

Ccn1 Cas9-CKO Strategy

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Design Date: 2019-12-11

Project Overview



Project Name

Ccn1

Project type

Cas9-CKO

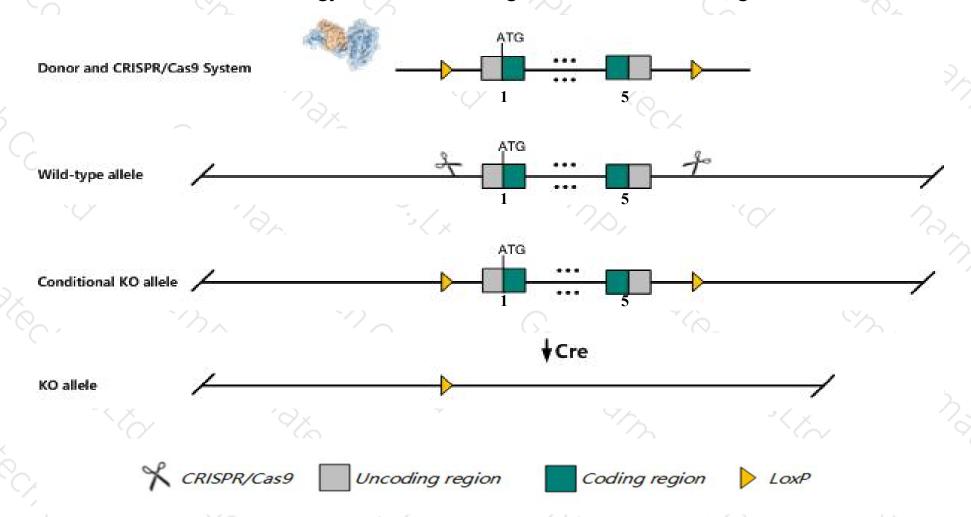
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Ccn1* gene. The schematic diagram is as follows:



Technical routes



- The *Ccn1* gene has 1 transcript. According to the structure of *Ccn1* gene, exon1-exon5 of *Ccn1-201* (ENSMUST00000029846.4) transcript is recommended as the knockout region. The region contains all of the coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Ccn1* 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, Targeted null mice die pre- or perinatally, and none survive beyond 24 hrs of birth. About 30% of embryos die by E10.5 from defects in chorioallantoic fusion, whereas 70% die from placental vascular defects, including impaired allantoic vessel bifurcation, and loss of large-vessel integrity.
- The KO region contains functional region of the Gm17501 gene. Knockout the region may affect the function of Gm17501 gene.
- The *Ccn1* gene is located on the Chr3. 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)



Ccn1 cellular communication network factor 1 [Mus musculus (house mouse)]

Gene ID: 16007, updated on 31-Jan-2019

Summary

☆ ?

Official Symbol Ccn1 provided by MGI

Official Full Name cellular communication network factor 1 provided by MGI

Primary source MGI:MGI:88613

See related Ensembl: ENSMUSG00000028195

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 Al325051, Cyr61, Igfbp10

Expression Broad expression in lung adult (RPKM 41.5), limb E14.5 (RPKM 37.8) and 26 other tissuesSee more

Orthologs human all

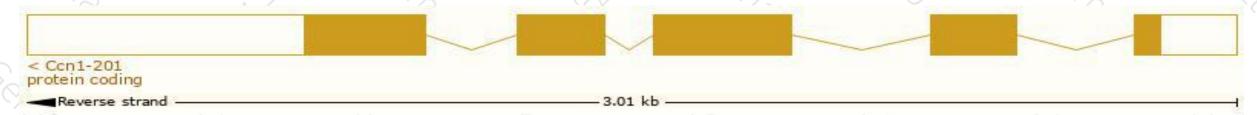
Transcript information (Ensembl)



The gene has 1 transcript, and the transcript is shown below:

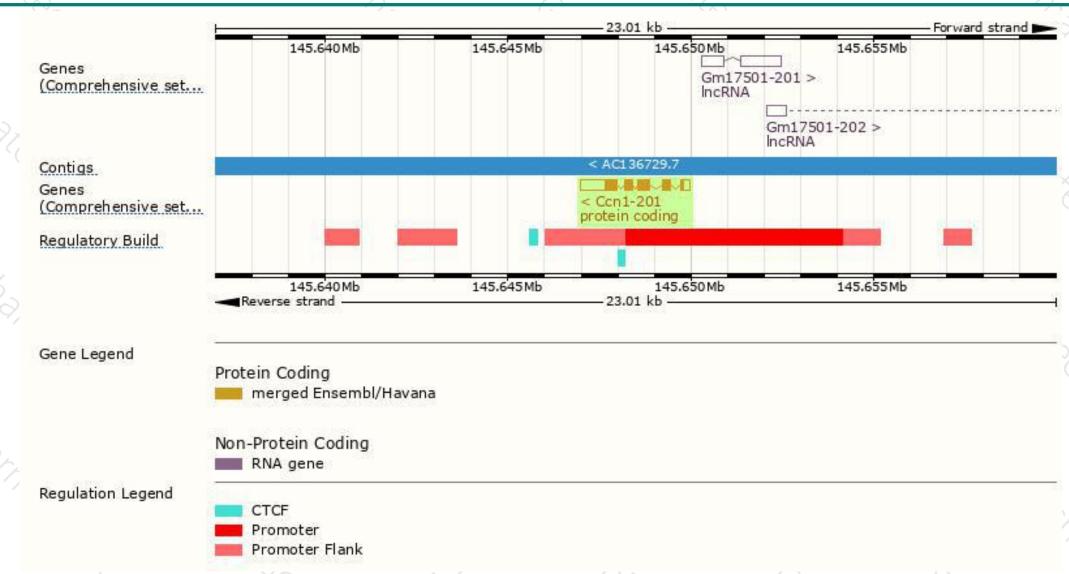
| Name | Transcript ID | bp | Protein | Biotype | CCDS | UniProt | Flags | |
|----------|----------------------|------|---------|----------------|-----------|---------------|-------------------------------|---|
| Ccn1-201 | ENSMUST00000029846.4 | 2019 | 379aa | Protein coding | CCDS17895 | P18406 Q3TX21 | TSL:1 GENCODE basic APPRIS P1 | 3 |

The strategy is based on the design of *Ccn1-201* transcript, The transcription is shown below



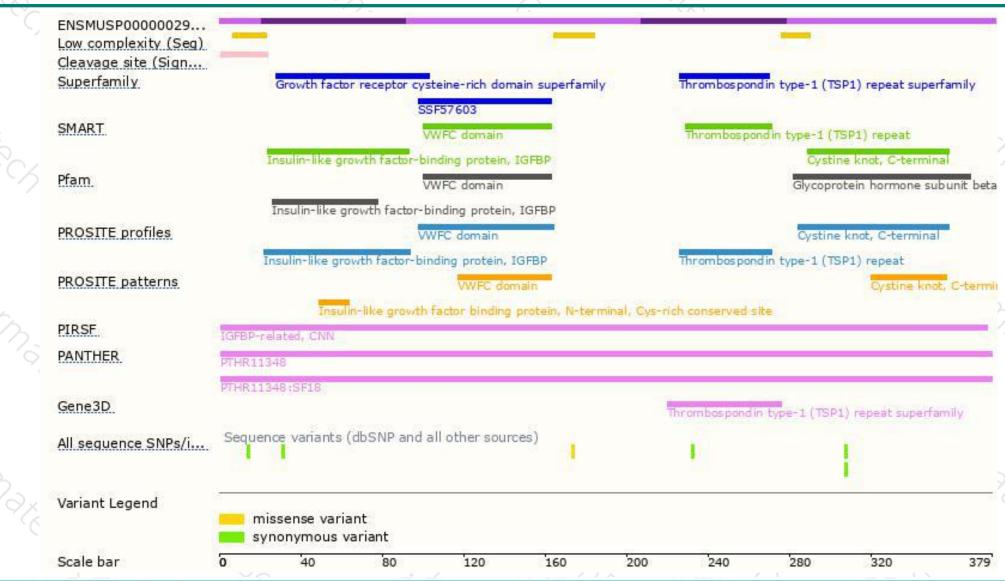
Genomic location distribution





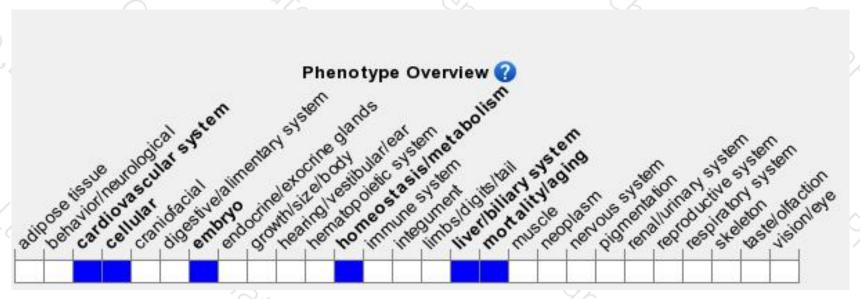
Protein domain





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, Targeted null mice die pre- or perinatally, and none survive beyond 24 hrs of birth. About 30% of embryos die by E10.5 from defects in chorioallantoic fusion, whereas 70% die from placental vascular defects, including impaired allantoic vessel bifurcation, and loss of large-vessel integrity.



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





