

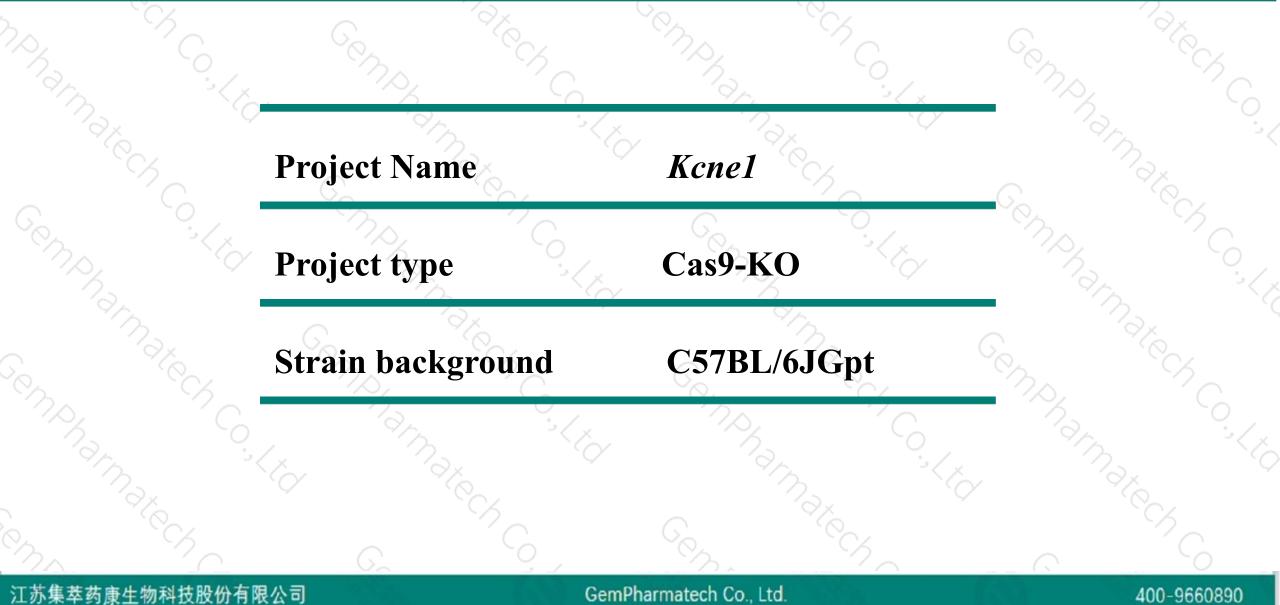
# Kcnel Cas9-KO Strategy

Designer:Xueting Zhang Reviewer:Yanhua Shen Date:2019-11-24

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# **Project Overview**

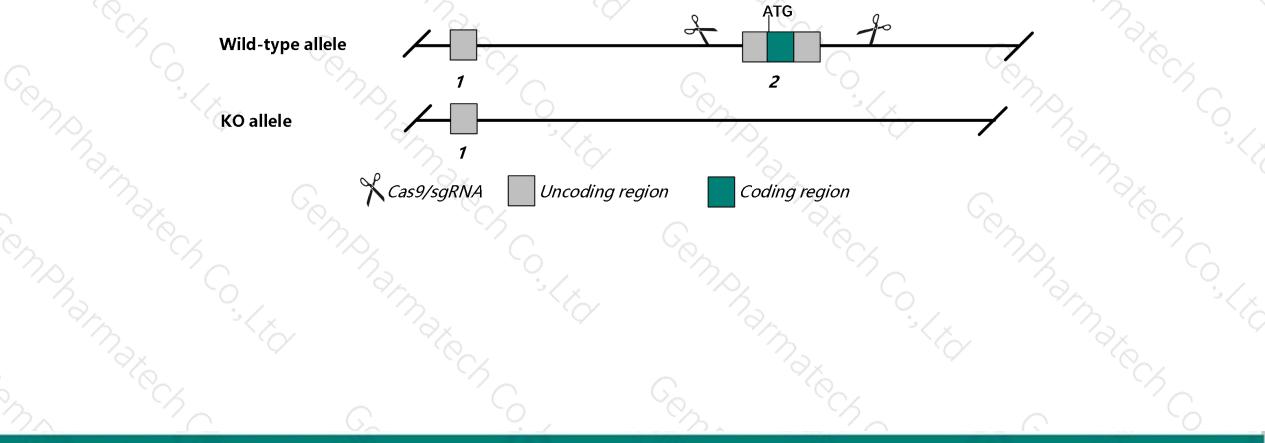






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This model will use CRISPR/Cas9 technology to edit the Kcnel gene. The schematic diagram is as follows:





- The Kcnel gene has 2 transcripts. According to the structure of Kcnel gene, exon2 of Kcnel-201 (ENSMUST00000051705.6) 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 Kcnel gene. The brief process is as follows: CRISPR/Cas9 system



- According to the existing MGI data, Homozygotes for targeted and spontaneous null mutations exhibit head-shaking, circling, ataxia, and severe deafness associated with inner ear defects. Older mutants show increased numbers of T cells. Study of cardiac myocytes in one line revealed physiologic defects.
- The Kcnel gene is located on the Chr16. 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)



Kcne1 potassium voltage-gated channel, lsk-related subfamily, member 1 [ Mus musculus (house mouse) ] Gene ID: 16509, updated on 24-Oct-2019 Summary ☆ ? Official Symbol Kcne1 provided by MGI Official Full Name potassium voltage-gated channel, Isk-related subfamily, member 1 provided by MGI Primary source MGI:MGI:96673 See related Ensembl:ENSMUSG00000039639 Gene type protein coding RefSeg 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 Isk; MinK; nmf190 Expression Biased expression in kidney adult (RPKM 8.7), heart adult (RPKM 4.7) and 2 other tissues See more Orthologs human all Genomic context **^** Location: 16 C4; 16 53.57 cM See Kcne1 in Genome Data Viewer Exon count: 3 Annotation release Chr Status Assembly Location current 108

 current
 GRCm38.p6 (GCF\_000001635.26)
 16
 NC\_000082.6 (92346001..92359468, complement)

 previous assembly
 MGSCv37 (GCF\_000001635.18)
 16
 NC\_000082.5 (92346246..92359713, complement)

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**Build 37.2** 

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# **Transcript information (Ensembl)**



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### The gene has 2 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Kcne1-201	ENSMUST00000051705.6	3174	<u>129aa</u>	Protein coding	CCDS28336	P23299 Q545H6	TSL:1 GENCODE basic APPRIS P1
Kcne1-202	ENSMUST00000166707.2	602	<u>129aa</u>	Protein coding	CCDS28336	P23299 Q545H6	TSL:1 GENCODE basic APPRIS P1

The strategy is based on the design of *Kcne1-201* transcript, The transcription is shown below

< Kcne1-201 protein coding

Reverse strand

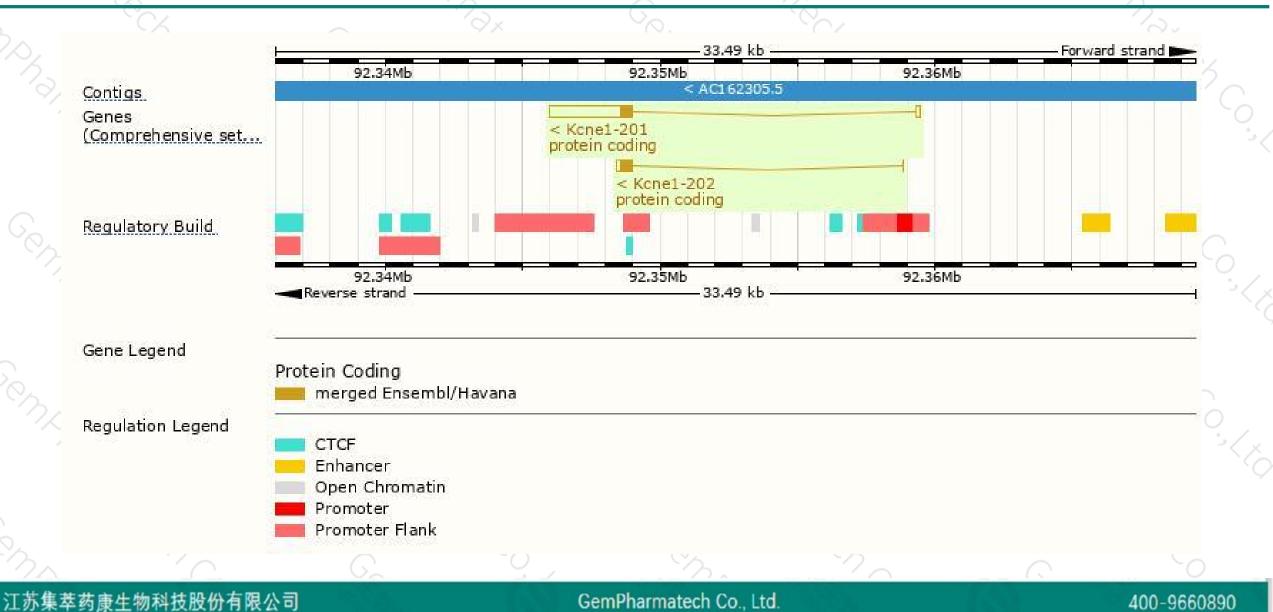
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13.49 kb

# **Genomic location distribution**





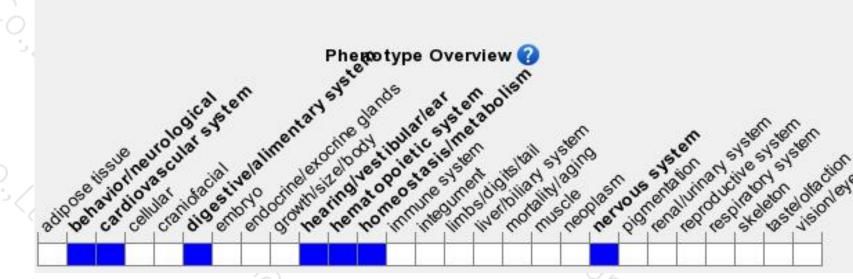
# **Protein domain**



		COND,	°°%	ns,	6	GON	
	ENSMUSP00000052 Transmembrane heli Prints		Potas	sium channel, voltage	-dependent, beta su	bunit, KCNE	2 0
Con	Pfam PANTHER	Potassium channel, volt Potassium channel, volt Potassium channel, voltag		ubunit, KCNE			6
	All sequence SNPs/i	Sequence variants (dbS	NP and all other sou	rces)	110		
S CNX	Variant Legend	missense variant	ant				0
	Scale bar	0 20	40	60	80	100	129
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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, Homozygotes for targeted and spontaneous null mutations exhibit head-shaking, circling, ataxia, and severe deafness associated with inner ear defects. Older mutants show increased numbers of T cells. Study of cardiac myocytes in one line revealed physiologic defects.

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



