

Tnfrsf19 Cas9-CKO Strategy

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Design Date: 2019-8-23

Project Overview



Project Name

Tnfrsf19

Project type

Cas9-CKO

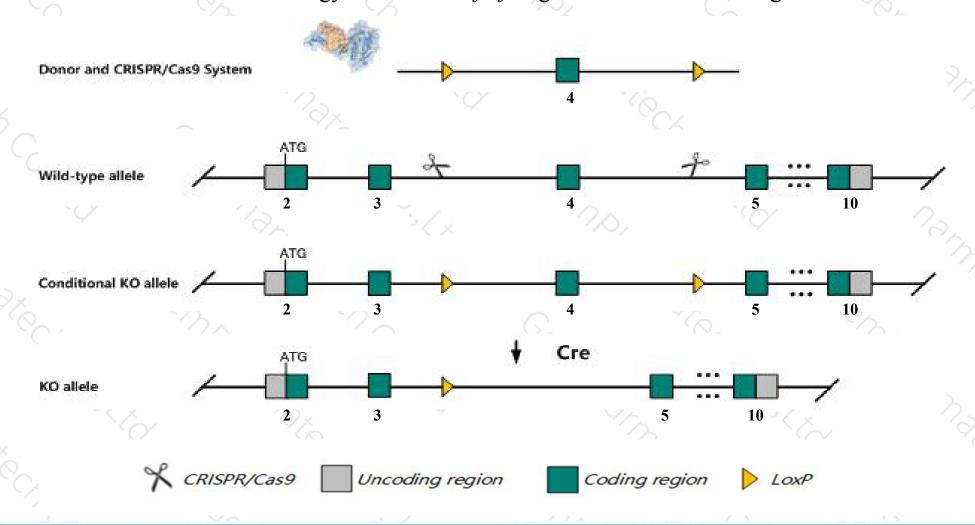
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Tnfrsf19* gene. The schematic diagram is as follows:



Technical routes



- The *Tnfrsf19* gene has 5 transcripts. According to the structure of *Tnfrsf19* gene, exon4 of *Tnfrsf19-201* (ENSMUST00000111234.9) transcript is recommended as the knockout region. The region contains 179bp coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Tnfrsf19* 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.

Notice



- ➤ According to the existing MGI data, Homozygous mutant mice exhibit no obvious physical abnormalities or alterations in behavior, locomotion, or fecundity, however neurons are more resistant to the suppressive action of myelin inhibitors.
- > The *Tnfrsf19* gene is located on the Chr14. 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)



Tnfrsf19 tumor necrosis factor receptor superfamily, member 19 [Mus musculus (house mouse)]

Gene ID: 29820, updated on 3-Feb-2019

Summary

☆ ?

Official Symbol Tnfrsf19 provided by MGI

Official Full Name tumor necrosis factor receptor superfamily, member 19 provided by MGI

Primary source MGI:MGI:1352474

See related Ensembl: ENSMUSG00000060548

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 AL023044, AW123854, TAJ, TAJ-ALPHA, TRADE, Troy

Expression Broad expression in adrenal adult (RPKM 14.8), CNS E11.5 (RPKM 12.6) and 21 other tissuesSee more

Orthologs <u>human</u> all

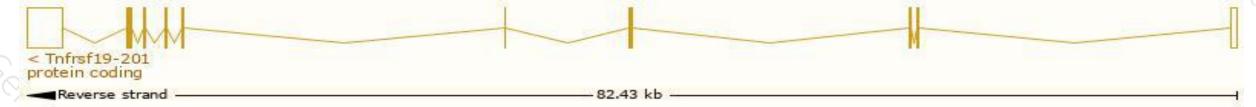
Transcript information (Ensembl)



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

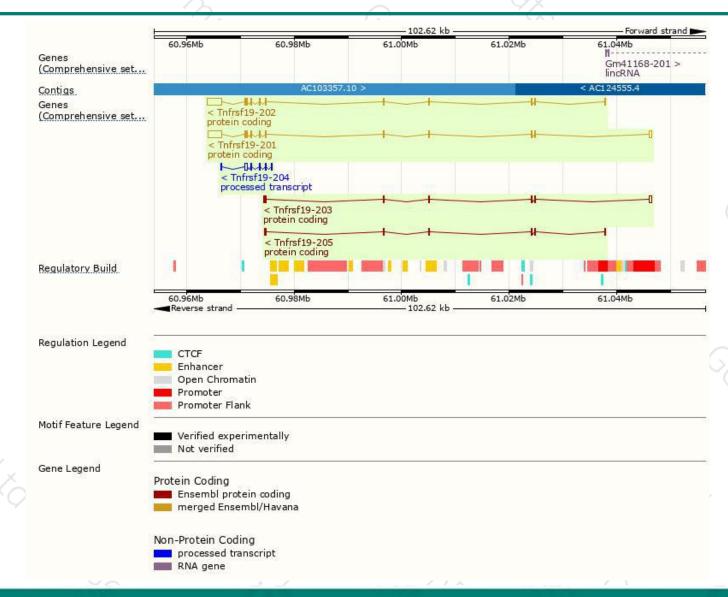
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Tnfrsf19-201	ENSMUST00000111234.9	4051	416aa	Protein coding	CCDS36941	Q9JLL3	TSL:1 GENCODE basic APPRIS P2
Tnfrsf19-202	ENSMUST00000111236.3	3958	<u>416aa</u>	Protein coding	CCDS36941	Q9JLL3	TSL:1 GENCODE basic APPRIS P2
Tnfrsf19-203	ENSMUST00000224371.1	1239	214aa	Protein coding	2	Q9JLL3	GENCODE basic APPRIS ALT2
Tnfrsf19-205	ENSMUST00000225730.1	744	214aa	Protein coding	24	Q9JLL3	GENCODE basic APPRIS ALT2
Tnfrsf19-204	ENSMUST00000225501.1	1087	No protein	Processed transcript	-	-	

The strategy is based on the design of *Tnfrsf19-201* transcript, The transcription is shown below



Genomic location distribution





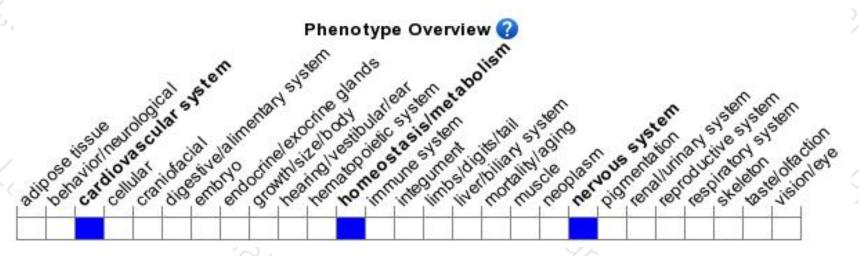
Protein domain





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, Homozygous mutant mice exhibit no obvious physical abnormalities or alterations in behavior, locomotion, or fecundity, however neurons are more resistant to the suppressive action of myelin inhibitors.



If you have any questions, you are welcome to inquire. Tel: 400-9660890





