

***Relb* Cas9-CKO Strategy**

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Design Date: 2019-8-15

Project Overview

Project Name

Relb

Project type

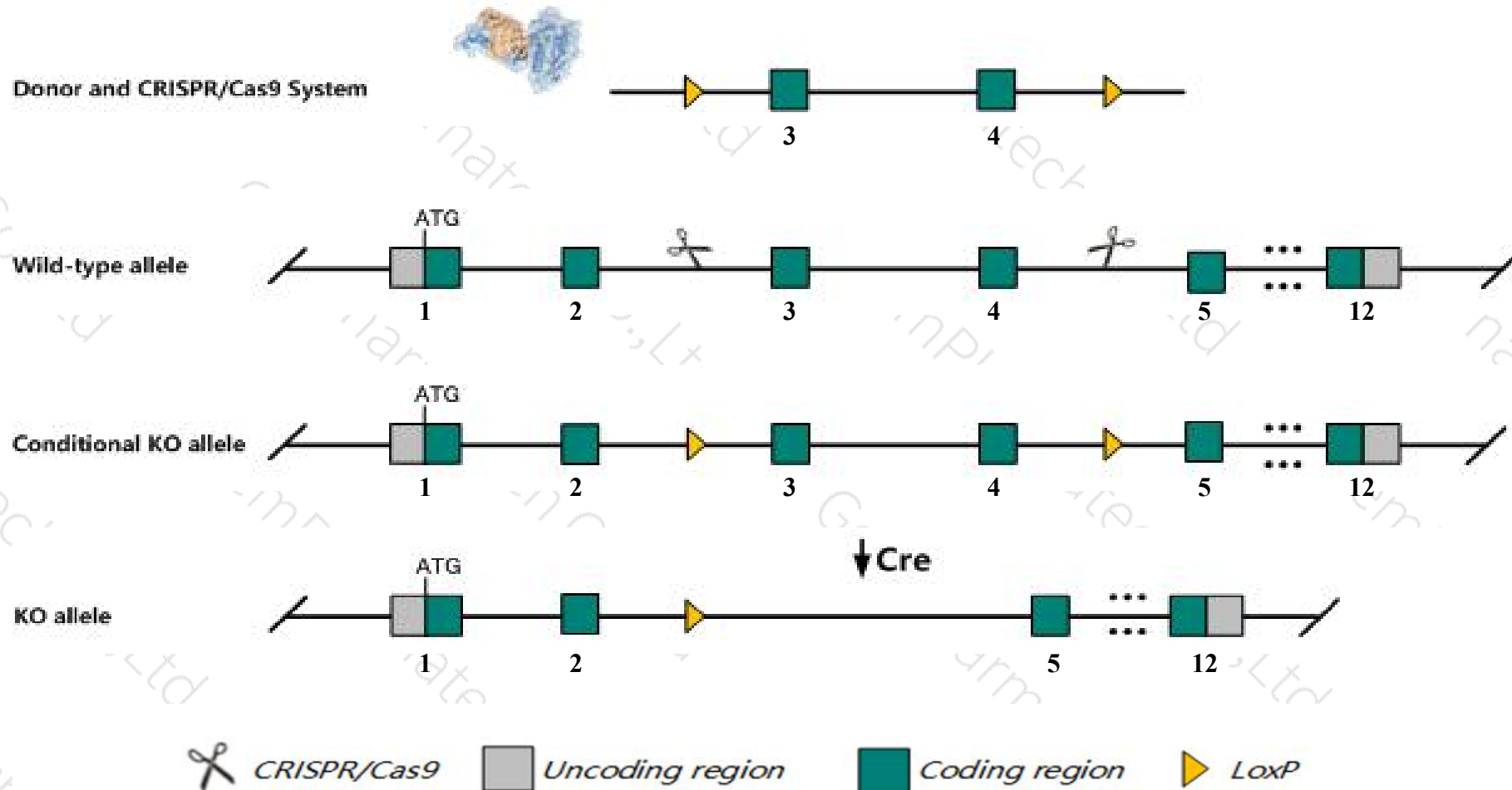
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

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



Technical routes

- The *Relb* gene has 10 transcripts. According to the structure of *Relb* gene, exon3-exon4 of *Relb*-202 (ENSMUST00000094762.9) transcript is recommended as the knockout region. The region contains 338bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Relb* 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.

- According to the existing MGI data, Mutant homozygotes die prematurely with phenotypes including inflammatory cell infiltration of organs, myeloid hyperplasia, splenomegaly, reduction in thymic dendritic cells, impaired cellular immunity, hyperkeratosis, epidermal hyperplasia, or hepatitis with mononuclear infiltration.
- The *Relb* 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)

Relb avian reticuloendotheliosis viral (v-rel) oncogene related B [Mus musculus (house mouse)]

Gene ID: 19698, updated on 9-Apr-2019

Summary



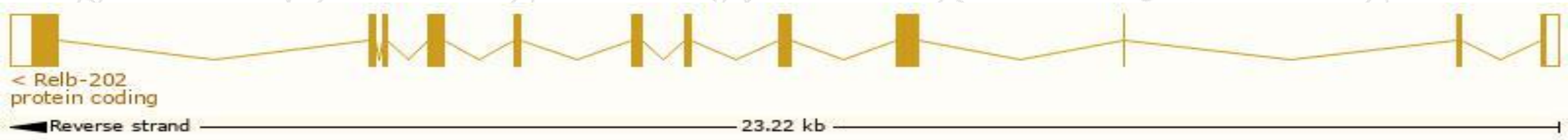
Official Symbol	Relb provided by MGI
Official Full Name	avian reticuloendotheliosis viral (v-rel) oncogene related B provided by MGI
Primary source	MGI:MGI:103289
See related	Ensembl:ENSMUSG000000002983
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	shep
Expression	Broad expression in spleen adult (RPKM 33.2), adrenal adult (RPKM 28.7) and 15 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

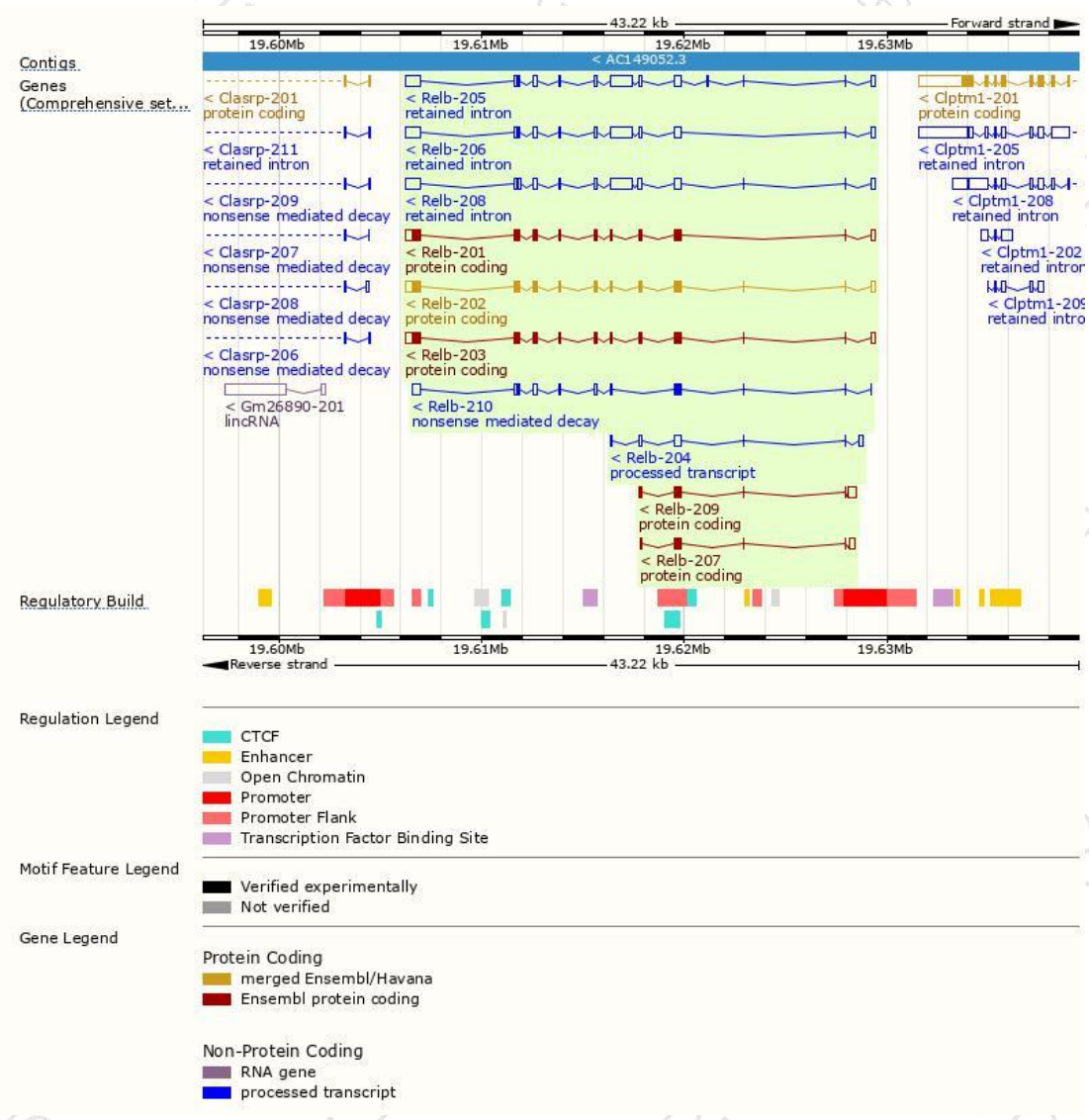
The gene has 10 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Relb-202	ENSMUST00000094762.9	2209	558aa	Protein coding	CCDS39799	Q04863	TSL:1 GENCODE basic APPRIS P3
Relb-201	ENSMUST00000049912.14	2200	555aa	Protein coding	CCDS71896	Q8K220	TSL:1 GENCODE basic APPRIS ALT2
Relb-203	ENSMUST00000098754.4	2203	558aa	Protein coding	-	Q04863	TSL:5 GENCODE basic APPRIS ALT2
Relb-209	ENSMUST00000153309.7	931	138aa	Protein coding	-	A0A140LI46	CDS 3' incomplete TSL:3
Relb-207	ENSMUST00000141586.1	727	116aa	Protein coding	-	A0A140LI24	CDS 3' incomplete TSL:5
Relb-210	ENSMUST00000208087.1	1525	147aa	Nonsense mediated decay	-	A0A140LJD6	TSL:1
Relb-204	ENSMUST00000130543.7	784	No protein	Processed transcript	-	-	TSL:5
Relb-208	ENSMUST00000148040.7	3273	No protein	Retained intron	-	-	TSL:1
Relb-205	ENSMUST00000131759.7	3210	No protein	Retained intron	-	-	TSL:1
Relb-206	ENSMUST00000137615.7	3166	No protein	Retained intron	-	-	TSL:1

The strategy is based on the design of *Relb-202* transcript,The transcription is shown below



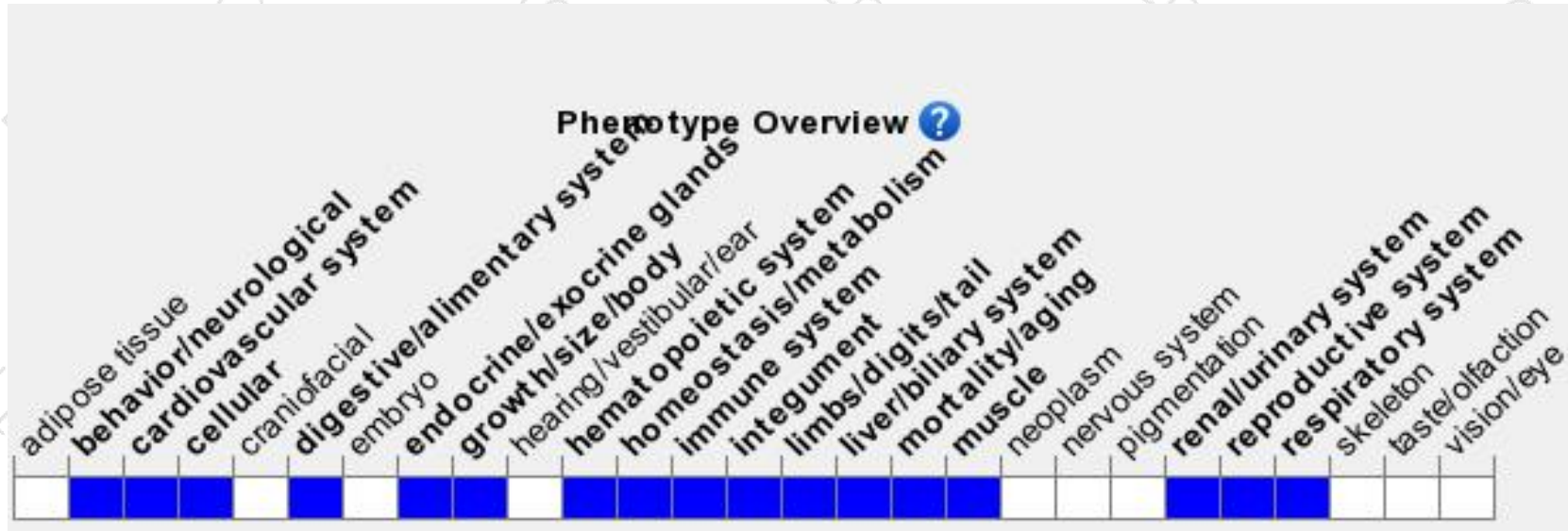
Genomic location distribution



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, Mutant homozygotes die prematurely with phenotypes including inflammatory cell infiltration of organs, myeloid hyperplasia, splenomegaly, reduction in thymic dendritic cells, impaired cellular immunity, hyperkeratosis, epidermal hyperplasia, or hepatitis with mononuclear infiltration.

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

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