

Proz Cas9-CKO Strategy

Designer: Xueting Zhang

Reviewer: Yanhua Shen

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Project Overview

Project Name

Proz

Project type

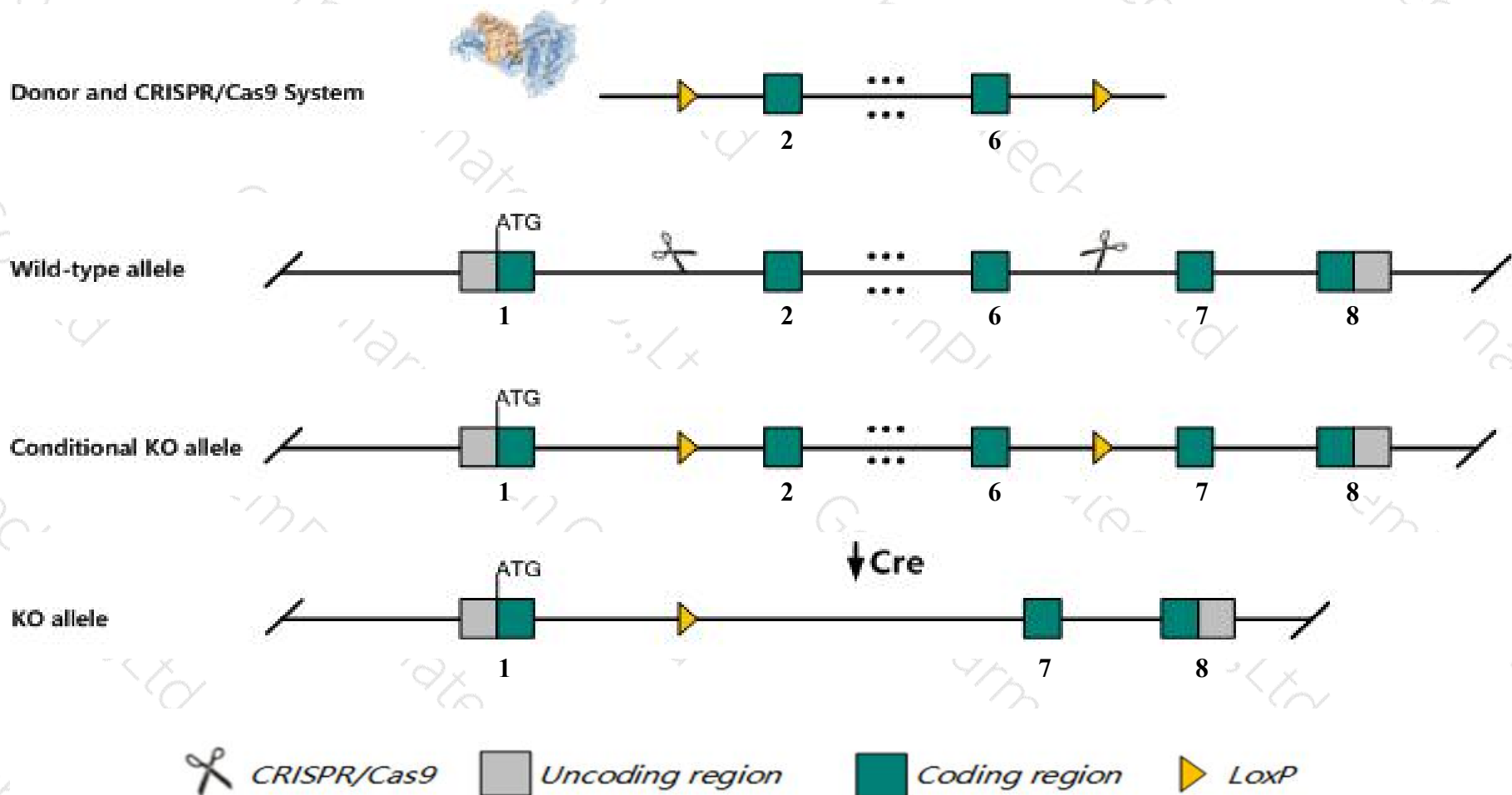
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

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



Technical routes

- The *Proz* gene has 4 transcripts. According to the structure of *Proz* gene, exon2-exon6 of *Proz-201* (ENSMUST00000033822.3) transcript is recommended as the knockout region. The region contains 518bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Proz* 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, When unchallenged, mice homozygous for a knock-out allele do not express an obvious phenotype; however, homozygotes exhibit significantly reduced survival following collagen/epinephrine-induced thromboembolism and develop enhanced thrombosis in the ferric chloride-induced arterial injury model.
- The *Proz* gene is located on the Chr8. 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)

Proz protein Z, vitamin K-dependent plasma glycoprotein [*Mus musculus* (house mouse)]

Gene ID: 66901, updated on 24-Oct-2019

Summary

- Official Symbol** Proz provided by [MGI](#)
- Official Full Name** protein Z, vitamin K-dependent plasma glycoprotein provided by [MGI](#)
- Primary source** [MGI:MGI:1860488](#)
- See related** [Ensembl:ENSMUSG00000031445](#)
- 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** 1300015B06Rik
- Expression** Biased expression in liver adult (RPKM 113.6), liver E18 (RPKM 53.9) and 3 other tissues [See more](#)
- Orthologs** [human](#) [all](#)

Genomic context

Location: 8; 8 A1.1 See Proz in [Genome Data Viewer](#)

Exon count: 9

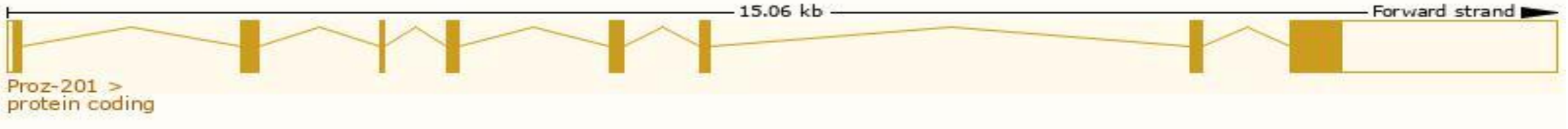
Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	8	NC_000074.6 (13060584..13076026)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	8	NC_000074.5 (13060908..13075006)

Transcript information (Ensembl)

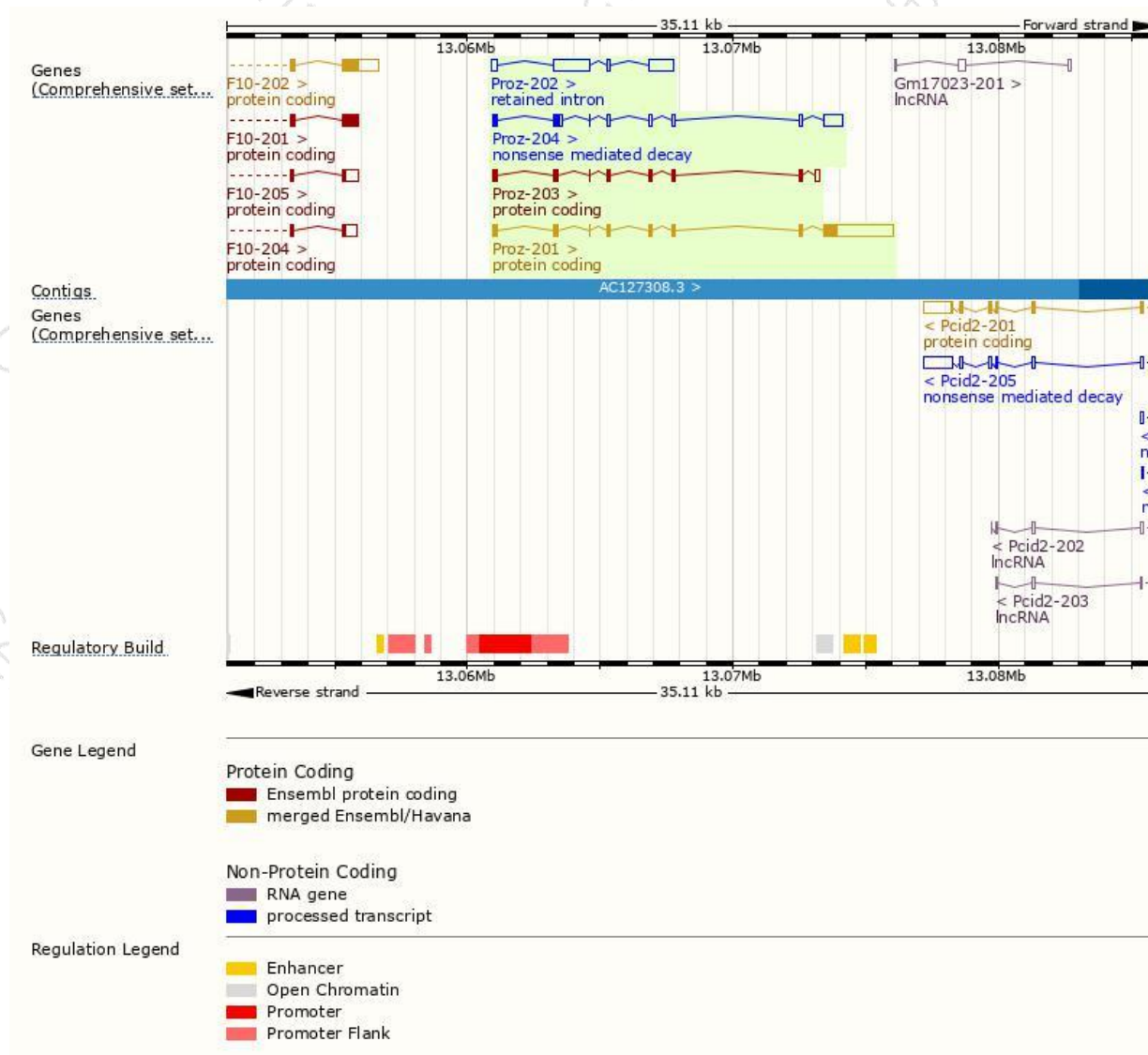
The gene has 4 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Proz-201	ENSMUST00000033822.3	3361	399aa	Protein coding	CCDS22105	Q9CQW3	TSL:1 GENCODE basic APPRIS P1
Proz-203	ENSMUST00000211363.1	984	241aa	Protein coding	-	Q8CI01	TSL:1 GENCODE basic
Proz-204	ENSMUST00000211453.1	1636	98aa	Nonsense mediated decay	-	A0A1B0GR07	TSL:1
Proz-202	ENSMUST00000210050.1	2618	No protein	Retained intron	-	-	TSL:5

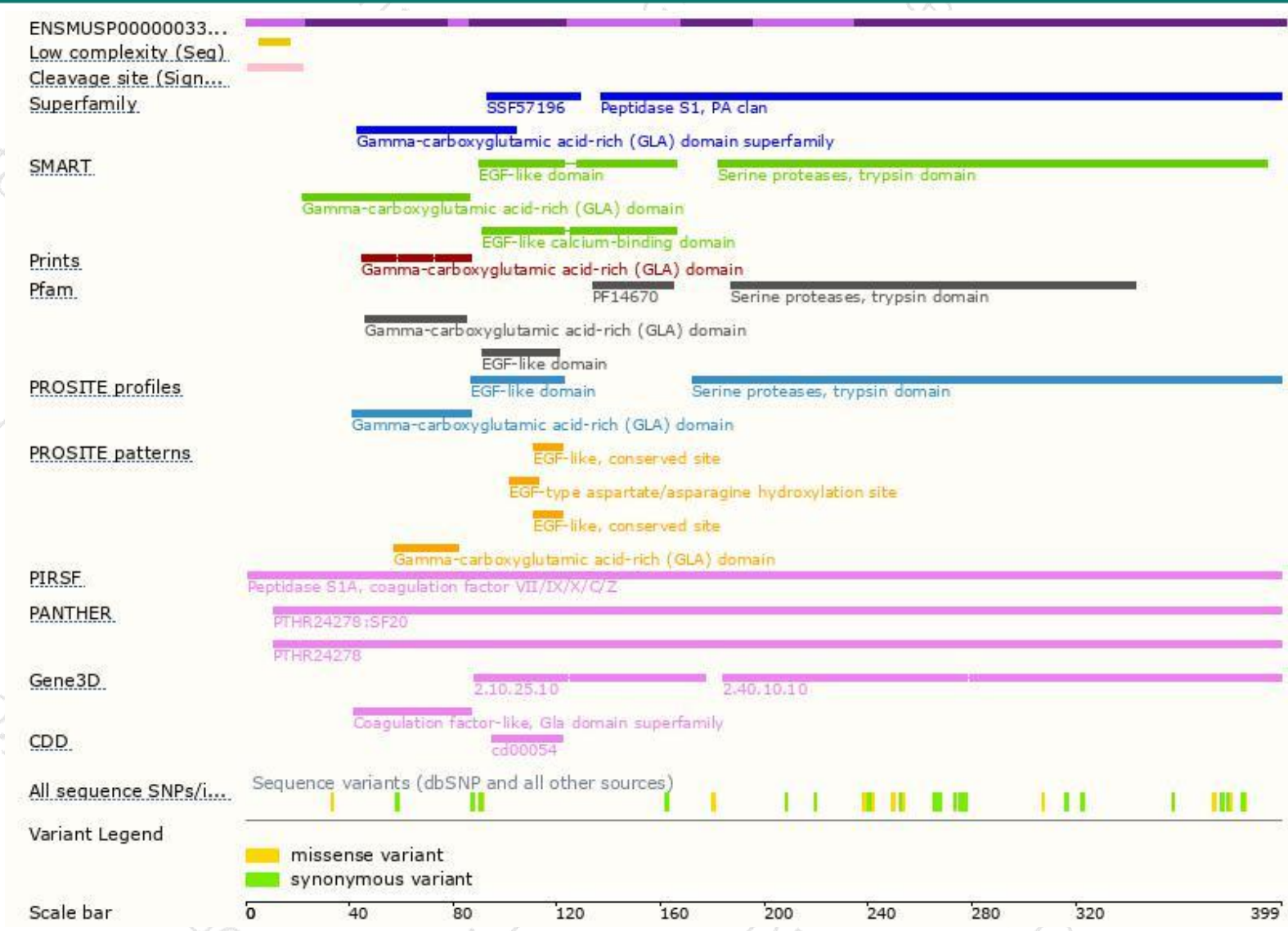
The strategy is based on the design of *Proz-201* transcript,The transcription is shown below



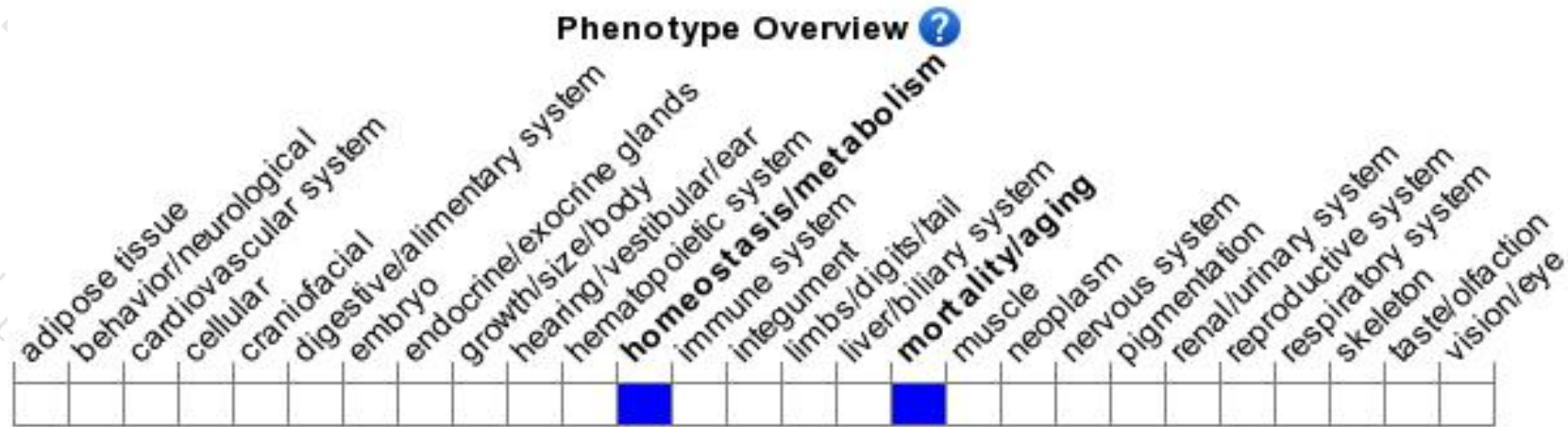
Genomic location distribution



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, When unchallenged, mice homozygous for a knock-out allele do not express an obvious phenotype; however, homozygotes exhibit significantly reduced survival following collagen/epinephrine-induced thromboembolism and develop enhanced thrombosis in the ferric chloride-induced arterial injury model.

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

Tel: 400-9660890

