

# Dynll1 Cas9-CKO Strategy

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Reviewer: Shilei Zhu

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## **Project Overview**



Project Name Dynll1

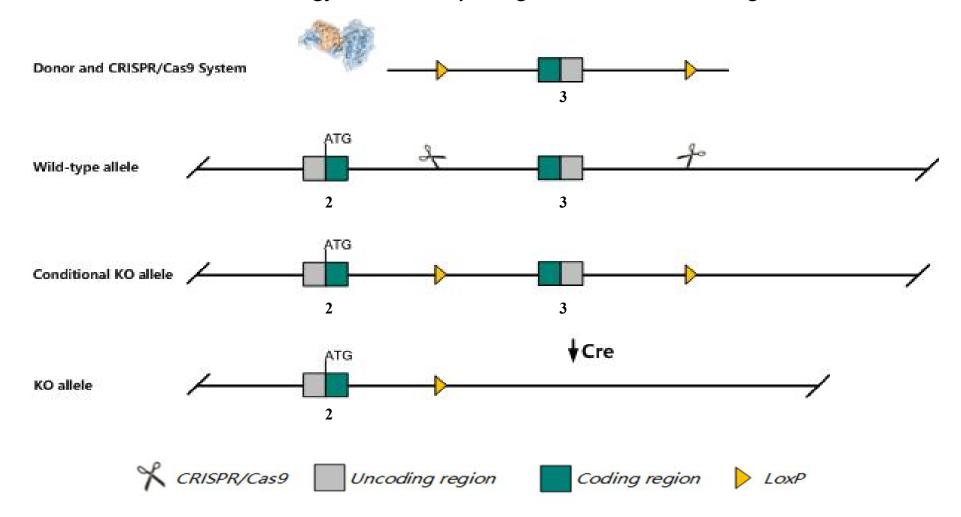
Project type Cas9-CKO

Strain background C57BL/6JGpt

## Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Dynll1* gene. The schematic diagram is as follows:



### **Technical routes**



The *Dynll1* gene has 6 transcripts. According to the structure of *Dynll1* gene, exon3 of *Dynll1*201(ENSMUST00000009157.3) transcript is recommended as the knockout region. The region contains 138bp coding sequence.

Knock out the region will result in disruption of protein function.

In this project we use CRISPR/Cas9 technology to modify *Dynll1* 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,mice carrying a knock-out mutation of this gene exhibit preweaning lethality. Mice homozygous for a gene trap allele exhibit heterotaxia, small or absent lungs and abnormal cilia.

The KO region contains functional region of the Dynll1 gene. Knockout the region may affect the function of Coq5 gene.

The *Dynll1* 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



#### Dynll1 dynein light chain LC8-type 1 [Mus musculus (house mouse)]

Gene ID: 56455, updated on 13-Mar-2020

#### Summary

☆ ?

Official Symbol Dynll1 provided by MGI

Official Full Name dynein light chain LC8-type 1 provided by MGI

Primary source MGI:MGI:1861457

See related Ensembl:ENSMUSG00000009013

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 Dlc8, Dnclc1, Pin

Expression Ubiquitous expression in testis adult (RPKM 89.2), CNS E18 (RPKM 88.6) and 25 other tissuesSee more

Orthologs <u>human all</u>

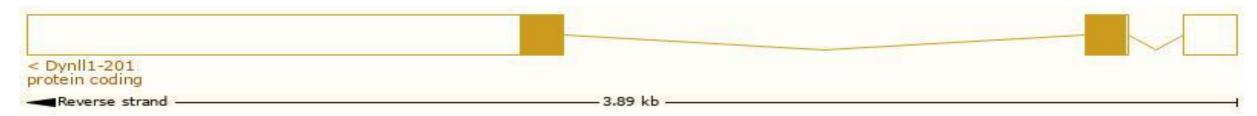
## Transcript information Ensembl



The gene has 6 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Dynll1-201	ENSMUST00000009157.3	2030	<u>89aa</u>	Protein coding	CCDS39227	P63168	TSL:1 GENCODE basic APPRIS P1
Dynll1-202	ENSMUST00000112090.1	729	<u>89aa</u>	Protein coding	CCDS39227	P63168	TSL:2 GENCODE basic APPRIS P1
Dynll1-203	ENSMUST00000123292.1	539	No protein	Processed transcript	16	2.1	TSL:3
Dynll1-205	ENSMUST00000127552.1	528	No protein	Processed transcript	15		TSL:3
Dynll1-206	ENSMUST00000202980.1	2242	No protein	Retained intron	12	62	TSL:NA
Dynll1-204	ENSMUST00000125959.1	478	No protein	Retained intron	59	8.	TSL:2

The strategy is based on the design of *Dynll1-201* transcript, the transcription is shown below:

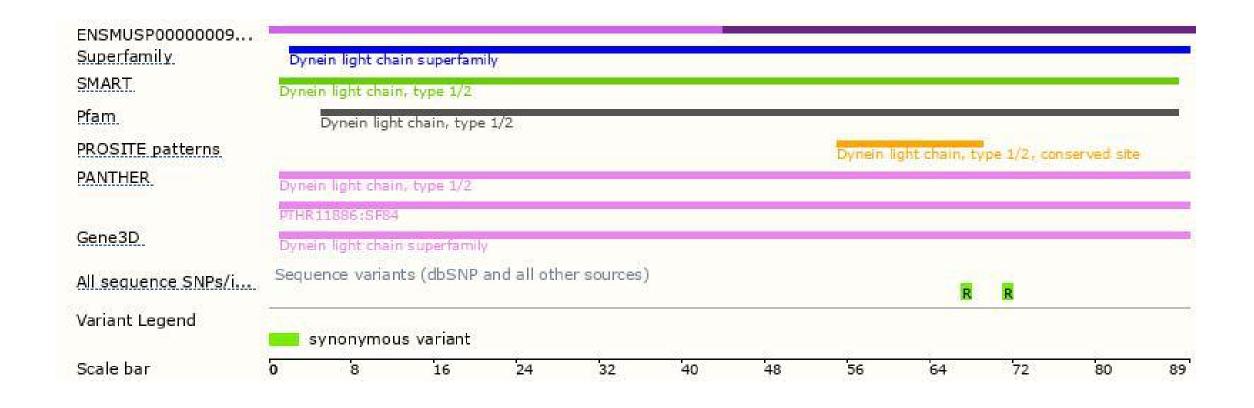


## Genomic location distribution



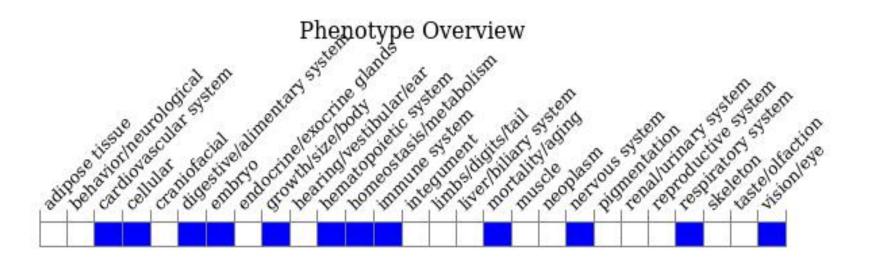
### Protein domain





## 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, mice carrying a knock-out mutation of this gene exhibit preweaning lethality.

Mice homozygous for a gene trap allele exhibit heterotaxia, small or absent lungs and abnormal cilia.



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





