

Serpinb1a Cas9-CKO Strategy

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Reviewer:

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Project Overview

Project Name

Serpinb1a

Project type

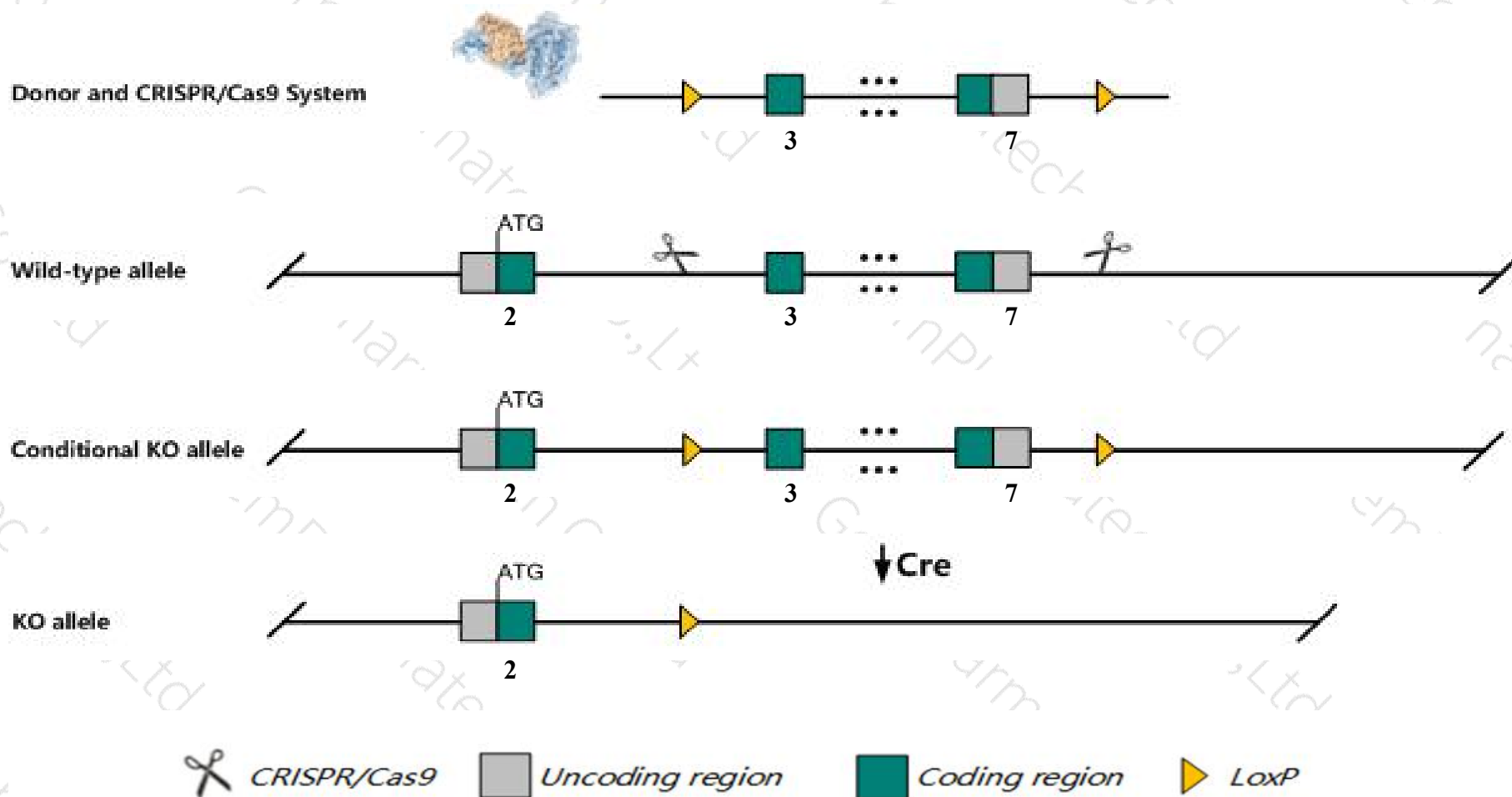
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Serpinb1a* gene. The schematic diagram is as follows:



Technical routes

- The *Serpinb1a* gene has 4 transcripts. According to the structure of *Serpinb1a* gene, exon3-exon7 of *Serpinb1a*-201 (ENSMUST00000076352.7) transcript is recommended as the knockout region. The region contains most of the coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Serpinb1a* 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, Homozygous null mice fail to clear *P. aeruginosa* lung infection and show increased mortality associated with late-onset failed bacterial clearance, partly due to elevated neutrophil necrosis, release of neutrophil protease activity, higher cytokine production and proteolysis of surfactant protein-D.
- The *Serpinbl1a* gene is located on the Chr13. 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)

Serpina1 serine (or cysteine) peptidase inhibitor, clade B, member 1a [Mus musculus (house mouse)]

Gene ID: 66222, updated on 31-Jan-2019

Summary



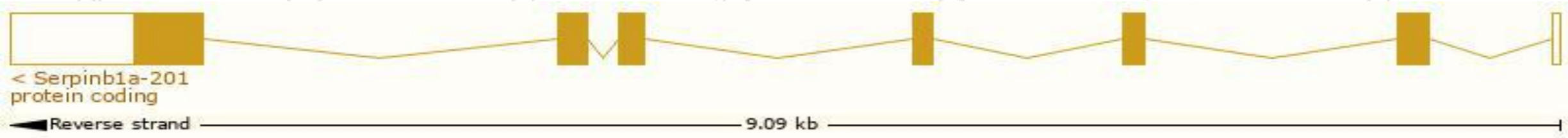
Official Symbol	Serpina1 provided by MGI
Official Full Name	serine (or cysteine) peptidase inhibitor, clade B, member 1a provided by MGI
Primary source	MGI:MGI:1913472
See related	Ensembl:ENSMUSG00000044734
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	1190005M04Rik, AI325983, EI, EIA, ELANH2, LEI, M/NEI, MNEI, PI2
Expression	Biased expression in large intestine adult (RPKM 90.3), duodenum adult (RPKM 21.7) and 10 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

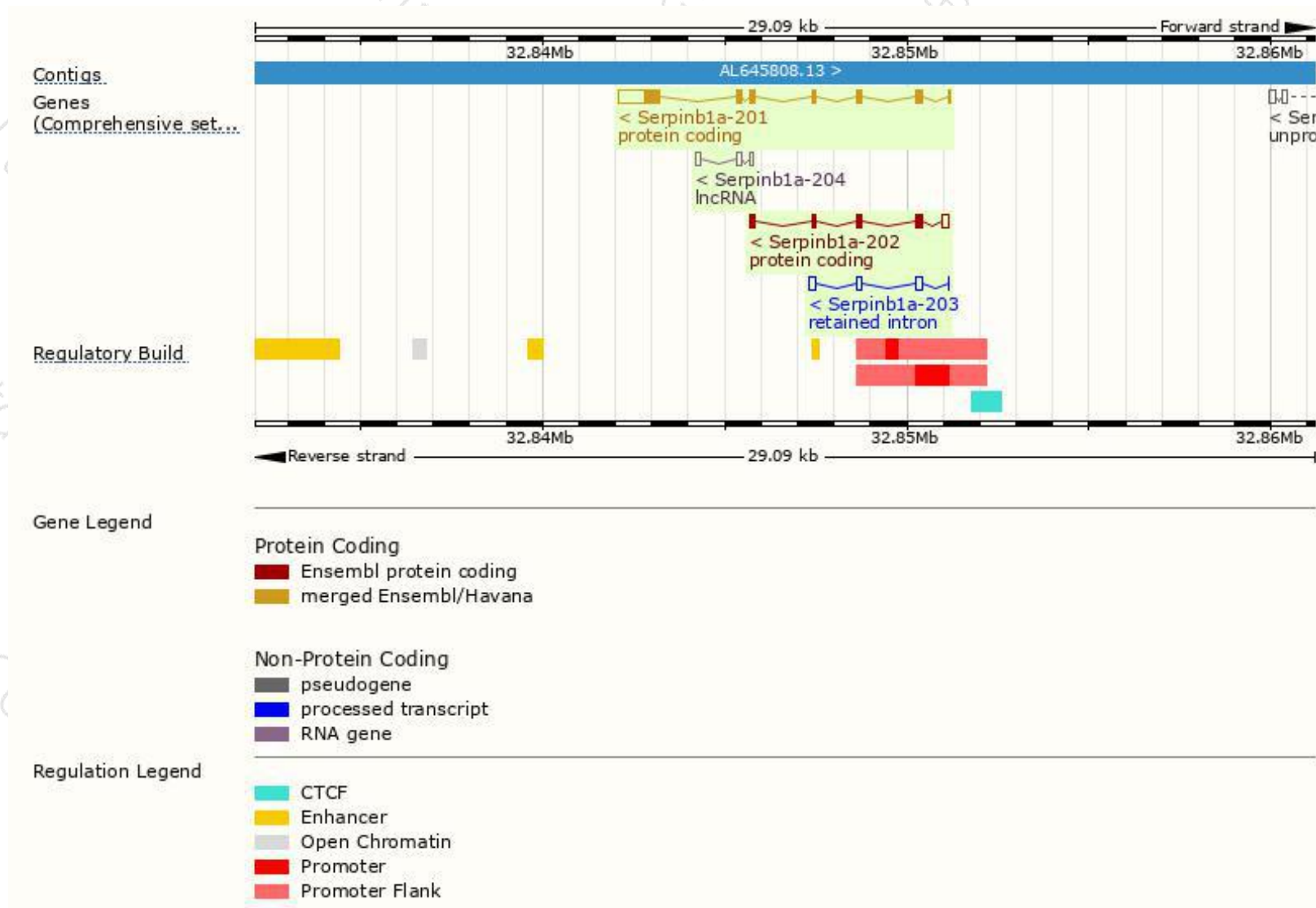
The gene has 4 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Serpina1a-201	ENSMUST00000076352.7	1918	379aa	Protein coding	CCDS26428	Q9D154	TSL:1 GENCODE basic APPRIS P1
Serpina1a-202	ENSMUST00000091668.12	759	188aa	Protein coding	-	Z4YK03	CDS 3' incomplete TSL:3
Serpina1a-203	ENSMUST00000221967.1	541	No protein	Retained intron	-	-	TSL:2
Serpina1a-204	ENSMUST00000223016.1	427	No protein	lncRNA	-	-	TSL:3

The strategy is based on the design of *Serpina1a-201* transcript,The transcription is shown below



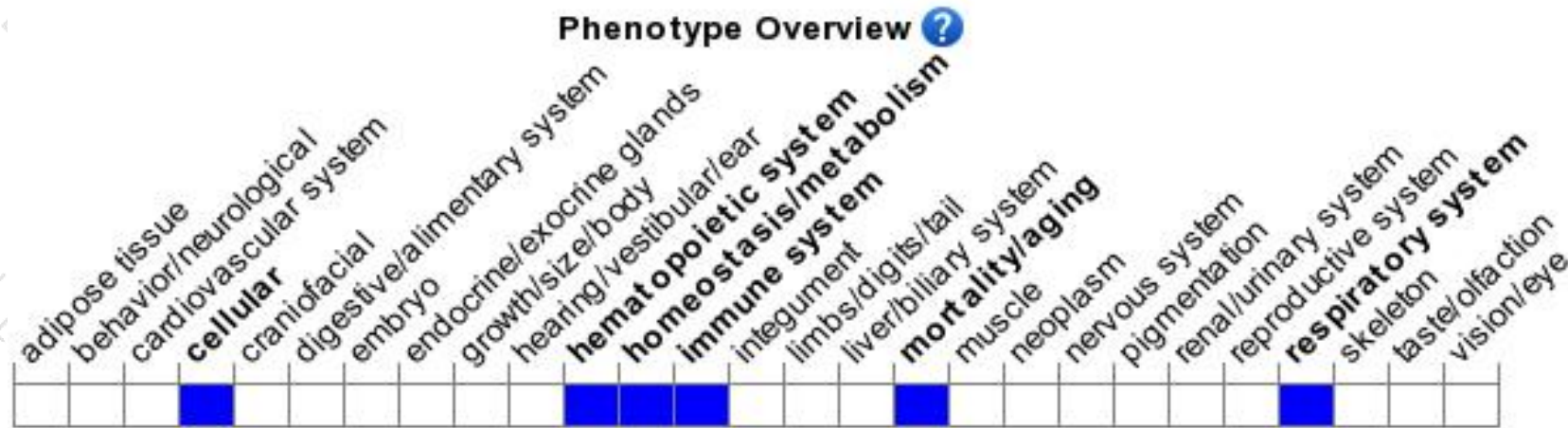
Genomic location distribution



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, Homozygous null mice fail to clear *P. aeruginosa* lung infection and show increased mortality associated with late-onset failed bacterial clearance, partly due to elevated neutrophil necrosis, release of neutrophil protease activity, higher cytokine production and proteolysis of surfactant protein-D.

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

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