



Slc9b2 Cas9-CKO Strategy

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Reviewer:

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Design Date:

2020-2-21

Project Overview

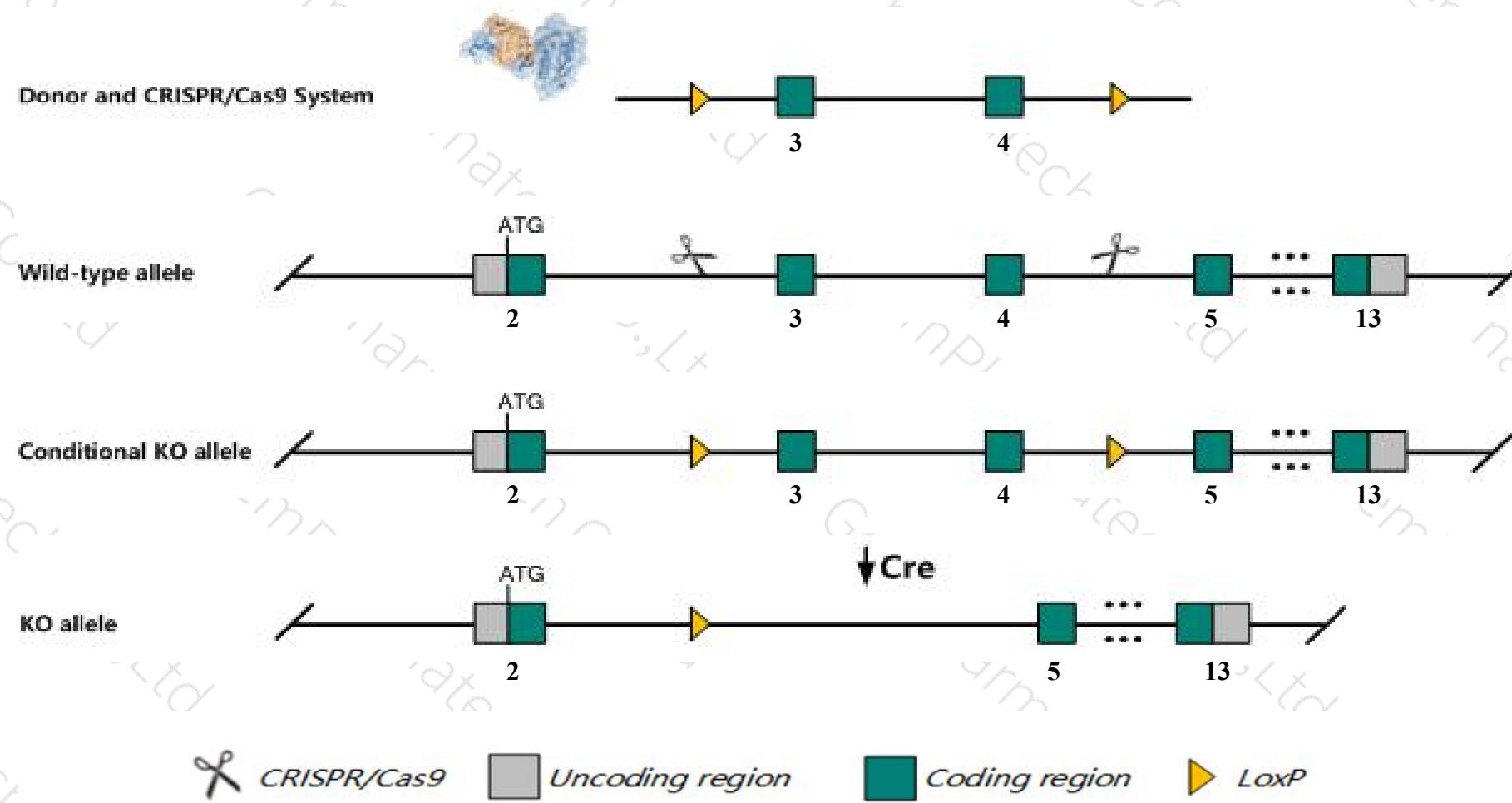
Project Name***Slc9b2***

Project type**Cas9-CKO**

Strain background**C57BL/6JGpt**

Conditional Knockout strategy

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



Technical routes

- 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 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.



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Notice

- 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological 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; Nhedc2; nha-oc
Expression	Low expression observed in reference dataset See more
Orthologs	human all

Genomic context

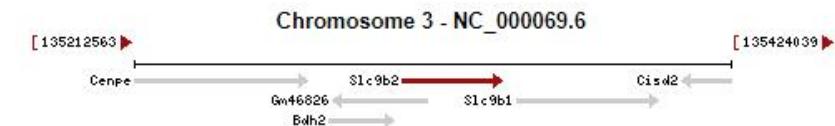


Location: 3; 3 G3

[See Slc9b2 in Genome Data Viewer](#)

Exon count: 15

Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	3	NC_000069.6 (135307651..135342845)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	3	NC_000069.5 (134970664..135005731)

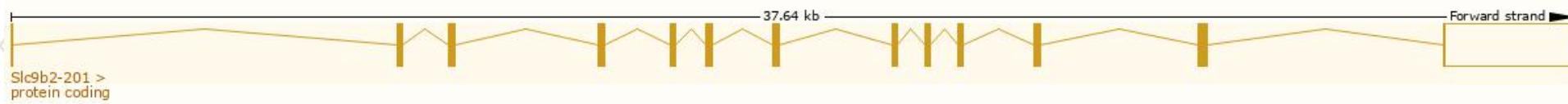


Transcript information (Ensembl)

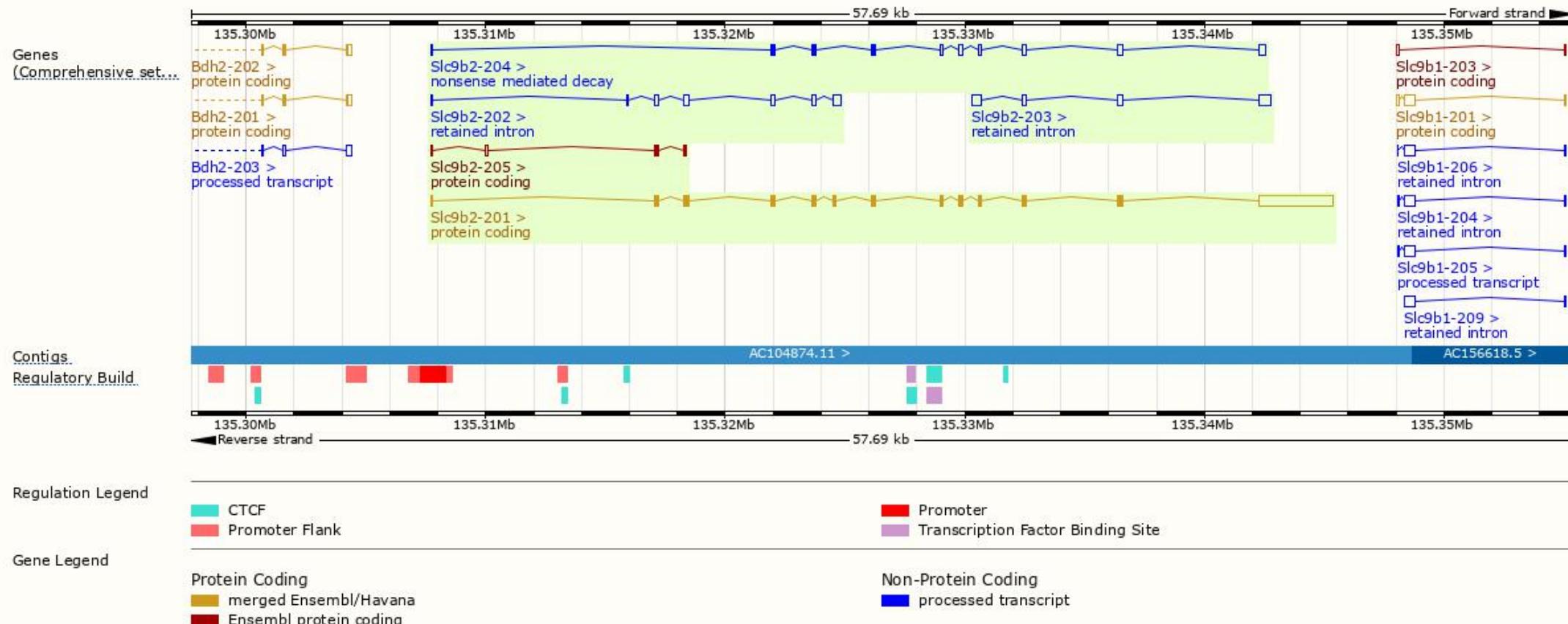
The gene has 5 transcripts, all transcripts are shown below:

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

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



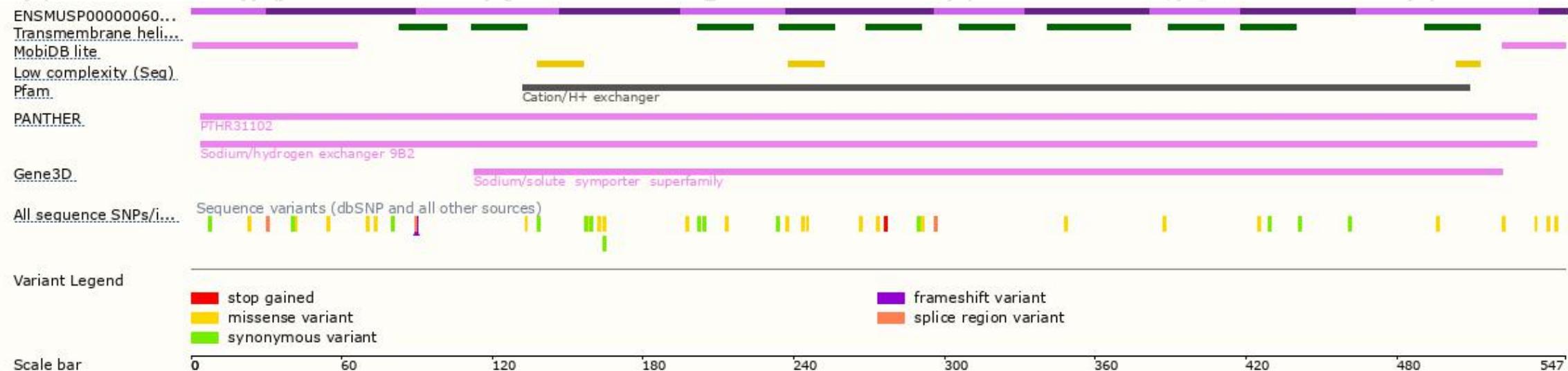
Genomic location distribution



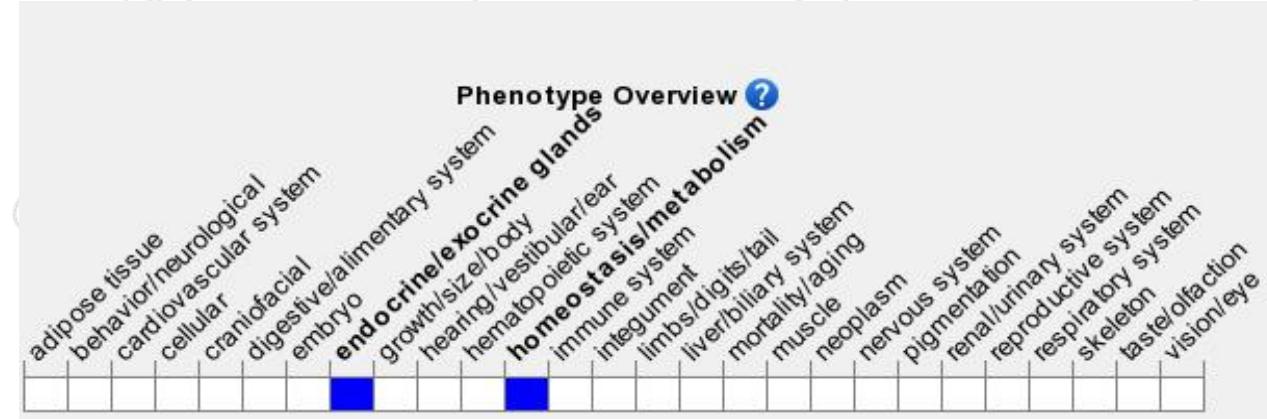


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Protein domain



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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