

Slc17a6 Cas9-CKO Strategy

Designer: Lingyan Wu

Reviewer: Miaomiao Cui

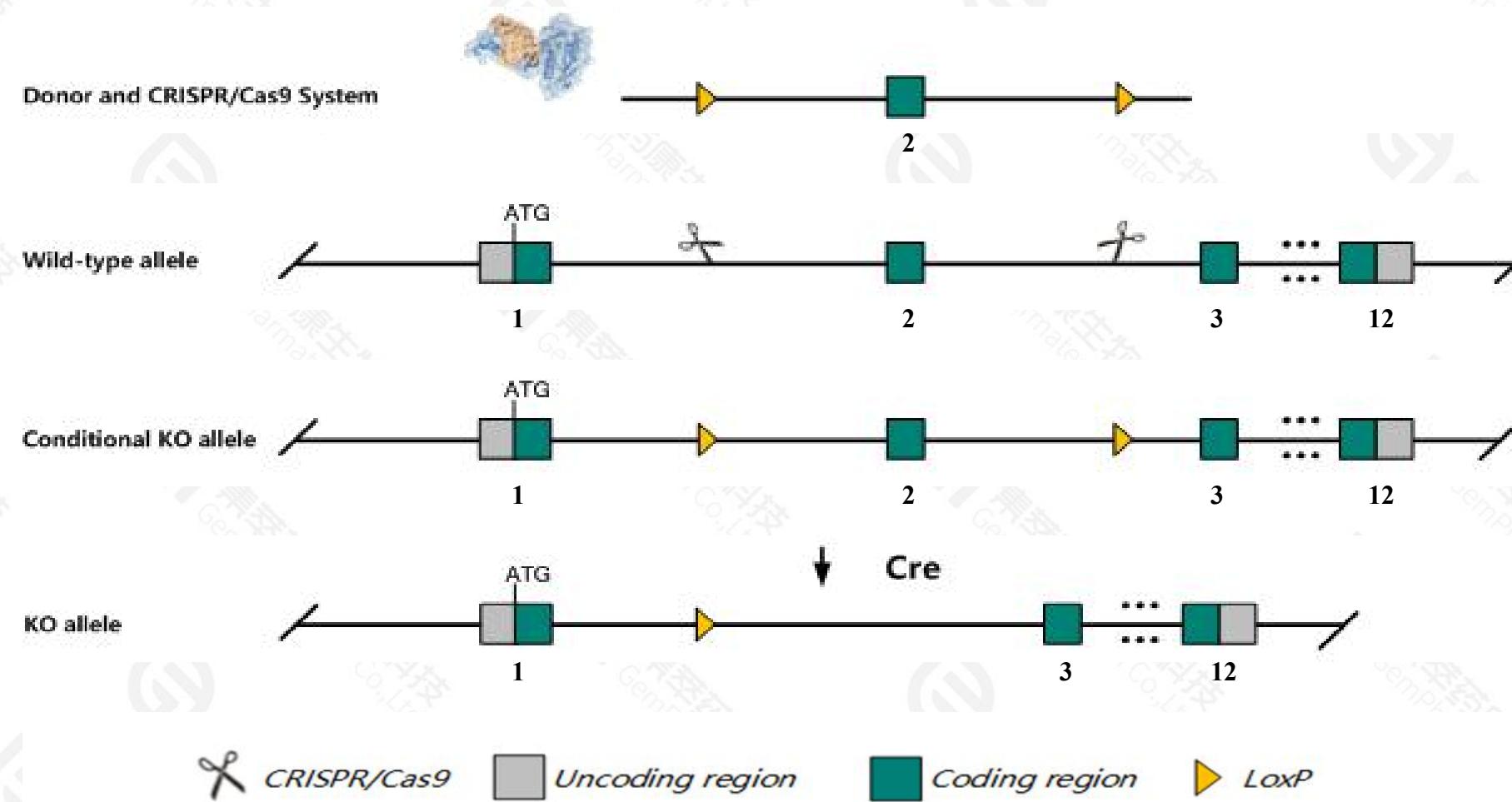
Design Date: 2021-4-23

Project Overview

Project Name	<i>Slc17a6</i>
Project type	Cas9-CKO
Strain background	C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc17a6* gene. The schematic diagram is as follows:



Technical routes

- The *Slc17a6* gene has 4 transcripts. According to the structure of *Slc17a6* gene, exon2 of *Slc17a6-201*(ENSMUST00000032710.7) transcript is recommended as the knockout region. The region contains 253bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Slc17a6* 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 null mutations display neonatal lethality, respiratory failure, and abnormal nervous system physiology. Heterozygous mice for one allele display abnormal miniature EPSC and reduced responses to neuropathic pain.
- The *Slc17a6* 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.

Gene information (NCBI)

Slc17a6 solute carrier family 17 (sodium-dependent inorganic phosphate cotransporter), member 6 [Mus musculus (house mouse)]

Gene ID: 140919, updated on 2-Mar-2021

Summary



Official Symbol Slc17a6 provided by MGI

Official Full Name solute carrier family 17 (sodium-dependent inorganic phosphate cotransporter), member 6 provided by MGI

Primary source MGI:MGU2156052

See related Ensembl:ENSMUSG00000030500

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 2900073D12Rik, DNPI, VGLU, VGLUT2

Expression Biased expression in CNS E18 (RPKM 12.2), whole brain E14.5 (RPKM 12.1) and 5 other tissues [See more](#)

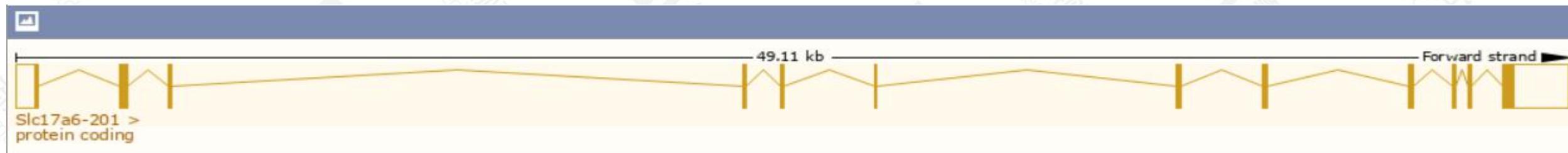
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

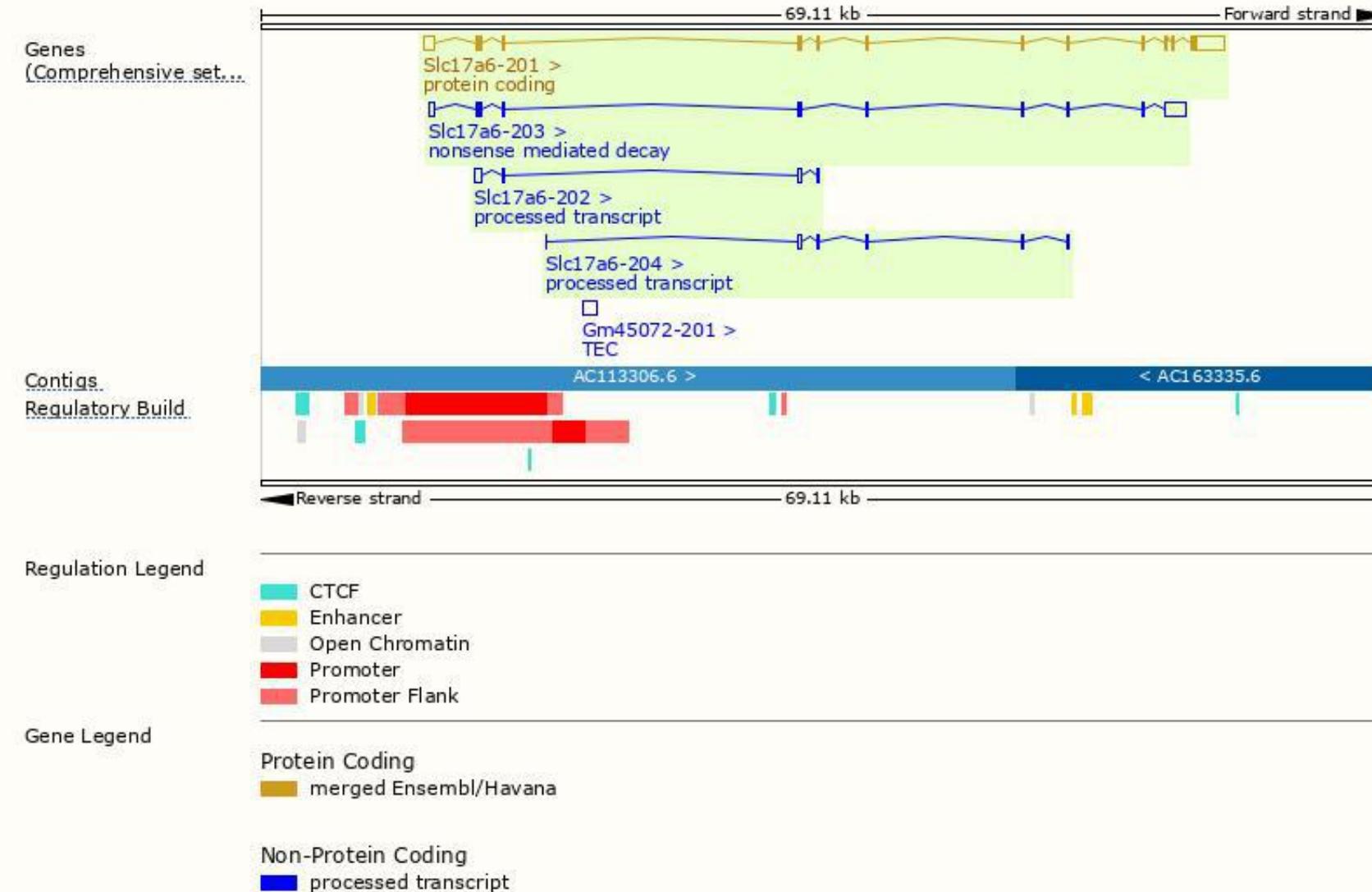
The gene has 4 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc17a6-201	ENSMUST00000032710.7	4137	<u>582aa</u>	Protein coding	CCDS39969		TSL:1 , GENCODE basic , APPRIS P1 ,
Slc17a6-203	ENSMUST00000207945.2	2710	<u>202aa</u>	Nonsense mediated decay	-		TSL:1 ,
Slc17a6-204	ENSMUST00000208597.2	663	No protein	Processed transcript	-		TSL:5 ,
Slc17a6-202	ENSMUST00000207375.2	655	No protein	Processed transcript	-		TSL:2 ,

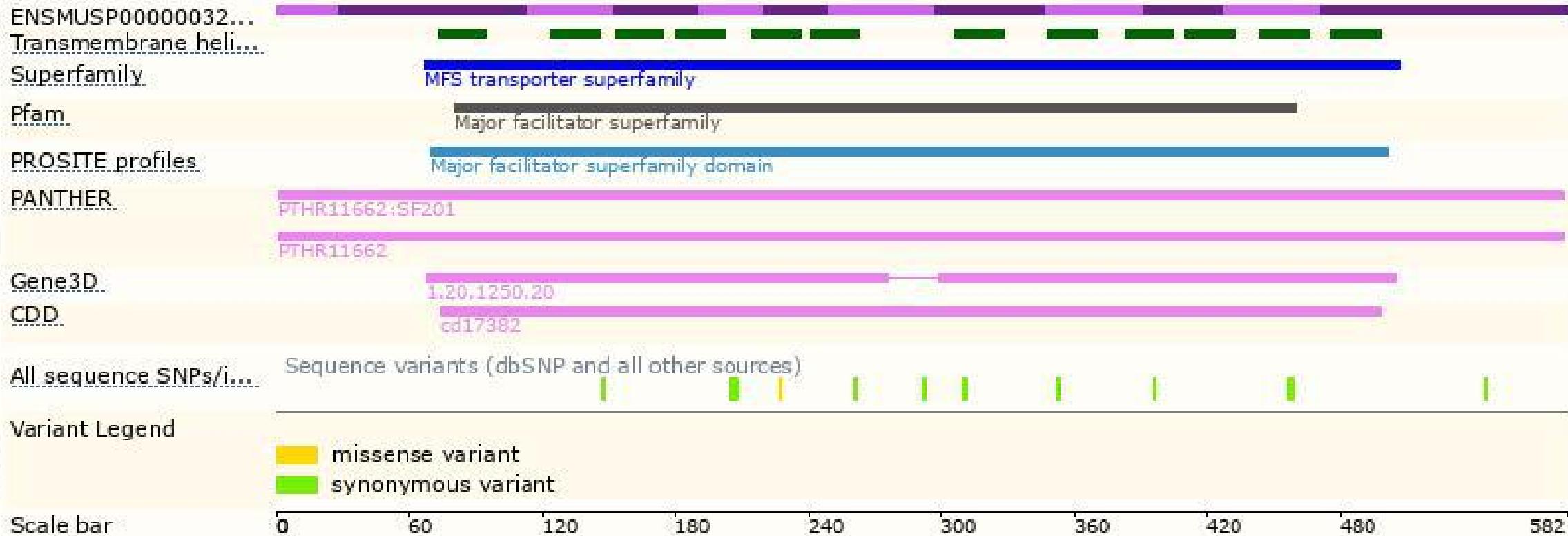
The strategy is based on the design of *Slc17a6-201* transcript, the transcription is shown below:



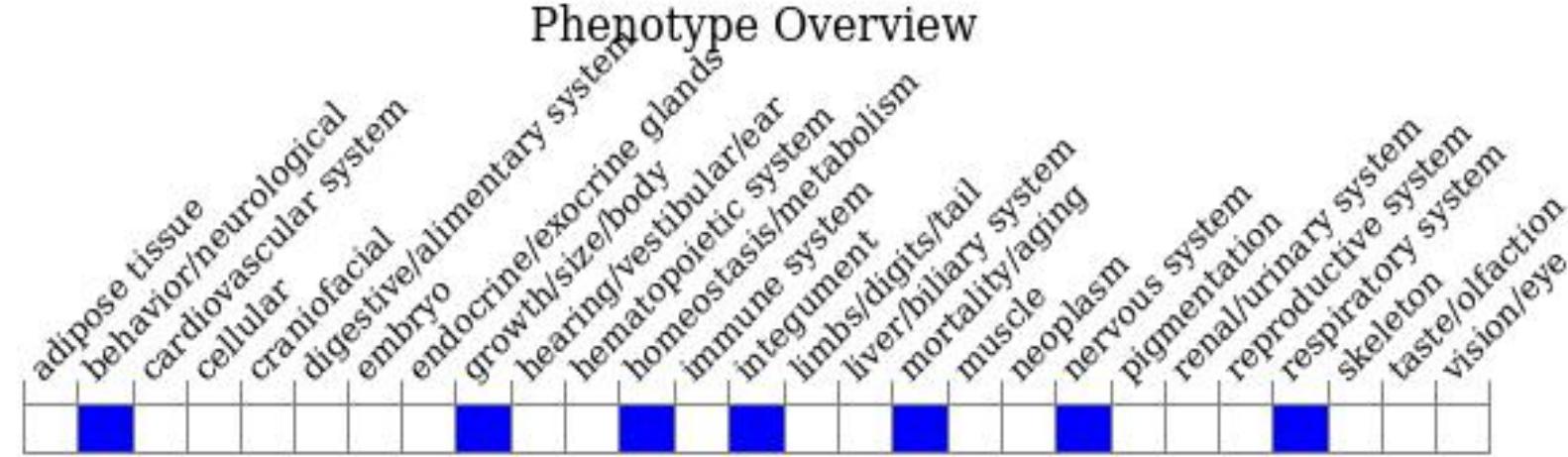
Genomic location distribution



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, mice homozygous for null mutations display neonatal lethality, respiratory failure, and abnormal nervous system physiology. Heterozygous mice for one allele display abnormal miniature EPSC and reduced responses to neuropathic pain.



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

