

# Psmb9 Cas9-CKO Strategy

**Designer:** 

**Reviewer:** 

**Design Date:** 

Ruirui Zhang

**Huimin Su** 

2019-9-21

# **Project Overview**



**Project Name** 

Psmb9

**Project type** 

Cas9-CKO

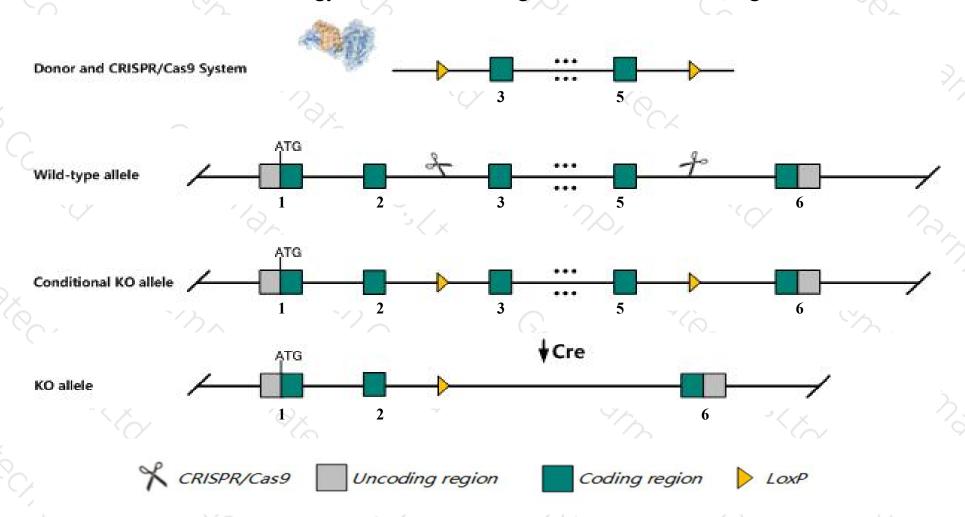
Strain background

C57BL/6JGpt

## Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Psmb9* gene. The schematic diagram is as follows:



### Technical routes



- ➤ The *Psmb9* gene has 7 transcripts. According to the structure of *Psmb9* gene, exon3-exon5 of *Psmb9-204*(ENSMUST00000174576.3) transcript is recommended as the knockout region. The region contains 404bp coding sequence.

  Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Psmb9* 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 disruptions in this gene have a grossly normal phenotype but suffer from increased susceptibility to some viruses and have an increased risk of tumor development.
- ➤ Knockout the region may affect the 5 terminal regulation function of *Tap1* gene.
- The *Psmb9* gene is located on the Chr17. 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)



Psmb9 proteasome (prosome, macropain) subunit, beta type 9 (large multifunctional peptidase 2) [

Mus musculus (house mouse)]

Gene ID: 16912, updated on 12-Aug-2019

#### Summary



Official Symbol Psmb9 provided by MGI

Official Full Name proteasome (prosome, macropain) subunit, beta type 9 (large multifunctional peptidase 2) provided by MGI

Primary source MGI:MGI:1346526

See related Ensembl:ENSMUSG00000096727

Gene type protein coding
RefSeg 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 Lmp2; Lmp-2

Expression Broad expression in large intestine adult (RPKM 97.4), thymus adult (RPKM 90.4) and 16 other tissues See more

Orthologs <u>human</u> all

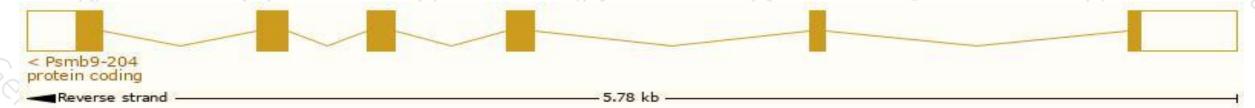
# Transcript information (Ensembl)



The gene has 7 transcripts, all transcripts are shown below:

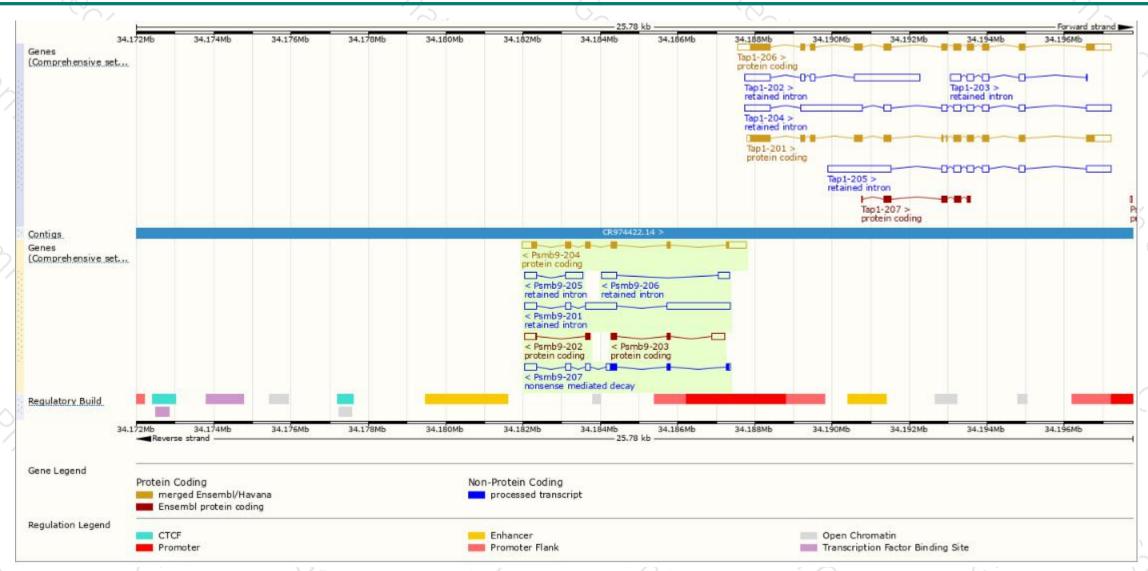
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Psmb9-204	ENSMUST00000174576.3	1352	219aa	Protein coding	CCDS28642@	<u>A0A0R4J256</u> €	TSL:1 GENCODE basic APPRIS P1
Psmb9-203	ENSMUST00000173831.2	529	63aa	Protein coding	-	G3UYK5₽	CDS 3' incomplete   TSL:3
Psmb9-202	ENSMUST00000171321.1	433	<u>44aa</u>	Protein coding	-	F6QXK7₽	CDS 5' incomplete TSL:2
Psmb9-207	ENSMUST00000237228.1	1025	93aa	Nonsense mediated decay	-	141	-
Psmb9-201	ENSMUST00000114230.2	2884	No protein	Retained intron	-	-	TSL:2
Psmb9-205	ENSMUST00000178857.1	783	No protein	Retained intron	-	141	TSL:2
Psmb9-206	ENSMUST00000179593.1	665	No protein	Retained intron	-		TSL:3

The strategy is based on the design of *Psmb9-204* transcript, The transcription is shown below



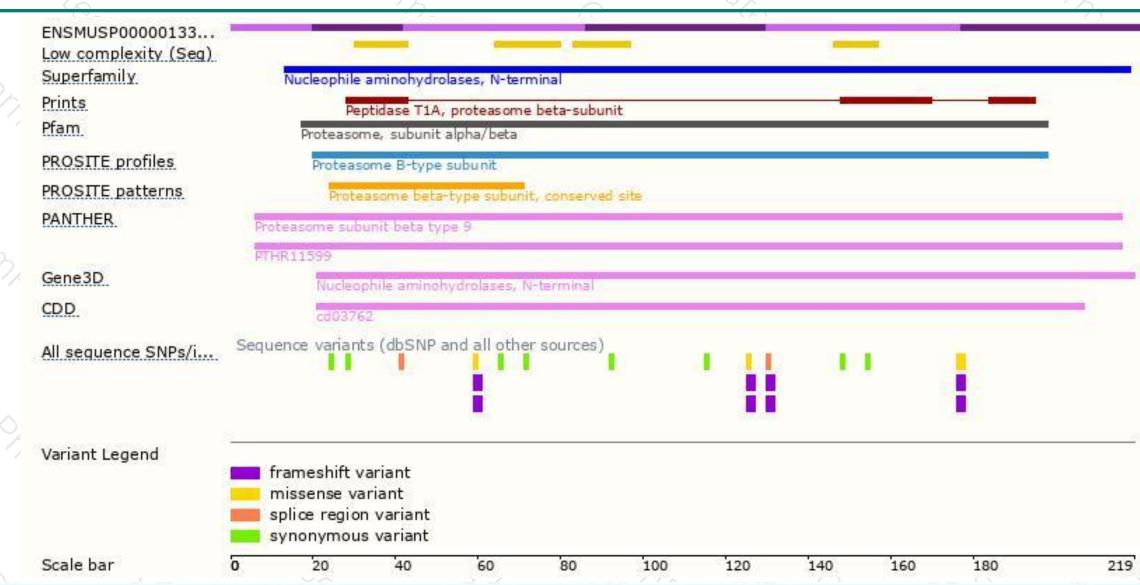
### Genomic location distribution





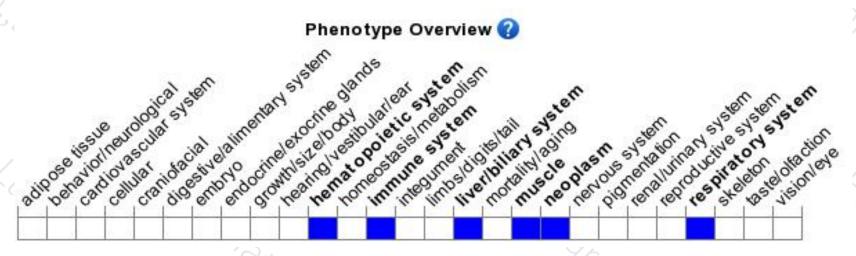
### Protein domain





# 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 disruptions in this gene have a grossly normal phenotype but suffer from increased susceptibility to some viruses and have an increased risk of tumor development.



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





