

# Slc35d1 Cas9-CKO Strategy

Designer: Yun Li

Reviewer: Longyun Hu

**Design Date: 2021-1-4** 

# **Project Overview**



Project Name Slc35d1

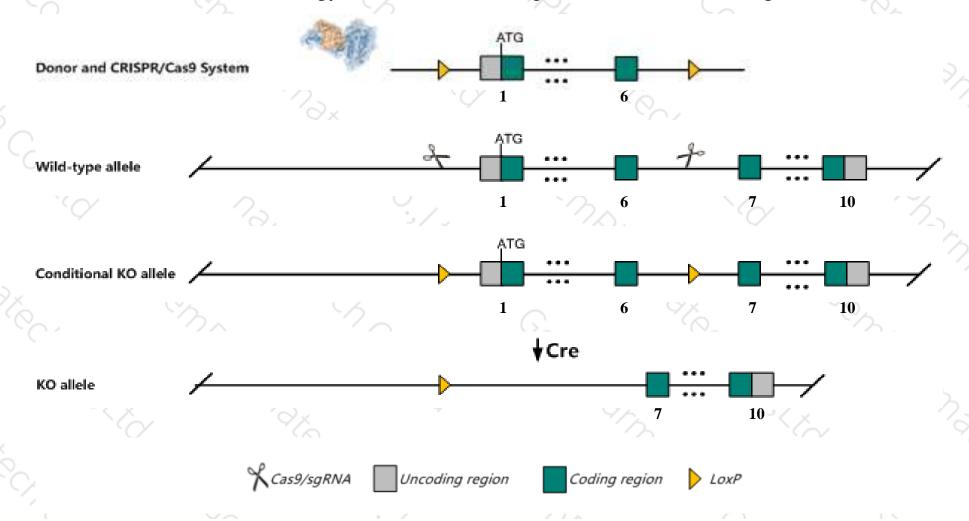
Project type Cas9-CKO

Strain background C57BL/6JGpt

## **Conditional Knockout strategy**



This model will use CRISPR/Cas9 technology to edit the *Slc35d1* gene. The schematic diagram is as follows:



#### **Technical routes**



- ➤ The *Slc35d1* gene has 5 transcripts. According to the structure of *Slc35d1* gene, exon1-exon6 of *Slc35d1*-203(ENSMUST00000150285.7) transcript is recommended as the knockout region. The region contains start codon ATG. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Slc35d1* 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.

#### **Notice**



- ➤ According to the existing MGI data, mice homozygous for a null allele exhibit neonatal lethality and chondrodystrophy associated with impaired chondroitin sulfate biosynthesis.
- ➤ The *Slc35d1* gene is located on the Chr4. 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)



#### Slc35d1 solute carrier family 35 (UDP-glucuronic acid/UDP-N-acetylgalactosamine dual transporter), member D1 [Mus musculus (house mouse)]

Gene ID: 242585, updated on 13-Mar-2020

#### Summary



Official Symbol Slc35d1 provided by MGI

Official Full Name solute carrier family 35 (UDP-glucuronic acid/UDP-N-acetylgalactosamine dual transporter), member D1 provided by MGI

Primary source MGI:MGI:2140361

See related Ensembl:ENSMUSG00000028521

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 Al834976, C330011J09, UGTREL7, mKIAA0260

Expression Ubiquitous expression in liver adult (RPKM 5.4), limb E14.5 (RPKM 3.4) and 28 other tissuesSee more

Orthologs <u>human</u> all

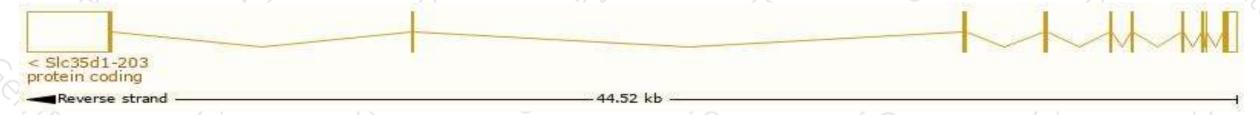
# Transcript information (Ensembl)



The gene has 5 transcripts, all transcripts are shown below:

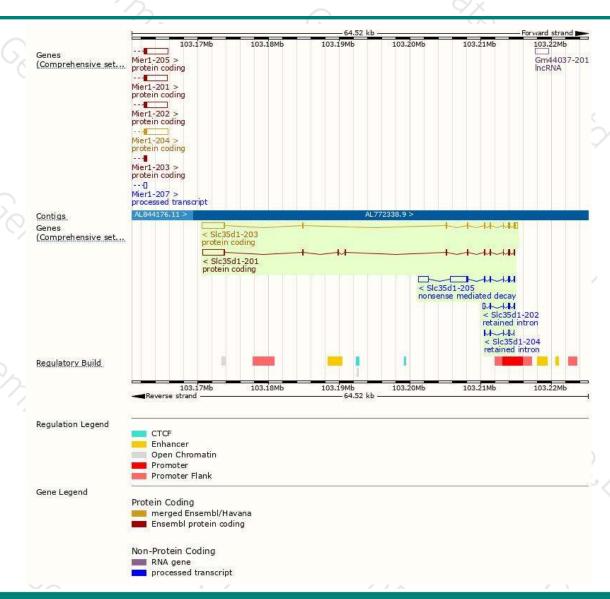
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc35d1-203	ENSMUST00000150285.7	4242	306aa	Protein coding	CCDS18409	A2AKQ0	TSL:1 GENCODE basic APPRIS P1
Slc35d1-201	ENSMUST00000036195.12	4021	<u>334aa</u>	Protein coding	5 <del>4</del> 5	A0A217FL58	CDS 5' incomplete TSL:1
Slc35d1-205	ENSMUST00000183432.5	4414	<u>211aa</u>	Nonsense mediated decay		V9GX08	CDS 5' incomplete TSL:5
Slc35d1-202	ENSMUST00000094947.4	663	No protein	Retained intron	170	-	TSL:1
Slc35d1-204	ENSMUST00000154845.1	416	No protein	Retained intron	121	28	TSL:5

The strategy is based on the design of *Slc35d1-203* transcript, the transcription is shown below:



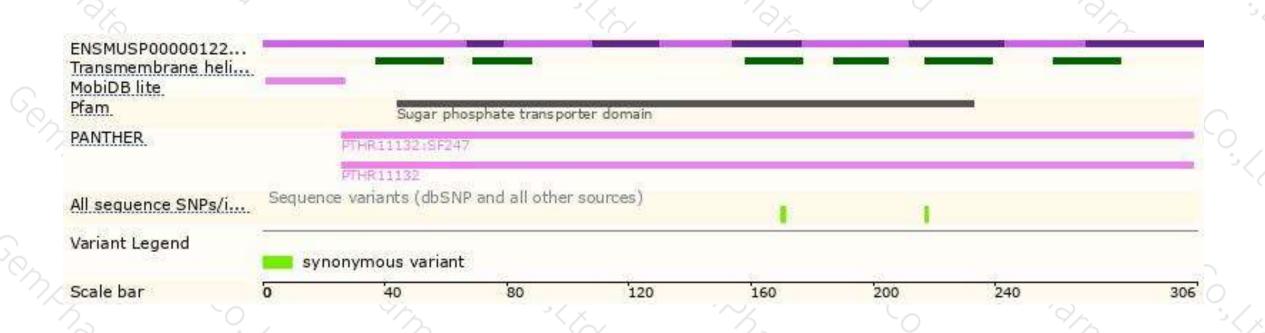
### Genomic location distribution





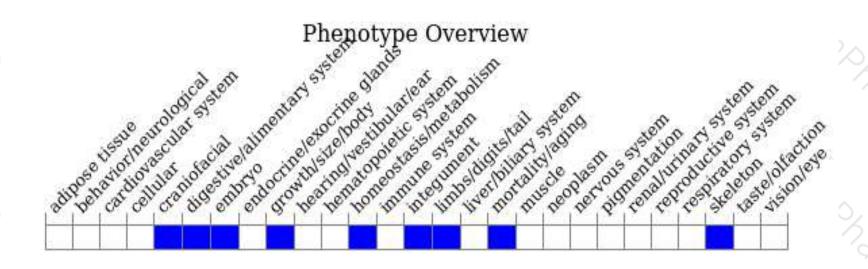
# Protein domain





# 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 null allele exhibit neonatal lethality and chondrodystrophy associated with impaired chondroitin sulfate biosynthesis.



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

Tel: 025-5864 1534





