

Rel Cas9-CKO Strategy

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Project Overview



Project Name Rel

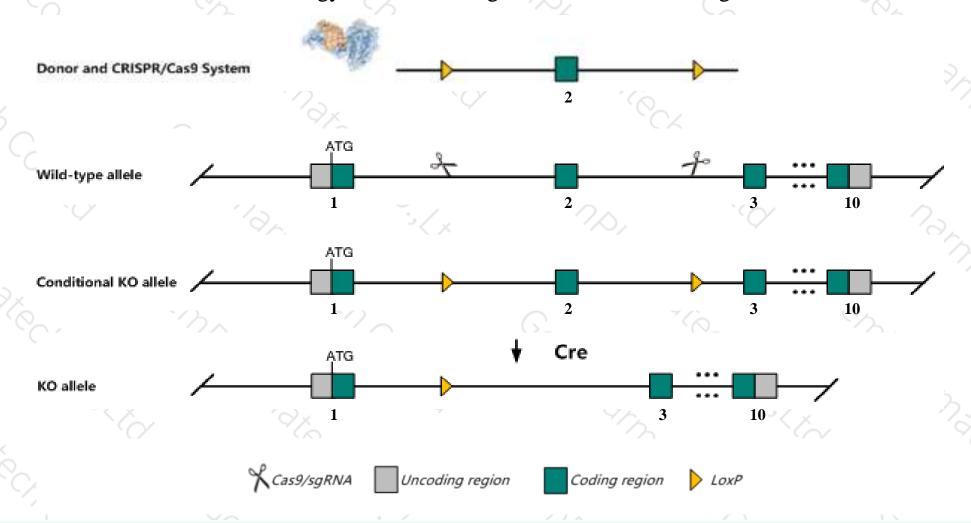
Project type Cas9-CKO

Strain background C57BL/6JGpt

Conditional Knockout strategy



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



Technical routes



- ➤ The *Rel* gene has 1 transcript. According to the structure of *Rel* gene, exon2 of *Rel-201*(ENSMUST00000102864.4) transcript is recommended as the knockout region. The region contains 143bp coding sequence.

 Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Rel* gene. The brief process is as follows:sgRNA was transcribed in vitro, donor vector was constructed.Cas9, sgRNA 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 was 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, Homozygous inactivation of this gene causes defects in lymphocyte proliferation, humoral immunity and cytokine production, and may lead to impaired Th1 responses and resistance to autoimmune disease. Mice lacking only the COOH-terminal region show severehemopoietic defects and lymphoid hyperplasia.
- ➤ The *Rel* gene is located on the Chr11. 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)



Rel reticuloendotheliosis oncogene [Mus musculus (house mouse)]

Gene ID: 19696, updated on 20-Feb-2019

Summary



Official Symbol Rel provided by MGI

Official Full Name reticuloendotheliosis oncogene provided by MGI

Primary source MGI:MGI:97897

See related Ensembl: ENSMUSG00000020275

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 c-Rel

Expression Broad expression in spleen adult (RPKM 6.1), thymus adult (RPKM 3.0) and 21 other tissues See more

Orthologs <u>human</u> <u>all</u>

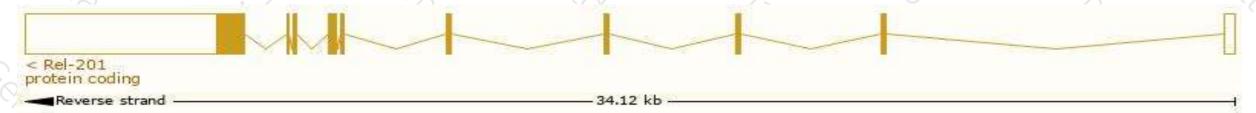
Transcript information (Ensembl)



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

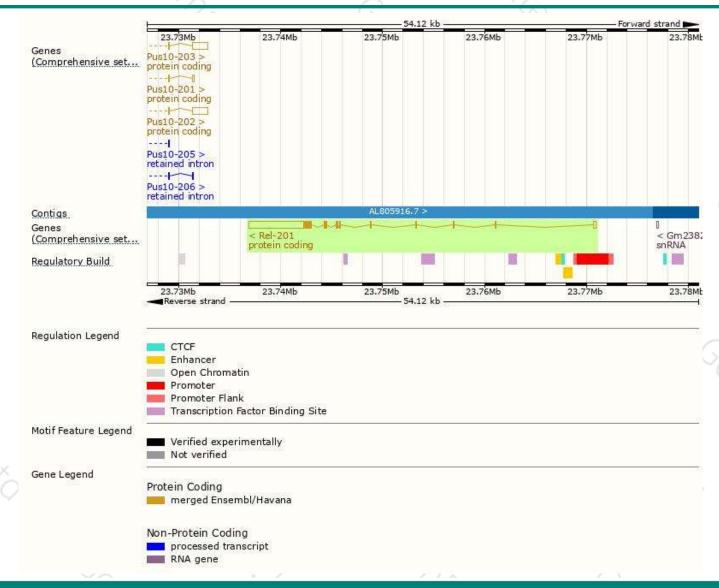
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags	
Rel-201	ENSMUST00000102864.4	7466	<u>588aa</u>	Protein coding	CCDS24480	A4QPD3	TSL:1 GENCODE basic APPRIS P1	1

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



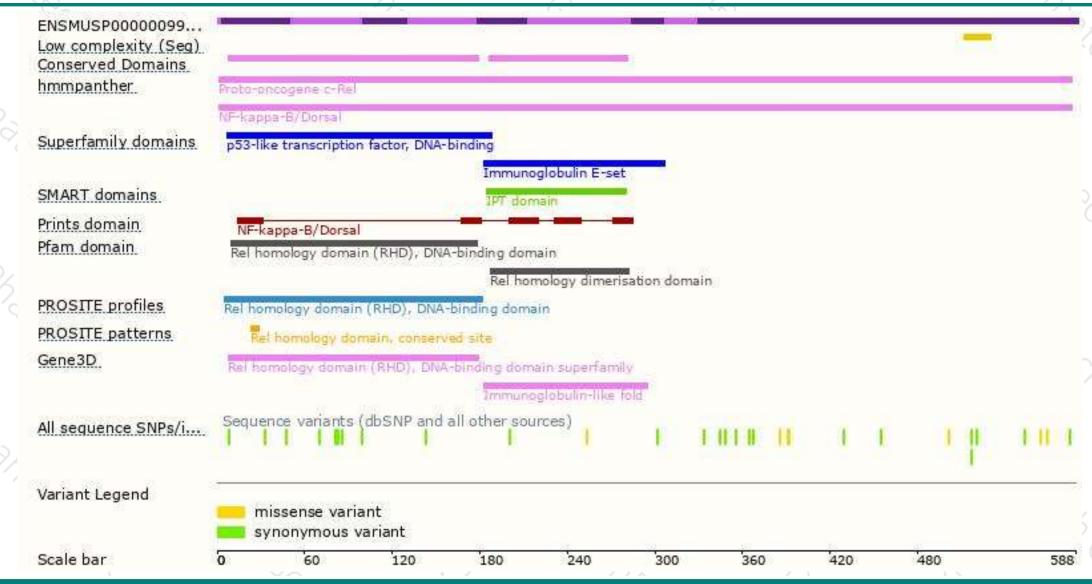
Genomic location distribution





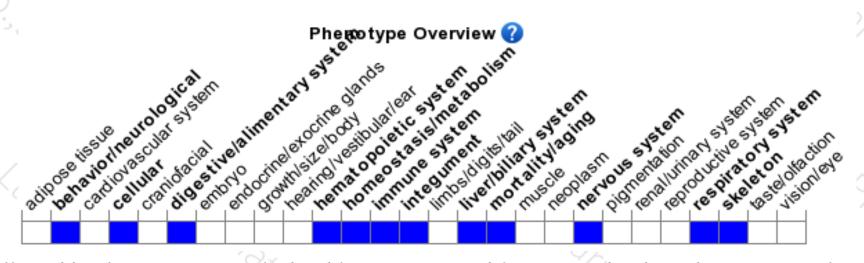
Protein domain





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, Homozygous inactivation of this gene causes defects in lymphocyte proliferation, humoral immunity and cytokine production, and may lead to impaired Th1 responses and resistance to autoimmune disease. Mice lacking only the COOH-terminal region show severehemopoietic defects and lymphoid hyperplasia.



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

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