

Kcna3 Cas9-KO Strategy

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Project Overview



Project Name

Kcna3

Project type

Cas9-KO

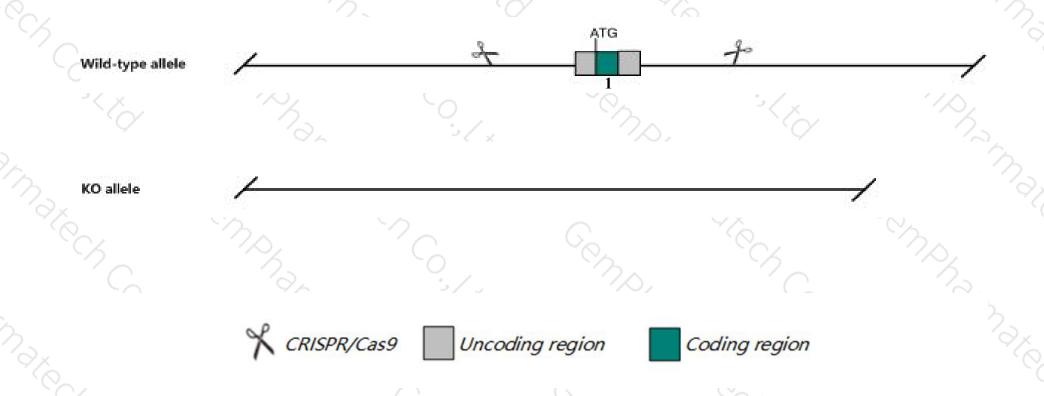
Strain background

C57BL/6JGpt

Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Kcna3 gene. The schematic diagram is as follows:



Technical routes



- ➤ The *Kcna3* gene has 1 transcript. According to the structure of *Kcna3* gene, exon1 of *Kcna3-201* (ENSMUST00000052718.4) 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.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Kcna3* gene. The brief process is as follows: gRNA was transcribed in vitro.Cas9 and gRNA 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.

Notice



- According to the existing MGI data, Mice homozygous for a knock-out allele display no apparent immune system defects. Mice homozygous for a different knock-out allele show reduced body weight, increased basal metabolic rate, resistance to diet-induced obesity, increased insulin sensitivity, and altered peripheral glucose metabolism.
- > The partial sequence of intron of Gm27008 gene will be deleted together in this strategy.
- ➤ The knockout region is near to the N-terminal of AI504432 gene, this strategy may influence the regulatory function of the N-terminal of AI504432 gene.
- The *Kcna3* 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)



Kcna3 potassium voltage-gated channel, shaker-related subfamily, member 3 [Mus musculus (house mouse)]

Gene ID: 16491, updated on 10-Oct-2019

Summary

☆ ?

Official Symbol Kcna3 provided by MGI

Official Full Name potassium voltage-gated channel, shaker-related subfamily, member 3 provided by MGI

Primary source MGI:MGI:96660

See related Ensembl:ENSMUSG00000047959

Gene type protein coding
RefSeq status VALIDATED
Organism <u>Mus musculus</u>

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

Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus

Also known as Mk-3; Kv1.3; Kca1-3

Orthologs human all

Genomic context



Location: 3 F2.3; 3 46.59 cM

See Kcna3 in Genome Data Viewer

Exon count: 1

Annotation release	Status	Assembly	Chr	Location	
108	current	GRCm38.p6 (GCF_000001635.26)	3	NC_000069.6 (107036162107038129)	
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	3	NC_000069.5 (106839080106841047)	

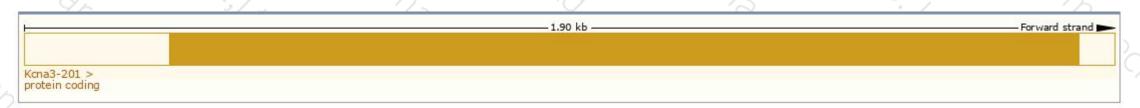
Transcript information (Ensembl)



The gene has 1 transcript, the transcript is shown below:

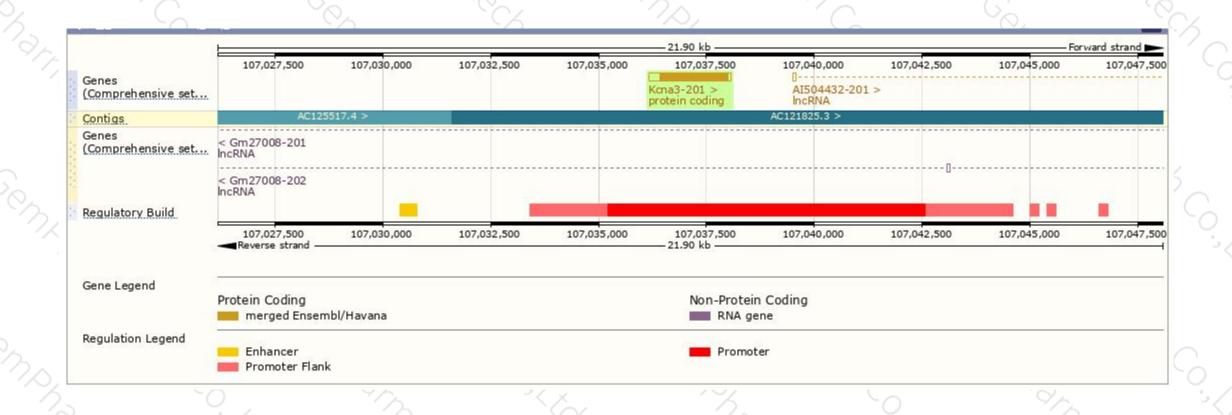
Name	Transcript ID 🍦	bp 🌲	Protein	Biotype 🍦	CCDS	UniProt	Flags		
Kcna3-201	ENSMUST00000052718.4	1902	<u>528aa</u>	Protein coding	CCDS17730 ₽	P16390 &	TSL:NA	GENCODE basic	APPRIS P1

The strategy is based on the design of *Kcna3-201* transcript, The transcription is shown below



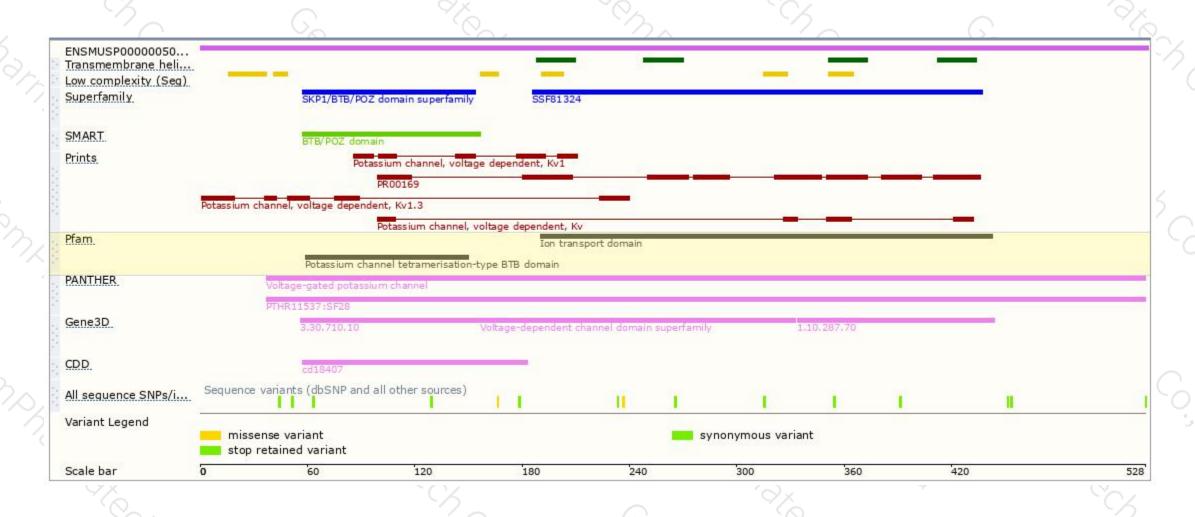
Genomic location distribution





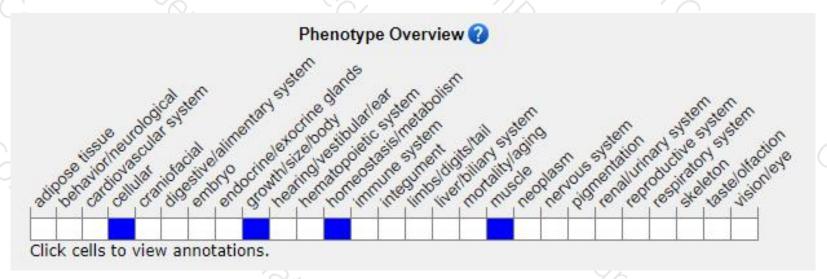
Protein domain





Mouse phenotype description(MGI)





Phenotypes affected by the gene are marked in blue.Data quoted from MGI database(http://www.informatics.jax.org/).

Mice homozygous for a knock-out allele display no apparent immune system defects. Mice homozygous for a different knock-out allele show reduced body weight, increased basal metabolic rate, resistance to diet-induced obesity, increased insulin sensitivity, and altered peripheral glucose metabolism.



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





