

Il17a Cas9-CKO Strategy

Designer:

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Design Date:

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Project Overview

Project Name

Il17a

Project type

Cas9-CKO

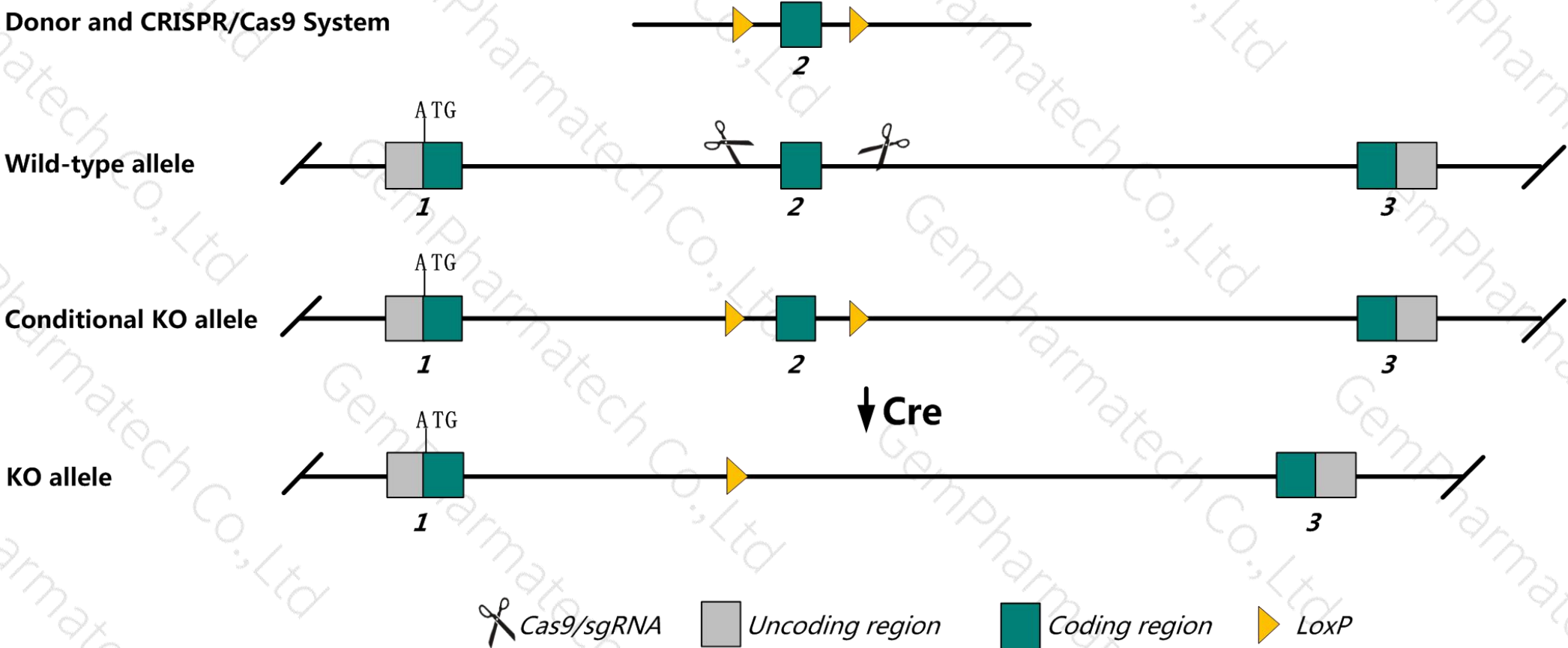
Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Il17a* gene. The schematic diagram is as follows:

Donor and CRISPR/Cas9 System



- The *Il17a* gene has 1 transcript. According to the structure of *Il17a* gene, exon2 of *Il17a*-201 transcript is recommended as the knockout region. The region contains 212bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Il17a* 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 , Homozygotes for a targeted null mutation exhibit reduced contact, delayed-type and airway hypersensitivity responses and impaired T-dependent antibody production.
- The *Il17a* 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 the loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

Gene information (NCBI)

IL17a interleukin 17A [*Mus musculus* (house mouse)]

Gene ID: 16171, updated on 20-Jul-2019

Summary



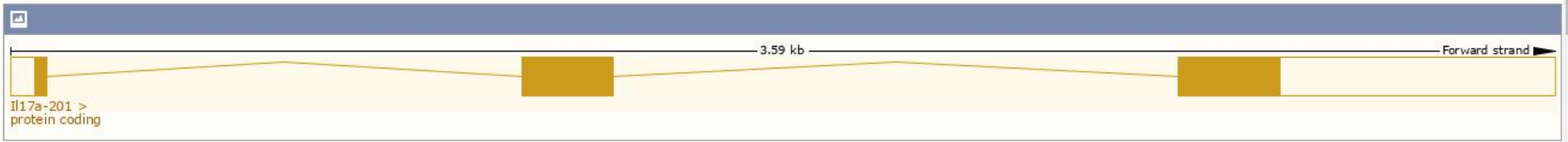
Official Symbol	IL17a provided by MGI
Official Full Name	interleukin 17A provided by MGI
Primary source	MGI:MGI:107364
See related	Ensembl:ENSMUSG000000025929
Gene type	protein coding
RefSeq status	REVIEWED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	IL17; CtlA8; IL-17; CtlA-8; IL-17A
Summary	This gene encodes a pro-inflammatory cytokine that is a member of the interleukin-17 family. The encoded protein plays a central role in host defense against diverse pathogens. The encoded protein is produced by activated T-cells and certain cell types of innate immune system. The active protein functions as either a homodimer with other interleukin-17 family members and signals through the interleukin-17 receptor to induce inflammatory cytokine production. Aberrant expression of this gene is associated with autoinflammatory diseases including rheumatoid arthritis, psoriasis and multiple sclerosis. [provided by RefSeq, Sep 2015]
Expression	Low expression observed in reference dataset See more
Orthologs	human all

Transcript information (Ensembl)

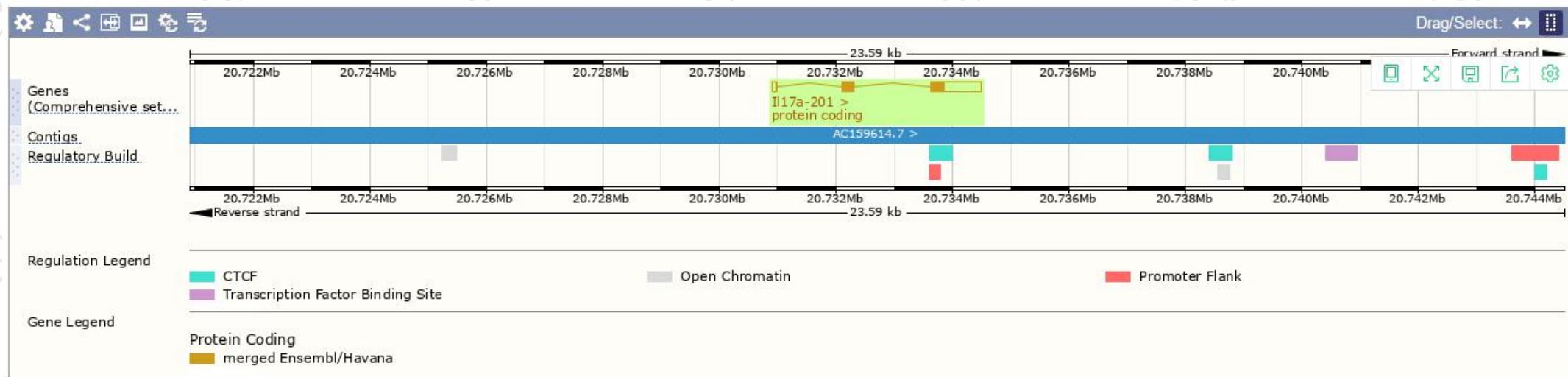
The gene has 1 transcript, and the transcript is shown below:

Show/hide columns (1 hidden)							Filter		
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags		
Il17a-201	ENSMUST00000027061.4	1171	158aa	Protein coding	CCDS14842	Q544E6 Q62386	TSL:1	GENCODE basic	APPRIS P1

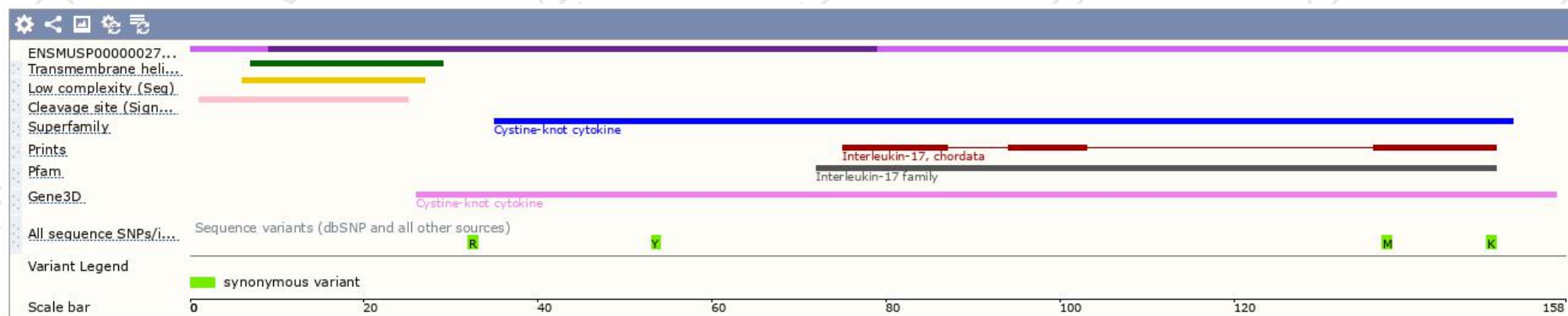
The strategy is based on the design of *Il17a*-201 transcript, The transcription is shown below



Genomic location distribution

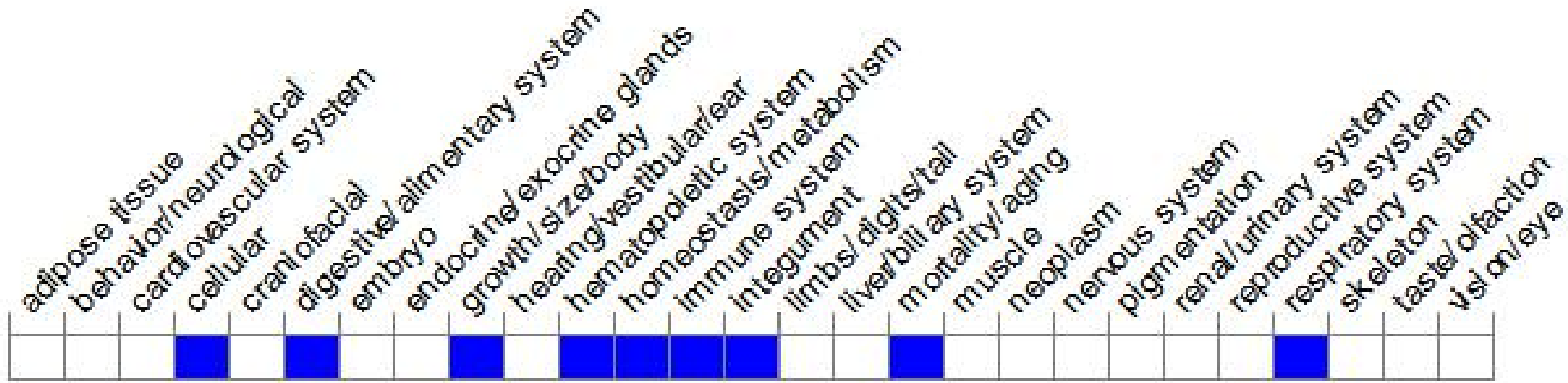


Protein domain



Mouse phenotype description(MGI)

Phenotype Overview ?



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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