Slc1a1 Cas9-CKO Strategy

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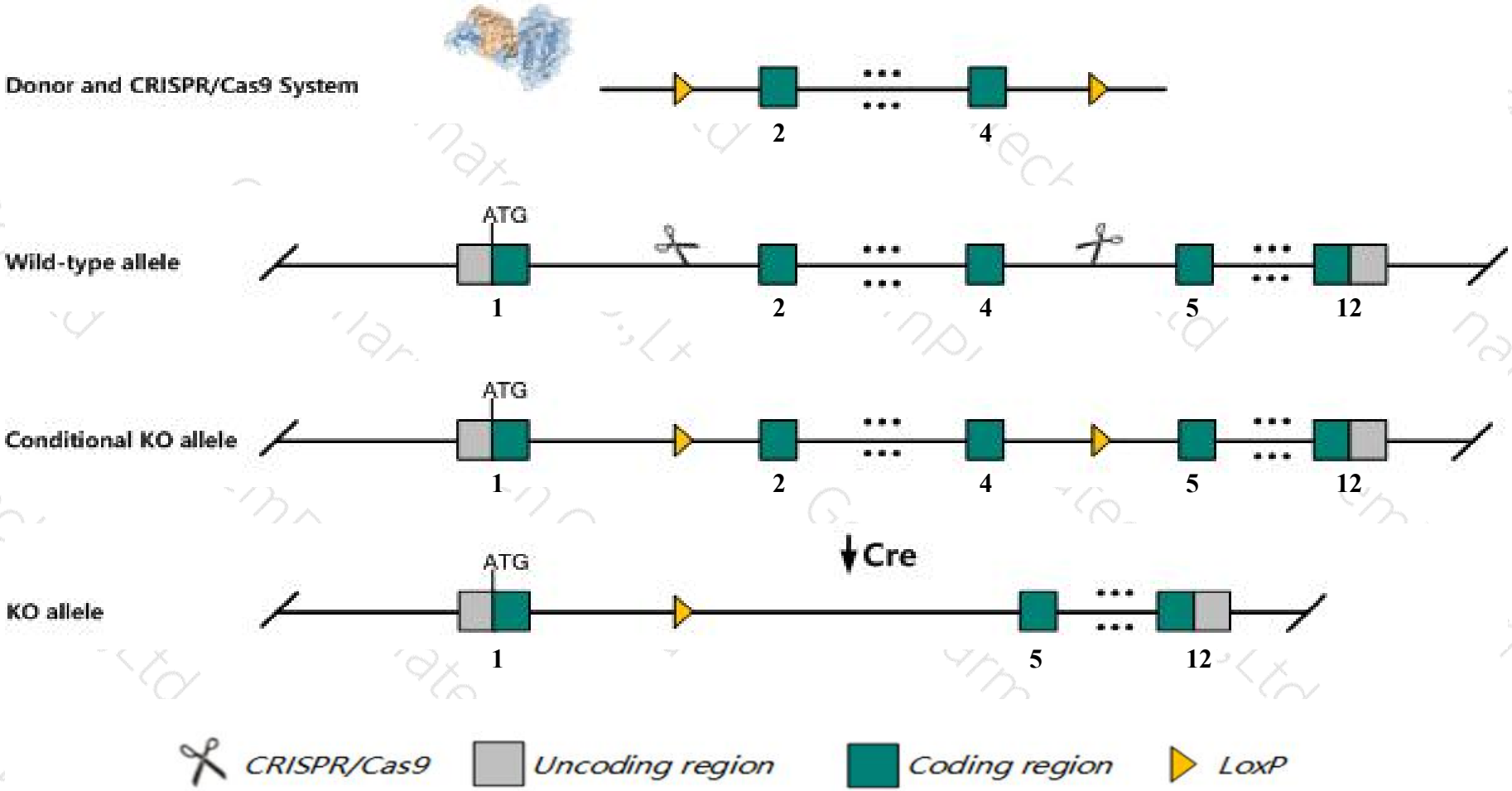
Project Overview

Project Name	***Slc1a1***
Project type	**Cas9-CKO**
Strain background	**C57BL/6JGpt**

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc1a1* gene. The schematic diagram is as follows:



Technical routes

- The *Slc1a1* gene has 6 transcripts. According to the structure of *Slc1a1* gene, exon2-exon4 of *Slc1a1-201* (ENSMUST00000025875.4) transcript is recommended as the knockout region.The region contains 349bp coding sequence. Knock out the region will result in disruption of protein function.

- In this project we use CRISPR/Cas9 technology to modify *Slc1a1* 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.

Notice

- According to the existing MGI data,Mice homozygous for disruptions in this gene display reduced locomotor activity and excessive excretion of glutamate and aspartate.
- The last exon of *4430402I18Rik* gene will be deleted in this strategy.
- Transcript *Slc1a1*-205 may not be affected.
- The *Slc1a1* gene is located on the Chr19. 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）

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Slc1a1 solute carrier family 1 (neuronal/epithelial high affinity glutamate transporter, system Xag), member 1 [*Mus musculus* (house mouse)]

Gene ID: 20510, updated on 5-Nov-2019

Summary

Official Symbol	Slc1a1 provided by MGI
Official Full Name	solute carrier family 1 (neuronal/epithelial high affinity glutamate transporter, system Xag), member 1 provided by MGI
Primary source	MGI:MGI:105083
See related	Ensembl:ENSMUSG00000024935
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	EAAC1; EAAC2; EAAT3; MEAAC1; D130048G10Rik
Expression	Biased expression in genital fat pad adult (RPKM 43.4), kidney adult (RPKM 33.9) and 13 other tissues See more
Orthologs	human all

Genomic context

Location: 19; 19 C1

See Slc1a1 in Genome Data Viewer

Exon count: 12

Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	19	NC_000085.6 (28835135..28913960)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	19	NC_000085.5 (28909656..28988450)

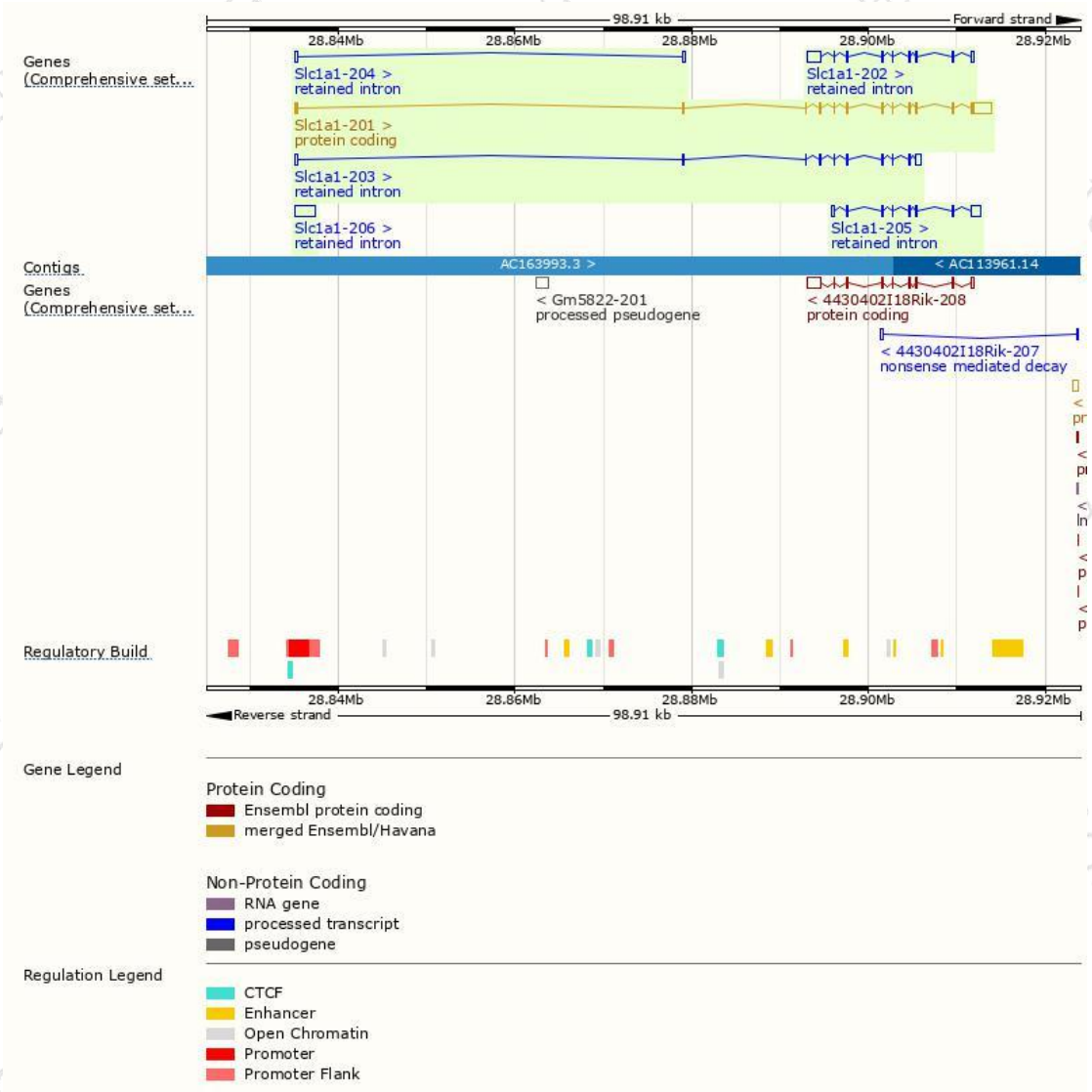
Transcript information（Ensembl）

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

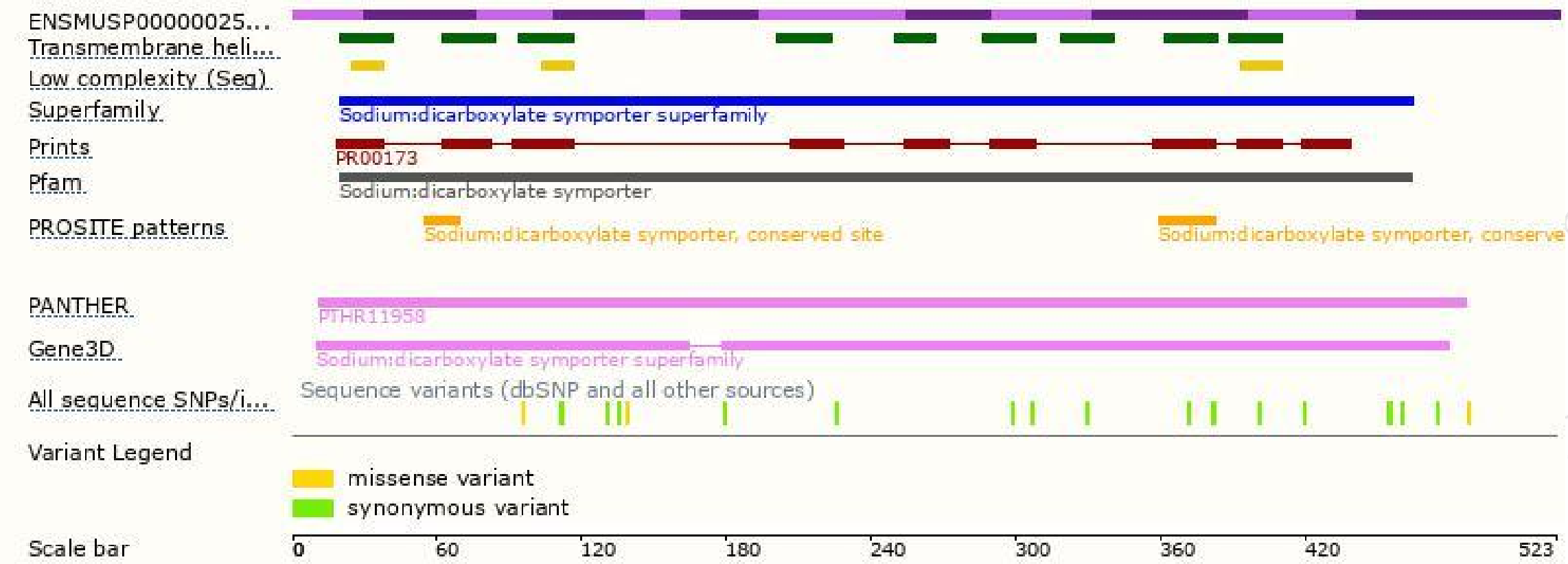
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc1a1-201	ENSMUST00000025875.4	3800	523aa	Protein coding	CCDS29727	P51906	TSL:1 GENCODE basic APPRIS P1
Slc1a1-202	ENSMUST00000160702.7	2665	No protein	Retained intron	-	-	TSL:5
Slc1a1-206	ENSMUST00000237545.1	2285	No protein	Retained intron	-	-	
Slc1a1-205	ENSMUST00000162189.1	2161	No protein	Retained intron	-	-	TSL:1
Slc1a1-203	ENSMUST00000161119.7	1762	No protein	Retained intron	-	-	TSL:1
Slc1a1-204	ENSMUST00000161340.1	573	No protein	Retained intron	-	-	TSL:2

The strategy is based on the design of *Slc1a1-201* transcript,The transcription is shown below

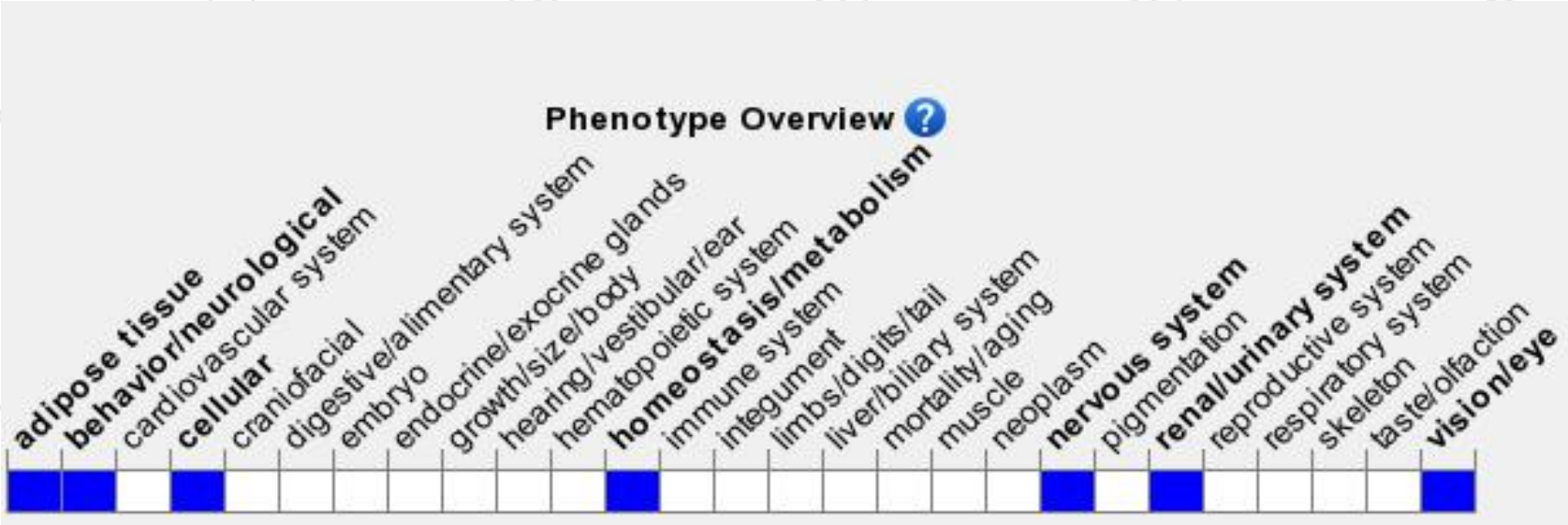
Genomic location distribution



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 disruptions in this gene display reduced locomotor activity and excessive excretion of glutamate and aspartate.

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

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