

Il18rap Cas9-CKO Strategy

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Project Overview

Project Name

Il18rap

Project type

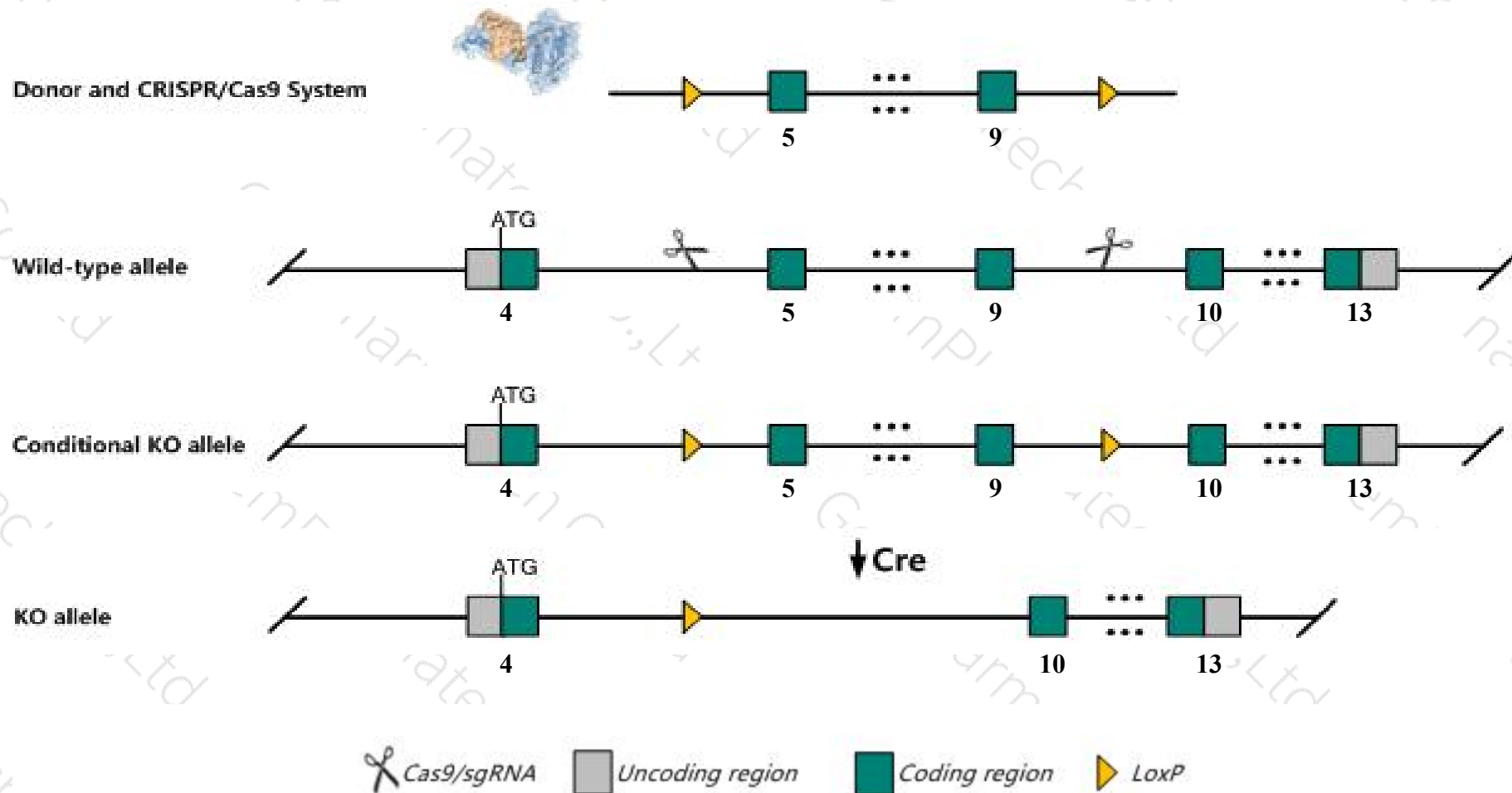
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Il18rap* gene. The schematic diagram is as follows:



Technical routes

- The *Il18rap* gene has 4 transcripts. According to the structure of *Il18rap* gene, exon5-exon9 of *Il18rap*-201 (ENSMUST00000027237.11) transcript is recommended as the knockout region. The region contains 847bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Il18rap* 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.

- According to the existing MGI data, Homozygous null mice exhibit defective IL-18-mediated immune responses such as the inability of splenocytes, T helper 1 cells and neutrophils to produce cytokines in response to IL-18.
- The *Il18rap* gene is located on the Chr1. 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)

Il18rap interleukin 18 receptor accessory protein [Mus musculus (house mouse)]

Gene ID: 16174, updated on 31-Jan-2019

Summary



Official Symbol Il18rap provided by [MGI](#)

Official Full Name interleukin 18 receptor accessory protein provided by [MGI](#)

Primary source [MGI:MGI:1338888](#)

See related [Ensembl:ENSMUSG00000026068](#)

Gene type protein coding

RefSeq status REVIEWED

Organism [Mus musculus](#)

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus

Also known as AcPL, IL-18R-beta, IL-18RAcP, IL-18Rbeta, IL-1RAcPL

Summary Interleukin-18 (or interferon-gamma inducing factor) is a proinflammatory cytokine that induces cell-mediated immunity following microbial infection. This gene encodes a member of the interleukin-1 receptor family. The encoded protein is an accessory subunit of the receptor for interleukin-18 and mediates signaling through this cytokine. Mice lacking this gene exhibit a defective cell-mediated immune response. [provided by RefSeq, Jan 2014]

Expression Biased expression in liver E18 (RPKM 1.1), spleen adult (RPKM 0.9) and 13 other tissues [See more](#)

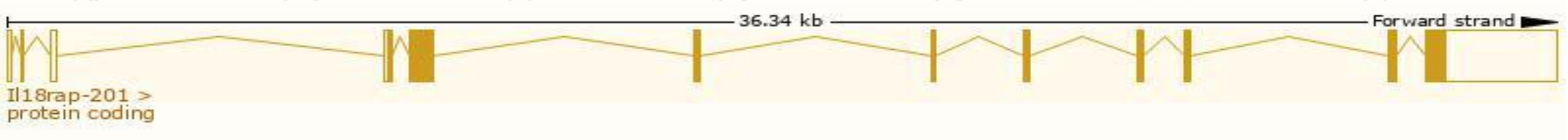
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

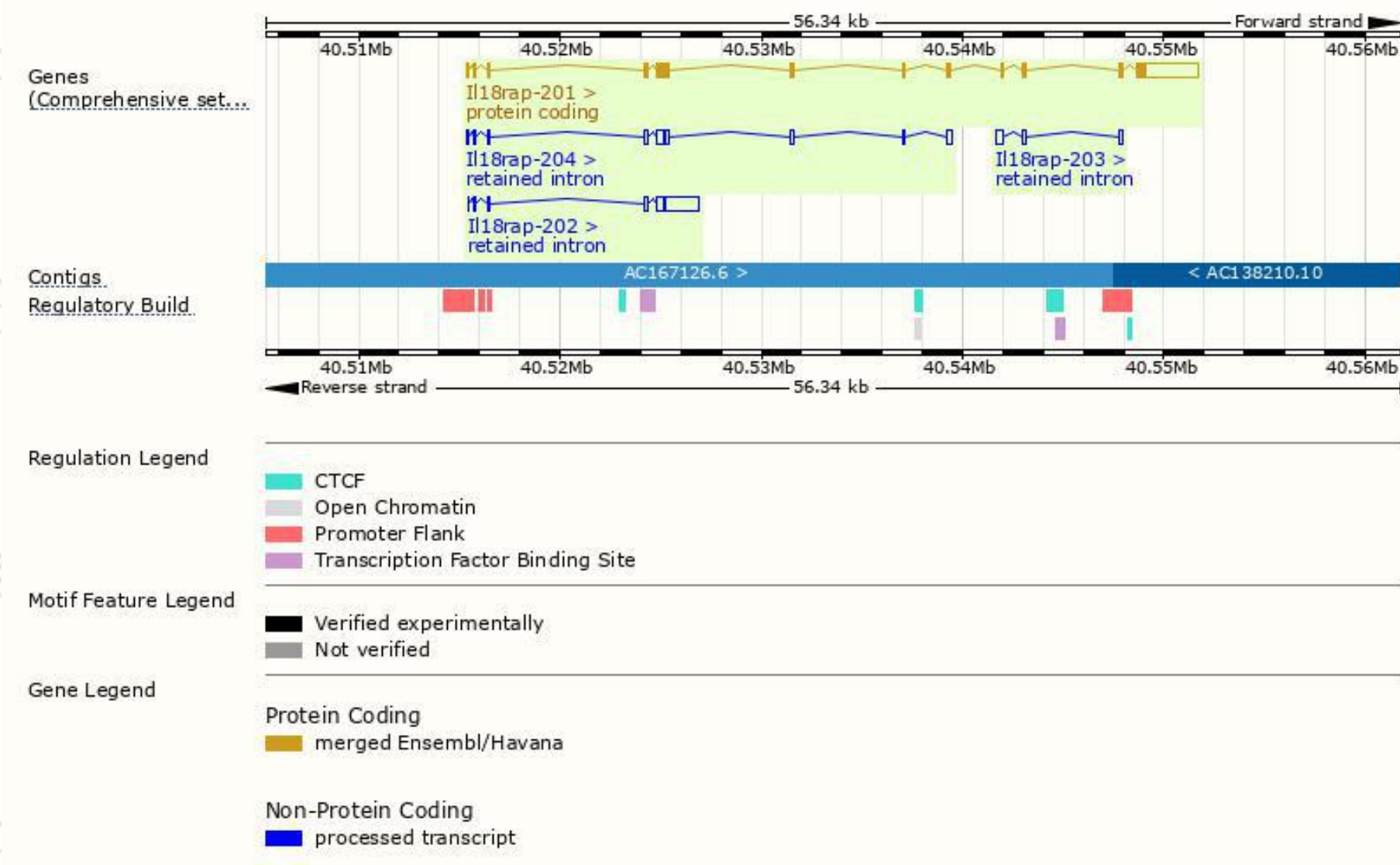
The gene has 4 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Il18rap-201	ENSMUST00000027237.11	4889	614aa	Protein coding	CCDS14912	Q0VBK3 Q9Z2B1	TSL:1 GENCODE basic APPRIS P1
Il18rap-202	ENSMUST00000159724.1	2447	No protein	Retained intron	-	-	TSL:2
Il18rap-204	ENSMUST00000163057.7	1439	No protein	Retained intron	-	-	TSL:2
Il18rap-203	ENSMUST00000160468.1	622	No protein	Retained intron	-	-	TSL:3

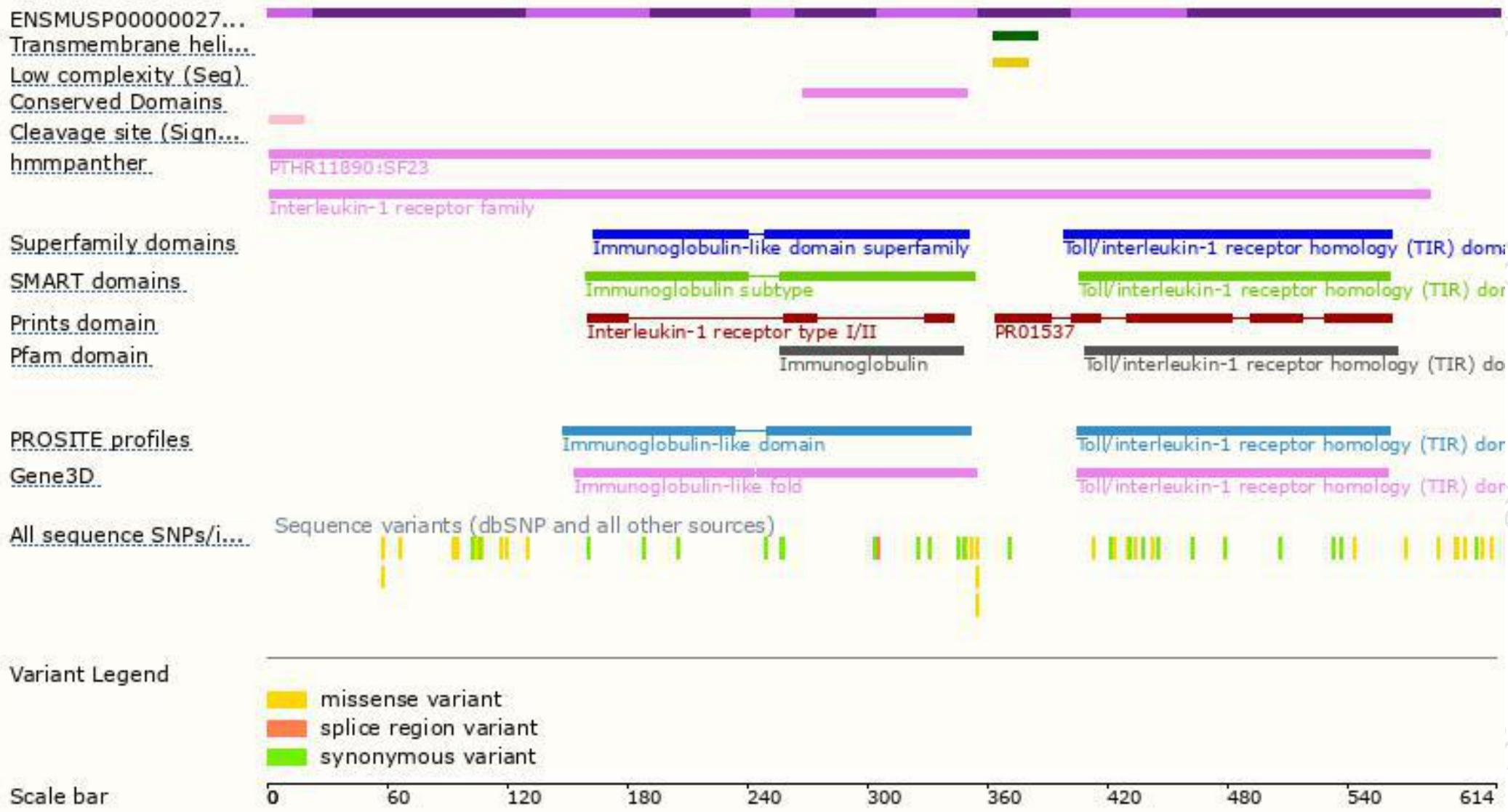
The strategy is based on the design of *Il18rap-201* transcript,The transcription is shown below



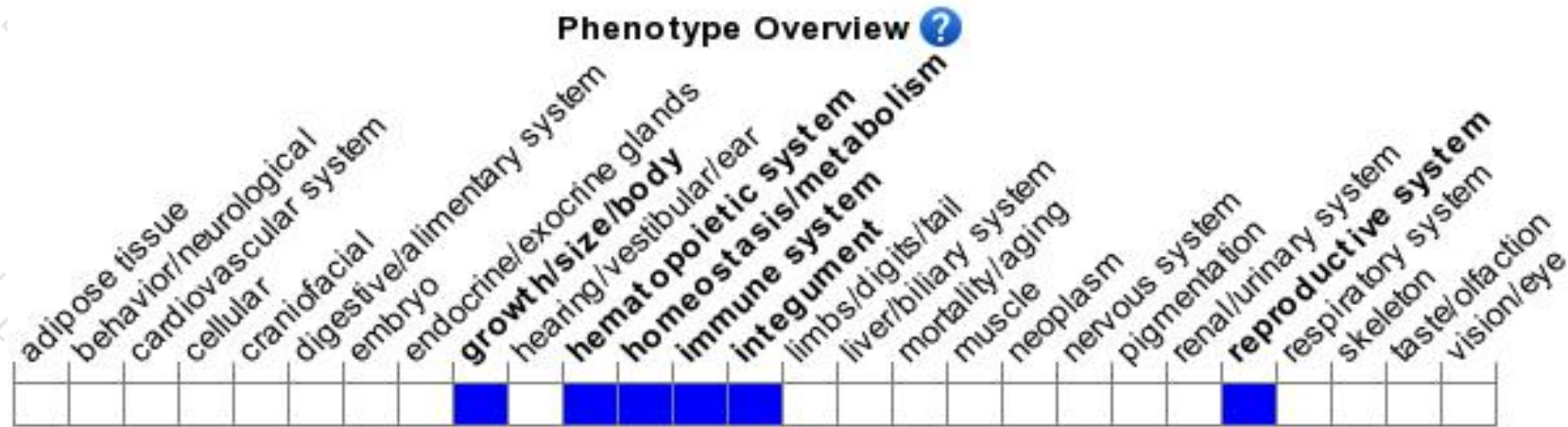
Genomic location distribution



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 null mice exhibit defective IL-18-mediated immune responses such as the inability of splenocytes, T helper 1 cells and neutrophils to produce cytokines in response to IL-18.

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

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