

# ***Klrk1* Cas9-KO Strategy**

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**Reviewer: Xiaojing Li**

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# Project Overview

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**Project Name**

***Klrk1***

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**Project type**

**Cas9-KO**

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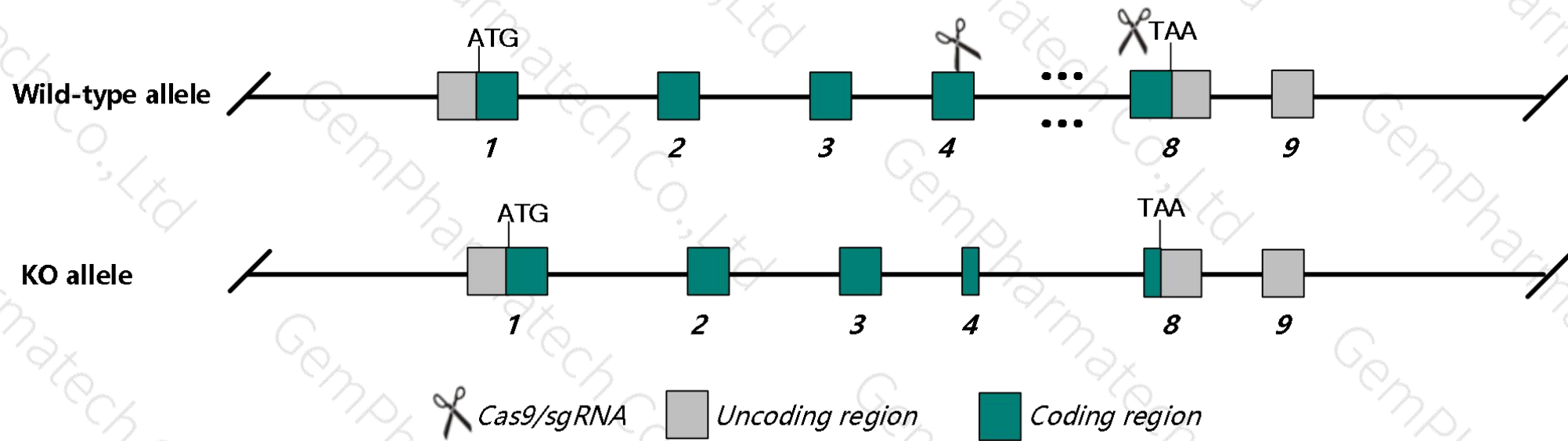
**Strain background**

**BALB/cJGpt**

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# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Klrk1* gene. The schematic diagram is as follows:



# Technical routes

- The *Klrk1* gene has 6 transcripts. According to the structure of *Klrk1* gene, exon4-exon8 of MGP\_BALBcJ\_T0080481.1 transcript is recommended as the knockout region. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Klrk1* gene. The brief process is as follows: sgRNA was transcribed in vitro. Cas9 and sgRNA were microinjected into the fertilized eggs of BALB/cJGpt 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 BALB/cJGpt mice.

- According to the existing MGI data, homozygous null mice have defects in Natural Killer (NK) cell development, diminished NK-mediated cytolysis of tumor cells, and resistance to MCMV infection.
- The *Klrkl* gene is located on the Chr6. 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)

## Klrk1 killer cell lectin-like receptor subfamily K, member 1 [Mus musculus (house mouse)]

Gene ID: 27007, updated on 31-Jan-2019

### Summary



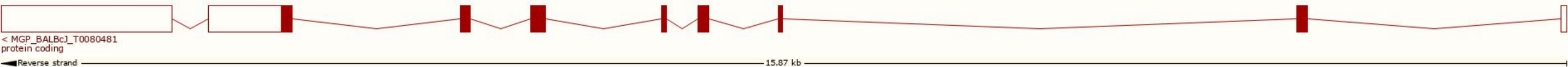
<b>Official Symbol</b>	Klrk1 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	killer cell lectin-like receptor subfamily K, member 1 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:1196250</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000030149</a>
<b>Gene type</b>	protein coding
<b>RefSeq status</b>	VALIDATED
<b>Organism</b>	<a href="#">Mus musculus</a>
<b>Lineage</b>	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
<b>Also known as</b>	D6H12S2489E, NKG2-D, Nkg2d
<b>Expression</b>	Low expression observed in reference dataset <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

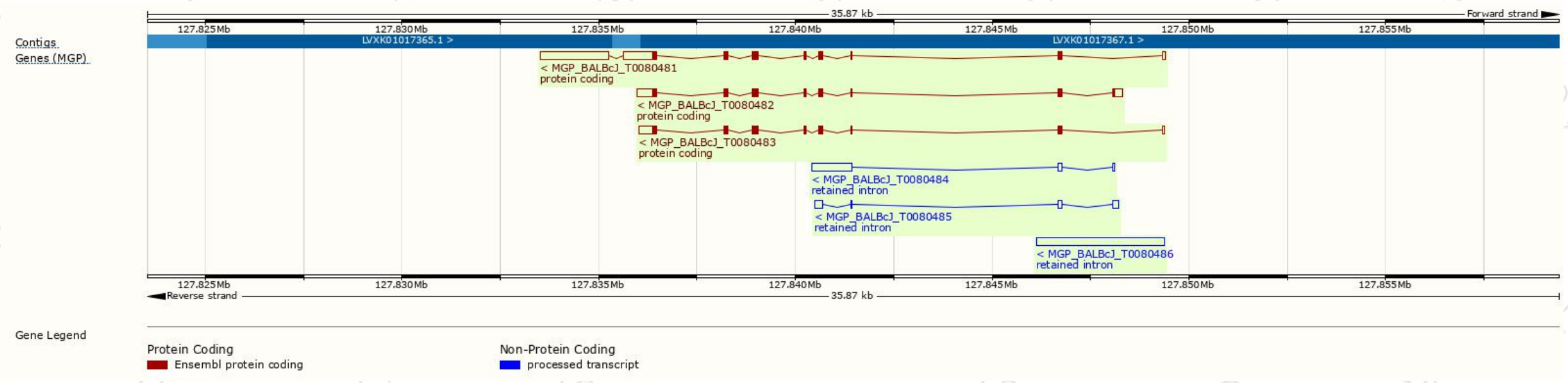
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
-	<a href="#">MGP_BALBcJ_T0080481.1</a>	3205	<a href="#">225aa</a>	Protein coding	<a href="#">CCDS39660</a> , <a href="#">CCDS39661</a> , <a href="#">CCDS71846</a>	<a href="#">O54709</a> , <a href="#">Q3TCW7</a> , <a href="#">Q4FJM0</a>	-
-	<a href="#">MGP_BALBcJ_T0080482.1</a>	1281	<a href="#">232aa</a>	Protein coding	-	-	-
-	<a href="#">MGP_BALBcJ_T0080483.1</a>	1030	<a href="#">219aa</a>	Protein coding	-	-	-
-	<a href="#">MGP_BALBcJ_T0080486.1</a>	3261	No protein	Retained intron	-	-	-
-	<a href="#">MGP_BALBcJ_T0080484.1</a>	1182	No protein	Retained intron	-	-	-
-	<a href="#">MGP_BALBcJ_T0080485.1</a>	501	No protein	Retained intron	-	-	-

The strategy is based on the design of *MGP\_BALBcJ\_T0080481.1* transcript,the transcription is shown below:

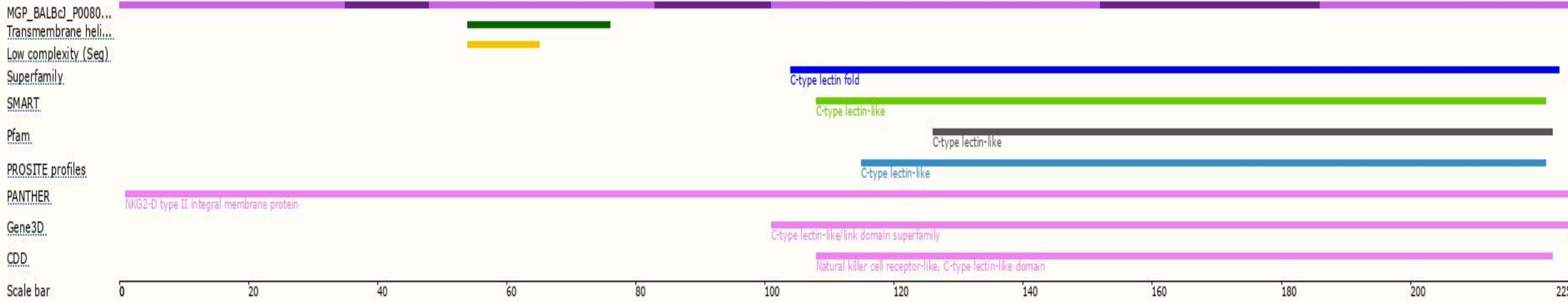


# Genomic location distribution

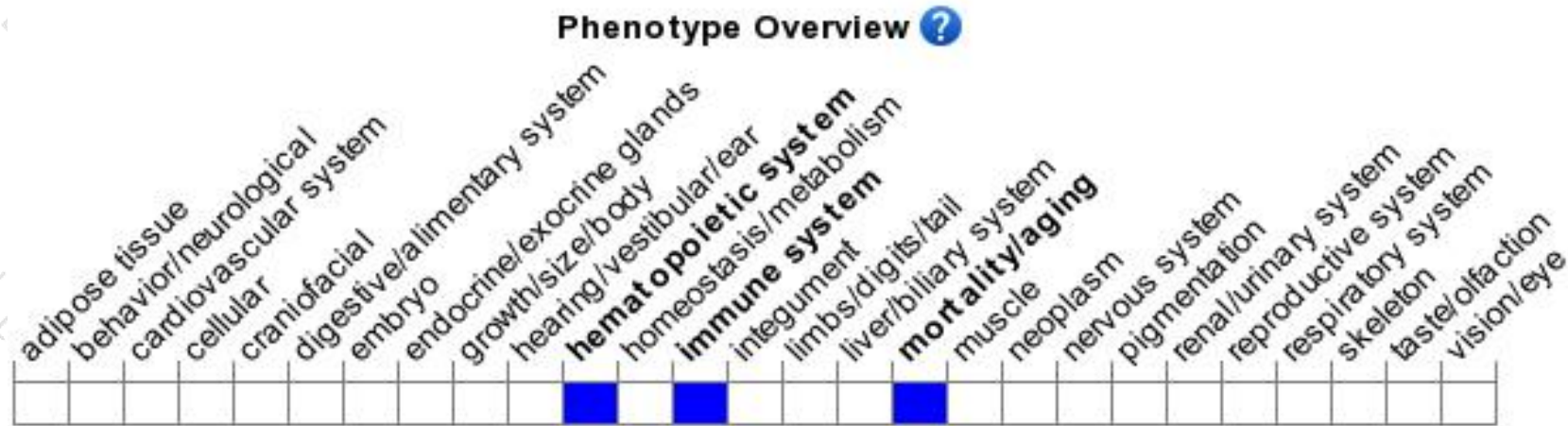




# Protein domain



# 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, homozygous null mice have defects in Natural Killer (NK) cell development, diminished NK-mediated cytolysis of tumor cells, and resistance to MCMV infection.

If you have any questions, you are welcome to inquire.

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