

Vezf1 Cas9-CKO Strategy

Designer: JiaYu

Reviewer: Xiaojing Li

Design Date: 2020-7-22

Project Overview



Project Name

Vezf1

Project type

Cas9-CKO

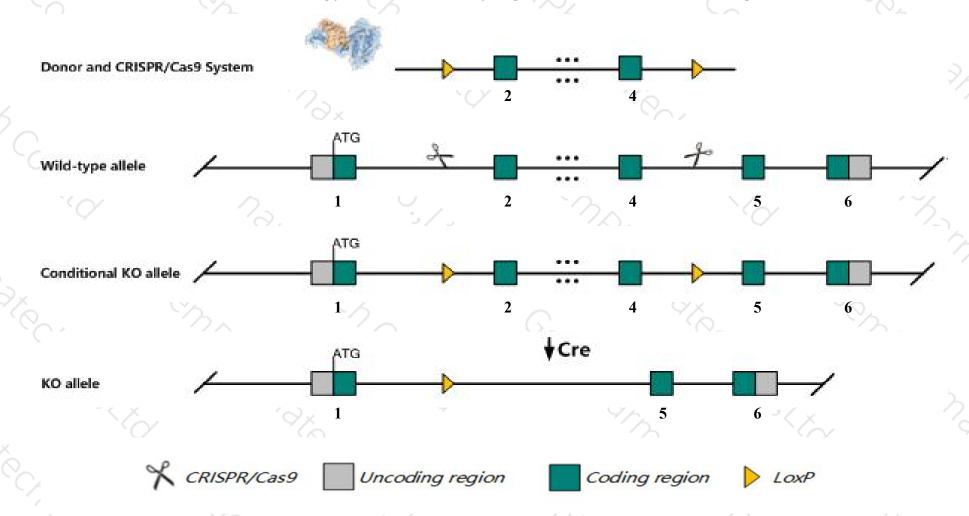
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Vezf1* gene. The schematic diagram is as follows:



Technical routes



- ➤ The *Vezf1* gene has 2 transcripts. According to the structure of *Vezf1* gene, exon2-exon4 of *Vezf1*201(ENSMUST00000018521.10) transcript is recommended as the knockout region. The region contains 943bp coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Vezf1* 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 null mice die at midgestation due to angiogenic remodeling defects and loss of vascular integrity leading to hemorrhaging in the head, heart and trunk. One fifth of heterozygous null embryos show lymphatic hypervascularization associated with hemorrhaging and edema in the jugular region.
- > The *Vezf1* gene is located on the Chr11. 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)



Vezf1 vascular endothelial zinc finger 1 [Mus musculus (house mouse)]

Gene ID: 22344, updated on 13-Mar-2020

Summary

☆ ?

Official Symbol Vezf1 provided by MGI

Official Full Name vascular endothelial zinc finger 1 provided by MGI

Primary source MGI:MGI:1313291

See related Ensembl: ENSMUSG00000018377

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 Al848691, AW046909, db1

Expression Ubiquitous expression in CNS E11.5 (RPKM 16.4), CNS E14 (RPKM 15.8) and 28 other tissuesSee more

Orthologs human all

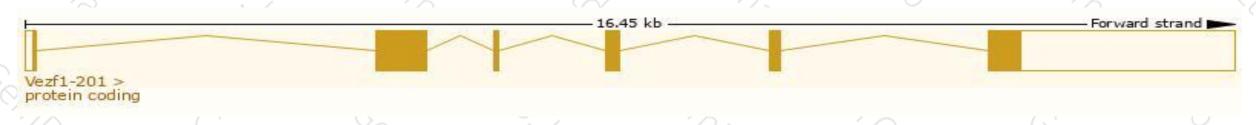
Transcript information (Ensembl)



The gene has 2 transcripts, all transcripts are shown below:

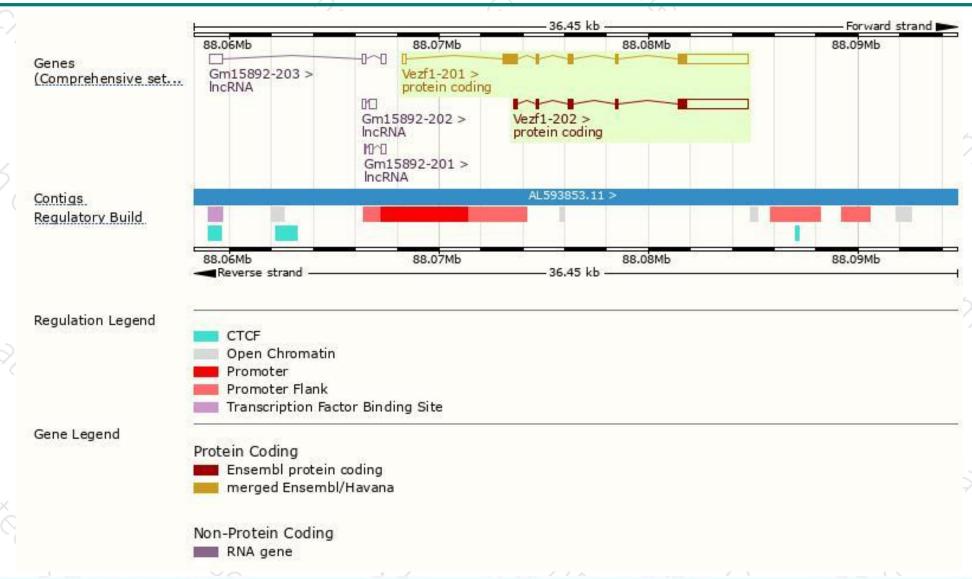
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Vezf1-201	ENSMUST00000018521.10	4583	518aa	Protein coding	CCDS48880	Q5SXC4	TSL:1 GENCODE basic APPRIS is a system to annotate alternatively spliced transcripts based on a range of computational methods to identify the most functionally important transcript(s) of a gene. APPRIS P
Vezf1-202	ENSMUST00000143052.1	3915	336aa	Protein coding	1-0	Q5SXC3	CDS 5' incomplete TSL:5

The strategy is based on the design of *Vezf1-201* transcript, the transcription is shown below:



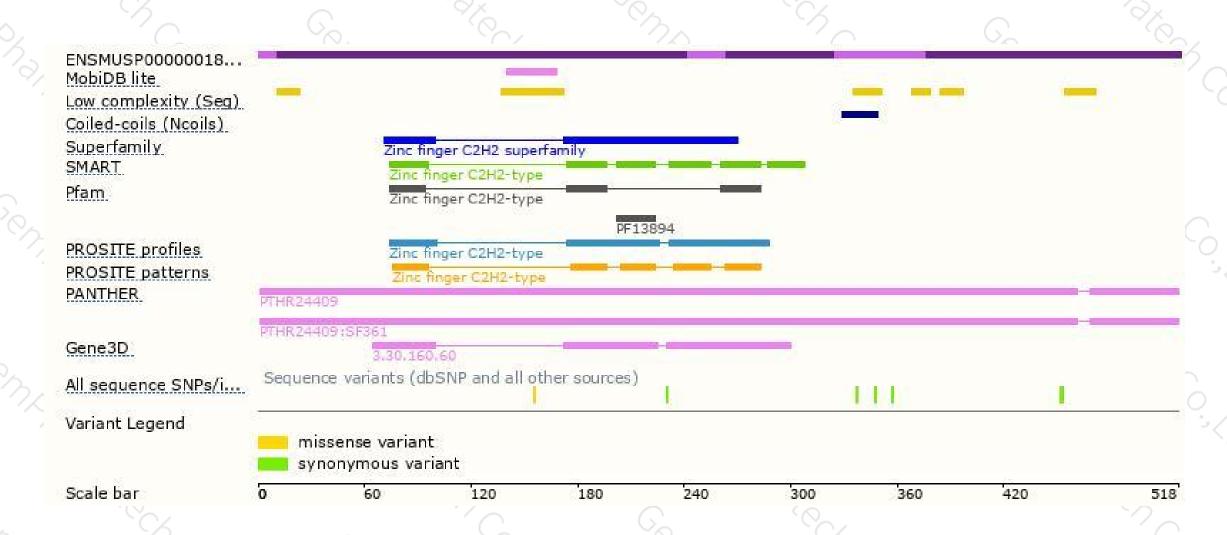
Genomic location distribution





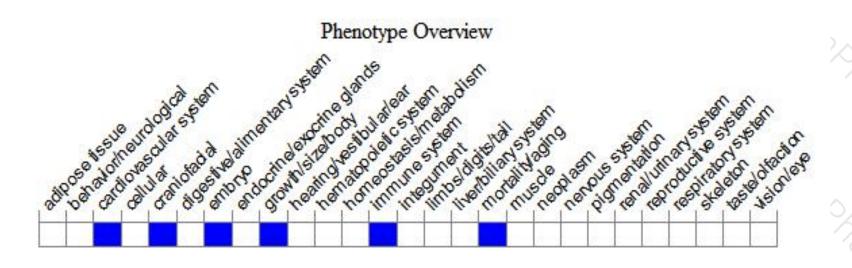
Protein domain





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 null mice die at midgestation due to angiogenic remodeling defects and loss of vascular integrity leading to hemorrhaging in the head, heart and trunk. One fifth of heterozygous null embryos show lymphatic hypervascularization associated with hemorrhaging and edema in the jugular region.



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





