

Myh10 Cas9-CKO Strategy

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Project Overview

Project Name

Myh10

Project type

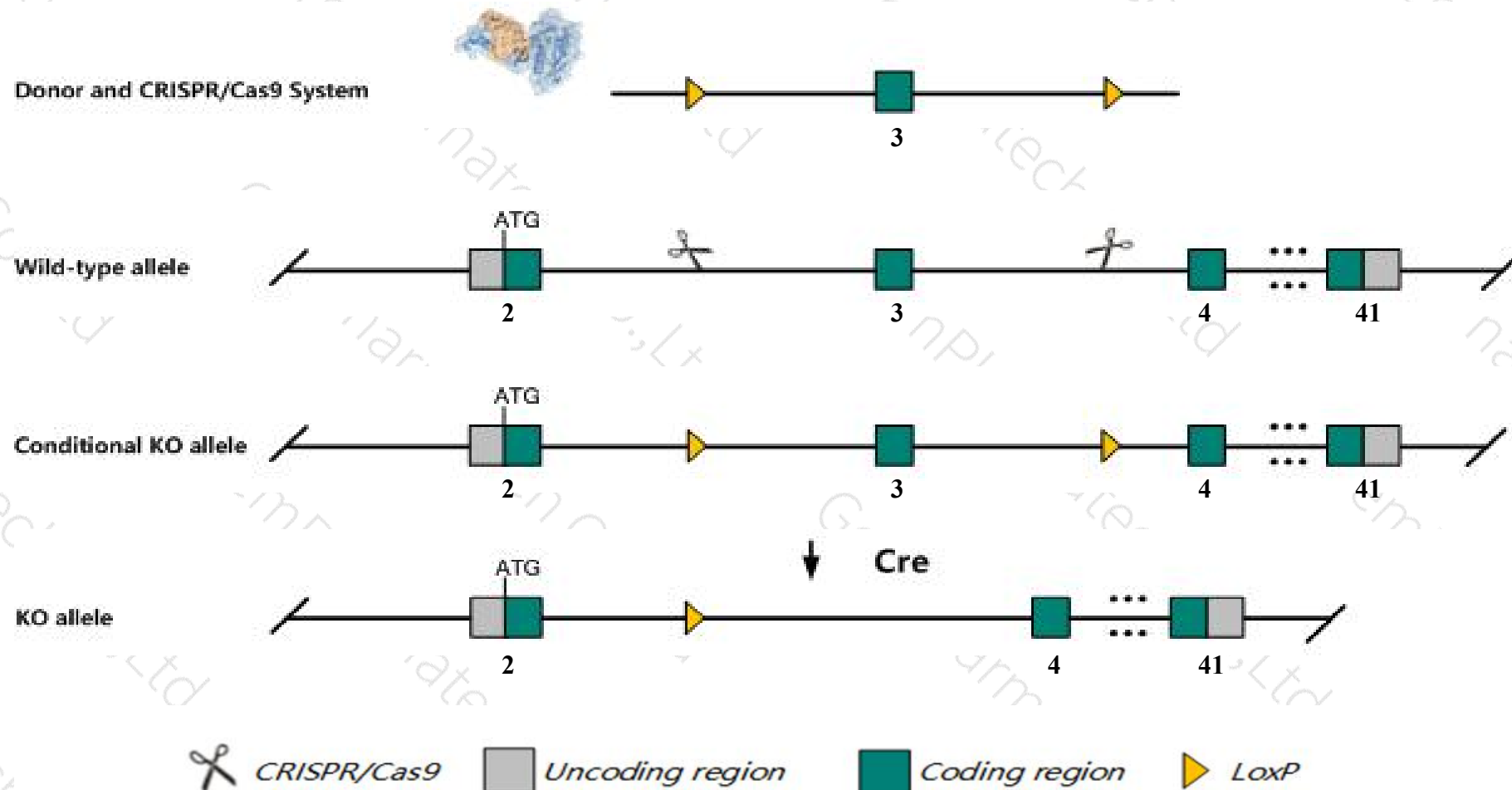
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Myh10* gene. The schematic diagram is as follows:



- The *Myh10* gene has 10 transcripts. According to the structure of *Myh10* gene, exon3 of *Myh10-203* (ENSMUST00000102611.9) transcript is recommended as the knockout region. The region contains 157bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Myh10* 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.

- According to the existing MGI data, Nullizygous mice show pre- and neonatal death, heart defects and hydrocephaly. Deletion of exon B1 disrupts migration of facial neurons, whereas deletion of exon B2 leads to Purkinje cell anomalies. Hypomorphs show hydrocephaly and defects in motor control, cerebellar foliation and neuron migration.
- The *Myh10* gene is located on the Chr11. 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)

Myh10 myosin, heavy polypeptide 10, non-muscle [Mus musculus (house mouse)]

Gene ID: 77579, updated on 7-Apr-2019

Summary



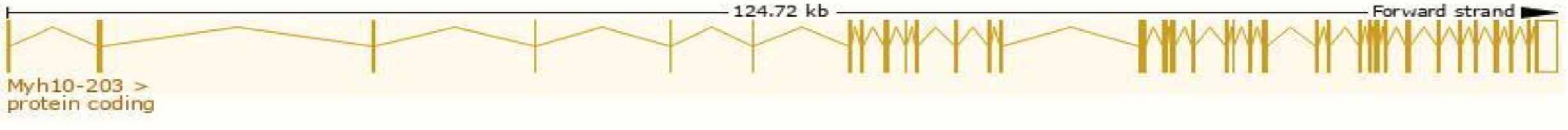
Official Symbol	Myh10 provided by MGI
Official Full Name	myosin, heavy polypeptide 10, non-muscle provided by MGI
Primary source	MGI:MGI:1930780
See related	Ensembl:ENSMUSG00000020900
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	5730504C04Rik, 9330167F11Rik, Fltn, Myhn-2, Myhn2, NMHC II-B, NMHC-B, NMHCII-B, NMMHC II-b, NMMHC-B, NMMHC-IIB, SMemb, mKIAA3005
Expression	Broad expression in CNS E11.5 (RPKM 43.2), CNS E14 (RPKM 26.8) and 19 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

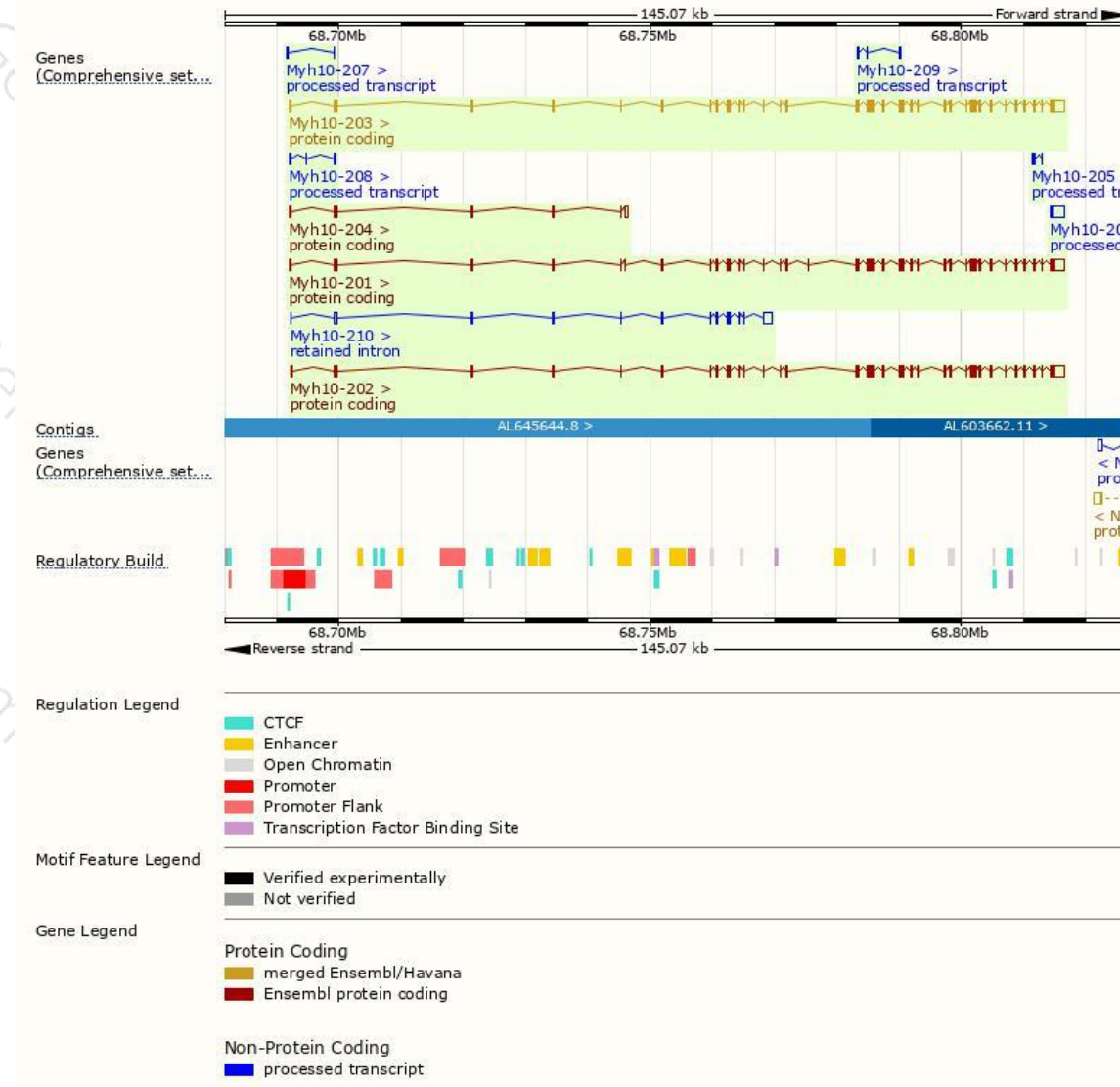
The gene has 10 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Myh10-203	ENSMUST00000102611.9	7791	1976aa	Protein coding	CCDS24869	Q61879	TSL:1 GENCODE basic APPRIS P2
Myh10-202	ENSMUST00000092984.5	7759	2013aa	Protein coding	-	Q3UH59	TSL:1 GENCODE basic
Myh10-201	ENSMUST00000018887.14	7667	2007aa	Protein coding	-	Q5SV64	TSL:5 GENCODE basic APPRIS ALT 1
Myh10-204	ENSMUST00000108673.7	1315	231aa	Protein coding	-	Q8BXF2	TSL:1 GENCODE basic
Myh10-206	ENSMUST00000139059.1	1811	No protein	Processed transcript	-	-	TSL:1
Myh10-205	ENSMUST00000124006.1	558	No protein	Processed transcript	-	-	TSL:2
Myh10-208	ENSMUST00000143032.1	376	No protein	Processed transcript	-	-	TSL:2
Myh10-207	ENSMUST00000141558.1	334	No protein	Processed transcript	-	-	TSL:3
Myh10-209	ENSMUST00000145408.1	278	No protein	Processed transcript	-	-	TSL:3
Myh10-210	ENSMUST00000155765.1	2906	No protein	Retained intron	-	-	TSL:5

The strategy is based on the design of *Myh10-203* 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 mice show pre- and neonatal death, heart defects and hydrocephaly.

Deletion of exon B1 disrupts migration of facial neurons, whereas deletion of exon B2 leads to Purkinje cell anomalies.

Hypomorphs show hydrocephaly and defects in motor control, cerebellar foliation and neuron migration.

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

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