

Setd7 Cas9-CKO Strategy

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Design Date: 2019-8-7

Project Overview



Project Name

Setd7

Project type

Cas9-CKO

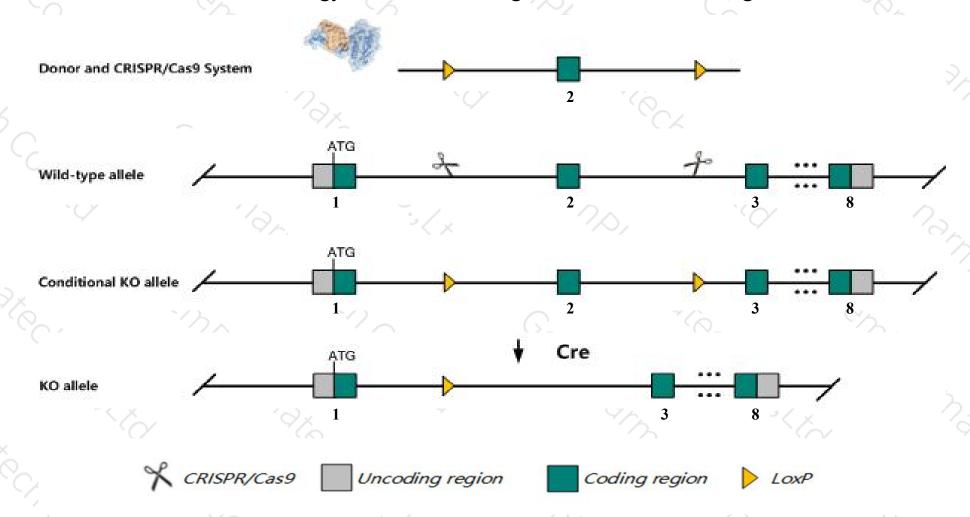
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Setd7 gene. The schematic diagram is as follows:



Technical routes



- ➤ The Setd7 gene has 4 transcripts. According to the structure of Setd7 gene, exon2 of Setd7-201

 (ENSMUST00000037141.8) transcript is recommended as the knockout region. The region contains 130bp coding sequence.

 Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Setd7* 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, Homozygotes for a knock-out allele exhibit partial prenatal lethality and failure of mouse embryonic fibroblasts and spleen cells to arrest after doxorubicin treatment.

 Homozygotes for a different knock-out allele show resistance to bleomycin- or adenovirus-TGFbeta-induced pulmonary fibrosis.
- > The Setd7 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)



Setd7 SET domain containing (lysine methyltransferase) 7 [Mus musculus (house mouse)]

Gene ID: 73251, updated on 19-Feb-2019

Summary

☆ ?

Official Symbol Setd7 provided by MGI

Official Full Name SET domain containing (lysine methyltransferase) 7 provided by MGI

Primary source MGI:MGI:1920501

See related Ensembl:ENSMUSG00000037111

Gene type protein coding
RefSeq status PROVISIONAL
Organism Mus musculus

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha;

Muroidea; Muridae; Murinae; Mus; Mus

Also known as 1600028F23Rik, H3K4MT, KMT7, Set7, Set7/9, mKIAA1717

Expression Ubiquitous expression in cerebellum adult (RPKM 24.0), bladder adult (RPKM 20.7) and 26 other tissuesSee more

Orthologs <u>human</u> all

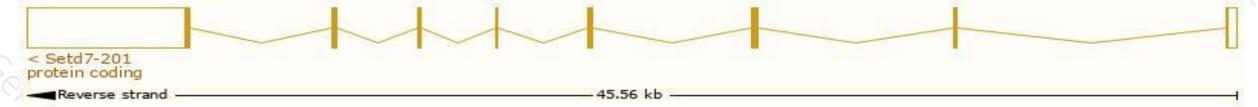
Transcript information (Ensembl)



The gene has 4 transcripts, all transcripts are shown below:

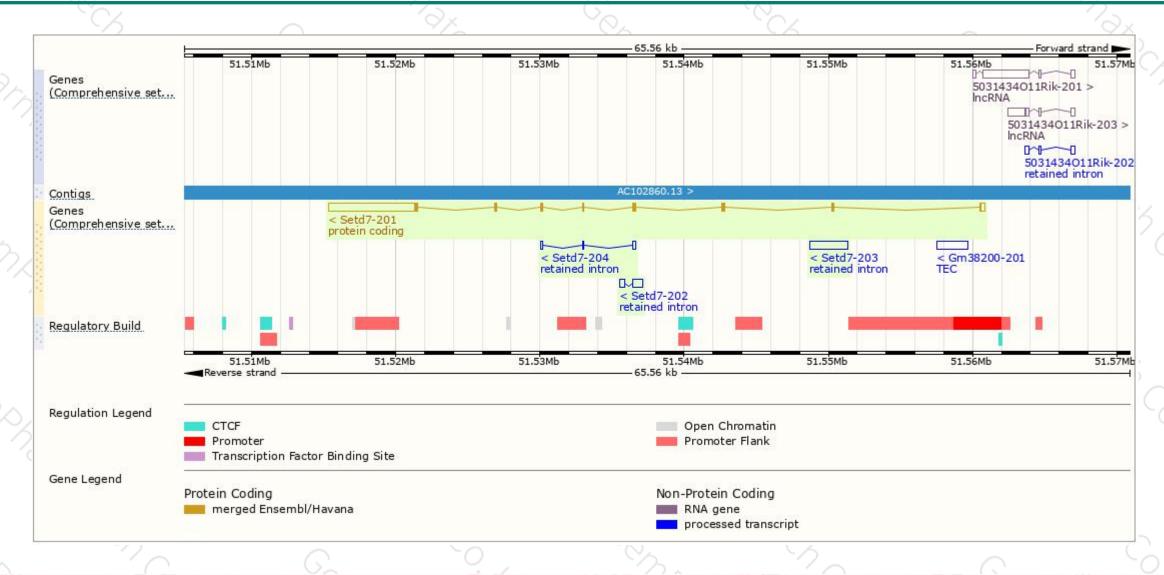
Name 🍦	Transcript ID 👙	bp 👙	Protein 🍦	Biotype 🍦	CCDS 🍦	UniProt 🌲	Flags
Setd7-201	ENSMUST00000037141.8	7411	366aa	Protein coding	CCDS17341 ₽	Q8VHL1₺	TSL:1 GENCODE basic APPRIS P1
Setd7-203	ENSMUST00000194828.1	2638	No protein	Retained intron	-		TSL:NA
Setd7-202	ENSMUST00000161755.2	1043	No protein	Retained intron	-	-	TSL:2
Setd7-204	ENSMUST00000195080.1	423	No protein	Retained intron	1921	3	TSL:3

The strategy is based on the design of Setd7-201 transcript, The transcription is shown below



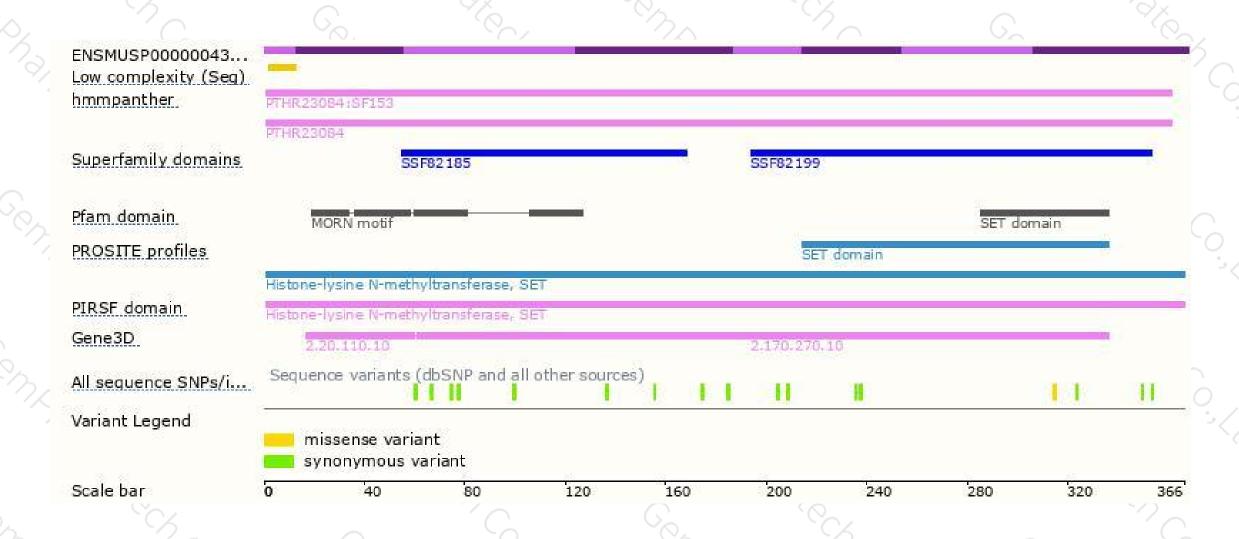
Genomic location distribution





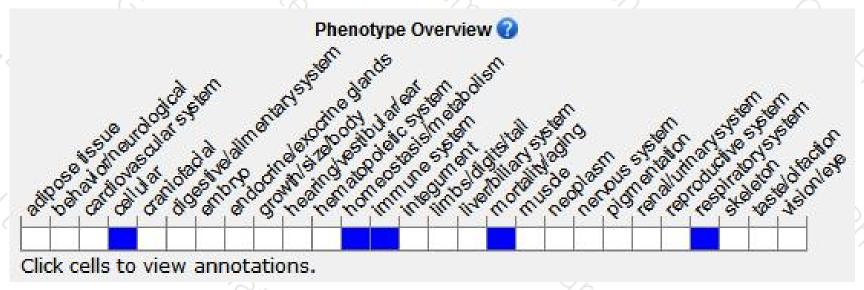
Protein domain





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, Homozygotes for a knock-out allele exhibit partial prenatal lethality and failure of mouse embryonic fibroblasts and spleen cells to arrest after doxorubicin treatment. Homozygotes for a different knock-out allele show resistance to bleomycin- or adenovirus-TGF beta-induced pulmonary fibrosis.



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





