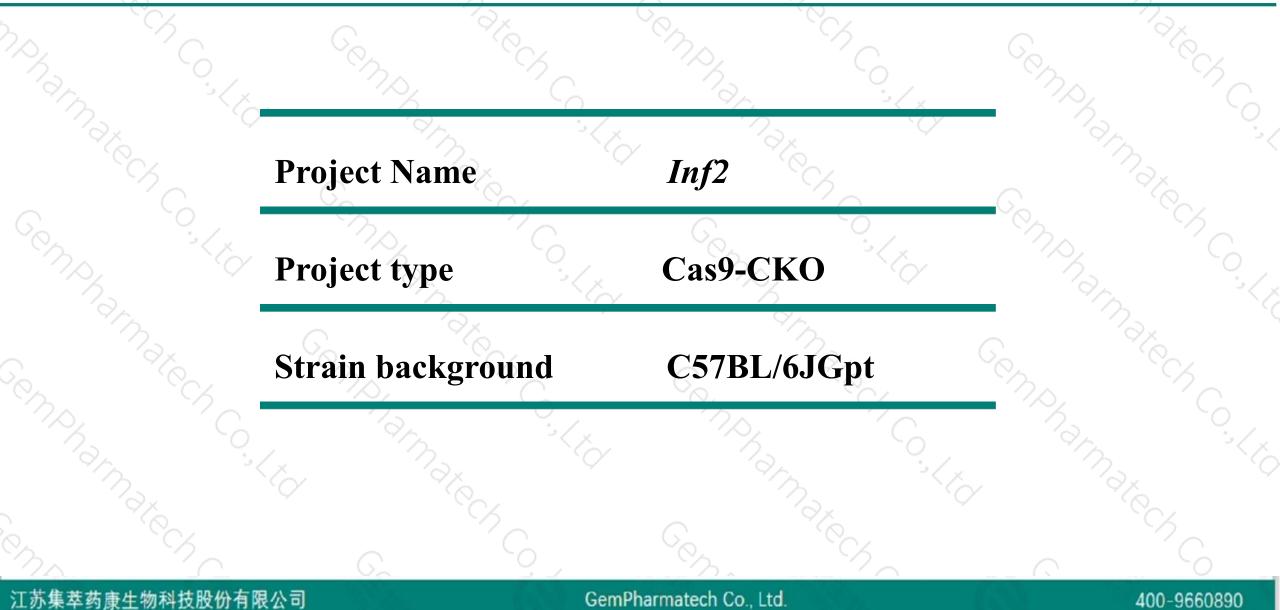


# Inf2 Cas9-CKO Strategy

Designer: Reviewer: Design Date: JiaYu Xiaojing Li 2020-2-13

## **Project Overview**



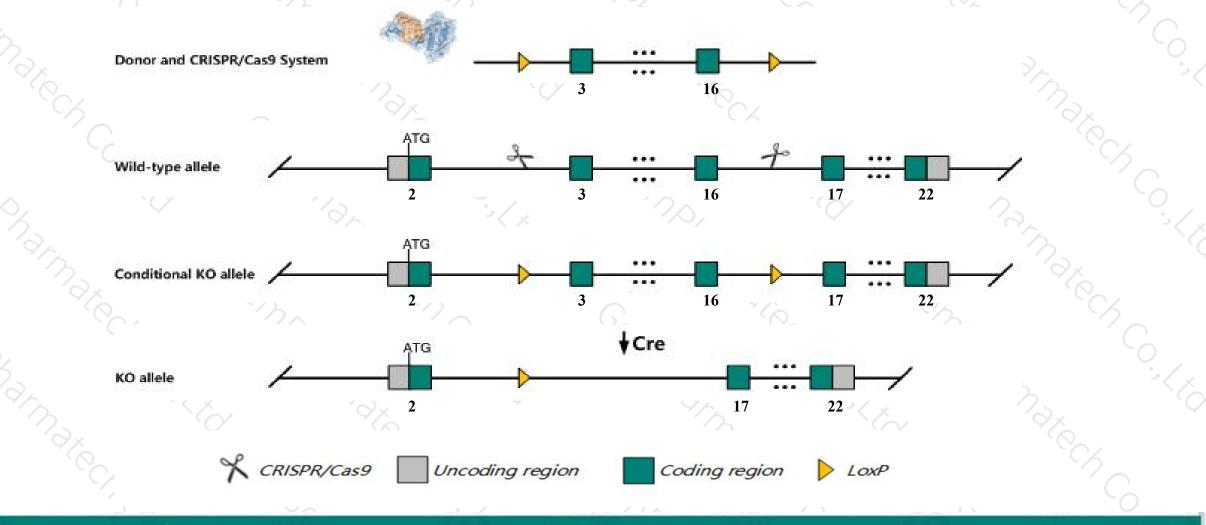


## **Conditional Knockout strategy**



400-9660890

This model will use CRISPR/Cas9 technology to edit the *Inf2* gene. The schematic diagram is as follows:



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The *Inf2* gene has 3 transcripts. According to the structure of *Inf2* gene, exon3-exon16 of *Inf2-201* (ENSMUST00000101029.3) transcript is recommended as the knockout region. The region contains 2191bp coding sequence. Knock out the region will result in disruption of protein function.

In this project we use CRISPR/Cas9 technology to modify *Inf2* 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 display placental vasculopathy, restricted fetal growth, increased gestational length and transient increase in maternal blood pressure in the late stages of pregnancy.
- Transcript 202 CDS 3' incomplete the influences is unknown.
- The Inf2 gene is located on the Chr12. 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.

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## **Gene information (NCBI)**



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#### Inf2 inverted formin, FH2 and WH2 domain containing [Mus musculus (house mouse)]

Gene ID: 70435, updated on 31-Jan-2019

#### Summary

Official Symbol	Inf2 provided by MGI					
Official Full Name	inverted formin, FH2 and WH2 domain containing provided by MGI					
Primary source	MGI:MGI:1917685					
See related	Ensembl:ENSMUSG0000037679					
Gene type	protein coding					
<b>RefSeq status</b>	VALIDATED					
Organism	Mus musculus					
Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rode						
	Muroidea; Muridae; Murinae; Mus; Mus					
Also known as	2610204M08Rik, AA589465, AW125550, EG629699					
Expression	Ubiquitous expression in cortex adult (RPKM 15.2), frontal lobe adult (RPKM 13.7) and 28 other tissues See more					
Orthologs	human all					

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## **Transcript information (Ensembl)**

#### The gene has 3 transcripts, all transcripts are shown below:

Name 🖕	Transcript ID	bp 🖕 F	Protein 🖕	Biotype 🍦	CCDS 🍦	UniProt 🖕	Flags		
Inf2-201	ENSMUST00000101029.3	4001	<u>1271aa</u>	Protein coding	<u>CCDS36571</u> ជ្	E9QLA5 &	TSL:	1 GENCODE basic	APPRIS P1
Inf2-203	ENSMUST00000222275.1	2795	<u>720aa</u>	Protein coding	17	<u>A0A1Y7VM80</u> 函		CDS 5' incomplete	TSL:1
Inf2-202	ENSMUST00000220786.1	352	<u>24aa</u>	Protein coding	17	A0A1Y7VLQ6@		CDS 3' incomplete	TSL:3

The strategy is based on the design of *Inf2-201* transcript, The transcription is shown below

Inf2-201 > protein coding

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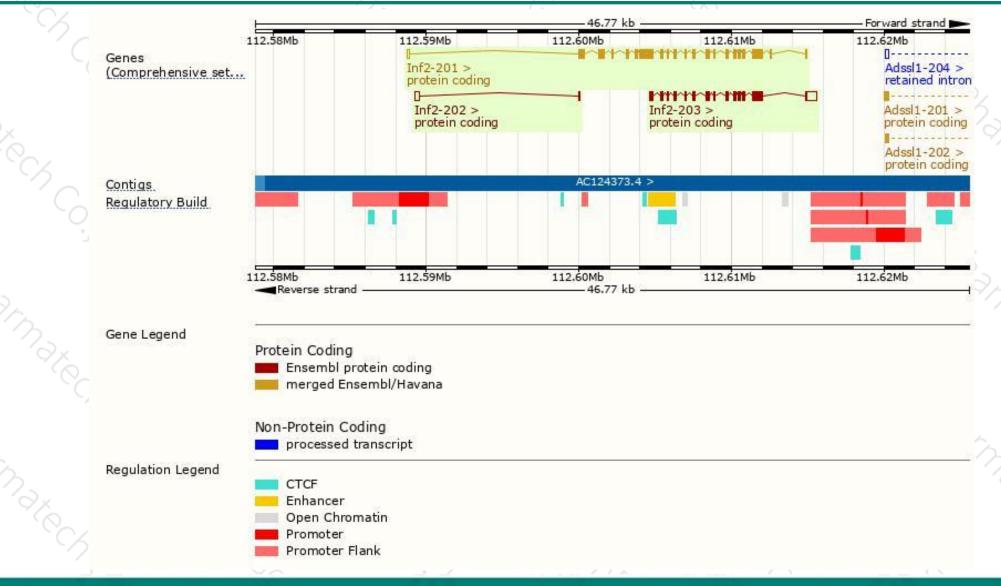
26.17 kb

#### 400-9660890

Forward strand

### **Genomic location distribution**



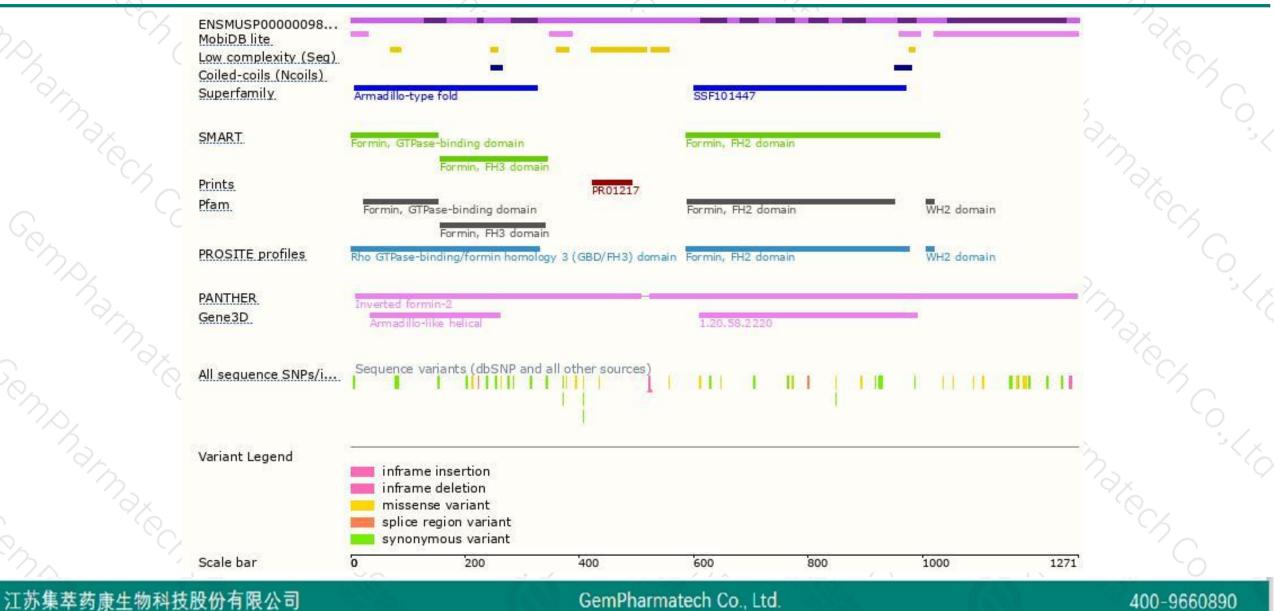


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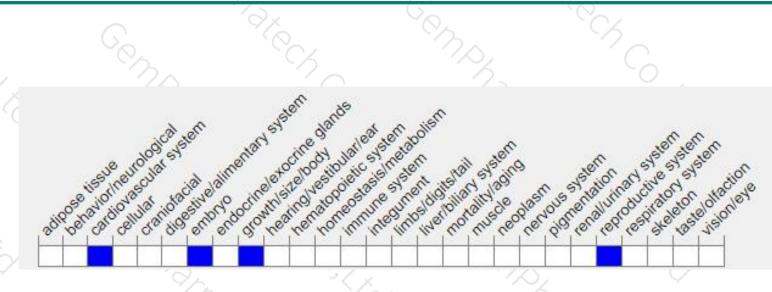
### **Protein domain**





## 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 a null allele display placental vasculopathy, restricted fetal growth, increased gestational length and transient increase in maternal blood pressure in the late stages of pregnancy.

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



