

# **Tnks Cas9-CKO Strategy**

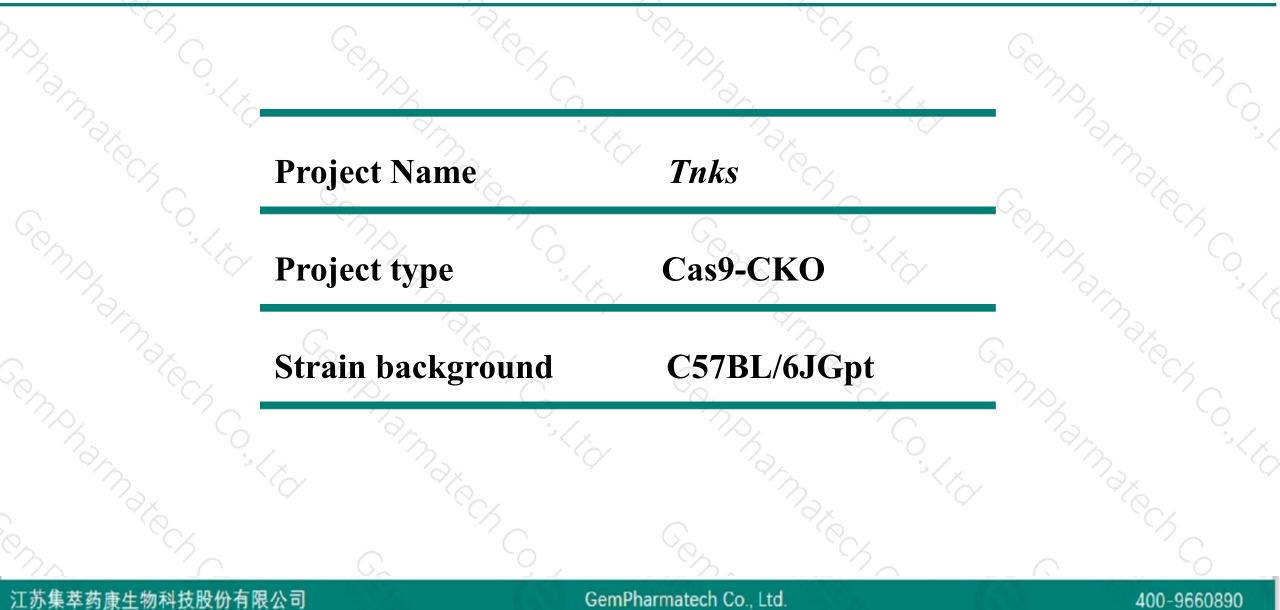
Designer: Reviewer:

**Design Date:** 

Huan Fan Huan Wang 2020-2-28

# **Project Overview**



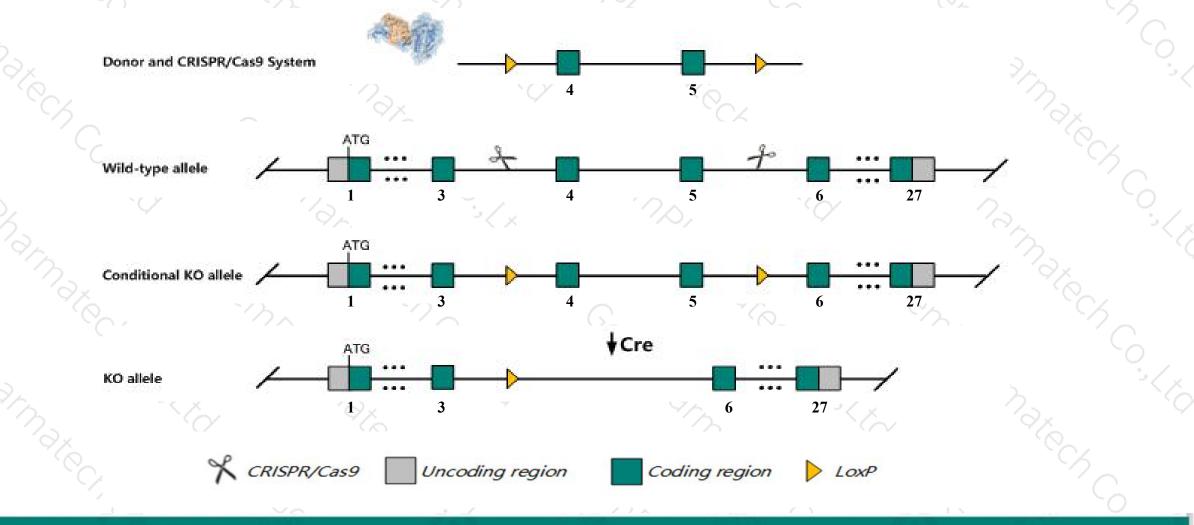


### **Conditional Knockout strategy**



400-9660890

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



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The *Tnks* gene has 6 transcripts. According to the structure of *Tnks* gene, exon4-exon5 of *Tnks-201* (ENSMUST00000033929.5) transcript is recommended as the knockout region. The region contains 113bp coding sequence. Knock out the region will result in disruption of protein function.

In this project we use CRISPR/Cas9 technology to modify *Tnks* 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 fail to exhibit any abonormalities.
  Male mice homozygous for a gene trapped allele exhibit decreased fat pad weight, increased metabolism, hyperinsulinemia, and hypoglycemia.
- > Transcript *Tnks-202* , 204 may not be affected.
- The *Tnks* 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)**



☆ ?

### Tnks tankyrase, TRF1-interacting ankyrin-related ADP-ribose polymerase [Mus musculus (house mouse)]

Gene ID: 21951, updated on 3-Feb-2019

#### Summary

Tnks provided by MGI
tankyrase, TRF1-interacting ankyrin-related ADP-ribose polymerase provided by MGI
MGI:MGI:1341087
Ensembl:ENSMUSG00000031529
protein coding
VALIDATED
Mus musculus
Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha;
Muroidea; Muridae; Murinae; Mus; Mus
4930554K12Rik, Al662855, ARTD5, C86528, D130072O21Rik, TANK1, mTNKS1
Ubiquitous expression in testis adult (RPKM 23.3), whole brain E14.5 (RPKM 14.7) and 28 other tissues See more
human all

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

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Tnks-201	ENSMUST00000033929.5	9163	<u>1320aa</u>	Protein coding	CCDS22242	Q6PFX9	TSL:1 GENCODE basic APPRIS P1
Tnks-204	ENSMUST00000210014.1	878	<u>151aa</u>	Protein coding	<del>.</del>	A0A1B0GRK1	CDS 5' incomplete TSL:3
Tnks-202	ENSMUST00000209632.1	688	<u>48aa</u>	Protein coding	45	A0A1B0GRH5	CDS 5' incomplete TSL:5
Tnks-205	ENSMUST00000210870.1	5265	No protein	Retained intron	20	1923	TSL:NA
Tnks-203	ENSMUST00000209904.1	3417	No protein	Retained intron	7.0	1.54	TSL:1
Tnks-206	ENSMUST00000211065.1	295	No protein	IncRNA	-8	6-0	TSL:5

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

< Tnks-201 protein coding

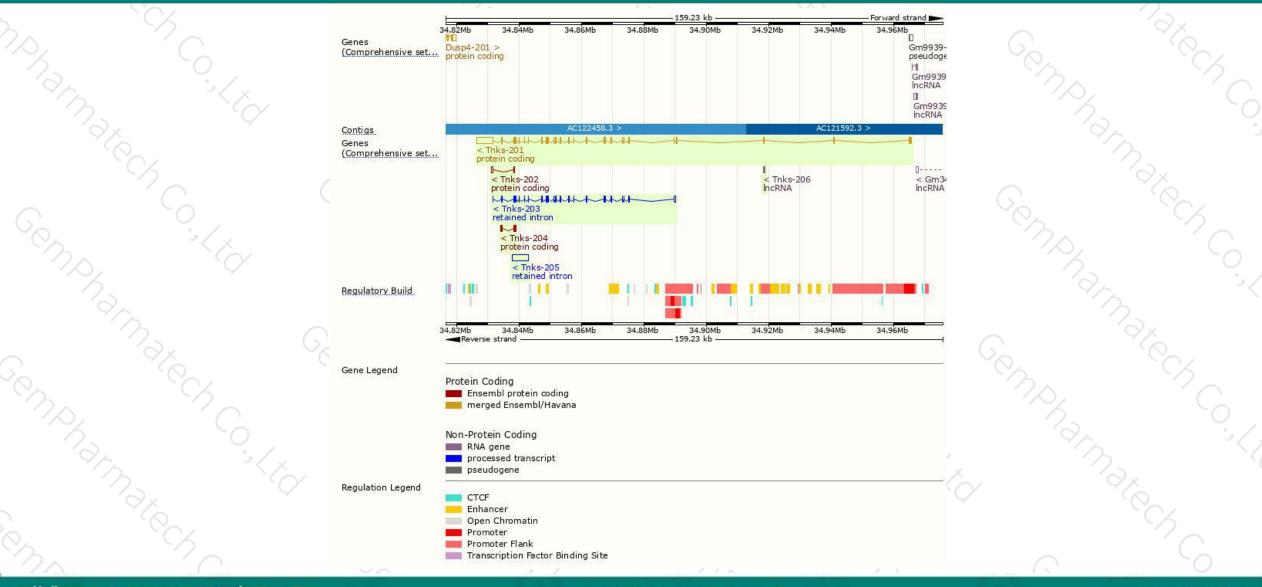
Reverse strand

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139.23 kb

### **Genomic location distribution**





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### **Protein domain**



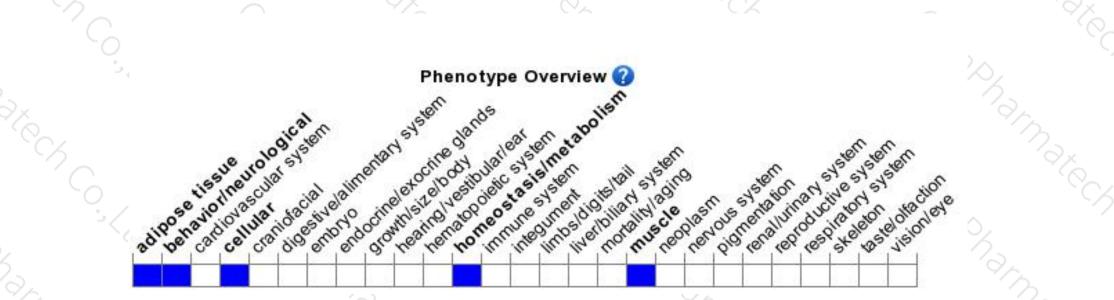


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### Mouse phenotype description(MGI)





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 fail to exhibit any abonormalities. Male mice homozygous for a gene trapped allele exhibit decreased fat pad weight, increased metabolism, hyperinsulinemia, and hypoglycemia.

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



