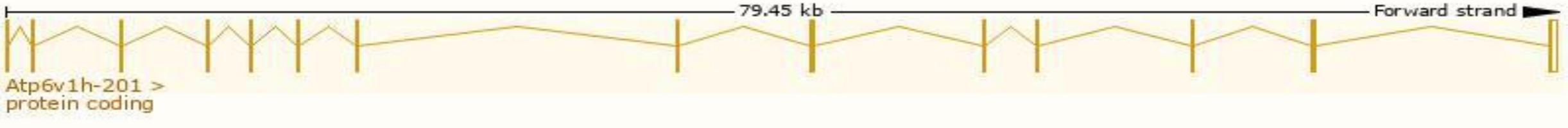
Official Symbol	Atp6v1h provided by MGI
Official Full Name	ATPase, H ⁺ transporting, lysosomal V1 subunit H provided by MGI
Primary source	MGI:MGI:1914864
See related	Ensembl:ENSMUSG00000033793
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	0710001F19Rik, AU022349, CGI-11, SFD, SFDalpha, SFDbeta, VMA13
Expression	Ubiquitous expression in placenta adult (RPKM 22.7), CNS E18 (RPKM 15.8) and 28 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

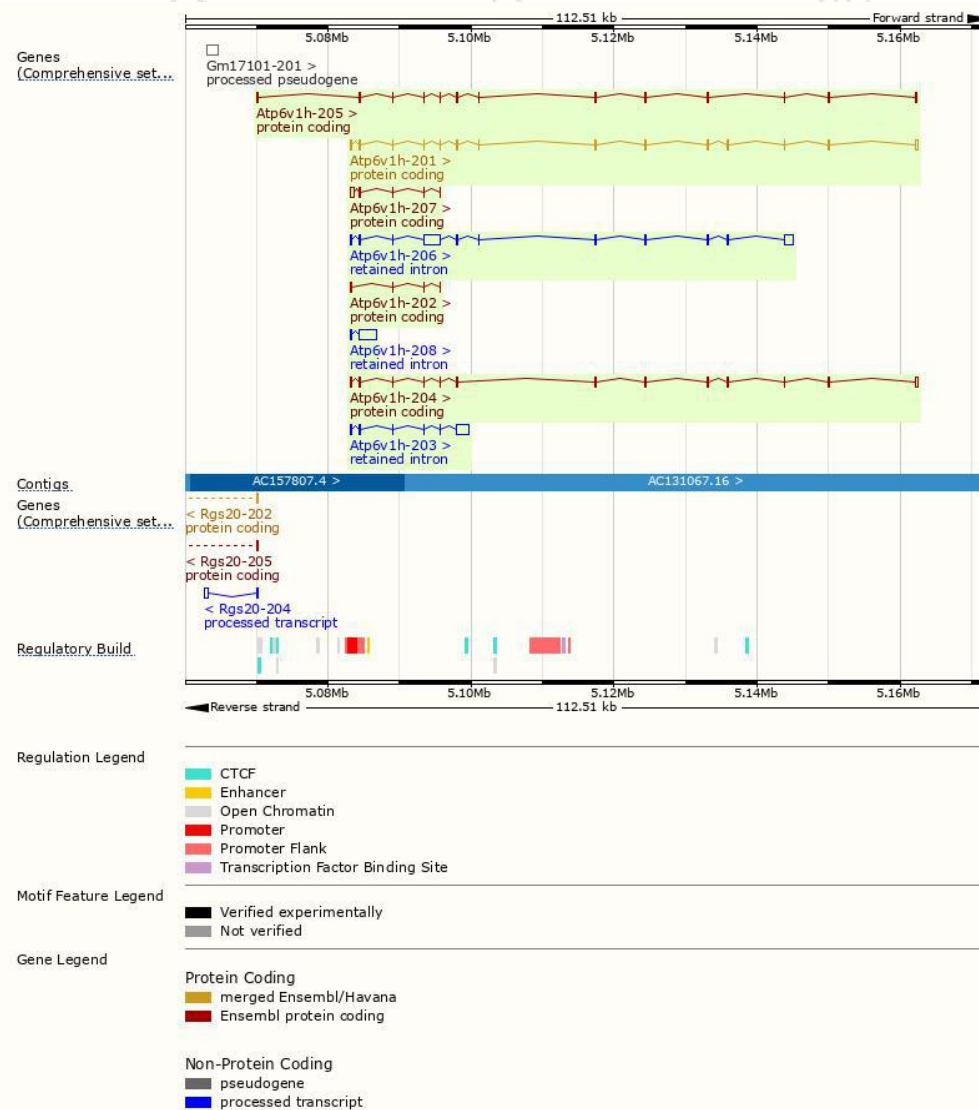
The gene has 8 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Atp6v1h-201	ENSMUST00000044369.12	2049	483aa	Protein coding	CCDS14808	Q8BVE3	TSL:1 GENCODE basic APPRIS P3
Atp6v1h-204	ENSMUST00000192698.2	1811	465aa	Protein coding	CCDS78541	A0A0A6YX18	TSL:5 GENCODE basic APPRIS ALT 1
Atp6v1h-205	ENSMUST00000192847.5	1662	441aa	Protein coding	-	A0A0A6YWP6	TSL:5 GENCODE basic
Atp6v1h-207	ENSMUST00000194676.5	970	140aa	Protein coding	-	A0A0A6YVU0	CDS 3' incomplete TSL:3
Atp6v1h-202	ENSMUST00000192029.5	385	77aa	Protein coding	-	A0A0A6YW86	CDS 3' incomplete TSL:3
Atp6v1h-206	ENSMUST00000194301.5	4829	No protein	Retained intron	-	-	TSL:5
Atp6v1h-208	ENSMUST00000194978.5	2510	No protein	Retained intron	-	-	TSL:1
Atp6v1h-203	ENSMUST00000192142.1	2318	No protein	Retained intron	-	-	TSL:1

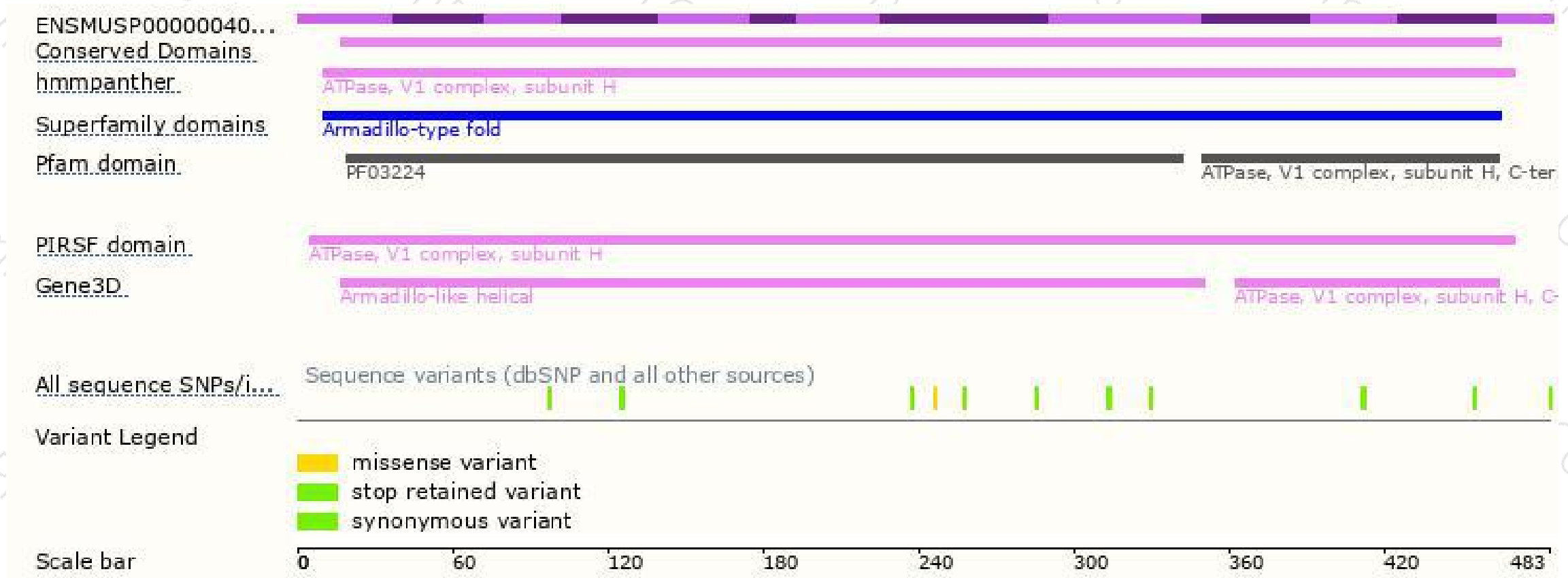
The strategy is based on the design of *Atp6v1h-201* transcript,The transcription is shown below



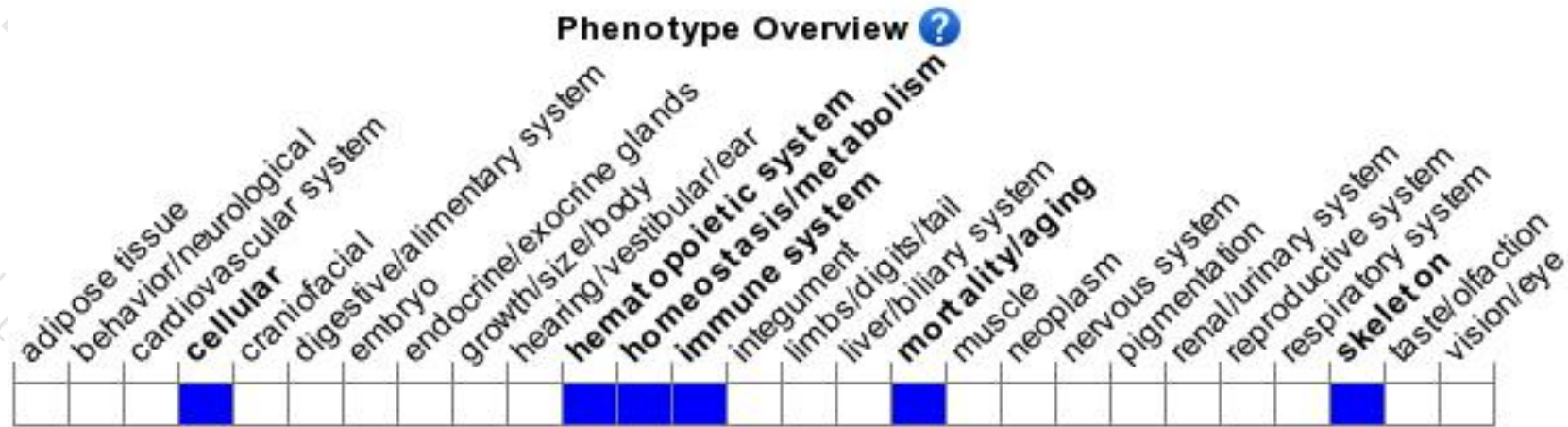
Genomic location distribution



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 homozygous for a knock-out allele exhibit embryonic lethality. Mice heterozygous for the allele exhibit bone loss with altered bone absorption and decreased bone formation.

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

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