

# ***Slc26a2 Cas9-KO Strategy***

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**Design Date:** 2019-7-22

# Project Overview

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<b>Project Name</b>	<b><i>Slc26a2</i></b>
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<b>Project type</b>	<b>Cas9-KO</b>
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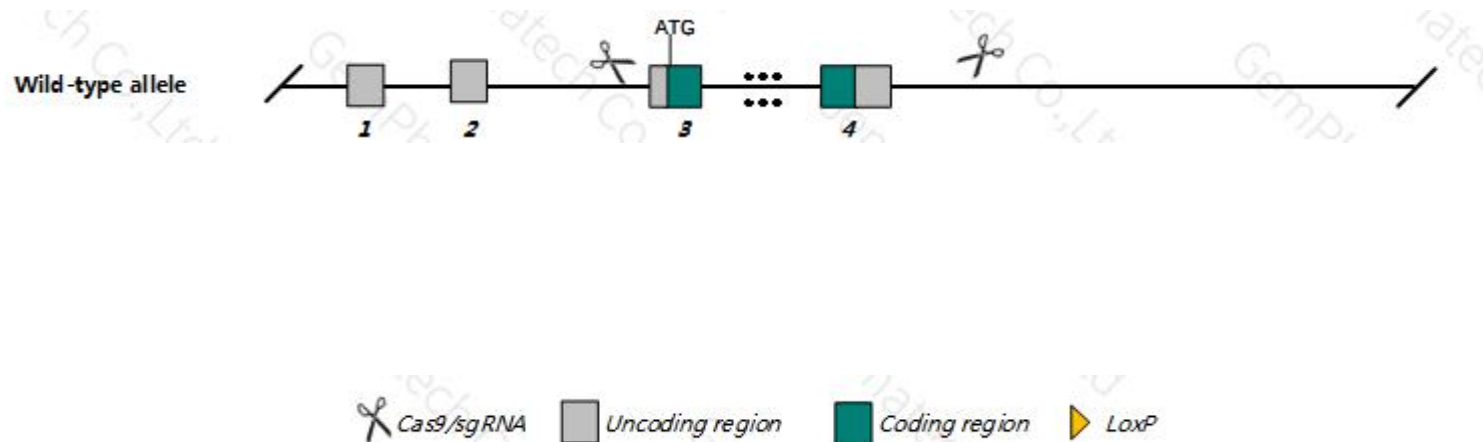
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<b>Strain background</b>	<b>C57BL/6JGpt</b>
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# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc26a2* gene. The schematic diagram is as follows:



- The *Slc26a2* gene has 4 transcripts. According to the structure of *Slc26a2* gene, exon3-4 of *Slc26a2*-202 (ENSMUST00000146409.7) transcript is recommended as the knockout region. The region contains all coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Slc26a2* gene. The brief process is as follows: gRNA was transcribed in vitro, donor was constructed. Cas9, gRNA 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.

- According to the existing MGI data Mice homozygous for a knock-in allele exhibit premature death, stunted growth, joint contractures, and skeletal dysplasia including kyphosis, shorter osteoporotic long bones, aberrant chondrocyte size, delayed endochondral bone ossification, and impaired chondrocyte proliferation and sulfate uptake.
- The *Slc26a2* gene is located on the Chr18. 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

**Slc26a2 solute carrier family 26 (sulfate transporter), member 2 [ *Mus musculus* (house mouse) ]**

Gene ID: 13521, updated on 2-Jul-2019

 **Summary**

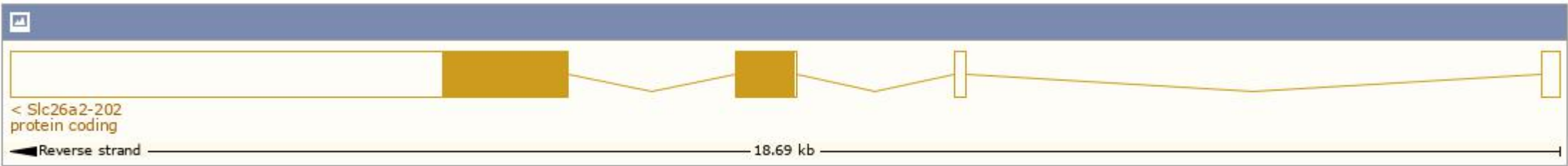
Official Symbol	Slc26a2 provided by <a href="#">MGI</a>
Official Full Name	solute carrier family 26 (sulfate transporter), member 2 provided by <a href="#">MGI</a>
Primary source	<a href="#">MGI:MGI:892977</a>
See related	<a href="#">Ensembl:ENSMUSG00000034320</a>
Gene type	protein coding
RefSeq status	VALIDATED
Organism	<a href="#">Mus musculus</a>
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Dtd; ST-OB
Expression	Biased expression in colon adult (RPKM 28.9), large intestine adult (RPKM 11.3) and 13 other tissues <a href="#">See more</a>
Orthologs	<a href="#">human</a> <a href="#">all</a>

# Transcript information      Ensembl

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

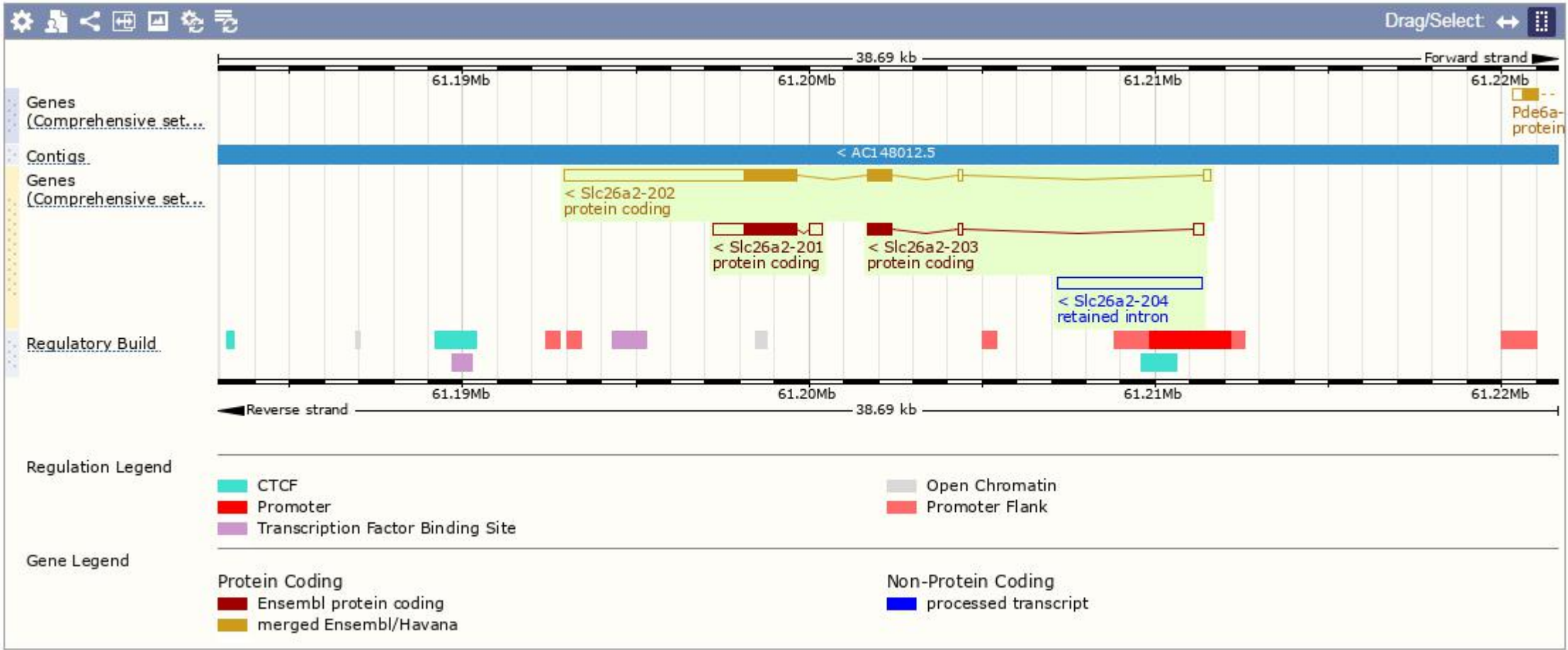
Show/hide columns (1 hidden)							Filter	
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags	
Slc26a2-202	<a href="#">ENSMUST00000146409.7</a>	7814	<a href="#">739aa</a>	Protein coding	<a href="#">CCDS29282</a>	<a href="#">Q62273</a>	TSL:1	GENCODE basic APPRIS P1
Slc26a2-201	<a href="#">ENSMUST00000037603.7</a>	2802	<a href="#">504aa</a>	Protein coding	-	<a href="#">F8WI08</a>	TSL:1	GENCODE basic
Slc26a2-203	<a href="#">ENSMUST00000148829.1</a>	1101	<a href="#">225aa</a>	Protein coding	-	<a href="#">E9PZ06</a>	CDS 3' incomplete	TSL:1
Slc26a2-204	<a href="#">ENSMUST00000181997.1</a>	4168	No protein	Retained intron	-	-	TSL:NA	

The strategy is based on the design of *Slc26a2*-202 transcript, The transcription is shown below

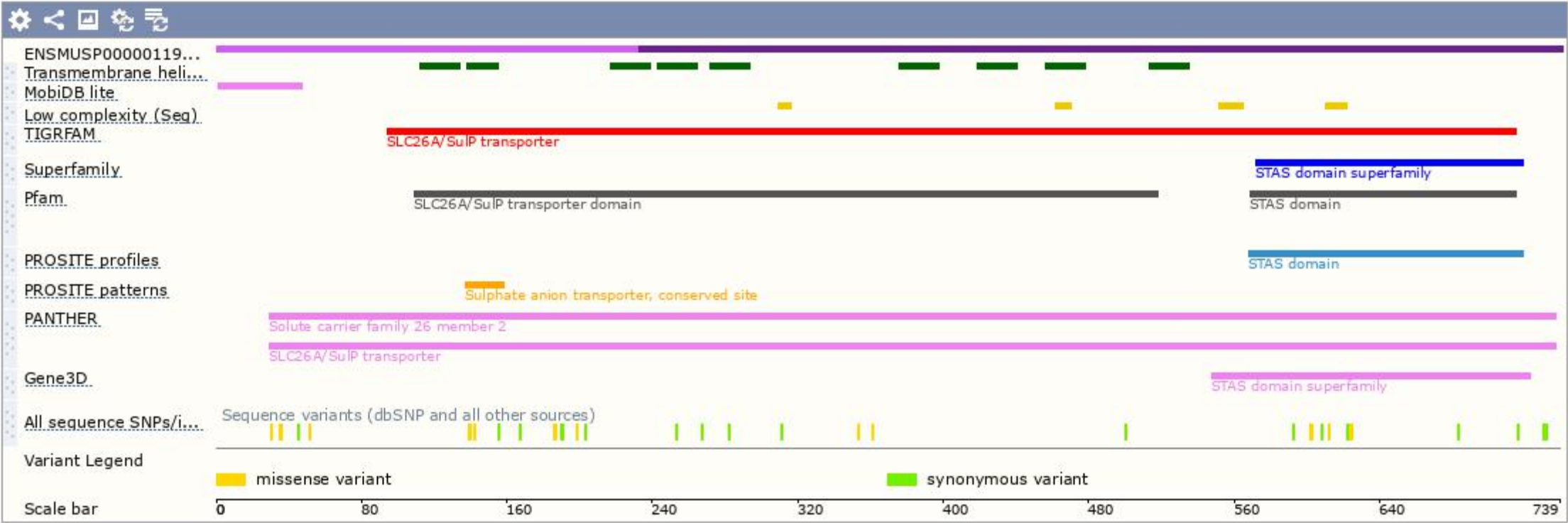


Statistics      Exons: 4, Coding exons: 2, Transcript length: 7,814 bps, Translation length: 739 residues

# 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/>) .*

Mice homozygous for a knock-in allele exhibit premature death, stunted growth, joint contractures, and skeletal dysplasia including kyphosis, shorter osteoporotic long bones, aberrant chondrocyte size, delayed endochondral bone ossification, and impaired chondrocyte proliferation and sulfate uptake.

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