

Ltb4r2 Cas9-CKO Strategy

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Reviewer:

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Design Date:

2019-12-18

Project Overview

Project Name

Ltb4r2

Project type

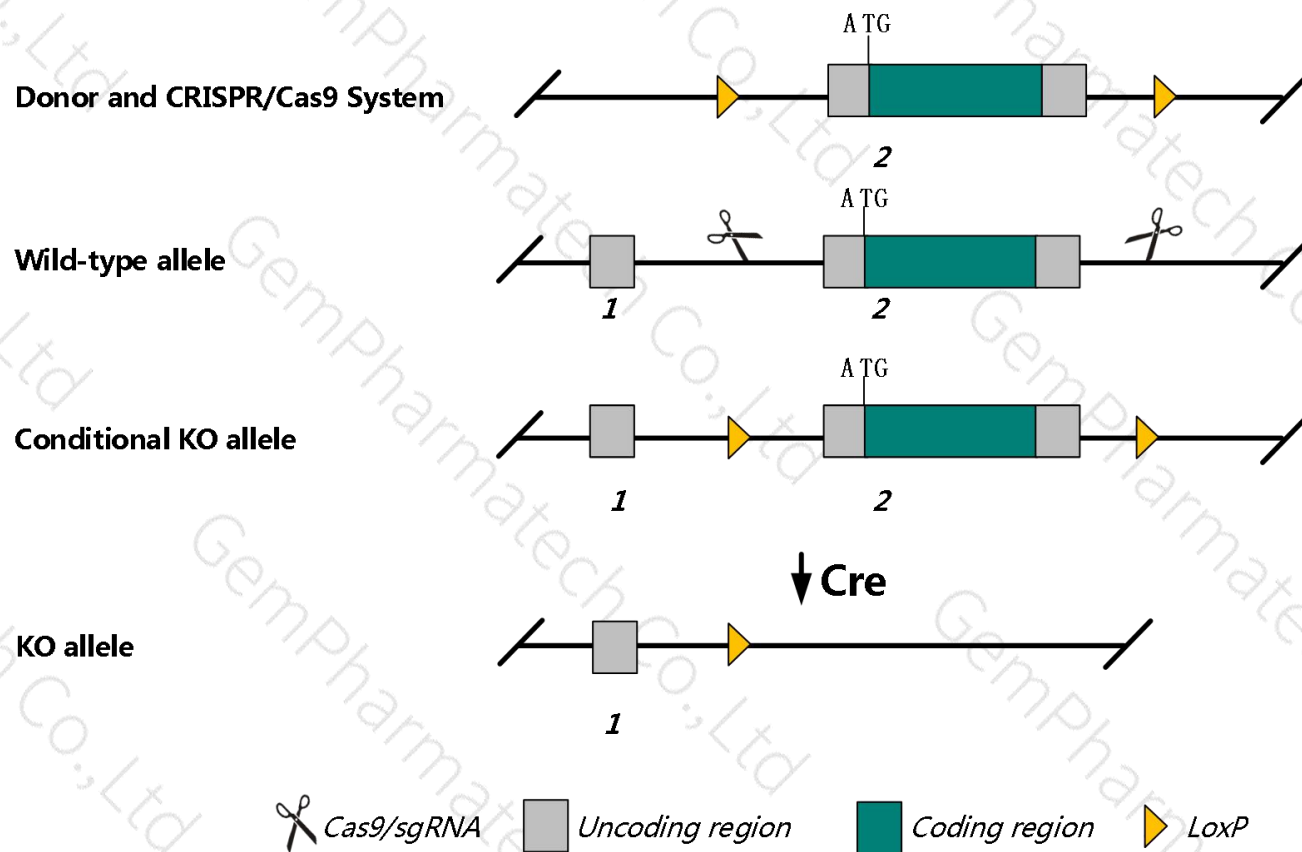
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Ltb4r2* gene. The schematic diagram is as follows:



- The *Ltb4r2* gene has 2 transcripts. According to the structure of *Ltb4r2* gene, exon 2 of *Ltb4r2*-201 (ENSMUST00000044554.4) 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 *Ltb4r2* gene. The brief process is as follows: gRNA was transcribed in vitro, donor was constructed. Cas9, gRNA 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 or cell types.

- According to the existing MGI data , Nullizygous mutations cause impaired Ltb4-driven chemotaxis and adhesion. Homozygous null phenotypes include attenuated autoAb-driven arthritis, adoptive transfer-induced uveitis, airway hyperresponsiveness and Th2-type immune responses, and reduced eosinophil recruitment in induced peritonitis.
- The CKO region contains functional region of the *Ltb4r1* gene. Knockout the region may affect the function of *Ltb4r1* gene.
- The *Ltb4r2* gene is located on the Chr 14. 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

Gene information (NCBI)

Ltb4r2 leukotriene B4 receptor 2 [*Mus musculus* (house mouse)]

Gene ID: 57260, updated on 12-Aug-2019

Summary

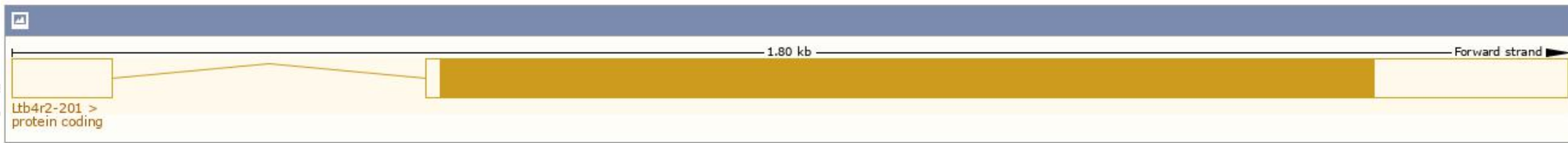
Official Symbol *Ltb4r2* provided by [MGI](#)
Official Full Name *leukotriene B4 receptor 2* provided by [MGI](#)
Primary source [MGI:MGI:1888501](#)
See related [Ensembl:ENSMUSG00000040432](#)
Gene type *protein coding*
RefSeq status *VALIDATED*
Organism [Mus musculus](#)
Lineage *Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus*
Also known as *BLT2; LTB4-R2; LTB4-R 2; LTB4-R-2; 5830462O07Rik*
Expression *Biased expression in duodenum adult (RPKM 18.1), small intestine adult (RPKM 13.4) and 6 other tissues* [See more](#)
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

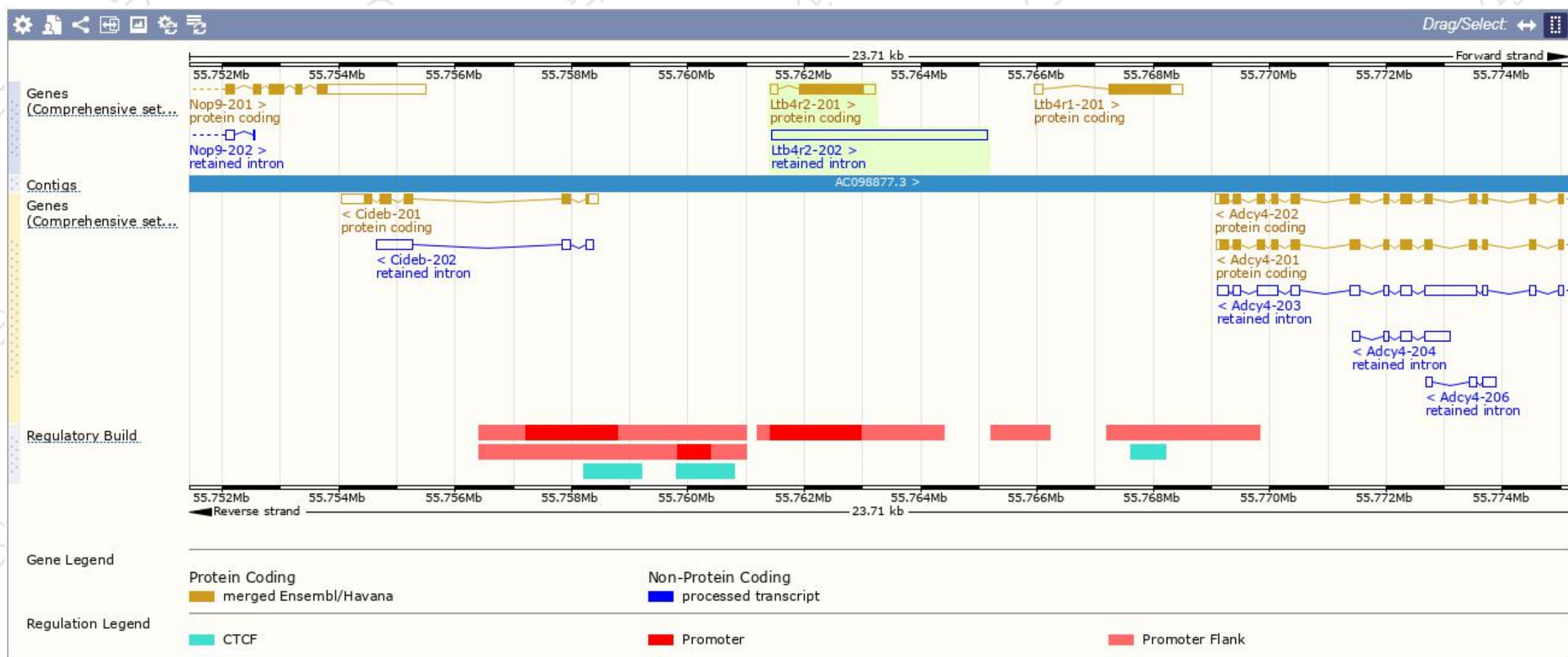
The gene has 2 transcripts, and all transcripts is shown below:

Show/hide columns (1 hidden)							Filter	
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags	
Ltb4r2-201	ENSMUST00000044554.4	1439	360aa	Protein coding	CCDS27128	Q9JJL9	TSL:1	GENCODE basic APPRIS P1
Ltb4r2-202	ENSMUST000000228302.1	3703	No protein	Retained intron	-	-	-	

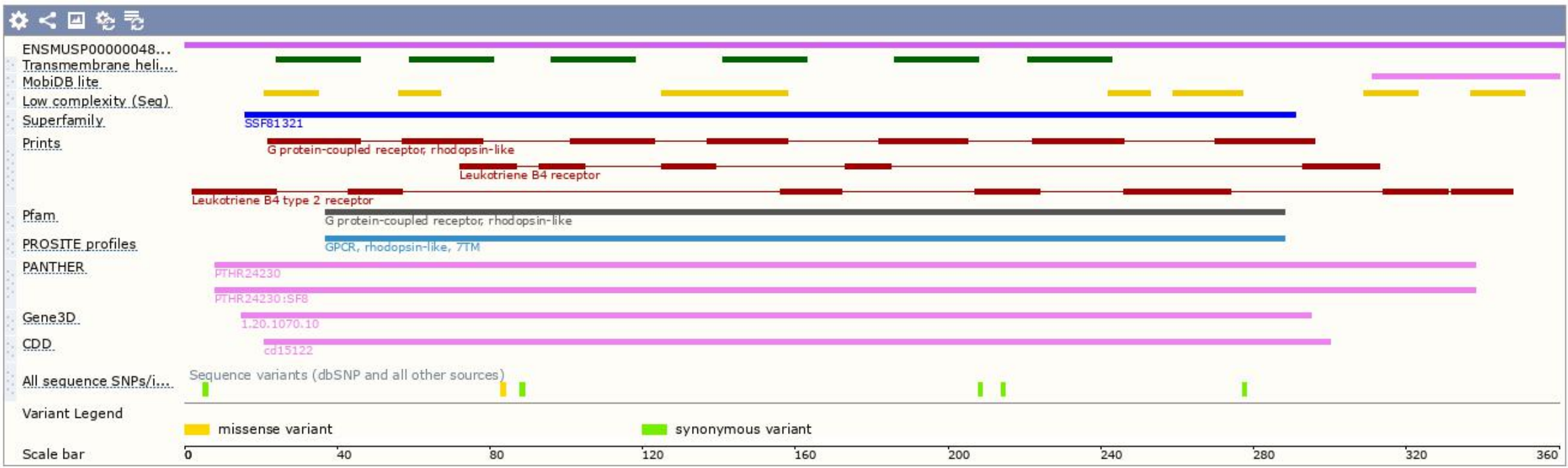
The strategy is based on the design of *Ltb4r2*-201 transcript, The transcription is shown below



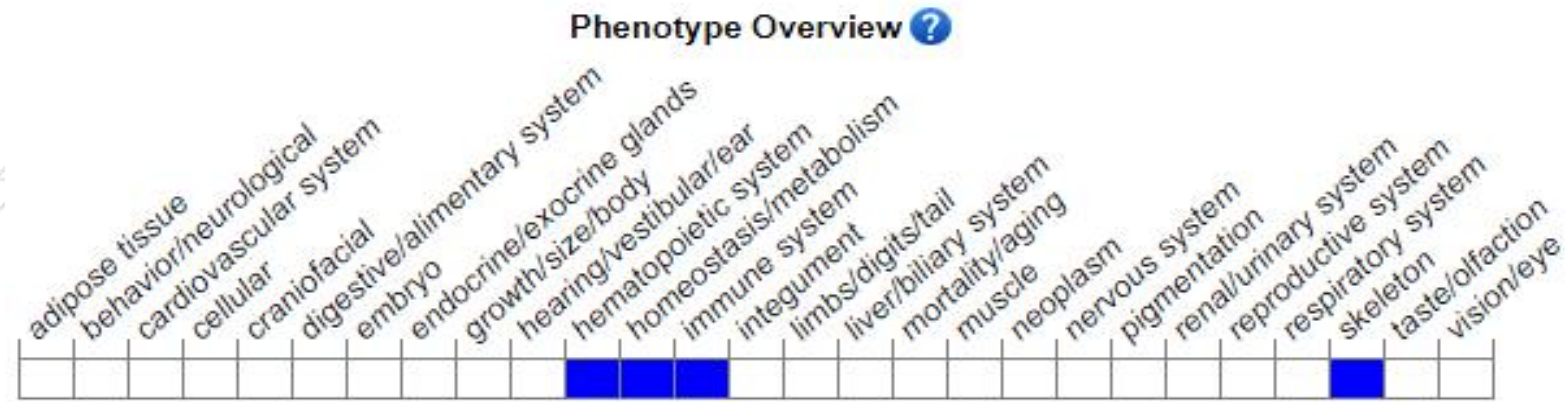
Genomic location distribution



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, Mutations in this locus affect cell-cycle regulation and apoptosis. Null homozygotes show high, early-onset tumor incidence; some have persistent hyaloid vasculature and cataracts. Truncated or temperature-sensitive alleles cause early aging phenotypes.

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