

***Slc44a2* Cas9-KO Strategy**

Designer: Xueting Zhang

Reviewer: Yanhua Shen

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Project Overview

Project Name

Slc44a2

Project type

Cas9-KO

Strain background

C57BL/6JGpt

Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc44a2* gene. The schematic diagram is as follows:



- The *Slc44a2* gene has 10 transcripts. According to the structure of *Slc44a2* gene, exon3-exon22 of *Slc44a2-201* (ENSMUST00000034697.7) transcript is recommended as the knockout region. The region contains most 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 *Slc44a2* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Mice homozygous for a knock-out allele exhibit cochlear hair cell loss, spiral ganglion degeneration, and progressive sensorineural hearing loss.
- The *Slc44a2* 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 the gene knockout on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

Gene information (NCBI)

Slc44a2 solute carrier family 44, member 2 [*Mus musculus* (house mouse)]

Gene ID: 68682, updated on 18-Feb-2020

Summary

- Official Symbol** Slc44a2 provided by MGI
- Official Full Name** solute carrier family 44, member 2 provided by MGI
- Primary source** MGI:MGI:1915932
- See related** Ensembl:ENSMUSG00000057193
- 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** CTL2; 1110028E10Rik
- Expression** Broad expression in thymus adult (RPKM 112.2), lung adult (RPKM 80.3) and 23 other tissues [See more](#)
- Orthologs** [human](#) [all](#)

Genomic context

Location: 9; 9 A3

Exon count: 25

See Slc44a2 in [Genome Data Viewer](#)

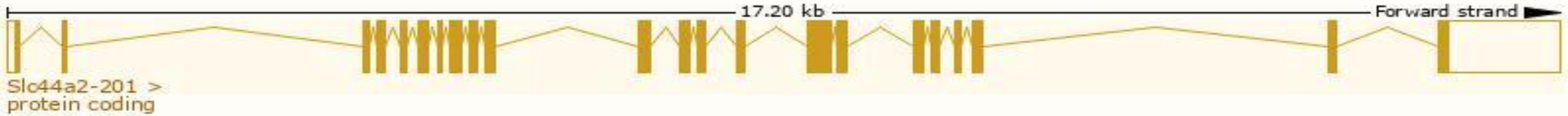
Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	9	NC_000075.6 (21320719..21355028)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	9	NC_000075.5 (21142296..21159472)

Transcript information (Ensembl)

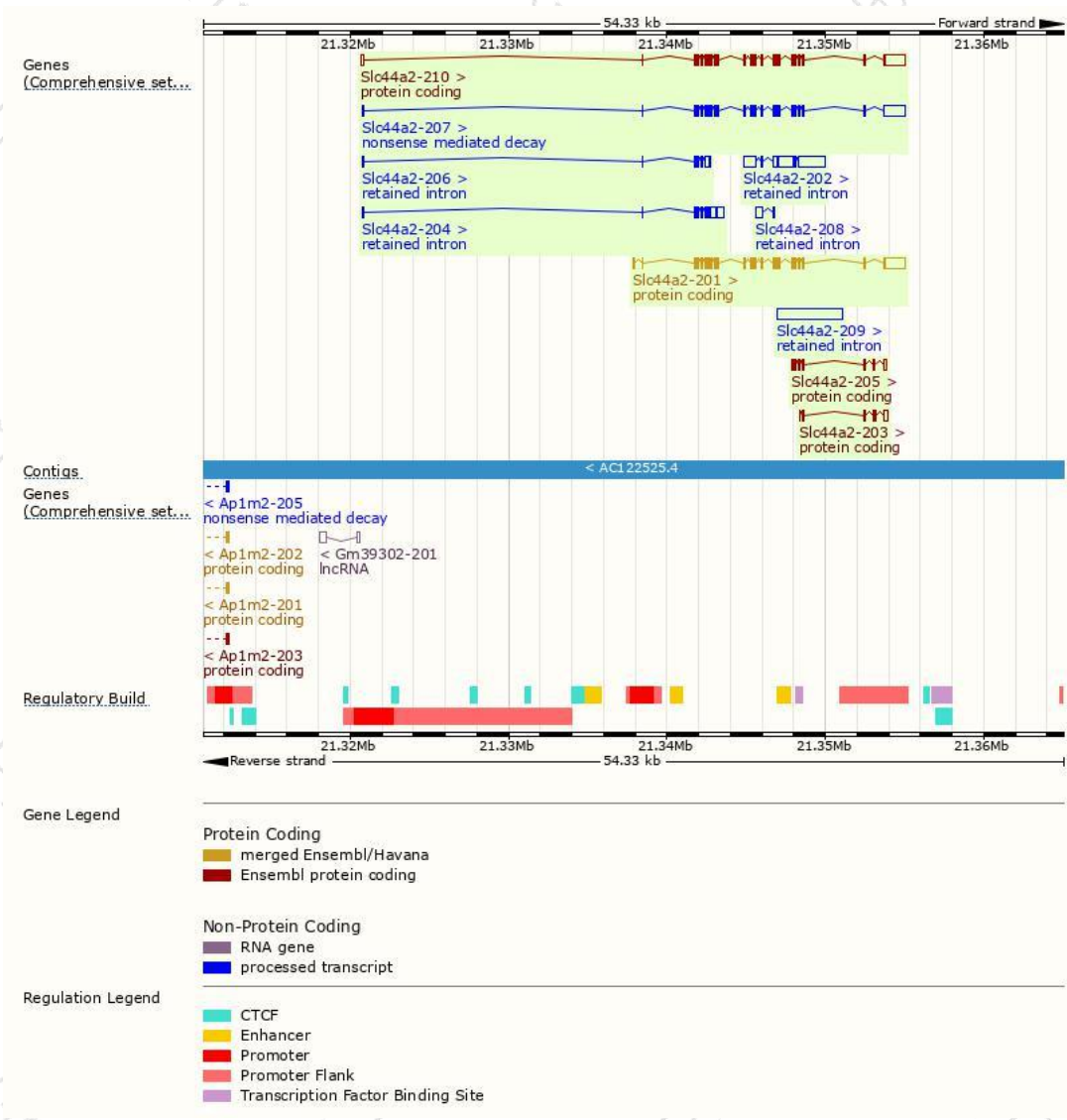
The gene has 10 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc44a2-201	ENSMUST00000034697.7	3456	706aa	Protein coding	CCDS22903	Q8BY89	TSL:1 GENCODE basic APPRIS P2
Slc44a2-210	ENSMUST00000217461.1	3478	704aa	Protein coding	-	Q8BY89	TSL:1 GENCODE basic APPRIS ALT 1
Slc44a2-205	ENSMUST00000214268.1	684	170aa	Protein coding	-	A0A1L1SU40	CDS 5' incomplete TSL:5
Slc44a2-203	ENSMUST00000213535.1	679	111aa	Protein coding	-	A0A1L1SS87	CDS 5' incomplete TSL:3
Slc44a2-207	ENSMUST00000215574.1	3428	554aa	Nonsense mediated decay	-	A0A1L1SVG6	TSL:1
Slc44a2-209	ENSMUST00000217453.1	4077	No protein	Retained intron	-	-	TSL:NA
Slc44a2-202	ENSMUST00000213499.1	3744	No protein	Retained intron	-	-	TSL:2
Slc44a2-204	ENSMUST00000213758.1	1315	No protein	Retained intron	-	-	TSL:1
Slc44a2-206	ENSMUST00000215528.1	713	No protein	Retained intron	-	-	TSL:5
Slc44a2-208	ENSMUST00000216266.1	515	No protein	Retained intron	-	-	TSL:1

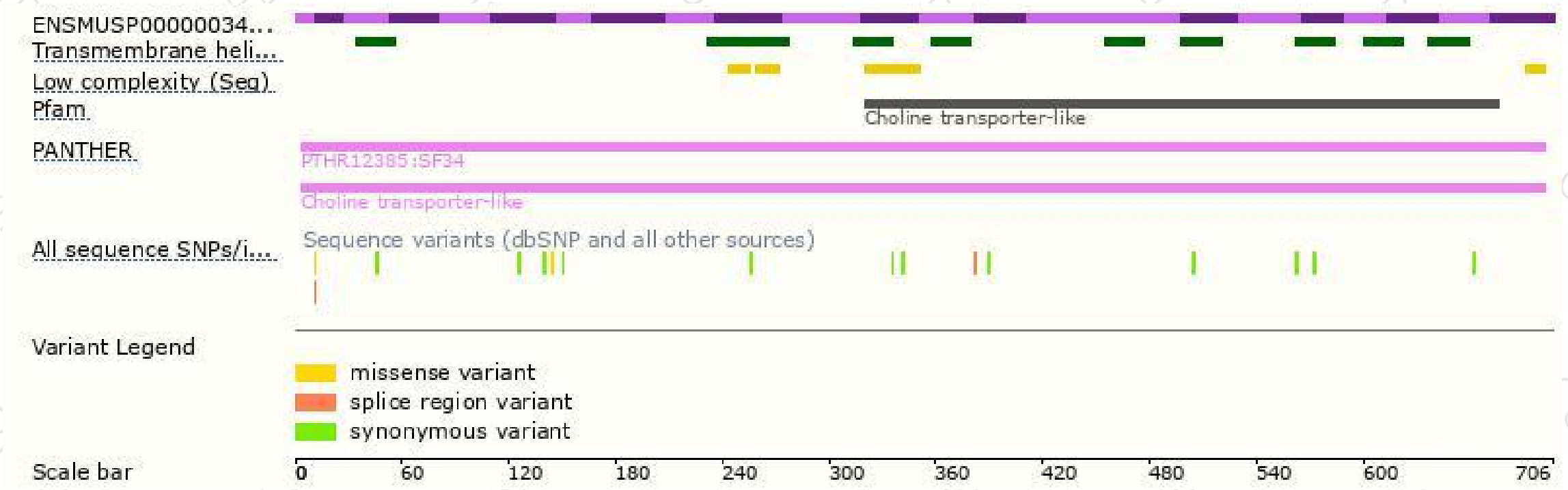
The strategy is based on the design of *Slc44a2-201* transcript,The transcription is shown below



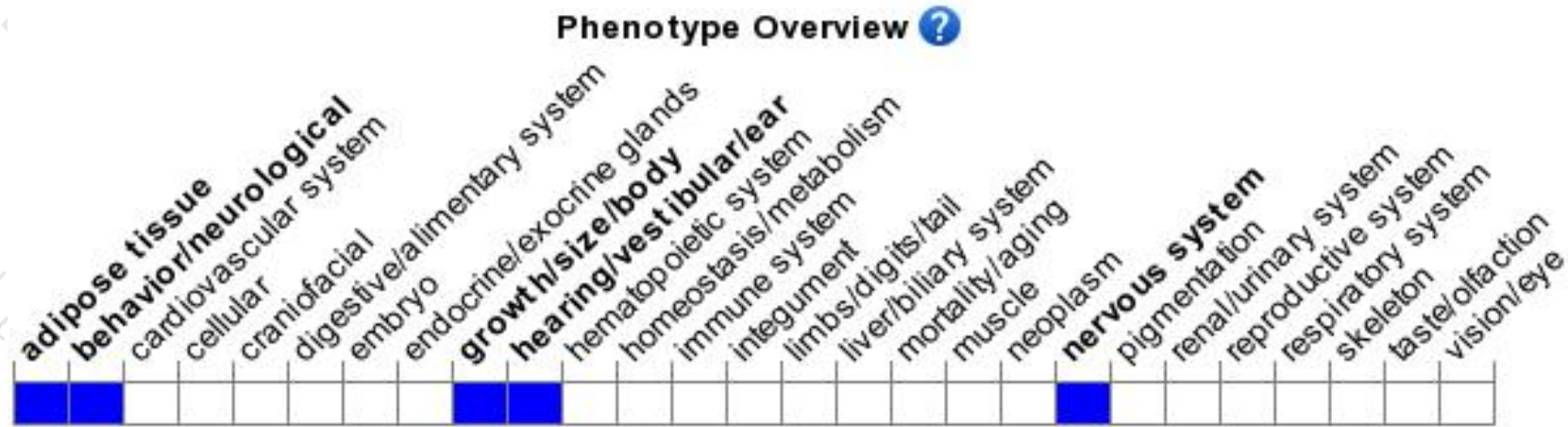
Genomic location distribution



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, Mice homozygous for a knock-out allele exhibit cochlear hair cell loss, spiral ganglion degeneration, and progressive sensorineural hearing loss.

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

Tel: 400-9660890

