

# ***Slc9b2*** Cas9-KO Strategy

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**Reviewer:**

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**Design Date:**

**2020-2-21**

# Project Overview

**Project Name**

*Slc9b2*

**Project type**

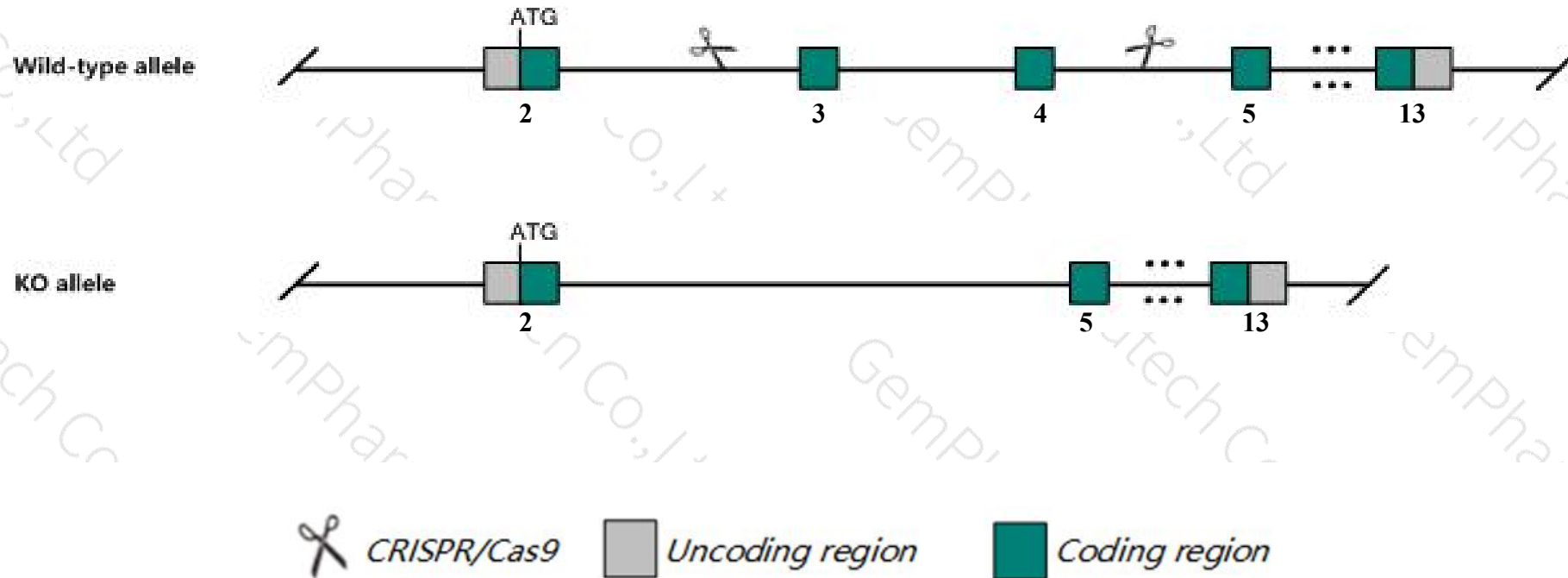
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc9b2* gene. The schematic diagram is as follows:



- The *Slc9b2* gene has 5 transcripts. According to the structure of *Slc9b2* gene, exon3-exon4 of *Slc9b2-201* (ENSMUST00000051849.9) transcript is recommended as the knockout region. The region contains 352bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Slc9b2* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Mice homozygous for a gene trapped allele are viable and overtly normal, with no detectable abnormalities in osteoclast differentiation and function.
- The *Slc9b2* gene is located on the Chr3. 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)

**Slc9b2** solute carrier family 9, subfamily B (NHA2, cation proton antiporter 2), member 2 [ *Mus musculus* (house mouse) ]

Gene ID: 97086, updated on 12-Aug-2019

**Summary**

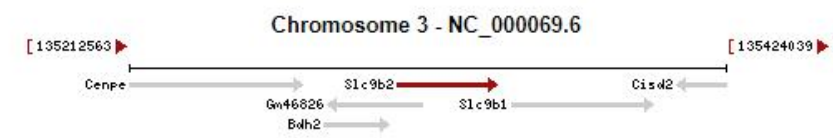
**Official Symbol** Slc9b2 provided by MGI  
**Official Full Name** solute carrier family 9, subfamily B (NHA2, cation proton antiporter 2), member 2 provided by MGI  
**Primary source** MGI:MGI:2140077  
**See related** Ensembl:ENSMUSG00000037994  
**Gene type** protein coding  
**RefSeq status** VALIDATED  
**Organism** *Mus musculus*  
**Lineage** Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus  
**Also known as** NHA2; NHE10; nhaoc; C80638; Nhdc2; nha-oc  
**Expression** Low expression observed in reference dataset [See more](#)  
**Orthologs** [human](#) [all](#)

**Genomic context**

**Location:** 3; 3 G3 [See Slc9b2 in Genome Data Viewer](#)

**Exon count:** 15

Annotation release	Status	Assembly	Chr	Location
<a href="#">108</a>	current	GRCm38.p6 ( <a href="#">GCF_000001635.26</a> )	3	NC_000069.6 (135307651..135342845)
Build 37.2	previous assembly	MGSCv37 ( <a href="#">GCF_000001635.18</a> )	3	NC_000069.5 (134970664..135005731)

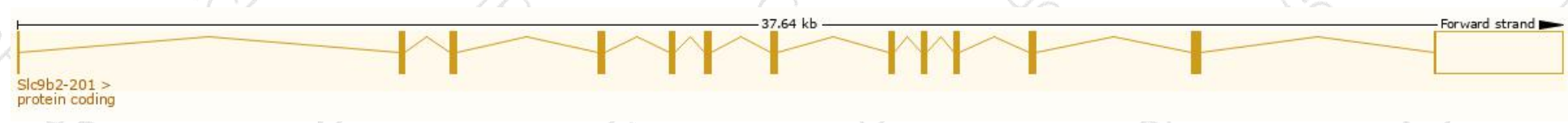


# Transcript information (Ensembl)

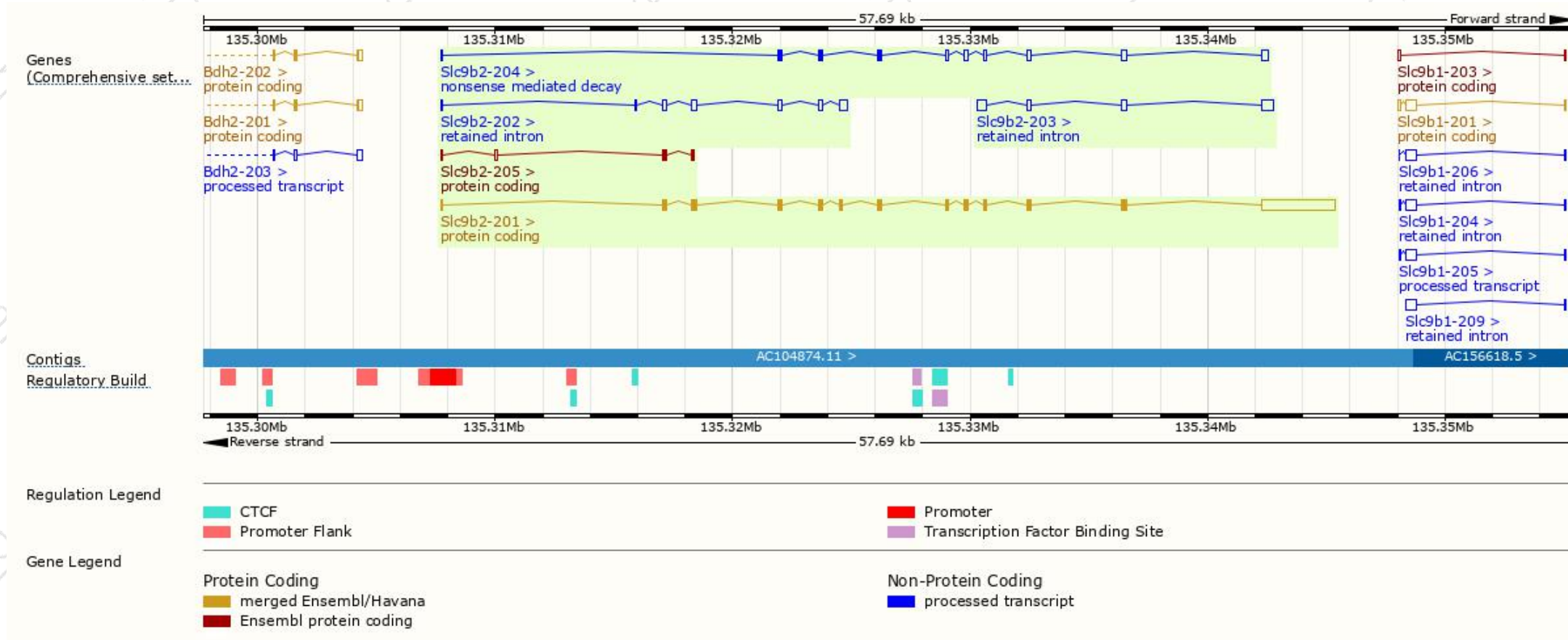
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc9b2-201	<a href="#">ENSMUST00000051849.9</a>	4818	<a href="#">547aa</a>	Protein coding	<a href="#">CCDS17854</a>	<a href="#">Q5BKR2</a>	TSL:1 GENCODE basic APPRIS P1
Slc9b2-205	<a href="#">ENSMUST00000149655.5</a>	356	<a href="#">58aa</a>	Protein coding	-	<a href="#">A0A0G2JEX4</a>	CDS 3' incomplete TSL:3
Slc9b2-204	<a href="#">ENSMUST00000145195.7</a>	1562	<a href="#">143aa</a>	Nonsense mediated decay	-	<a href="#">D6RDC5</a>	TSL:1
Slc9b2-203	<a href="#">ENSMUST00000132405.1</a>	1242	No protein	Retained intron	-	-	TSL:2
Slc9b2-202	<a href="#">ENSMUST00000126034.3</a>	1081	No protein	Retained intron	-	-	TSL:1

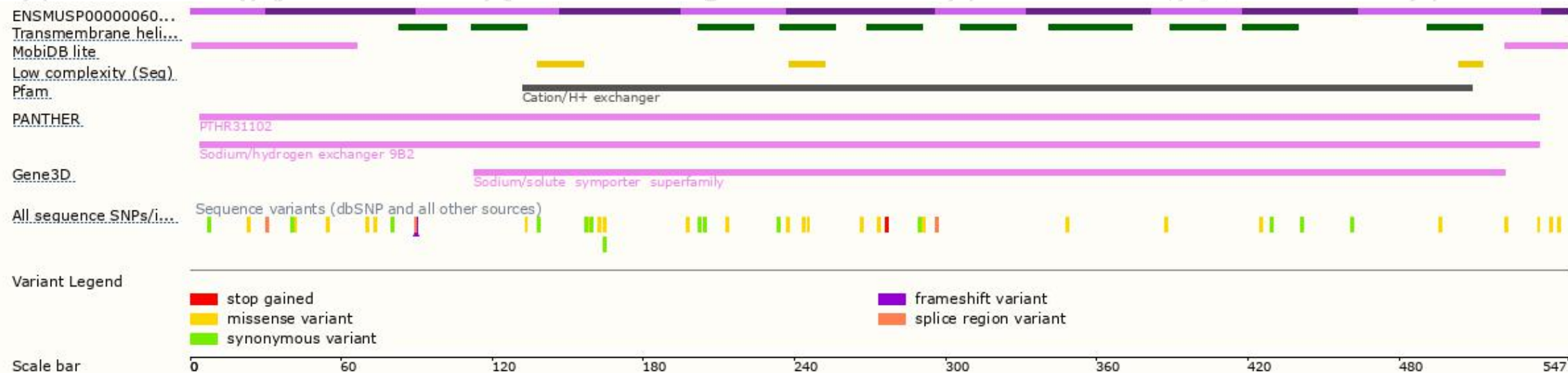
The strategy is based on the design of *Slc9b2-201* transcript,The transcription is shown below



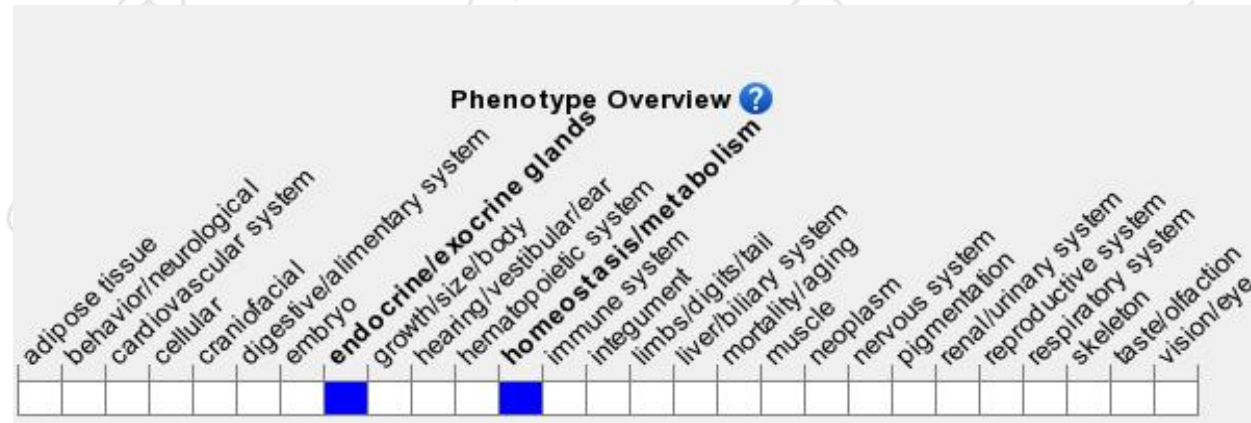
# Genomic location distribution



# 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 gene trapped allele are viable and overtly normal, with no detectable abnormalities in osteoclast differentiation and function.

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

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