

# F3 Cas9-KO Strategy

**Designer:** 

**Reviewer:** 

**Design Date:** 

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## **Project Overview**



**Project Name** 

*F3* 

**Project type** 

Cas9-KO

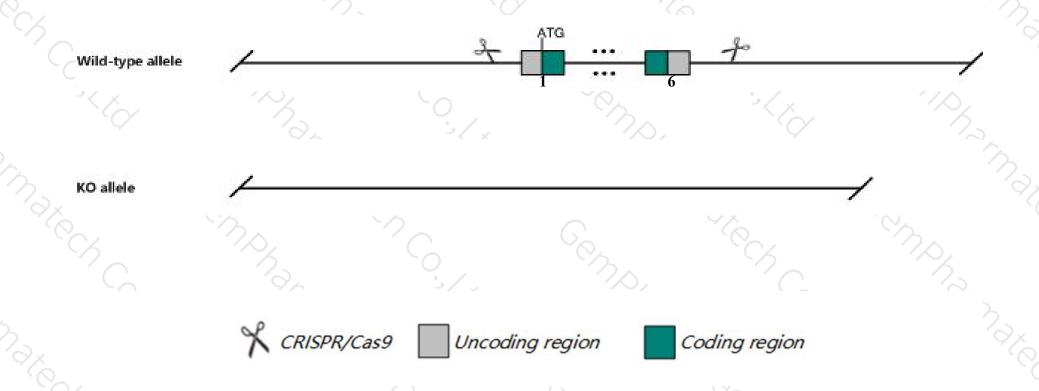
Strain background

C57BL/6JGpt

# **Knockout strategy**



This model will use CRISPR/Cas9 technology to edit the F3 gene. The schematic diagram is as follows:



### **Technical routes**



- ➤ The F3 gene has 4 transcripts. According to the structure of F3 gene, exon1-exon6 of F3-201 (ENSMUST00000029771.12) transcript is recommended as the knockout region. The region contains all of the coding sequence. Knock out the region will result in disruption of protein function.
- $\succ$  In this project we use CRISPR/Cas9 technology to modify F3 gene. The brief process is as follows: CRISPR/Cas9 system we

### **Notice**



- ➤ According to the existing MGI data, Homozygotes for targeted null mutations exhibit impaired blood vessel development, retarded growth, and, in most cases, midgestational lethality. On a mixed background, some mutants survive to birth and appear to be normal.
- The KO region contains Gm43823-201 gene. Knockout the region may affect the function of Gm43823-201 gene.
- $\gt$  The F3 gene is located on the Chr3. 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 the gene knockout on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

### Gene information (NCBI)



#### F3 coagulation factor III [ Mus musculus (house mouse) ]

Gene ID: 14066, updated on 21-Aug-2019

#### Summary

☆ ? 🗅

Official Symbol F3 provided by MGI

Official Full Name coagulation factor III provided by MGI

Primary source MGI:MGI:88381

See related Ensembl:ENSMUSG00000028128

Gene type protein coding
RefSeq status REVIEWED
Organism Mus musculus

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha;

Muroidea; Muridae; Murinae; Mus; Mus

Also known as TF; Cf3; Cf-3; CD142; AA409063

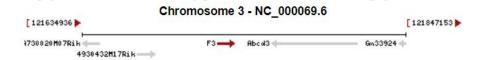
Summary This gene encodes a membrane-bound glycoprotein that forms the primary physiological initiator of the blood coagulation process

following vascular damage. The encoded protein binds to coagulation factor VIIa and the ensuing complex catalyzes the proteolytic activation of coagulation factors IX and X. Mice lacking encoded protein die in utero resulting from massive hemorrhaging in both extraembryonic and embryonic vessels. A severe deficiency of the encoded protein in mice results in impaired uterine homeostasis,

shorter life spans due to spontaneous fatal hemorrhages and cardiac fibrosis. [provided by RefSeq, Aug 2015]

Expression Broad expression in bladder adult (RPKM 29.5), subcutaneous fat pad adult (RPKM 20.6) and 21 other tissues See more

Orthologs human all



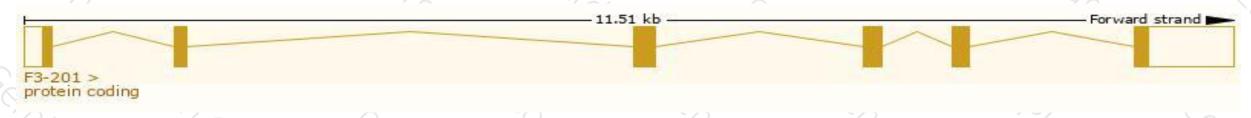
# Transcript information (Ensembl)



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

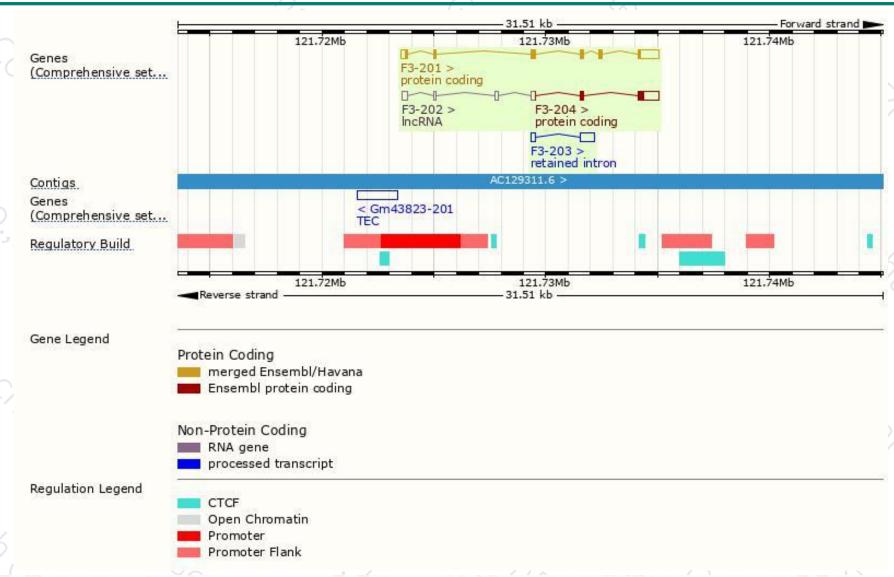
Name 🍦	Transcript ID 👙	bp 🍦	Protein	Translation ID	Biotype	CCDS	UniProt	Flags
F3-201	ENSMUST00000029771.12	1872	294aa	ENSMUSP00000029771.8	Protein coding	CCDS17805 ₺	A0A0R4J088₺	TSL:1 GENCODE basic APPRIS P1
F3-204	ENSMUST00000199997.1	1130	157aa	ENSMUSP00000143678.1	Protein coding	29	A0A0G2JGS5 ₺	CDS 5' incomplete TSL:5
F3-203	ENSMUST00000197731.1	890	No protein	<u>u</u>	Retained intron	2	2	TSL:2
F3-202	ENSMUST00000196746.1	700	No protein	<u>@</u>	IncRNA	n	<u>u</u>	TSL:3

The strategy is based on the design of F3-201 transcript, The transcription is shown below



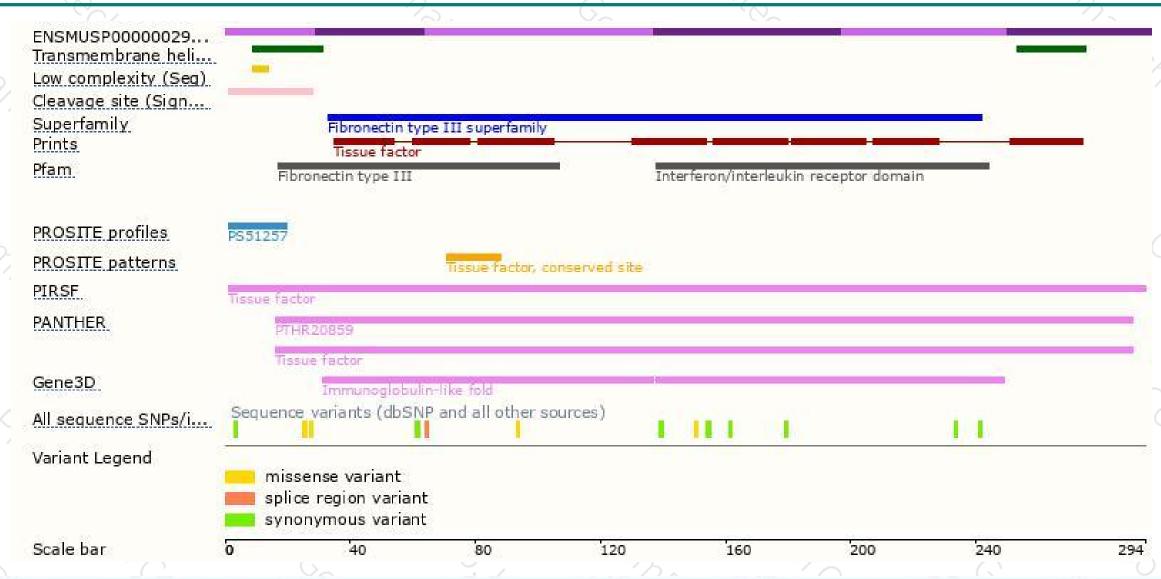
### Genomic location distribution





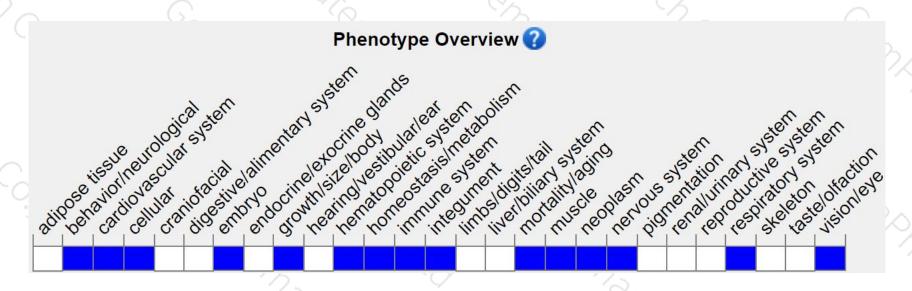
### Protein domain





# 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, Homozygotes for targeted null mutations exhibit impaired blood vessel development, retarded growth, and, in most cases, midgestational lethality. On a mixed background, some mutants survive to birth and appear to be normal.



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





