

S1pr2 Cas9-CKO Strategy

Designer:

Daohua Xu

Reviewer:

Huimin Su

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Project Overview

Project Name

S1pr2

Project type

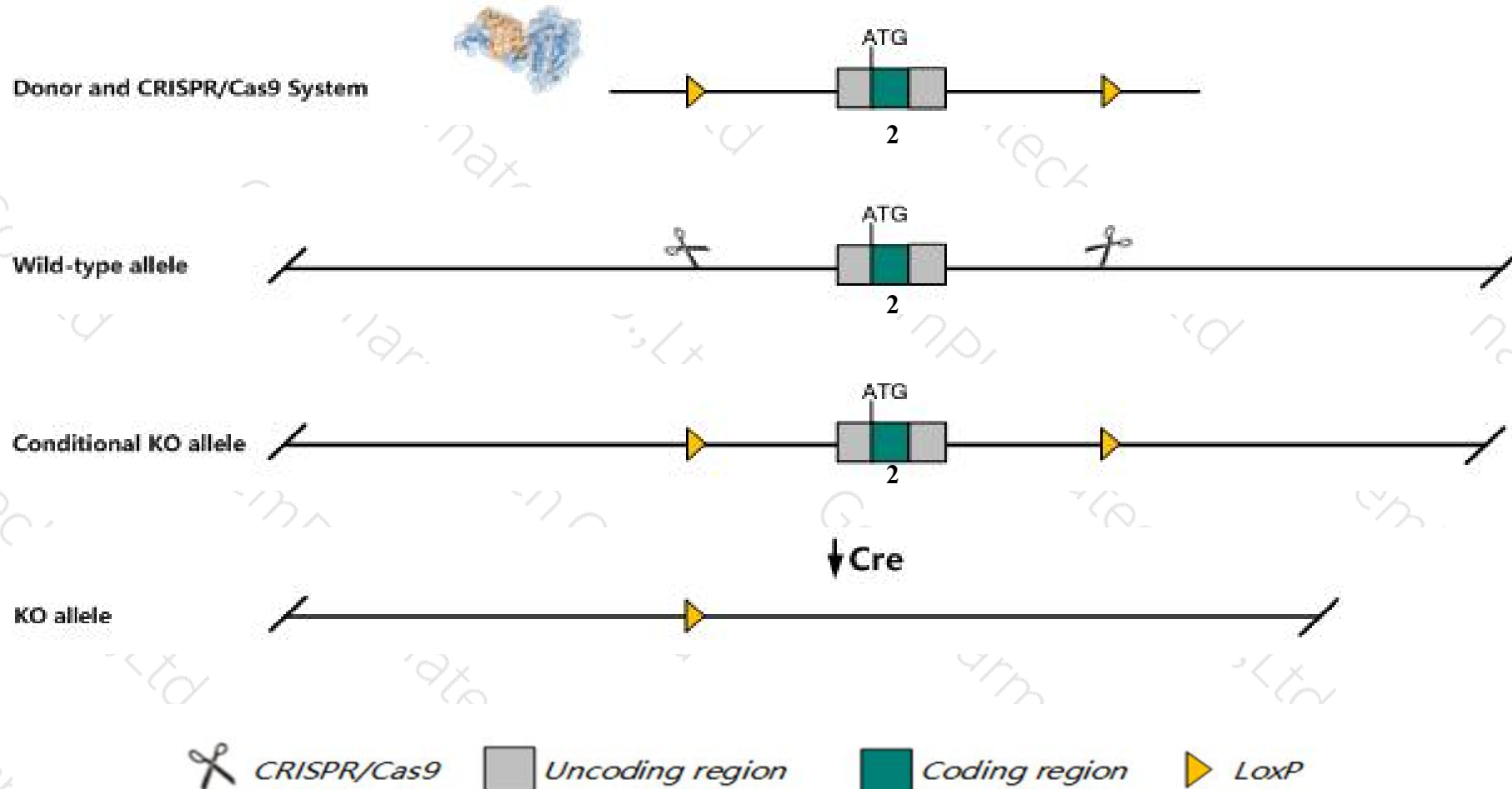
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slpr2* gene. The schematic diagram is as follows:



Technical routes

- The *Slpr2* gene has 4 transcripts. According to the structure of *Slpr2* gene, exon2 of *Slpr2-201* (ENSMUST00000054197.6) 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 *Slpr2* 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, Homozygous null mutations in this gene may lead to impaired auditory and vestibular function, multiple inner ear pathologies, deafness, altered neuronal excitability, lethal seizures, altered physiology of germinal center B cells, small litter size, and enhanced tumor angiogenesis and tumor growth.
- This strategy may affect the 5-terminal regulation of the *Dnmt1* gene.
- The *Slpr2* gene is located on the Chr9. 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)

S1pr2 sphingosine-1-phosphate receptor 2 [Mus musculus (house mouse)]

Gene ID: 14739, updated on 12-Mar-2019

Summary



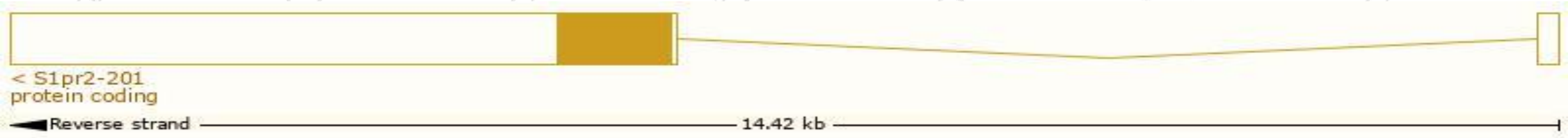
Official Symbol	S1pr2 provided by MGI
Official Full Name	sphingosine-1-phosphate receptor 2 provided by MGI
Primary source	MGI:MGI:99569
See related	Ensembl:ENSMUSG00000043895
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	1100001A16Rik, Edg5, Gpcr13, H218, LPb2, S1P2
Expression	Broad expression in lung adult (RPKM 19.6), limb E14.5 (RPKM 17.0) and 24 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

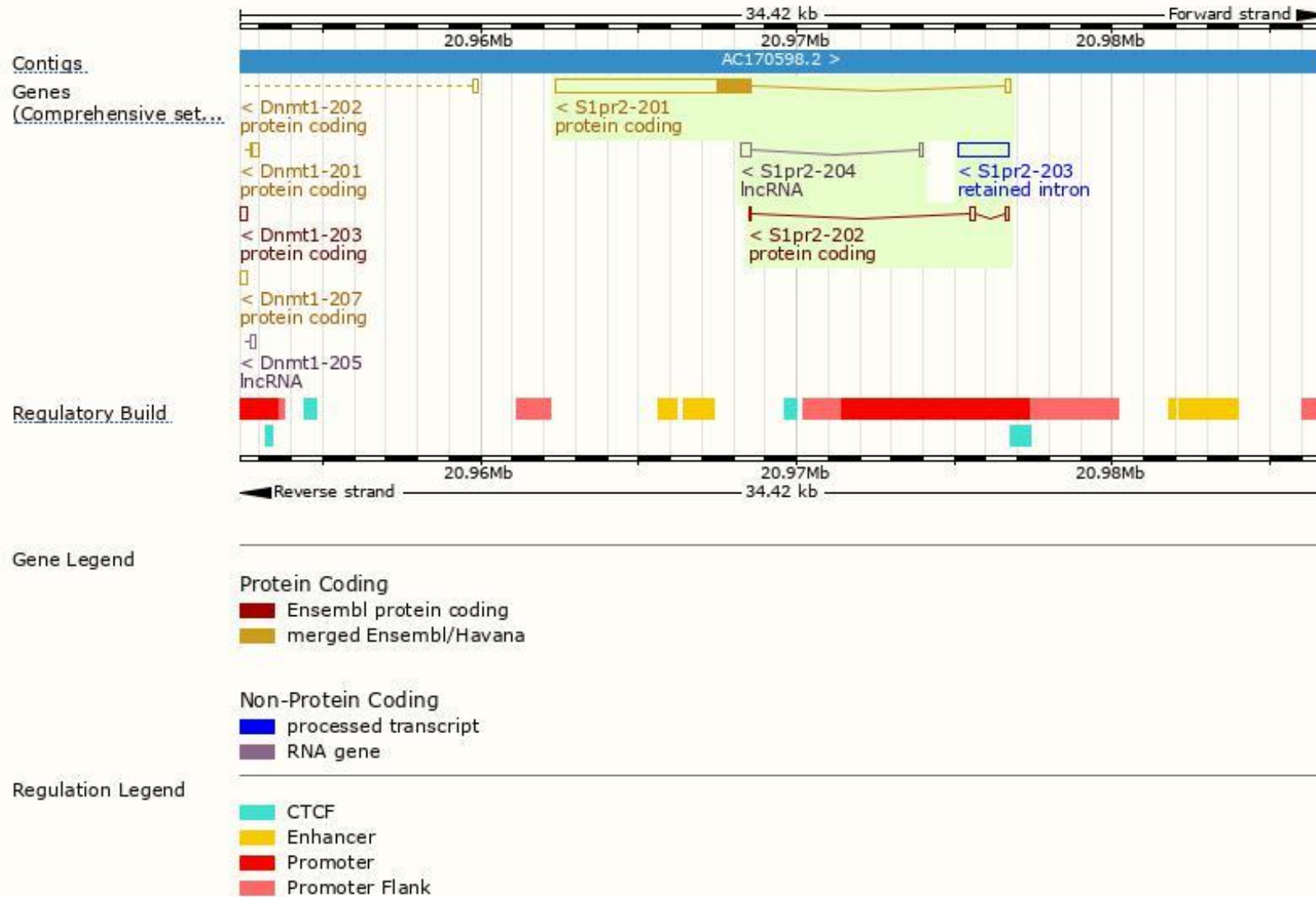
The gene has 4 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
S1pr2-201	ENSMUST00000054197.6	6394	352aa	Protein coding	CCDS22888	P52592	TSL:1 GENCODE basic APPRIS P1
S1pr2-202	ENSMUST00000214218.1	379	16aa	Protein coding	-	A0A1L1ST05	CDS 3' incomplete TSL:3
S1pr2-204	ENSMUST00000216499.1	419	No protein	Processed transcript	-	-	TSL:2
S1pr2-203	ENSMUST00000214333.1	1587	No protein	Retained intron	-	-	TSL:NA

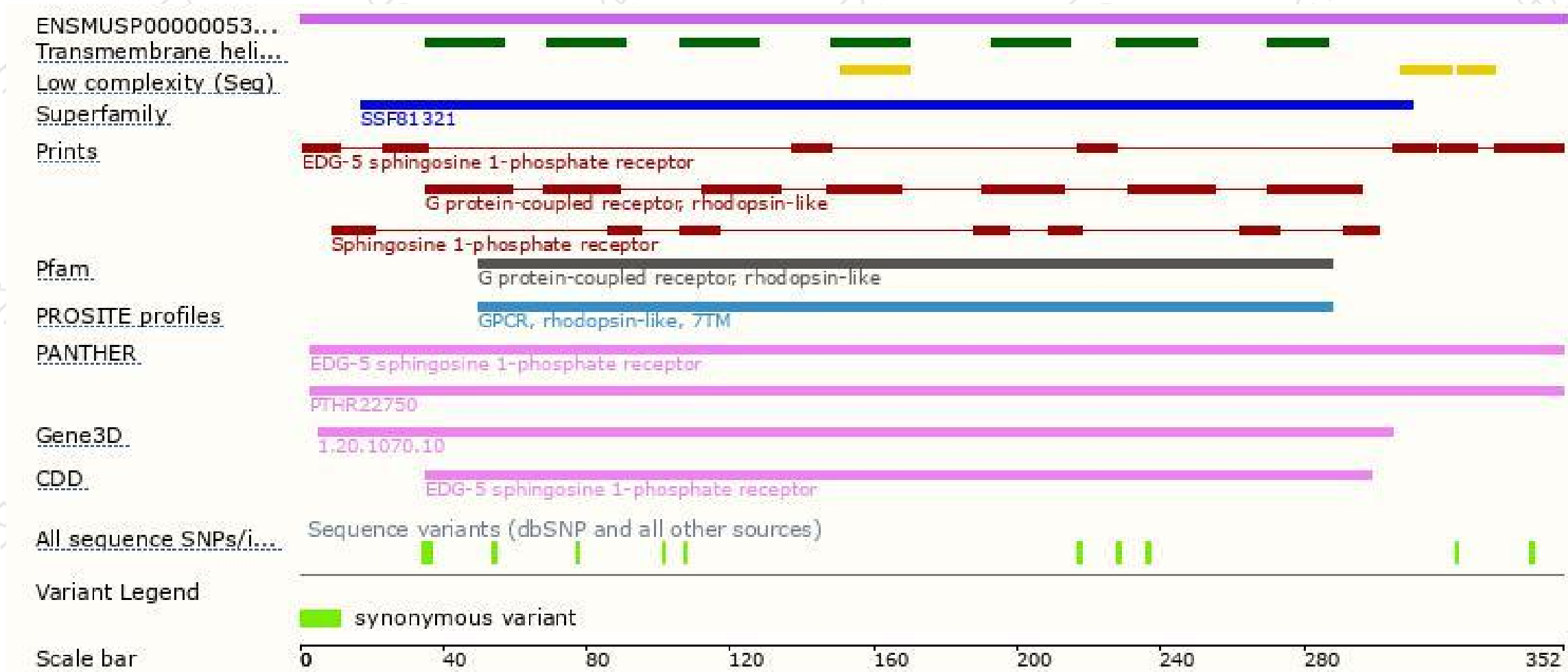
The strategy is based on the design of *S1pr2-201* transcript,The transcription is shown below



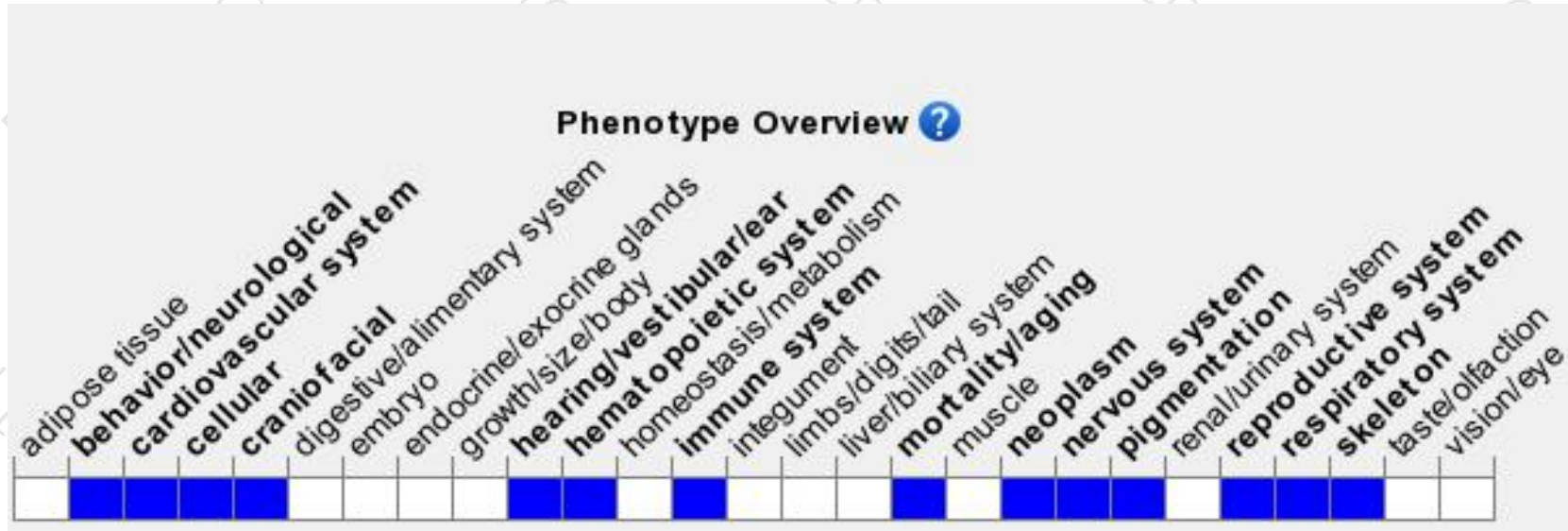
Genomic location distribution



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 mutations in this gene may lead to impaired auditory and vestibular function, multiple inner ear pathologies, deafness, altered neuronal excitability, lethal seizures, altered physiology of germinal center B cells, small litter size, and enhanced tumor angiogenesis and tumor growth.

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

Tel: 400-9660890

