

Grin3a Cas9-CKO Strategy

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Project Overview

Project Name

Grin3a

Project type

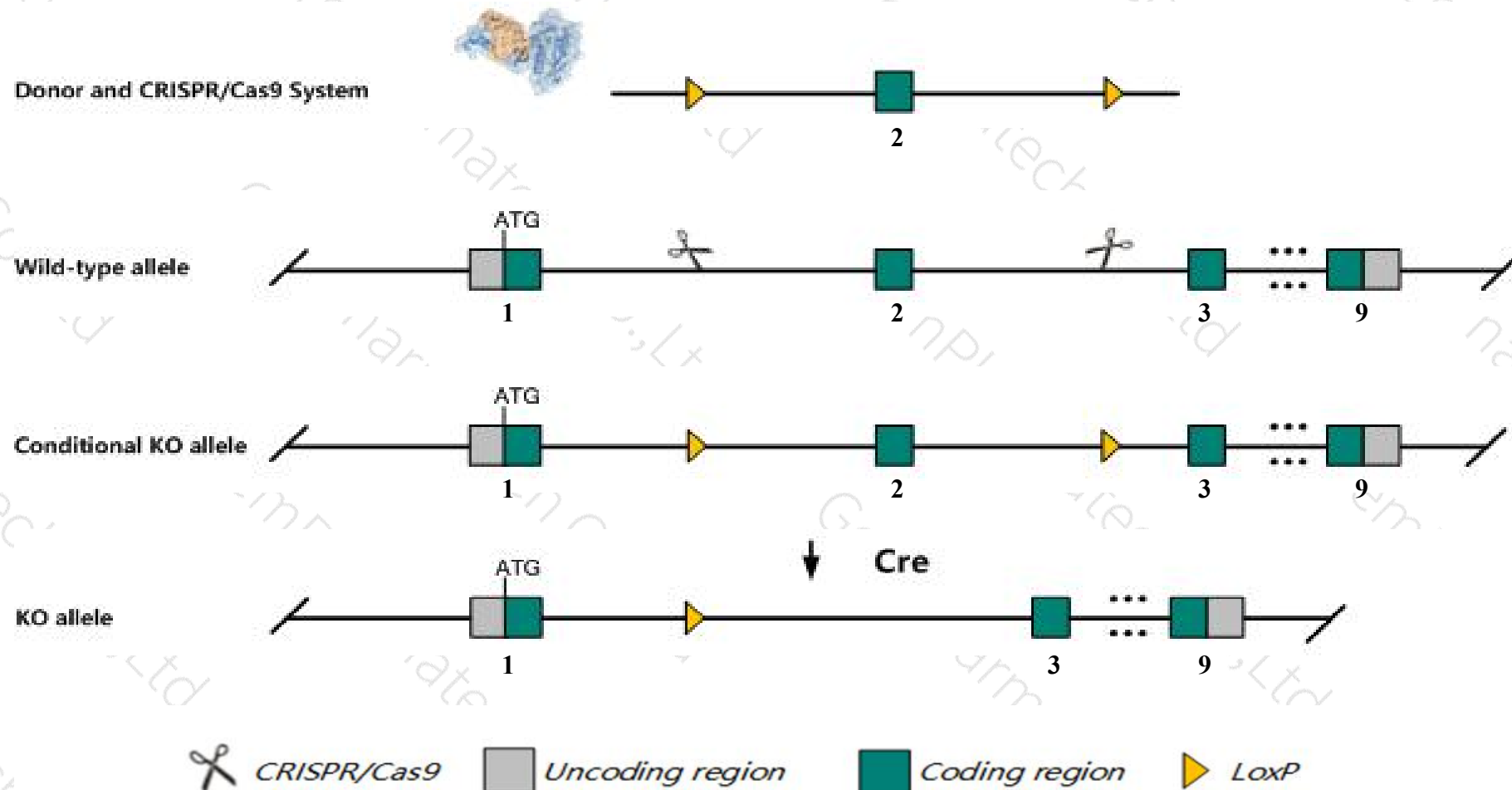
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Grin3a* gene. The schematic diagram is as follows:



Technical routes

- The *Grin3a* gene has 4 transcripts. According to the structure of *Grin3a* gene, exon2 of *Grin3a-202* (ENSMUST00000093859.10) transcript is recommended as the knockout region. The region contains 605bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Grin3a* 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.

- According to the existing MGI data, Mice homozygous for a disruption in this gene display increased current densities in some cerebrocortical neurons of the brain, increased levels of prepulse inhibition, and altered dendritic spine morphology. Otherwise, they display a normal phenotype.
- Transcript *Grin3a*-204 may not be affected
- The N-terminal of *Grin3a* gene will remain 233aa, it may remain the partial function of *Grin3a* gene.
- The *Grin3a* 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)

Grin3a glutamate receptor ionotropic, NMDA3A [Mus musculus (house mouse)]

Gene ID: 242443, updated on 31-Jan-2019

Summary



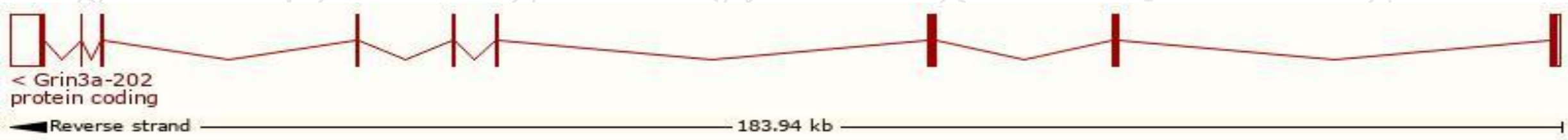
Official Symbol	Grin3a provided by MGI
Official Full Name	glutamate receptor ionotropic, NMDA3A provided by MGI
Primary source	MGI:MGI:1933206
See related	Ensembl:ENSMUSG00000039579
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	6430537F04, A830097C19Rik, NMDAR-L, NR3A, mKIAA1973
Expression	Biased expression in CNS E18 (RPKM 8.2), whole brain E14.5 (RPKM 3.6) and 6 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

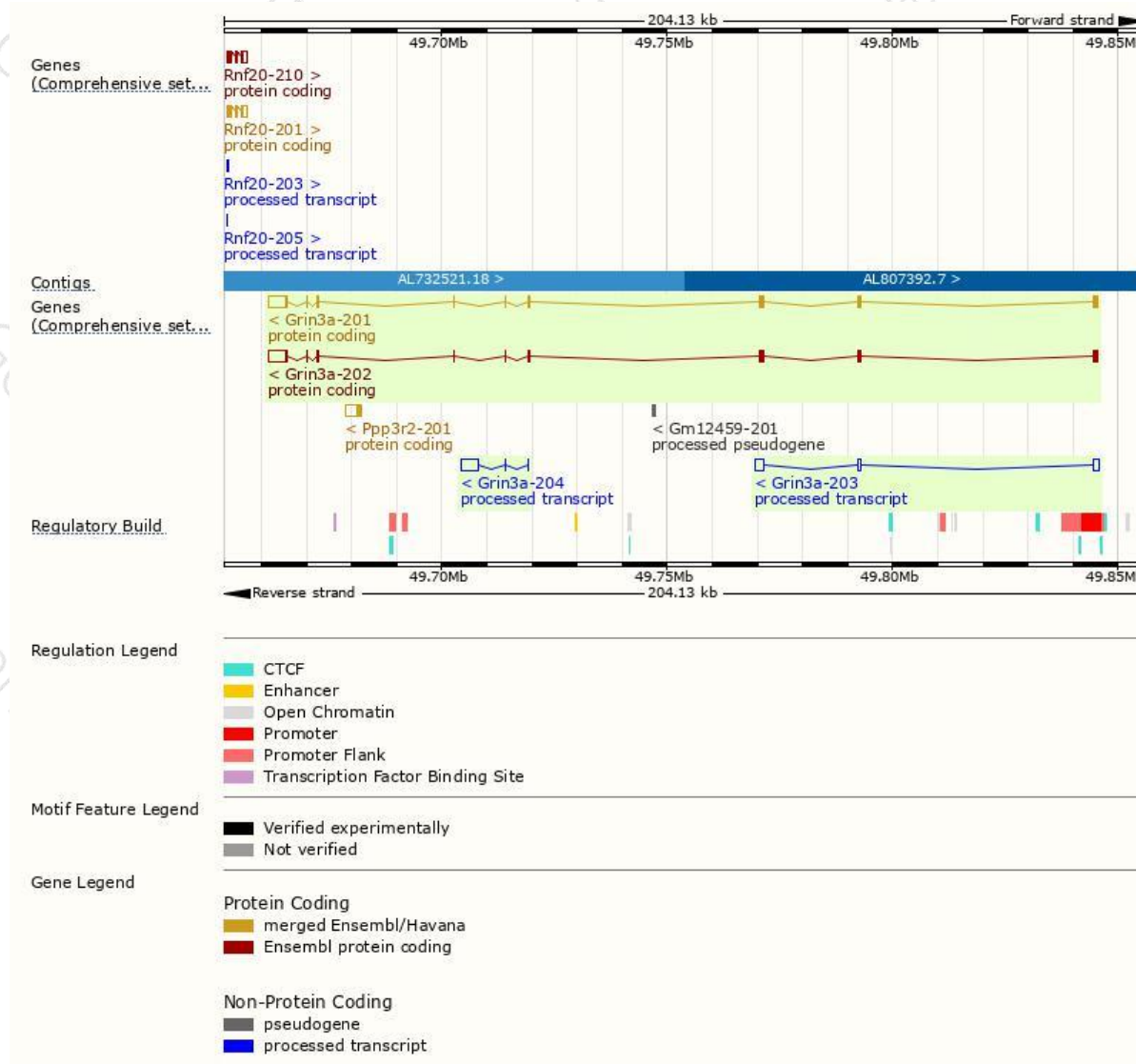
The gene has 4 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Grin3a-202	ENSMUST00000093859.10	7491	1135aa	Protein coding	CCDS71385	A2AIR5	TSL:1 GENCODE basic
Grin3a-201	ENSMUST00000076674.3	7431	1115aa	Protein coding	CCDS51175	A2AIR4	TSL:1 GENCODE basic APPRIS P1
Grin3a-204	ENSMUST00000149059.1	3925	No protein	Processed transcript	-	-	TSL:5
Grin3a-203	ENSMUST00000131797.1	3799	No protein	Processed transcript	-	-	TSL:1

The strategy is based on the design of *Grin3a-202* transcript,The transcription is shown below



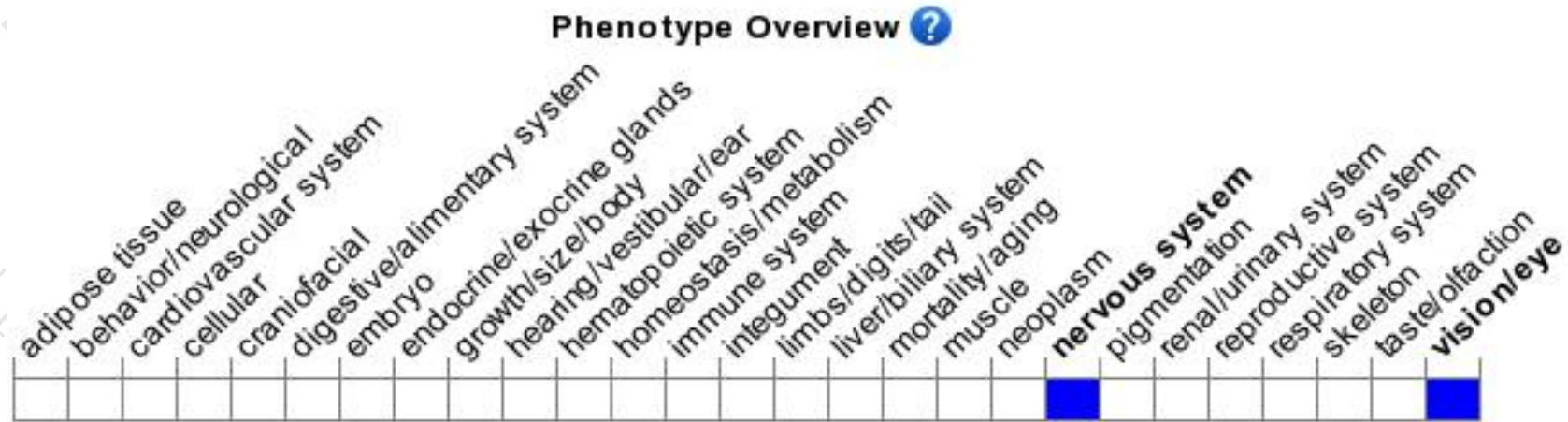
Genomic location distribution



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 disruption in this gene display increased current densities in some cerebrocortical neurons of the brain, increased levels of prepulse inhibition, and altered dendritic spine morphology. Otherwise, they display a normal phenotype.

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

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