

Tacc3 Cas9-CKO Strategy

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Project Overview

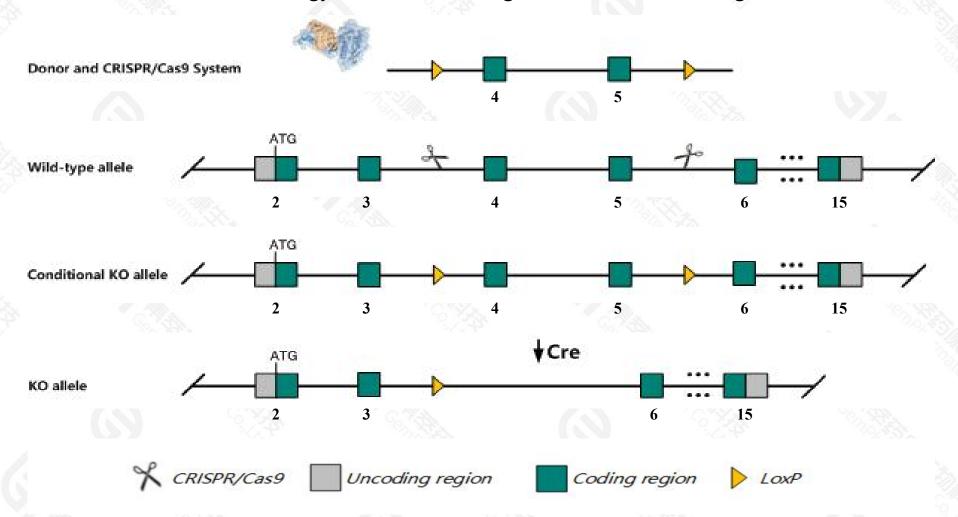


Project Name	Tacc3
Project type	Cas9-CKO
Strain background	C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Tacc3* gene. The schematic diagram is as follows:



Technical routes



- ➤ The *Tacc3* gene has 8 transcripts. According to the structure of *Tacc3* gene, exon4-exon5 of *Tacc3*201(ENSMUST00000074849.13) transcript is recommended as the knockout region. The region contains 671bp coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Tacc3* 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, nullizygous mutations cause embryonic growth delay and prenatal death.

 Homozygotes for a null allele show hematopoietic deficiencies and severe facial clefts. Homozygotes for a hypomorphic allele die neonatally with malformed axial skeletons due to failed mitosis in mesenchymal sclerotome cells.
- > The *Tacc3* gene is located on the Chr5. 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)



Tacc3 transforming, acidic coiled-coil containing protein 3 [Mus musculus (house mouse)]

Gene ID: 21335, updated on 2-Mar-2021

Summary



Official Symbol Tacc3 provided by MGI

Official Full Name transforming, acidic coiled-coil containing protein 3 provided by MGI

Primary source MGI:MGI:1341163

See related Ensembl:ENSMUSG00000037313

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 A, Aint, C86661, Eric1

Expression Broad expression in CNS E11.5 (RPKM 34.6), liver E14 (RPKM 25.7) and 18 other tissuesSee more

Orthologs <u>human all</u>

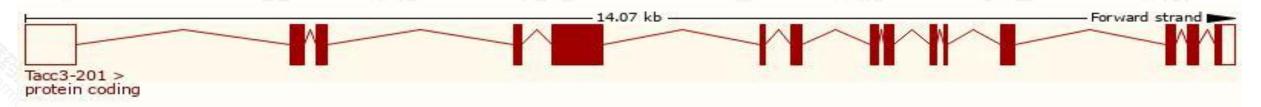
Transcript information (Ensembl)



The gene has 8 transcripts, all transcripts are shown below:

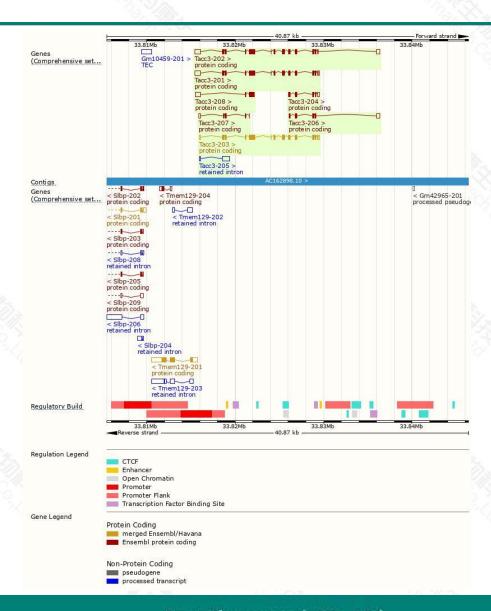
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Tacc3-202	ENSMUST00000079534.11	2899	630aa	Protein coding	CCDS39065		TSL:1 , GENCODE basic , APPRIS P3 ,
Tacc3-201	ENSMUST00000074849.13	2669	<u>637aa</u>	Protein coding	CCDS80258		TSL:1 , GENCODE basic , APPRIS ALT2 ,
Tacc3-203	ENSMUST00000114426.10	2187	<u>630aa</u>	Protein coding	CCDS39065		TSL:1 , GENCODE basic , APPRIS P3 ,
Tacc3-208	ENSMUST00000201633.2	1242	206aa	Protein coding	-		CDS 3' incomplete , TSL:2 ,
Tacc3-206	ENSMUST00000139888.5	823	<u>158aa</u>	Protein coding	(2)		CDS 5' incomplete , TSL:3 ,
Tacc3-204	ENSMUST00000138240.5	679	<u>136aa</u>	Protein coding	450		CDS 5' incomplete , TSL:3 ,
Tacc3-207	ENSMUST00000152847.8	440	<u>55aa</u>	Protein coding	(F)		CDS 3' incomplete , TSL:3 ,
Tacc3-205	ENSMUST00000139453.2	888	No protein	Retained intron	121		TSL:1,
-	VOV. SCHOOL STATE			2001	TV.	7-7-1	

The strategy is based on the design of *Tacc3-201* transcript, the transcription is shown below:



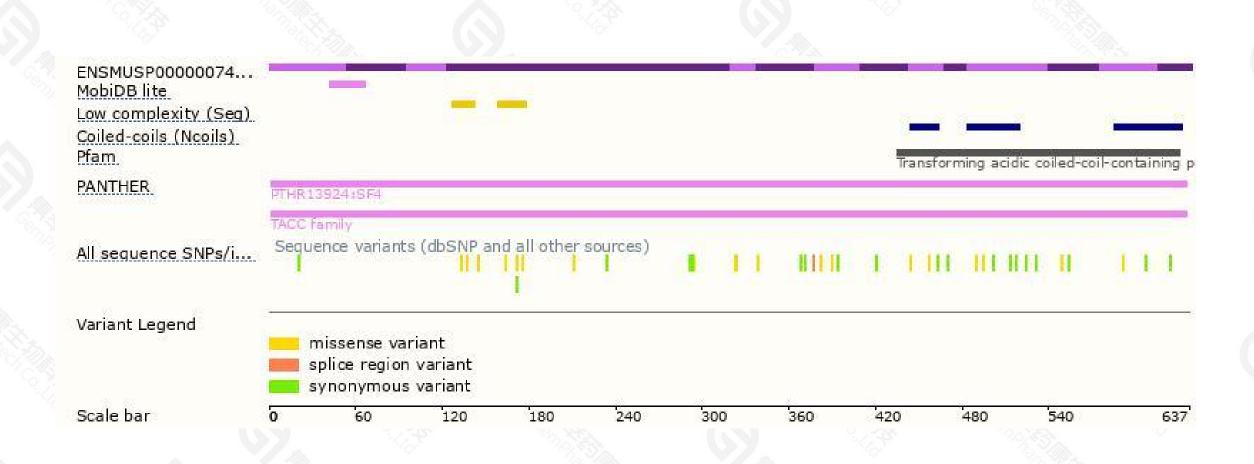
Genomic location distribution





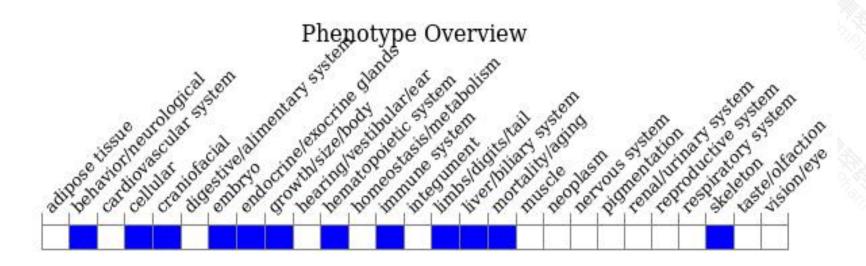
Protein domain





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,nullizygous mutations cause embryonic growth delay and prenatal death. Homozygotes for a null allele show hematopoietic deficiencies and severe facial clefts. Homozygotes for a hypomorphic allele die neonatally with malformed axial skeletons due to failed mitosis in mesenchymal sclerotome cells.



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

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