

Atoh7 Cas9-CKO Strategy

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Project Overview



Project Name

Atoh7

Project type

Cas9-CKO

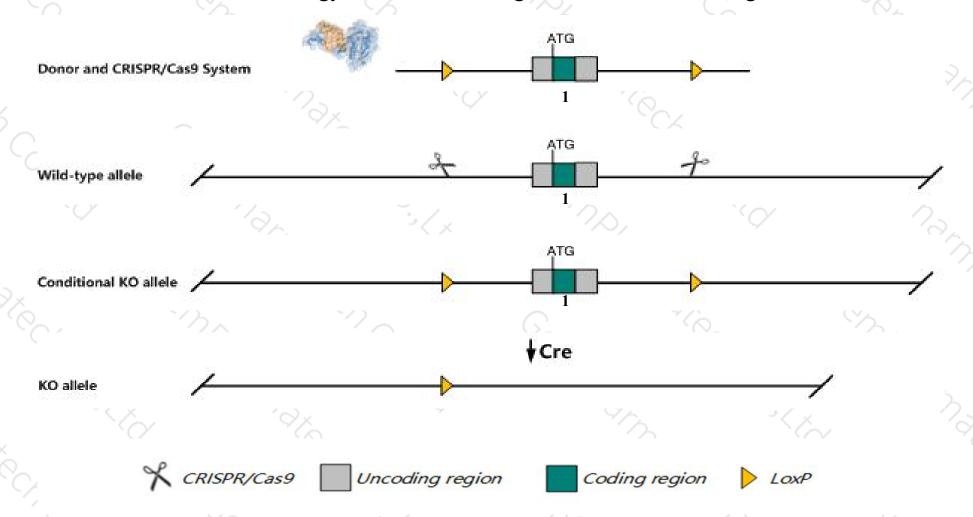
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Atoh7* gene. The schematic diagram is as follows:



Technical routes



- The *Atoh7* gene has 2 transcripts. According to the structure of *Atoh7* gene, exon1 of *Atoh7-201* (ENSMUST00000044059.4) 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 *Atoh7* 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 mutation of this gene results in impaired differentiation of retinal ganglion cells resulting in an increase of amacrine cells. Mice show impaired optic nerve formation and one allele shows loss of circadian photoentrainment.
- > Gm38063 gene will be deleted together in this strategy.
- > The *Atoh7* gene is located on the Chr10. 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)



Atoh7 atonal bHLH transcription factor 7 [Mus musculus (house mouse)]

Gene ID: 53404, updated on 5-Nov-2019

Summary

↑ ?

Official Symbol Atoh7 provided by MGI

Official Full Name atonal bHLH transcription factor 7 provided by MGI

Primary source MGI:MGI:1355553

See related Ensembl: ENSMUSG00000036816

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 Math5; bHLHa13

Expression Biased expression in cerebellum adult (RPKM 1.7), cortex adult (RPKM 0.3) and 4 other tissues See more

Orthologs human all

Genomic context



Location: 10 B4; 10 32.54 cM

See Atoh7 in Genome Data Viewer

Exon count: 1

Annotation release	Status	Assembly	Chr	Location	
108	current	GRCm38.p6 (GCF_000001635.26)	10	NC_000076.6 (6309978563101280)	
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	10	NC_000076.5 (6256290462563353)	

Transcript information (Ensembl)



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

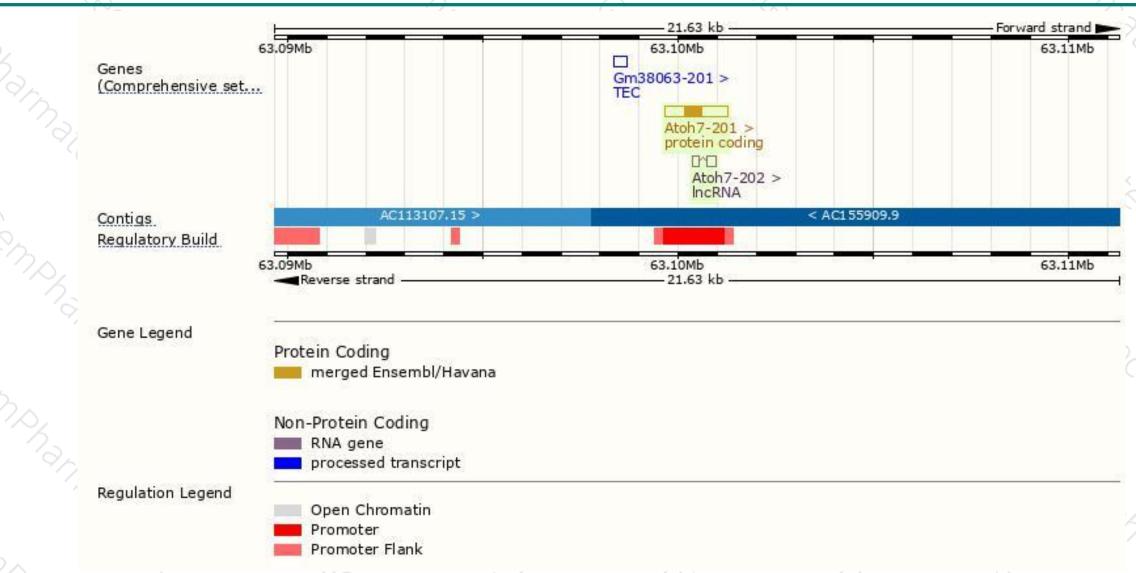
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Atoh7-201	ENSMUST00000044059.4	1629	<u>149aa</u>	Protein coding	CCDS23896	Q9Z2E5	TSL:NA GENCODE basic APPRIS P1
Atoh7-202	ENSMUST00000219964.1	416	No protein	IncRNA	-	÷8	TSL:1

The strategy is based on the design of Atoh7-201 transcript, The transcription is shown below

Atoh7-201 > protein coding

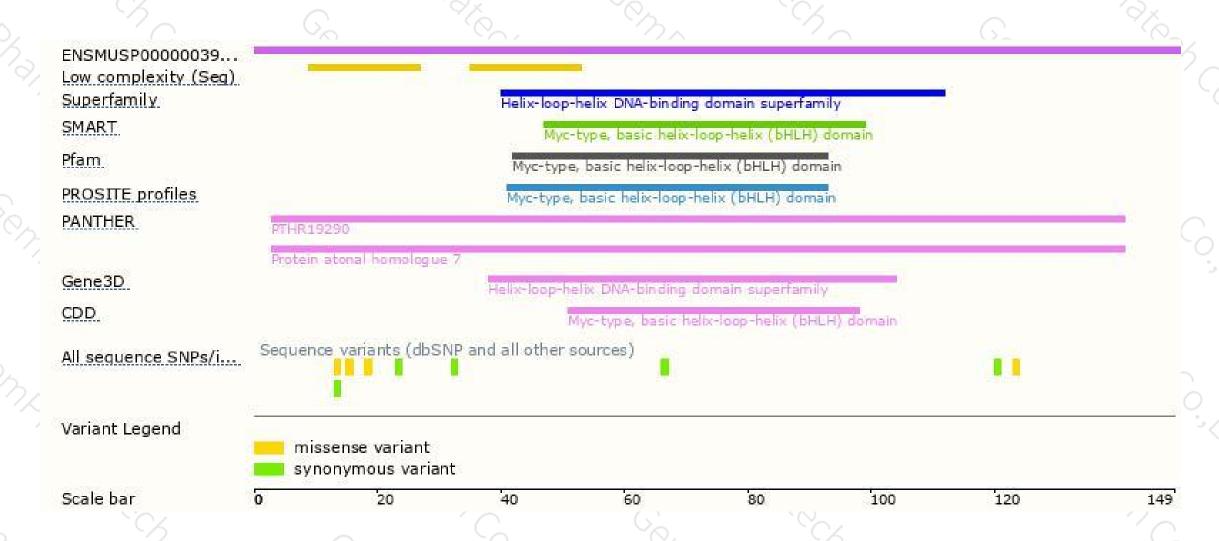
Genomic location distribution





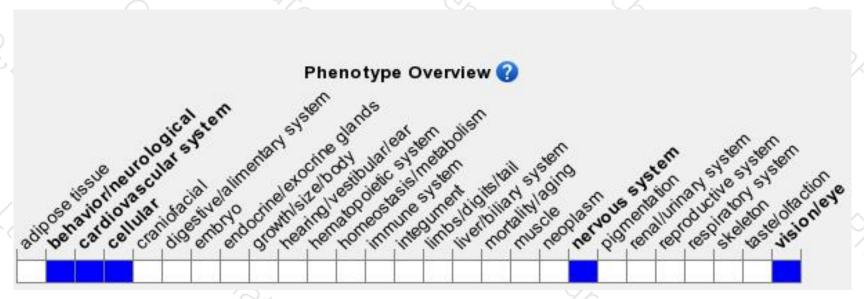
Protein domain





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 mutation of this gene results in impaired differentiation of retinal ganglion cells resulting in an increase of amacrine cells. Mice show impaired optic nerve formation and one allele shows loss of circadian photoentrainment.



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





