GemPharmatech™

# Slc33a1 Cas9-CKO Strategy

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Design Date: 2024/1/25

# Overview

## Target Gene Name

- Slc33a1

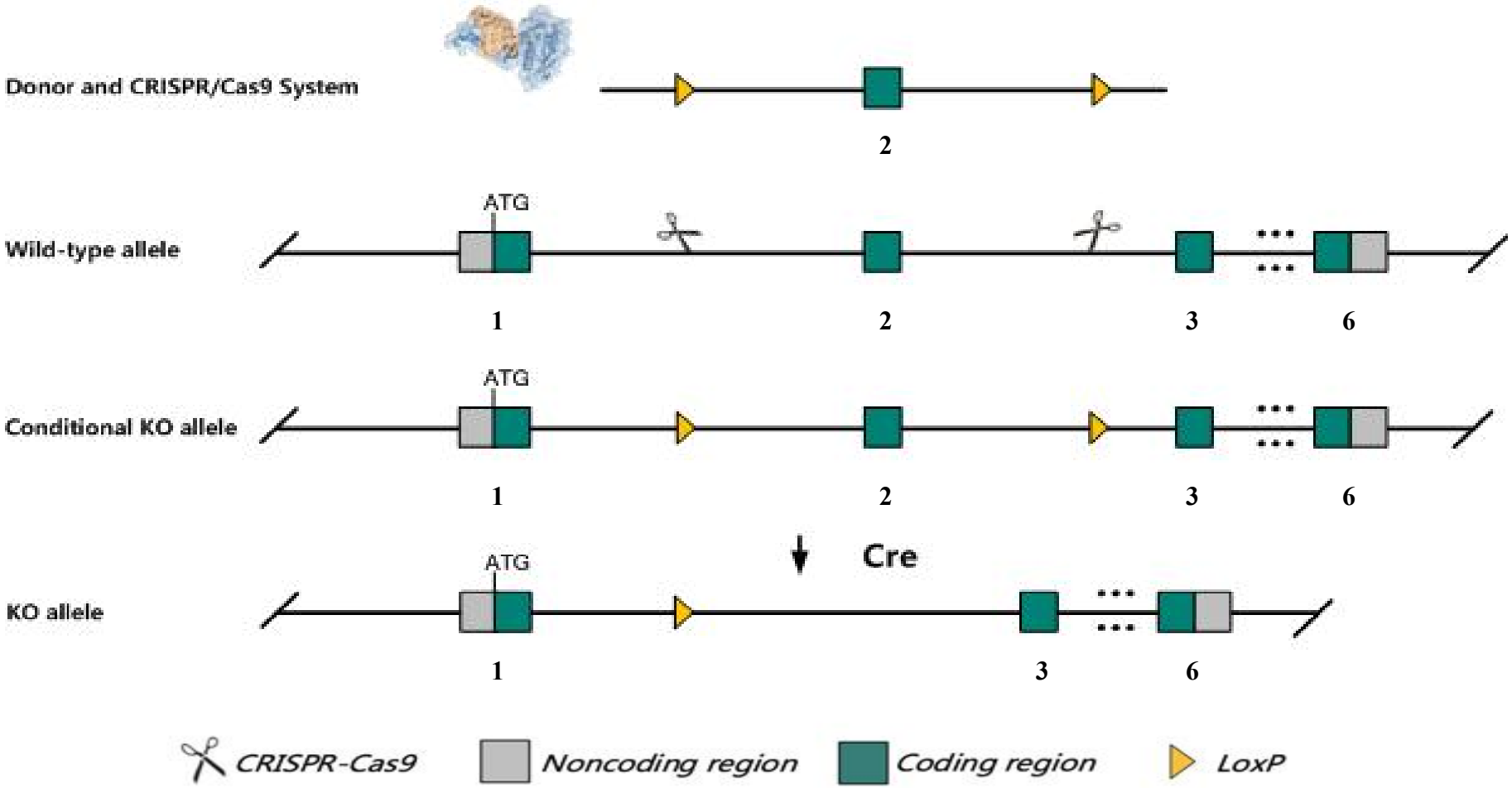
## Project Type

- Cas9-CKO

## Genetic Background

- C57BL/6JGpt

# Strain Strategy

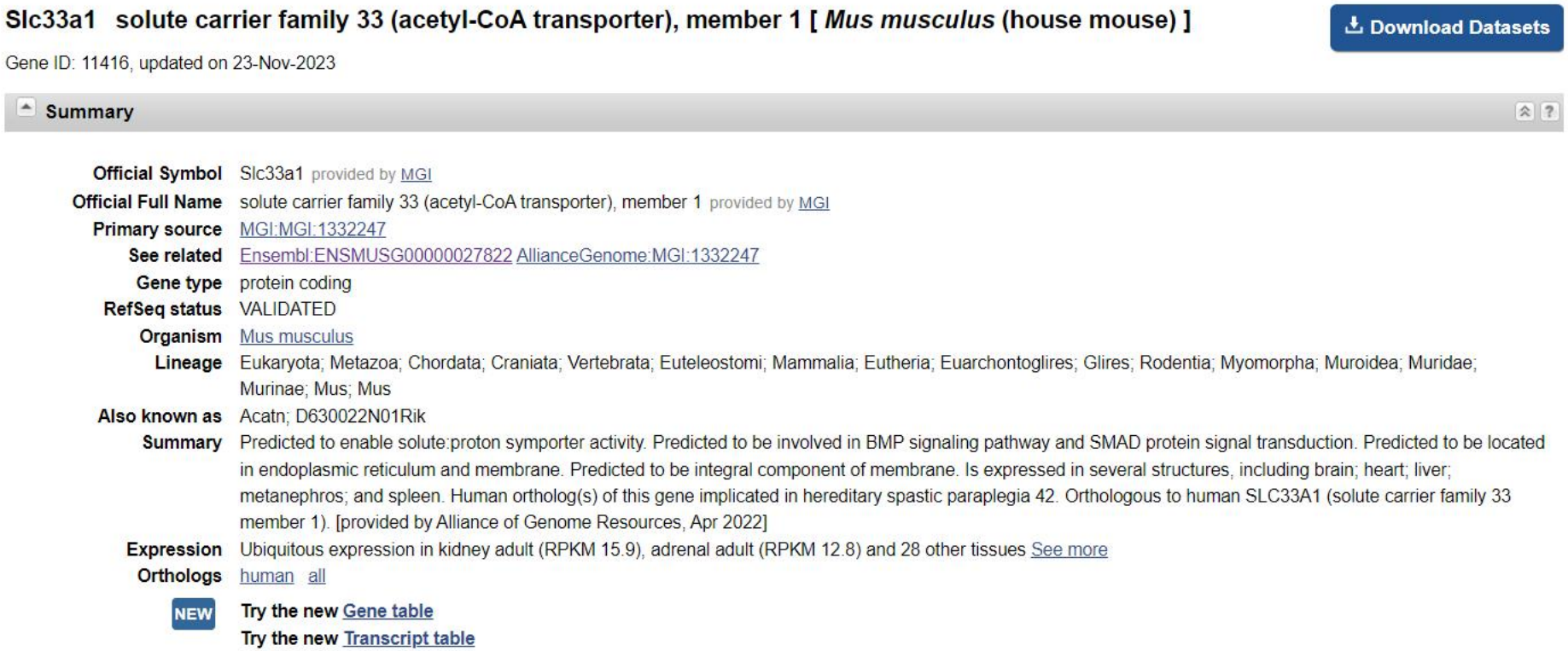



Schematic representation of CRISPR-Cas9 engineering used to edit the *Slc33a1* gene.

# Technical Information

- The *Slc33a1* gene has 3 transcripts. According to the structure of *Slc33a1* gene, exon 2 of *Slc33a1*-201 (ENSMUST00000029402.15) transcript is recommended as the knockout region. The region contains 191 bp coding sequence. Knocking out the region will result in disruption of protein function.
- In this project we use CRISPR-Cas9 technology to modify *Slc33a1* 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 on-target amplicon sequencing. A stable F1-generation mouse strain was obtained by mating positive F0-generation mice with C57BL/6JGpt mice and confirmation of the desired mutant allele was carried out by PCR and on-target amplicon sequencing.
- 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.

# Gene Information



**Slc33a1 solute carrier family 33 (acetyl-CoA transporter), member 1 [ *Mus musculus* (house mouse) ]**

Download Datasets

Gene ID: 11416, updated on 23-Nov-2023

**Summary**

| | |
|---|---|
| **Official Symbol** | Slc33a1 provided by MGI |
| **Official Full Name** | solute carrier family 33 (acetyl-CoA transporter), member 1 provided by MGI |
| **Primary source** | MGI:MGI:1332247 |
| **See related** | Ensembl:ENSMUSG00000027822 AllianceGenome:MGI:1332247 |
| **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** | Acatn; D630022N01Rik |
| **Summary** | Predicted to enable solute:proton symporter activity. Predicted to be involved in BMP signaling pathway and SMAD protein signal transduction. Predicted to be located in endoplasmic reticulum and membrane. Predicted to be integral component of membrane. Is expressed in several structures, including brain; heart; liver; metanephros; and spleen. Human ortholog(s) of this gene implicated in hereditary spastic paraplegia 42. Orthologous to human SLC33A1 (solute carrier family 33 member 1). [provided by Alliance of Genome Resources, Apr 2022] |
| **Expression** | Ubiquitous expression in kidney adult (RPKM 15.9), adrenal adult (RPKM 12.8) and 28 other tissues See more |
| **Orthologs** | human all |

NEW **Try the new Gene table**

**Try the new Transcript table**

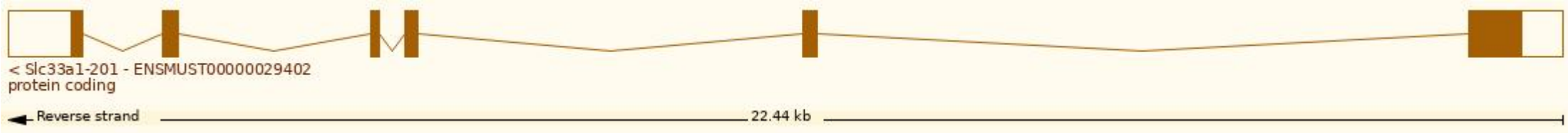
Source: https://www.ncbi.nlm.nih.gov/

# Transcript Information

The gene has 3 transcripts,all transcripts are shown below:

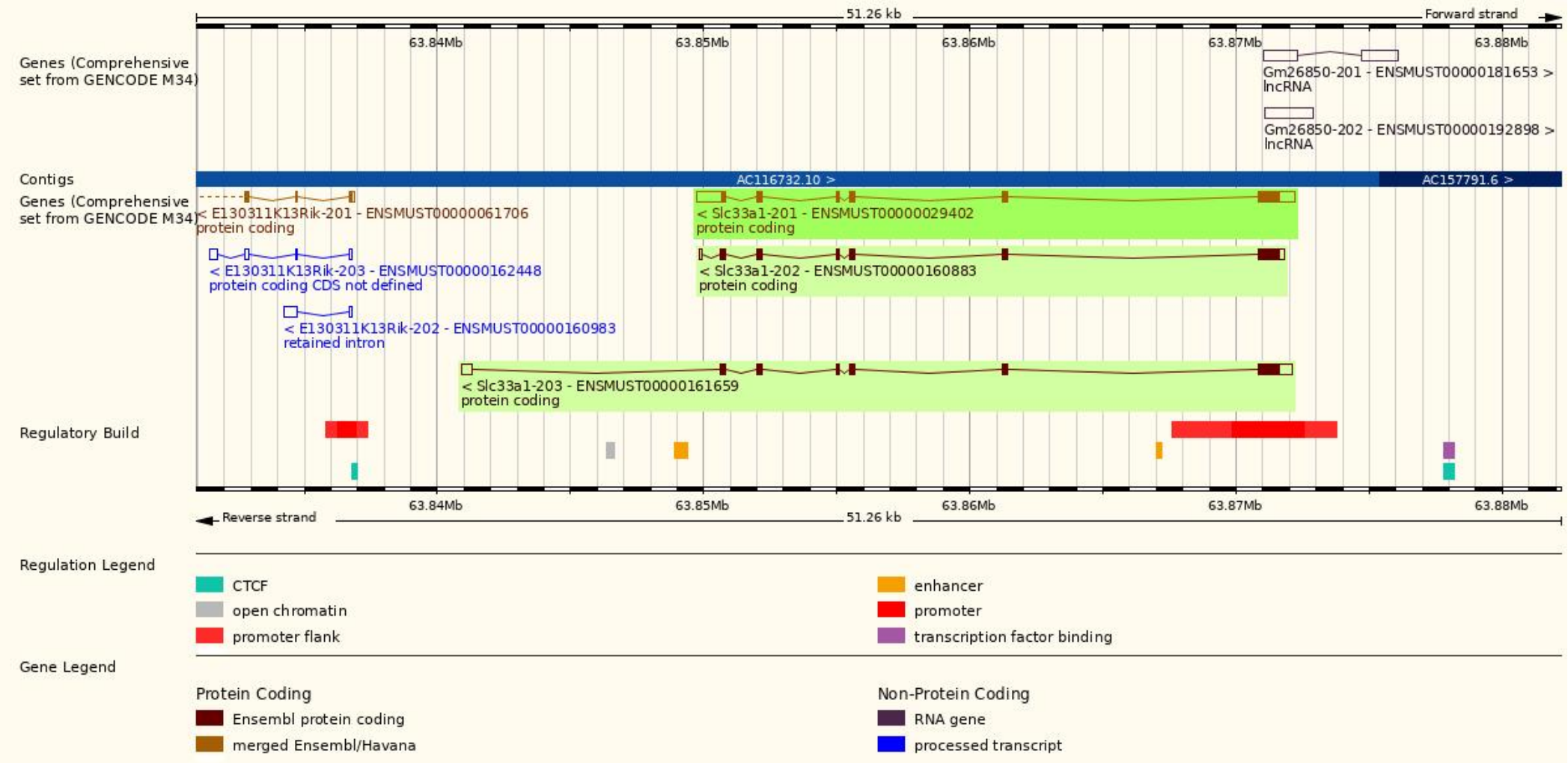
| Transcript ID | Name | bp | Protein | Biotype | CCDS | UniProt Match | Flags |
|---|---|---|---|---|---|---|---|
| ENSMUST00000029402.15 | Slc33a1-201 | 3150 | 550aa | Protein coding | CCDS17382 | Q99J27 | Ensembl Canonical GENCODE basic APPRIS P1 TSL:1 |
| ENSMUST00000161659.8 | Slc33a1-203 | 2554 | 550aa | Protein coding | CCDS17382 | Q99J27 | GENCODE basic APPRIS P1 TSL:1 |
| ENSMUST00000160883.2 | Slc33a1-202 | 1978 | 550aa | Protein coding | CCDS17382 | Q99J27 | GENCODE basic APPRIS P1 TSL:5 |

The strategy is based on the design of *Slc33a1*-201 transcript, the transcription is shown below:




Source: https://www.ensembl.org

# Genomic Information

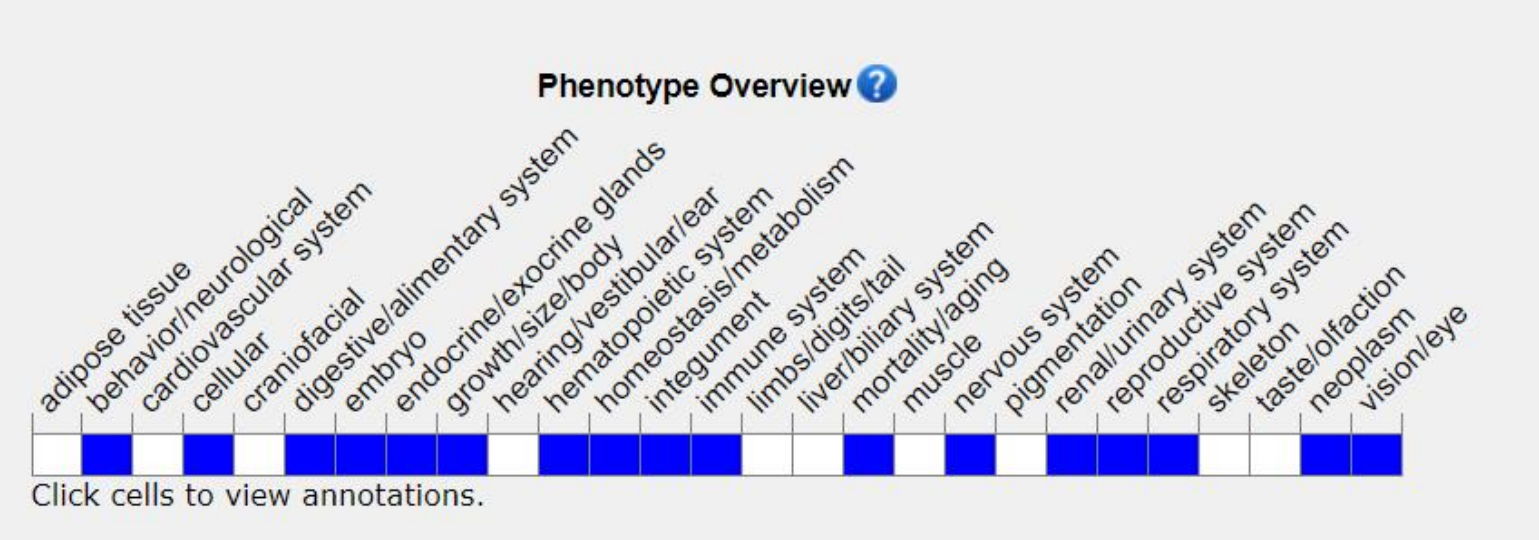



Source: : https://www.ensembl.org

# Protein Information


Source: : https://www.ensembl.org

# Mouse Phenotype Information (MGI)




- Mice homozygous for a serine to arginine substitution at amino acid 113 show early embryonic growth arrest. Adult heterozygotes display aberrant inflammatory response, increased propensity to infections and malignancies, degenerative features of the PNS and CNS, and abnormal induction of autophagy.



Source: https://www.informatics.jax.org

# References

| | | |
|---|---|---|
| **Mutation description** | **Allele Type:** | Targeted (Conditional ready) |
| | **Mutation:** | Insertion |
| | | Mutation details: The L1L2_Bact_P cassette was inserted at position 63860885 of Chromosome 3 upstream of the critical exon(s) (Build GRCm39). The cassette is composed of an FRT site followed by lacZ sequence and a loxP site. This first loxP site is followed by a neomycin resistance gene under the control of the human beta-actin promoter, SV40 polyA, a second FRT site and a second loxP site. A third loxP site is inserted downstream of the targeted exon(s) at position 63861873. The critical exon(s) is/are thus flanked by loxP sites. A "conditional ready" (floxed) allele was created by flp recombinase expression in mice carrying this allele to remove the lacZ sequence and neo selection cassette, leaving loxP sites flanking the critical exon(s). Further information on targeting strategies used for this and other IKMC alleles can be found at http://www.informatics.jax.org/mgihome/nomen/IKMC_schematics.shtml (*J:302198*) |

https://www.informatics.jax.org/allele/MGI:6507953

# Important Information

- According to the existing MGI data，mice homozygous for a serine to arginine substitution at amino acid 113 show early embryonic growth arrest. Adult heterozygotes display aberrant inflammatory response, increased propensity to infections and malignancies, degenerative features of the PNS and CNS, and abnormal induction of autophagy.
- *Slc33a1* is located on Chr 3. If the knockout mice are crossed with other mouse strains to obtain double homozygous mutant offspring, please avoid the situation that the second gene is 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 the existing technology level.