

Il1rn Cas9-CKO Strategy

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Reviewer:

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Design Date:

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Project Overview

Project Name

Il1rn

Project type

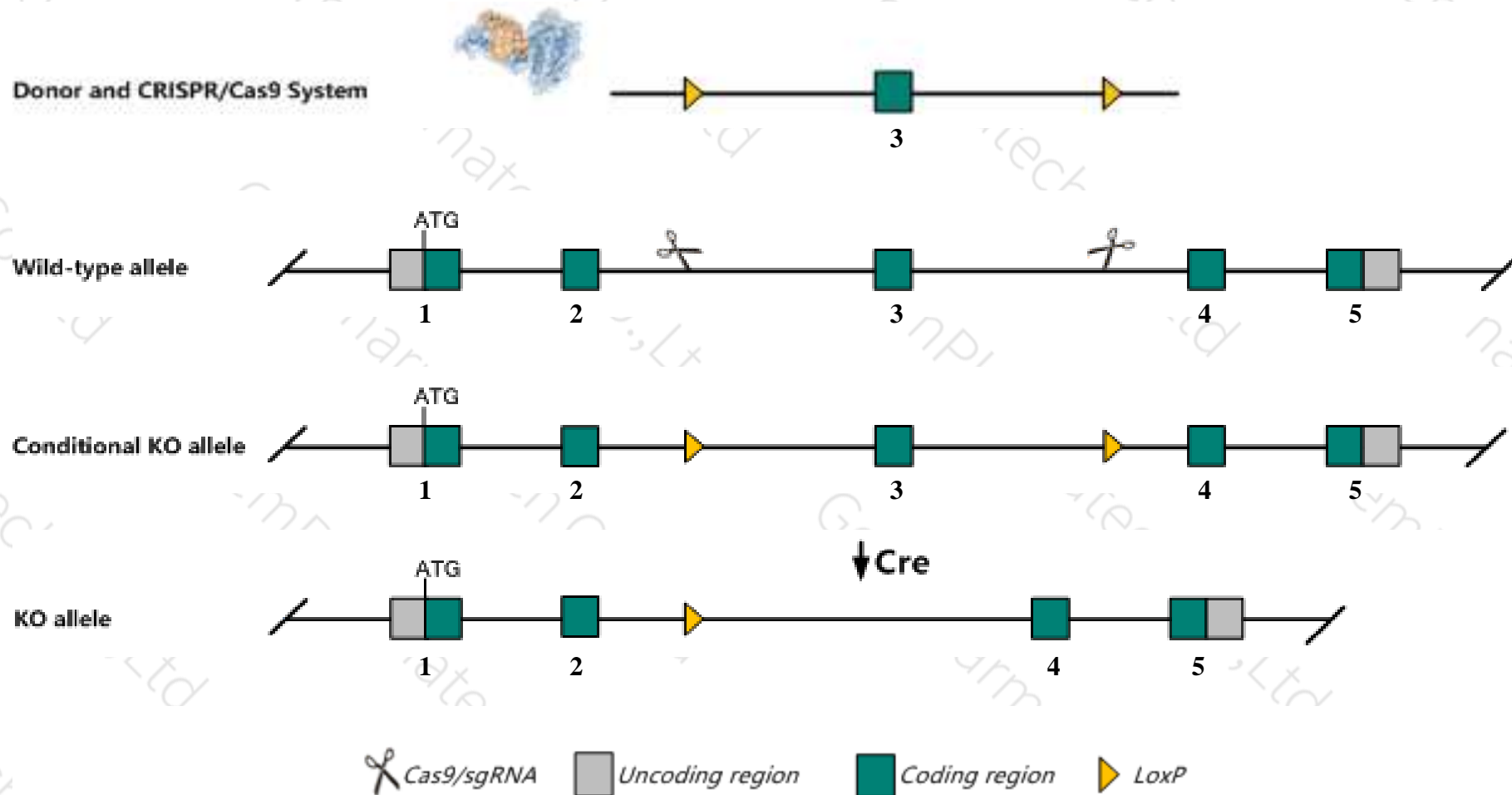
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Il1rn* gene. The schematic diagram is as follows:



- The *Il1rn* gene has 6 transcripts. According to the structure of *Il1rn* gene, exon3 of *Il1rn-204* (ENSMUST00000114487.8) transcript is recommended as the knockout region. The region contains 89bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Il1rn* 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, Nullizygous mutations of this gene may result in decreased body weight, increased inflammatory response to turpentine and LPS, decreased susceptibility to bacterial infection, psoriasis, aortitis, rheumatoid arthritis, and abnormal dendritic and CD4-positive T cell morphology.
- Transcript *Il1rn*-203/206 may not be affected.
- The *Il1rn* gene is located on the Chr2. 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)

Il1rn interleukin 1 receptor antagonist [*Mus musculus* (house mouse)]

Gene ID: 16181, updated on 13-Aug-2019

Summary

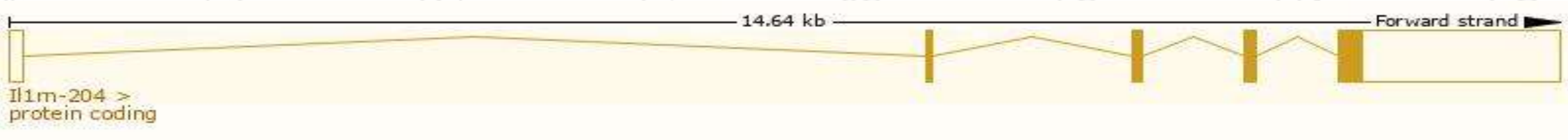
Official Symbol	Il1rn provided by MGI
Official Full Name	interleukin 1 receptor antagonist provided by MGI
Primary source	MGI:MGI:96547
See related	Ensembl:ENSMUSG00000026981
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	IL-1ra; F630041P17Rik
Expression	Broad expression in liver E18 (RPKM 5.5), colon adult (RPKM 5.1) and 18 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

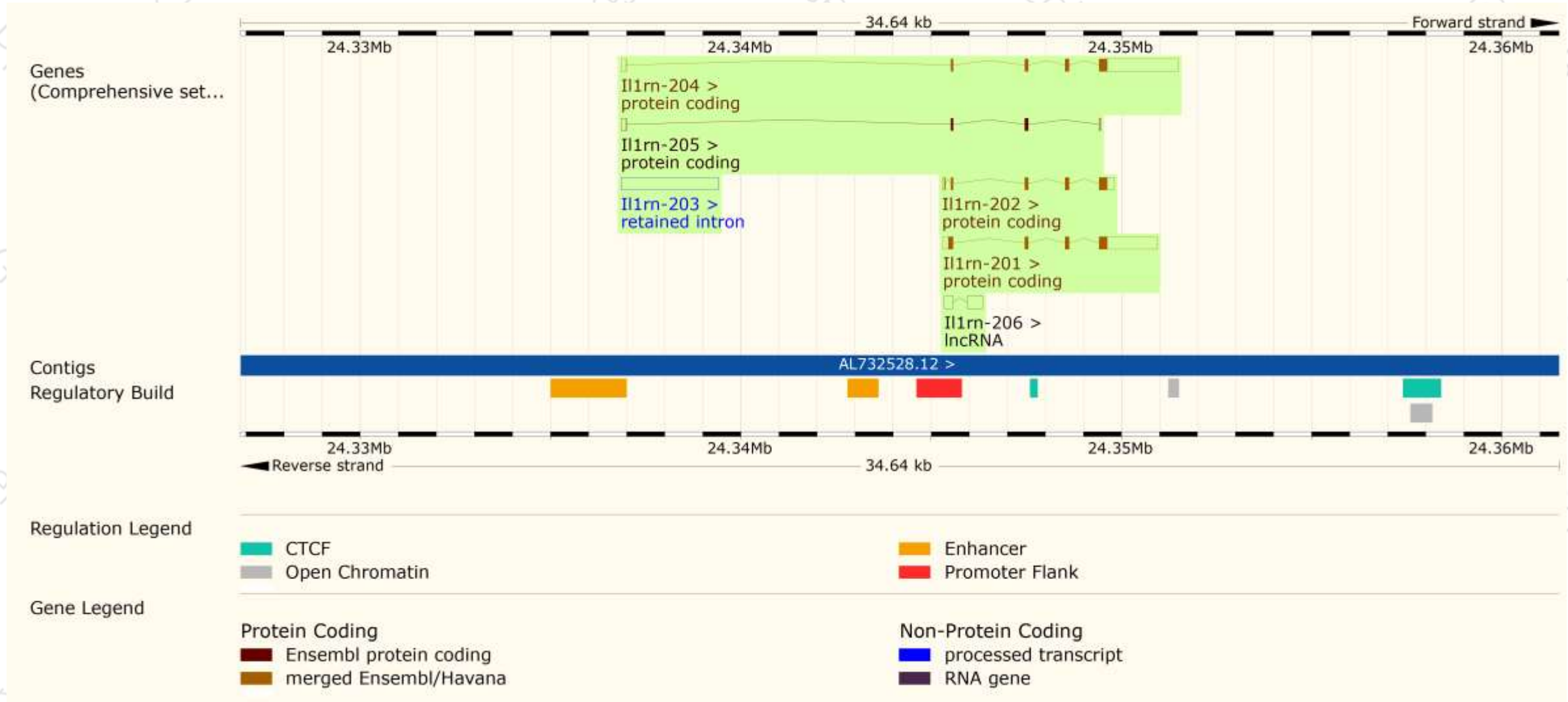
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Il1rn-204	ENSMUST00000114487.8	2474	159aa	Protein coding	CCDS15736	P25085 Q542W1	TSL:1 GENCODE basic APPRIS P3
Il1rn-201	ENSMUST00000114482.2	1986	178aa	Protein coding	CCDS38064	P25085 Q542C7	TSL:1 GENCODE basic APPRIS ALT 1
Il1rn-202	ENSMUST00000114485.8	728	162aa	Protein coding	CCDS50520	Q3TBV5	TSL:1 GENCODE basic
Il1rn-205	ENSMUST00000142093.6	321	52aa	Protein coding	-	A0A0A6YVU4	TSL:3 GENCODE basic
Il1rn-206	ENSMUST00000143423.1	636	No protein	Processed transcript	-	-	TSL:2
Il1rn-203	ENSMUST00000114486.3	2554	No protein	Retained intron	-	-	TSL:NA

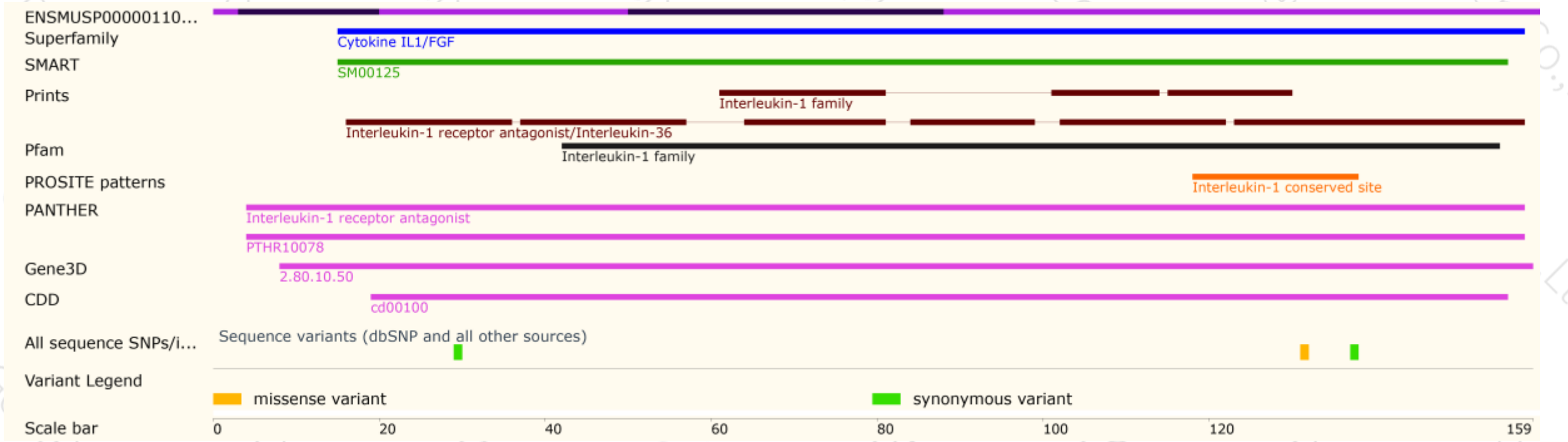
The strategy is based on the design of *Il1rn-204* transcript,The transcription is shown below



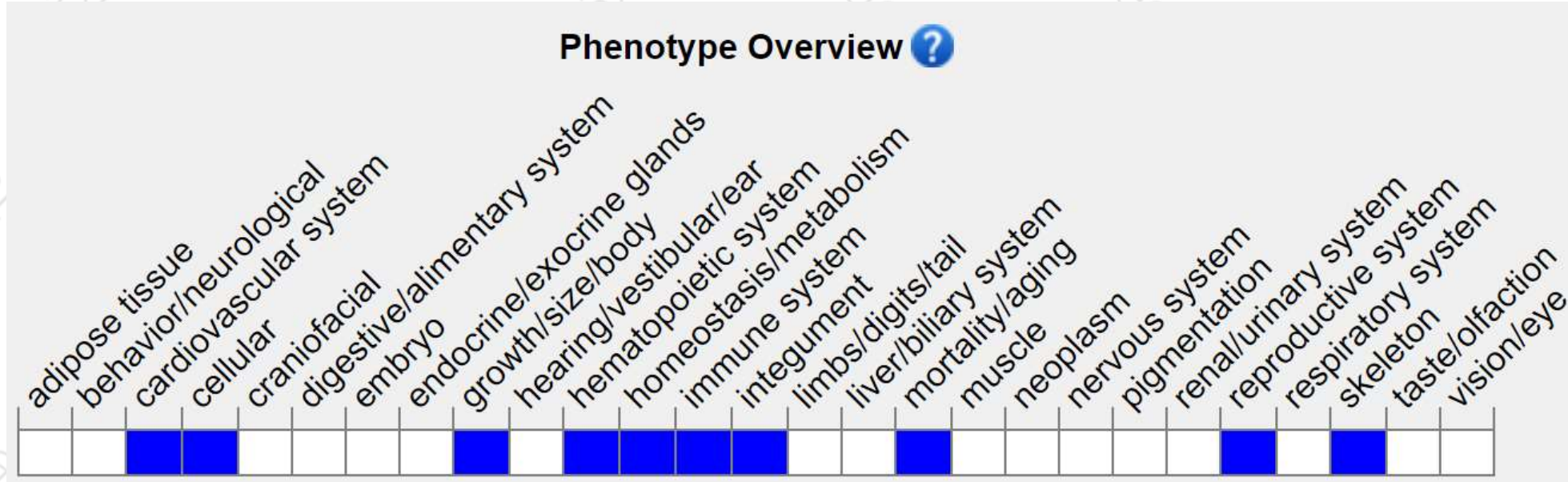
Genomic location distribution



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, Nullizygous mutations of this gene may result in decreased body weight, increased inflammatory response to turpentine and LPS, decreased susceptibility to bacterial infection, psoriasis, aortitis, rheumatoid arthritis, and abnormal dendritic and CD4-positive T cell morphology.

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

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