

# Acpl Cas9-CKO Strategy

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Design Date: 2020-1-22

Reviewer: JiaYu

## **Project Overview**



**Project Name** 

**Project type** 

Cas9-CKO

Acp1

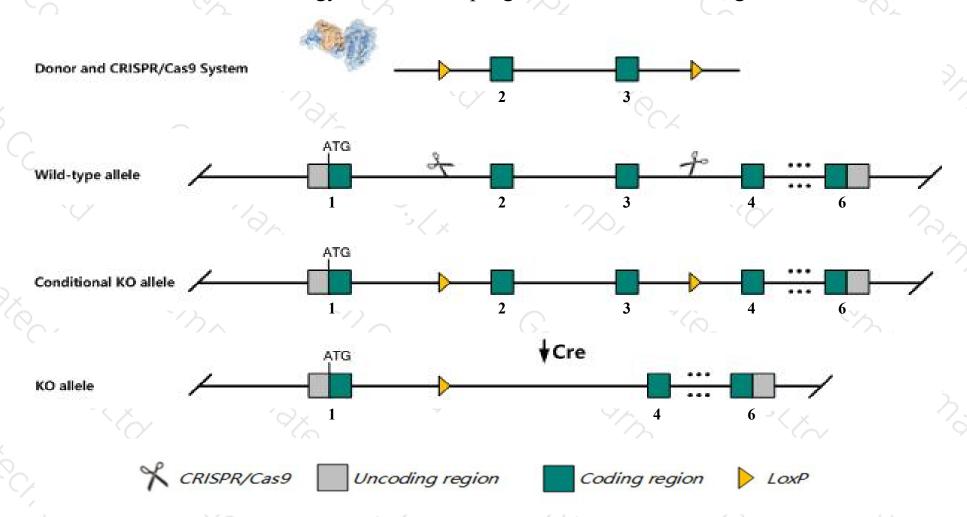
Strain background

C57BL/6JGpt

## Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Acp1 gene. The schematic diagram is as follows:



### Technical routes



- ➤ The *Acp1* gene has 6 transcripts. According to the structure of *Acp1* gene, exon2-exon3 of *Acp1-201*(ENSMUST00000062740.14) transcript is recommended as the knockout region. The region contains 188bp coding sequence.

  Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Acp1* 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 null allele show an increased mean serum IL-6 response to LPS challenge. Male homozygotes are smaller than controls whereas female homozygotes show an increased mean skin fibroblast proliferation rate. Males homozygous for a different null allele show decreased response of heart to induced stress.
- The *Acp1* gene is located on the Chr12. 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)



#### Acp1 acid phosphatase 1, soluble [ Mus musculus (house mouse) ]

Gene ID: 11431, updated on 5-Jan-2020

#### Summary

Official Symbol Acp1 provided by MGI

Official Full Name acid phosphatase 1, soluble provided by MGI

Primary source MGI:MGI:87881

See related Ensembl: ENSMUSG00000044573

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 Acp-1; Lmptp; LMW-PTP; Al427468; 4632432E04Rik

Expression Ubiquitous expression in liver E14 (RPKM 16.3), liver E14.5 (RPKM 13.9) and 27 other tissues See more

Orthologs human all

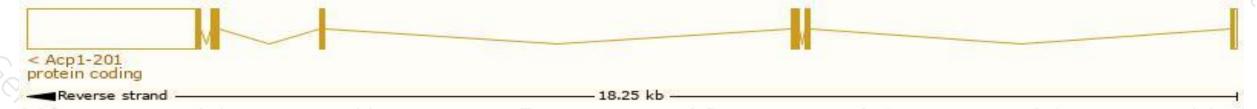
## Transcript information (Ensembl)



The gene has 6 transcripts, all transcripts are shown below:

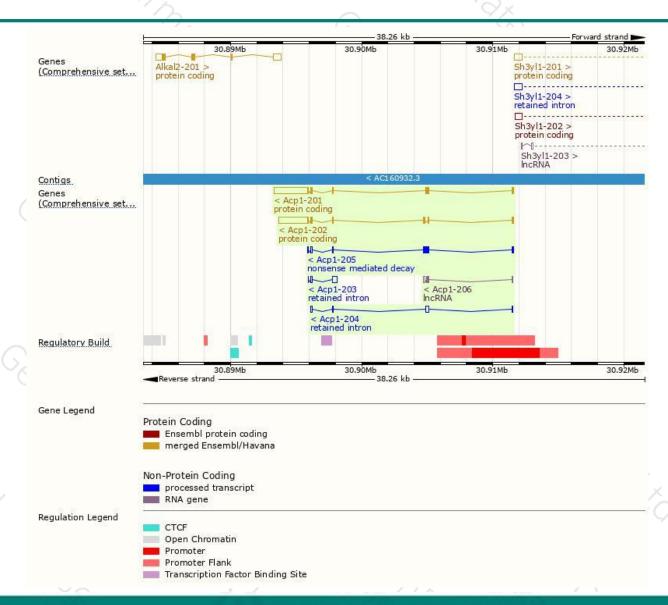
***		V				
Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
ENSMUST00000062740.14	3066	<u>158aa</u>	Protein coding	CCDS36427	Q4VAI2 Q9D358	TSL:1 GENCODE basic APPRIS P3
ENSMUST00000074038.6	2754	<u>158aa</u>	Protein coding	CCDS49045	Q561M1	TSL:1 GENCODE basic APPRIS ALT1
ENSMUST00000219697.1	668	<u>114aa</u>	Nonsense mediated decay	140	A0A1W2P7X3	TSL:5
ENSMUST00000218615.1	531	No protein	Retained intron	150	2	TSL:2
ENSMUST00000218696.1	499	No protein	Retained intron	1783		TSL:3
ENSMUST00000222791.1	307	No protein	IncRNA	657		TSL:5
	ENSMUST00000062740.14 ENSMUST00000074038.6 ENSMUST00000219697.1 ENSMUST00000218615.1 ENSMUST00000218696.1	ENSMUST00000062740.14 3066 ENSMUST00000074038.6 2754 ENSMUST00000219697.1 668 ENSMUST00000218615.1 531 ENSMUST00000218696.1 499	ENSMUST00000062740.14 3066 158aa ENSMUST00000074038.6 2754 158aa ENSMUST00000219697.1 668 114aa ENSMUST00000218615.1 531 No protein ENSMUST00000218696.1 499 No protein	ENSMUST00000062740.14         3066         158aa         Protein coding           ENSMUST00000074038.6         2754         158aa         Protein coding           ENSMUST00000219697.1         668         114aa         Nonsense mediated decay           ENSMUST00000218615.1         531         No protein         Retained intron           ENSMUST000000218696.1         499         No protein         Retained intron	ENSMUST00000062740.14         3066         158aa         Protein coding         CCDS36427           ENSMUST00000074038.6         2754         158aa         Protein coding         CCDS49045           ENSMUST00000219697.1         668         114aa         Nonsense mediated decay         -           ENSMUST00000218615.1         531         No protein         Retained intron         -           ENSMUST00000218696.1         499         No protein         Retained intron         -	ENSMUST00000062740.14         3066         158aa         Protein coding         CCDS36427         Q4VAI2 Q9D358           ENSMUST00000074038.6         2754         158aa         Protein coding         CCDS49045         Q561M1           ENSMUST00000219697.1         668         114aa         Nonsense mediated decay         -         A0A1W2P7X3           ENSMUST00000218615.1         531         No protein         Retained intron         -         -           ENSMUST00000218696.1         499         No protein         Retained intron         -         -

The strategy is based on the design of Acp1-201 transcript, The transcription is shown below



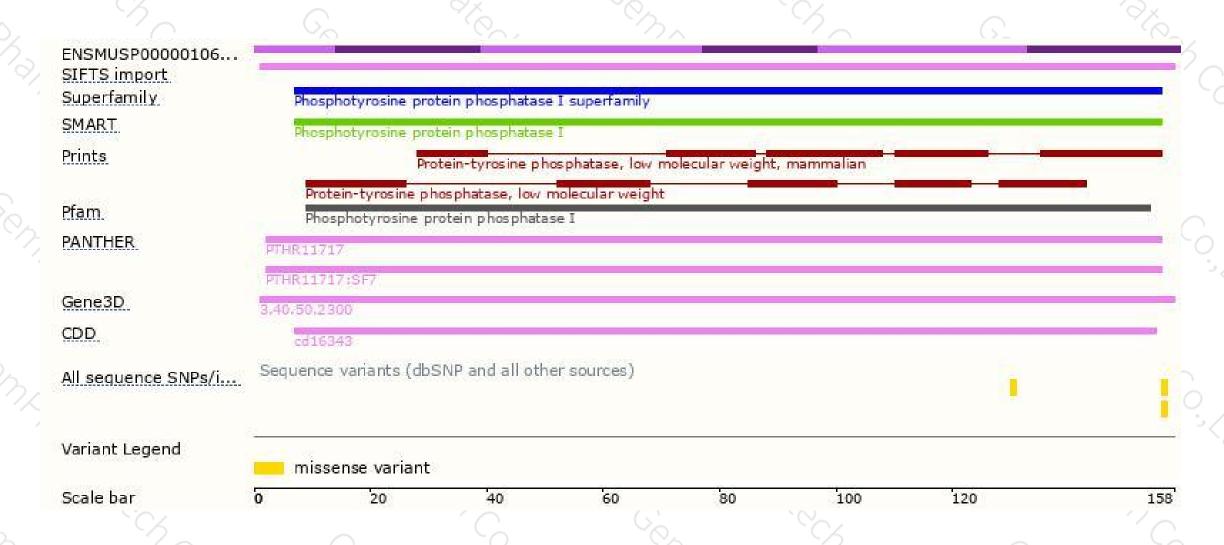
## Genomic location distribution





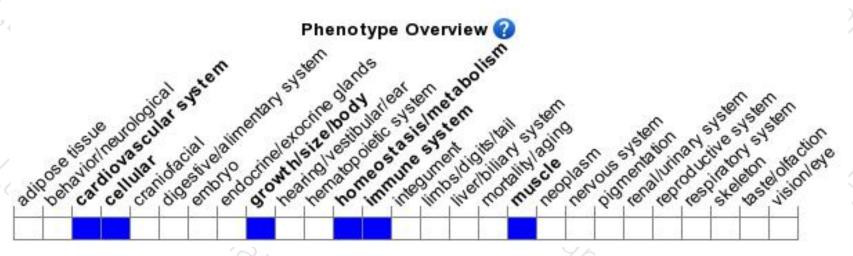
### Protein domain





## 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 null allele show an increased mean serum IL-6 response to LPS challenge. Male homozygotes are smaller than controls whereas female homozygotes show an increased mean skin fibro proliferation rate. Males homozygous for a different null allele show decreased response of heart to induced stress.



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





