

Eif2s2 Cas9-CKO Strategy

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Reviewer Shilei Zhu

Design Date: 2018/10/13

Project Overview



Project Name Eif2s2

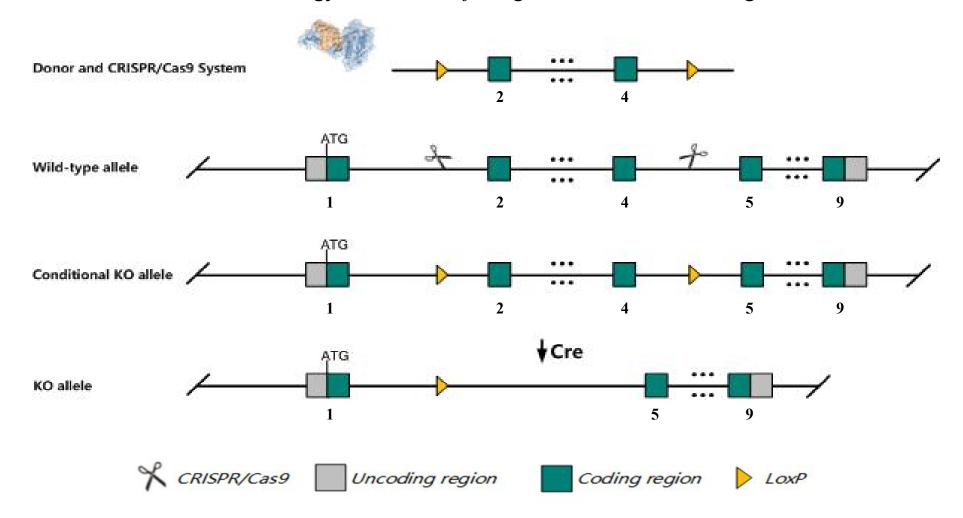
Project type Cas9-CKO

Strain background C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Eif2s2* gene. The schematic diagram is as follows:



Technical routes



The *Eif2s2* gene has 5 transcripts. According to the structure of *Eif2s2* gene, exon2-exon4 of *Eif2s2-201* (ENSMUST00000099173.10) transcript is recommended as the knockout region. The region contains 418bp coding sequence. Knock out the region will result in disruption of protein function.

In this project we use CRISPR/Cas9 technology to modify *Eif2s2* 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 gene trap allele exhibit embryonic lethallity prior to E8.5. Mice heterozygous for a gene trap allele exhibit reduced incidence of testicular germ cell tumors.

The *Eif2s2* gene is located on the Chr2. 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



Eif2s2 eukaryotic translation initiation factor 2, subunit 2 (beta) [Mus musculus (house mouse)]

Gene ID: 67204, updated on 31-Jan-2019

Summary

☆ ?

Official Symbol Eif2s2 provided by MGI

Official Full Name eukaryotic translation initiation factor 2, subunit 2 (beta) provided by MGI

Primary source MGI:MGI:1914454

See related Ensembl:ENSMUSG00000074656

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 2810026E11Rik, 38kDa, AA408636, AA571381, AA986487, AW822225, D2Ertd303e, EIF2, EIF2B

Expression Broad expression in CNS E11.5 (RPKM 37.7), placenta adult (RPKM 37.6) and 22 other tissuesSee more

Orthologs <u>human all</u>

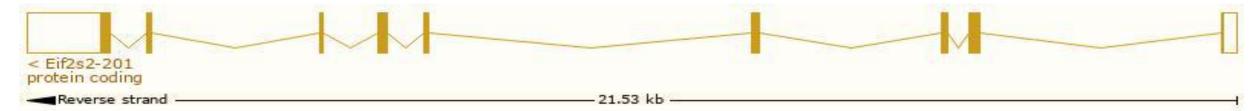
Transcript information Ensembl



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

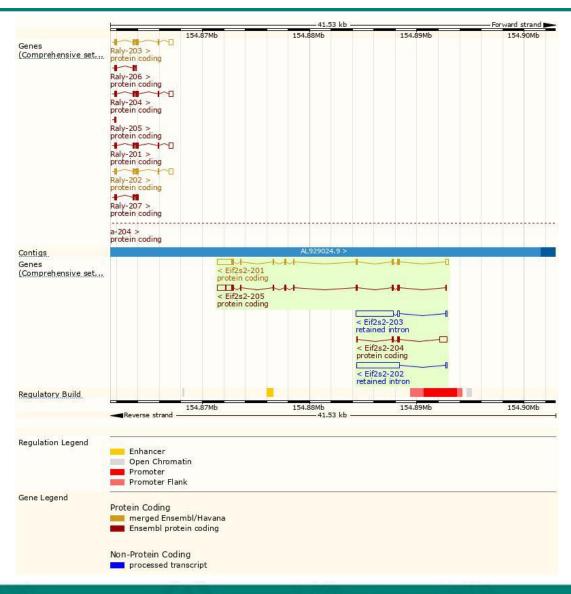
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Eif2s2-201	ENSMUST00000099173.10	2542	<u>331aa</u>	Protein coding	CCDS38292	Q3ULL5 Q99L45	TSL:1 GENCODE basic APPRIS P1
Eif2s2-205	ENSMUST00000166171.7	2312	<u>331aa</u>	Protein coding	CCDS38292	Q3ULL5 Q99L45	TSL:1 GENCODE basic APPRIS P1
Eif2s2-204	ENSMUST00000161172.2	1041	118aa	Protein coding	120	E0CXJ3	CDS 3' incomplete TSL:2
Eif2s2-202	ENSMUST00000135524.1	4102	No protein	Retained intron	725	24	TSL:1
Eif2s2-203	ENSMUST00000147136.1	3785	No protein	Retained intron	-	-	TSL:1

The strategy is based on the design of *Eif2s2-201* transcript, The transcription is shown below



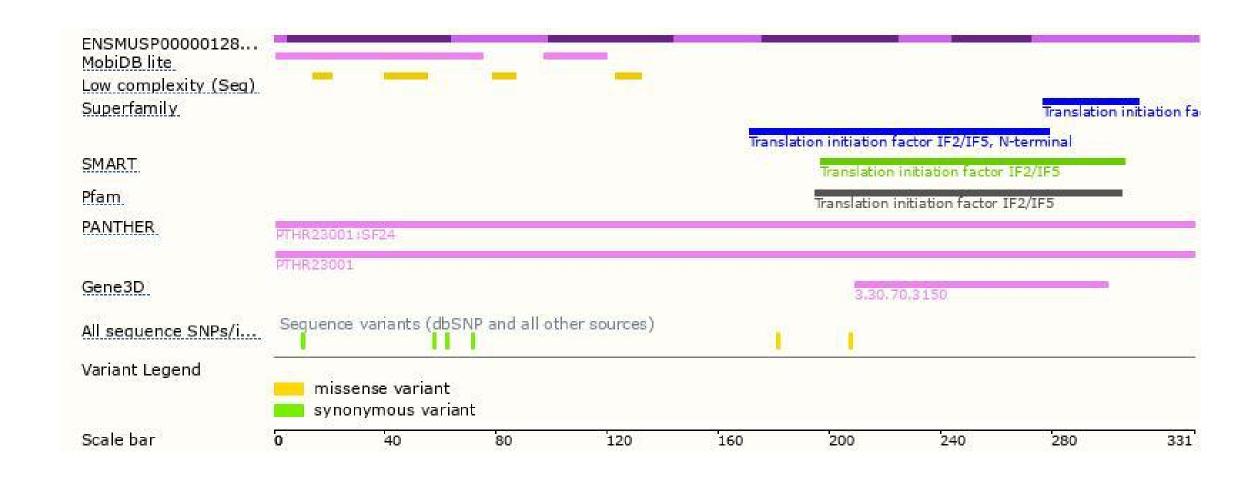
Genomic location distribution





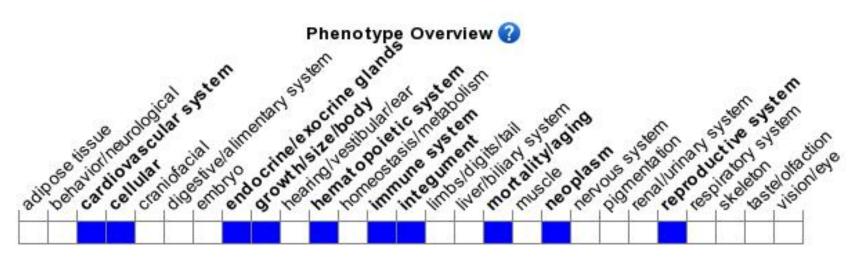
Protein domain





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 trap allele exhibit embryonic lethallity prior to E8.5. Mice heterozygous for a gene trap allele exhibit reduced incidence of testicular germ cell tumors.



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





