

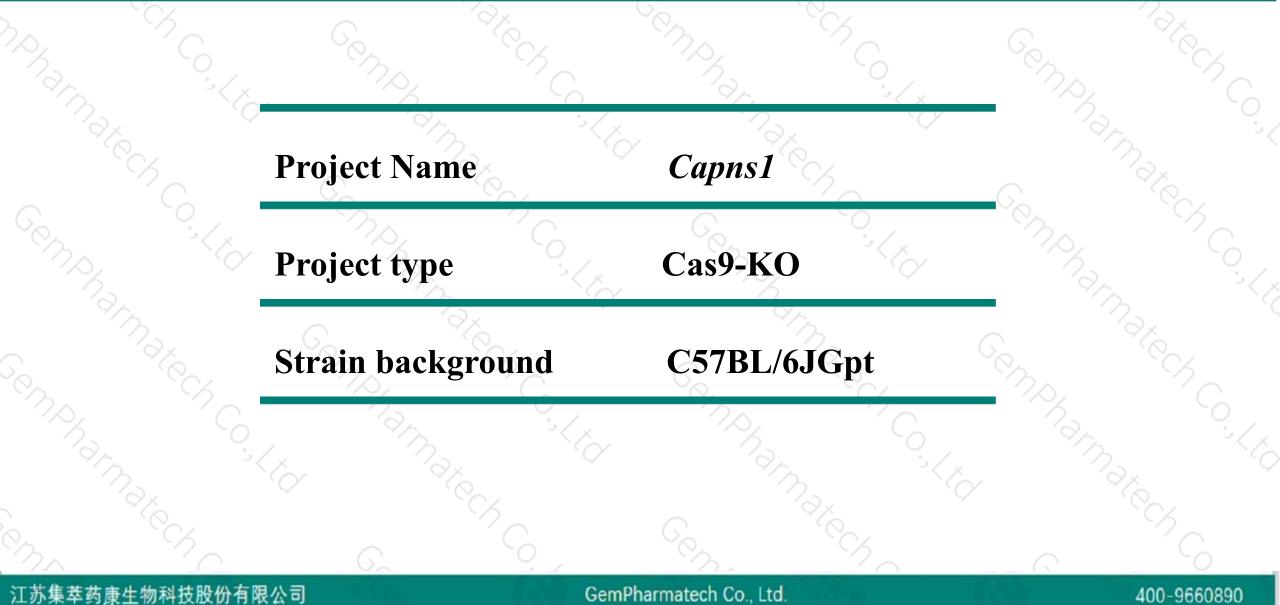
Capns1 Cas9-KO Strategy

Designer: Reviewer: Design Date: Ruirui Zhang Huimin Su

2020-1-22

Project Overview

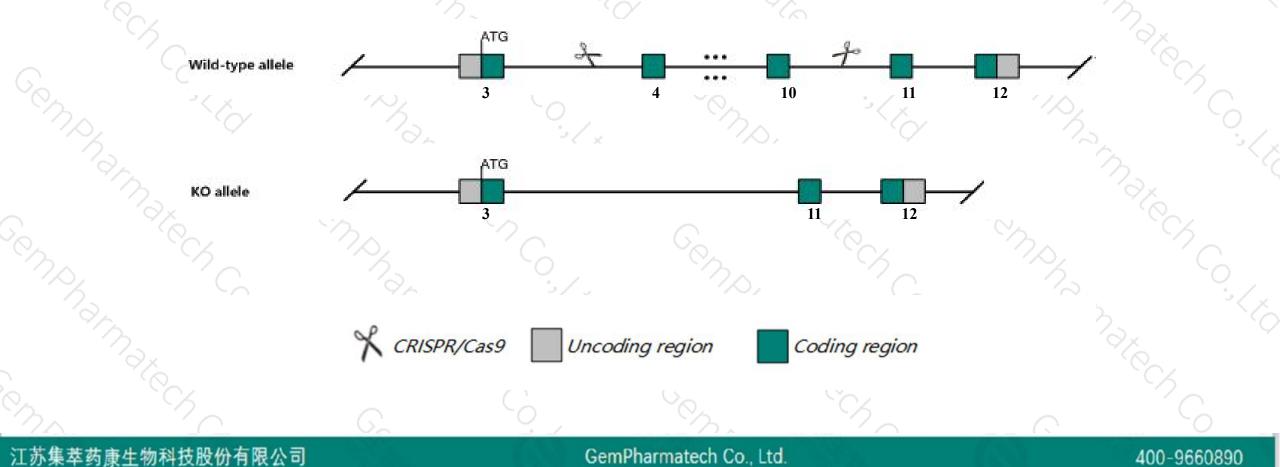




Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Capns1 gene. The schematic diagram is as follows:





- The Capns1 gene has 8 transcripts. According to the structure of Capns1 gene, exon4-exon10 of Capns1-201 (ENSMUST0000001845.12) transcript is recommended as the knockout region. The region contains 512bp coding sequence. Knock out the region will result in disruption of protein function.
- > In this project we use CRISPR/Cas9 technology to modify Capns1 gene. The brief process is as follows: CRISPR/Cas9 system

Notice



- According to the existing MGI data, Homozygous mutation of this gene results in embryonic lethality around E11.5. Mutant embryos exhibit cardiac developmental defects, reduced yolk sac vasculature, hemorrhaging in the area between the embryo and amnion, and accumulation of nucleated erythroid cells in the heart chambers, blood vessels, and developing liver.
 - This strategy may affect the 5-terminal regulation function of *Gm26810* gene.
- The Capns1 gene is located on the Chr7. 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.

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Gene information (NCBI)



Capns1 calpain, small subunit 1 [Mus musculus (house mouse)]

Gene ID: 12336, updated on 24-Sep-2019

Summary

Official Symbol	Capns1 provided by MGI
Official Full Name	calpain, small subunit 1 provided by MGI
Primary source	MGI:MGI:88266
See related	Ensembl:ENSMUSG0000001794
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	Cdps; Css1; Capa4; Capa4; Capa-4; D7Ertd146e
Expression	Ubiquitous expression in adrenal adult (RPKM 274.2), mammary gland adult (RPKM 214.7) and 28 other tissues See more
Orthologs	human all
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400-9660890

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Transcript information (Ensembl)



Name 🖕	Transcript ID	bp 🖕	Protein 🖕	Biotype 🖕	CCDS	UniProt 🖕	Flags		
Capns1-201	ENSMUST0000001845.12	1600	<u>268aa</u>	Protein coding	CCDS21082	A0A0R4IZW8	TSL:5	GENCODE basic	APPRIS P2
Capns1-203	ENSMUST00000126116.2	1429	<u>268aa</u>	Protein coding	CCDS21082	<u>A0A0R4IZW8</u> &	TSL:1	GENCODE basic	APPRIS P2
Capns1-202	ENSMUST00000108196.7	1192	<u>200aa</u>	Protein coding	-	A0A0R4J1C2	TSL:1	GENCODE basic	APPRIS ALT2
Capns1-204	ENSMUST00000129761.7	1157	No protein	Processed transcript	2	2	TSL:5 TSL:1 TSL:2 TSL:2 TSL:2		
Capns1-208	ENSMUST00000207082.1	1241	No protein	Retained intron	-	-			
Capns1-207	ENSMUST00000148973.7	600	No protein	Retained intron	-	-			
Capns1-206	ENSMUST00000146852.1	587	No protein	Retained intron	-	-			
Capns1-205	ENSMUST00000141851.1	507	No protein	Retained intron	2	<u>-</u>			

The strategy is based on the design of Capns1-201 transcript, The transcription is shown below

< Capns1-201 protein coding

Reverse strand -

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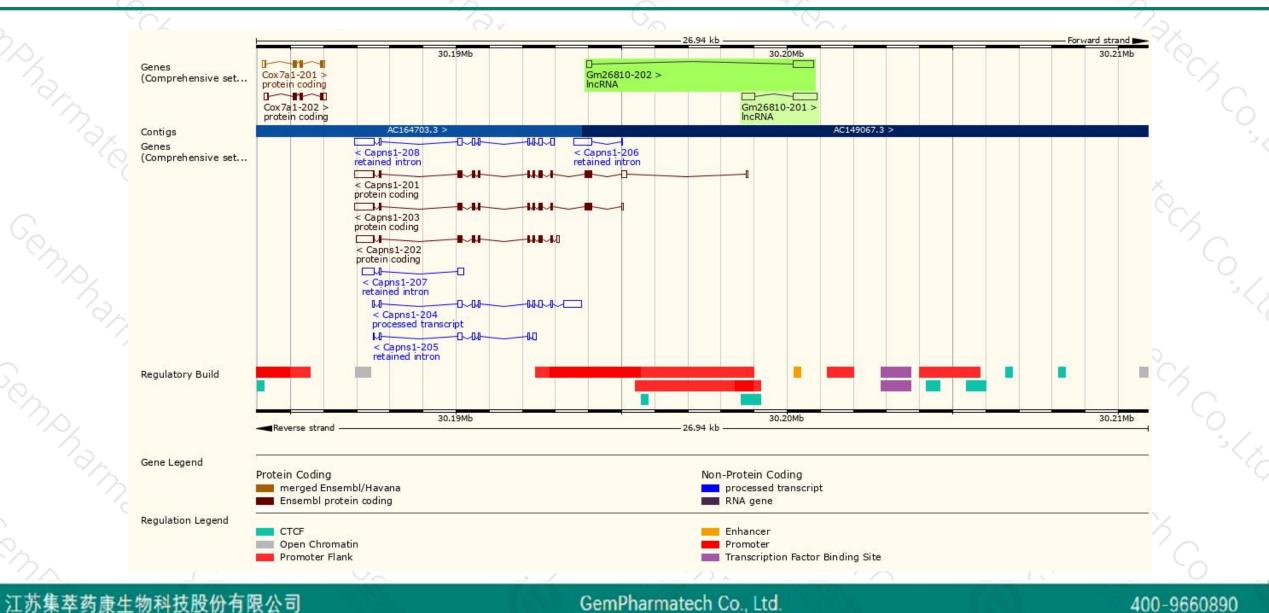
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11.87 kb

Genomic location distribution

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公司



集萃

集卒药康 GemPharmatech

Protein domain



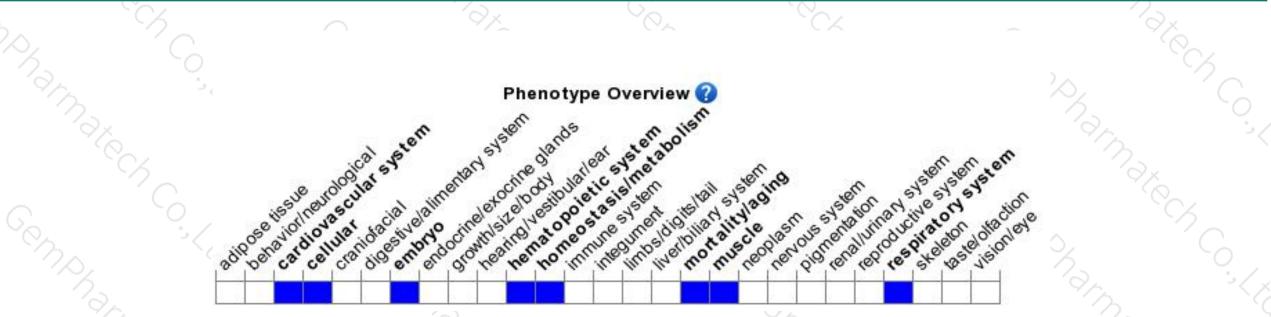
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Mouse phenotype description(MGI)





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, Homozygous mutation of this gene results in embryonic lethality around E11.5. Mutant embryos exhibit cardiac developmental defects, reduced yolk sac vasculature, hemorrhaging in the area between the embryo and amnion, and accumulation of nucleated erythroid cells in the heart chambers, blood vessels, and developing liver.

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



