

***Stk36* Cas9-CKO Strategy**

Designer: Huimin Su

Reviewer: Ruiuri Zhang

Design Date: 2020-7-23

Project Overview

Project Name

Stk36

Project type

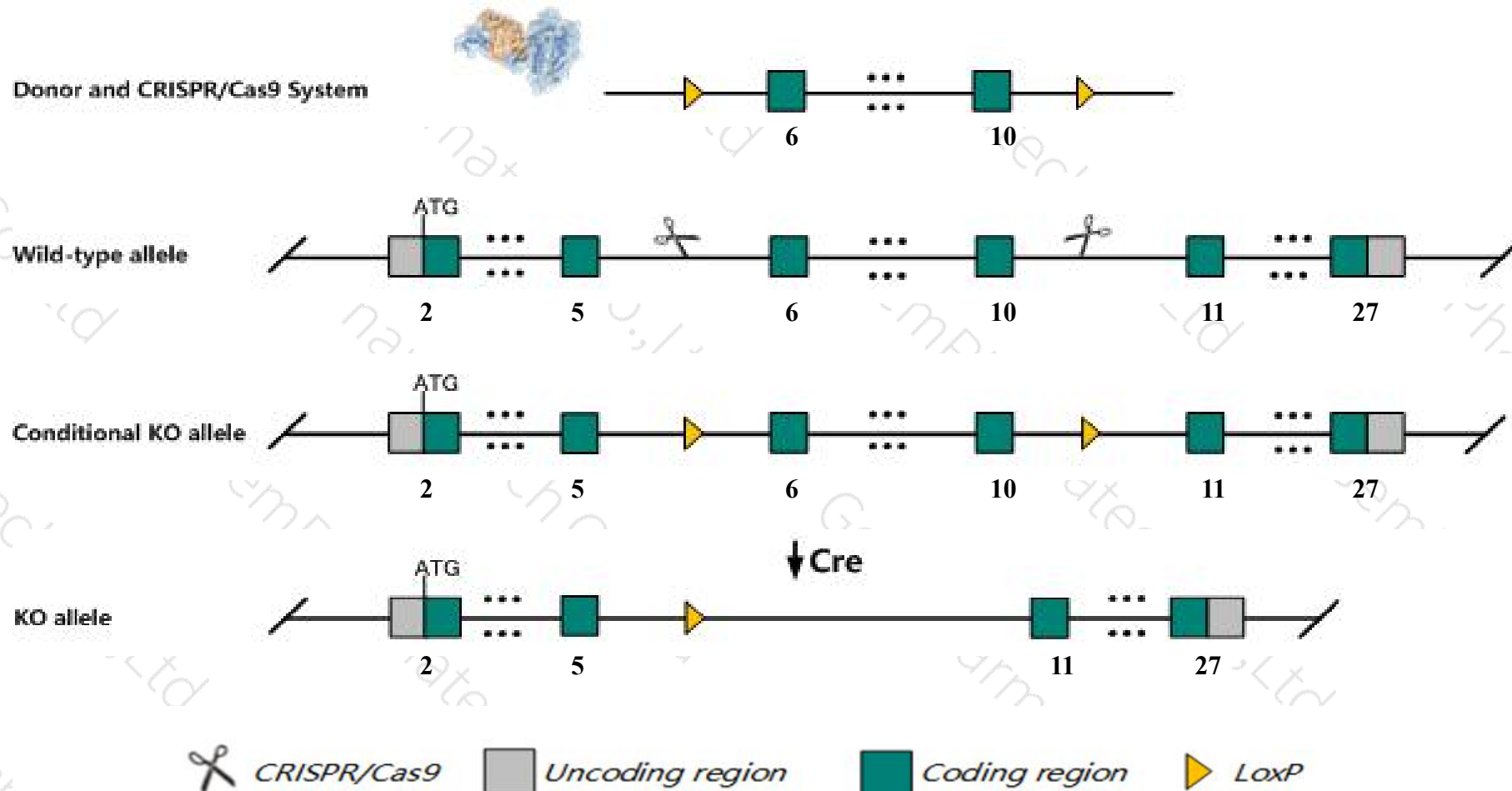
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Stk36* gene. The schematic diagram is as follows:



Technical routes

- The *Stk36* gene has 9 transcripts. According to the structure of *Stk36* gene, exon6-exon10 of *Stk36*-201(ENSMUST00000087183.10) transcript is recommended as the knockout region. The region contains 802bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Stk36* 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 mutations cause postnatal growth defects and lethality. Homozygotes for a null allele show hydrocephaly, cranial defects, otitis media and sterility. Homozygotes for another null allele show additional defects in lung and renal development, thymus and spleen atrophy, rhinitis and ataxia.
- Transcript *Stk36-203* is incomplete, so the effect on it is unknown.
- The *Stk36* gene is located on the Chr1. 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)

Stk36 serine/threonine kinase 36 [*Mus musculus* (house mouse)]

Gene ID: 269209, updated on 26-Jun-2020

Summary

Official Symbol	Stk36 provided by MGI
Official Full Name	serine/threonine kinase 36 provided by MGI
Primary source	MGI:MGI:1920831
See related	Ensembl:ENSMUSG00000033276
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	FU; Fused; mKIAA1278; B930045J24; 1700112N14Rik
Expression	Biased expression in testis adult (RPKM 49.4), whole brain E14.5 (RPKM 6.3) and 6 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

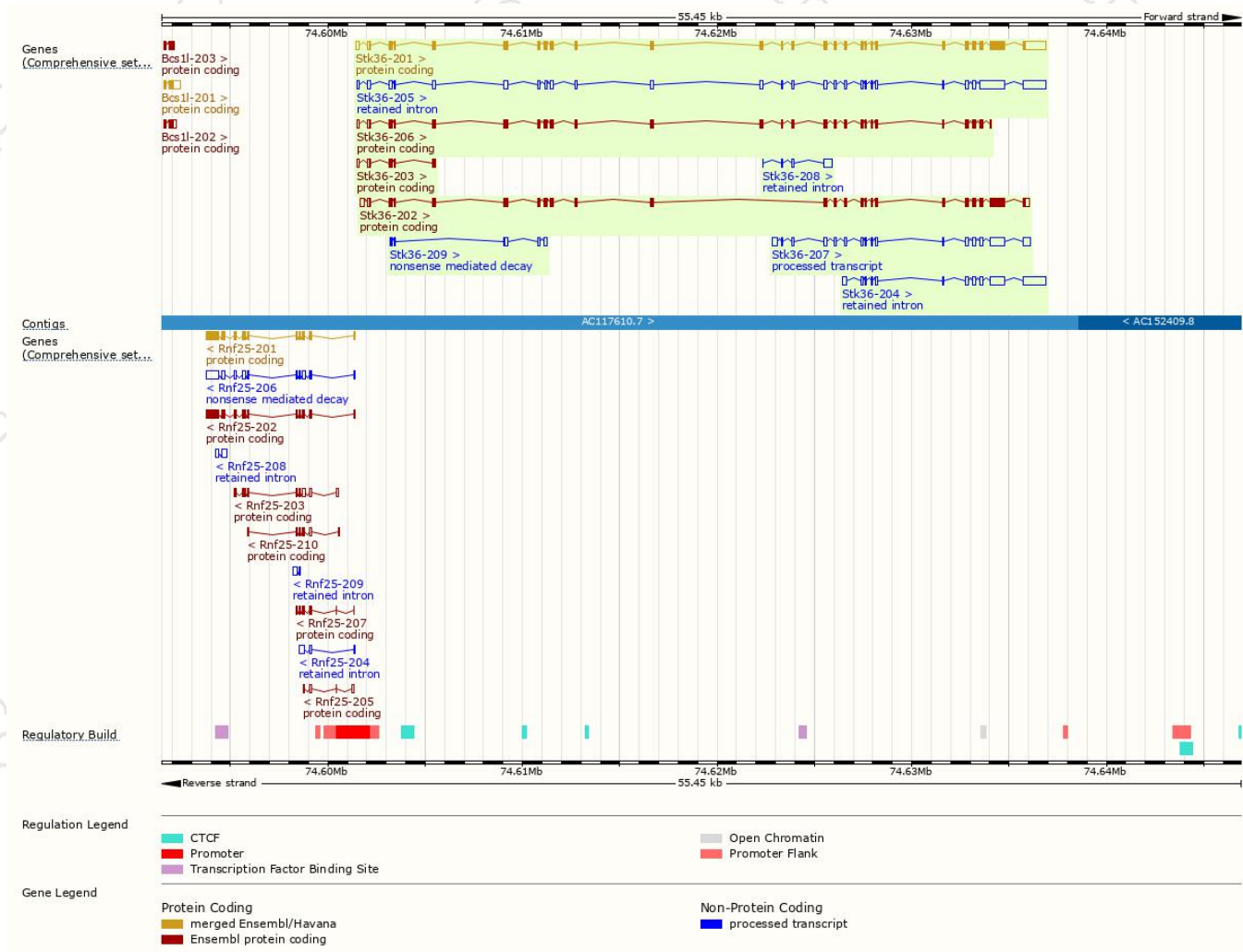
The gene has 9 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Stk36-209	ENSMUST00000189830.1	659	79aa	Nonsense mediated decay	-	A0A087WS61	TSL:3
Stk36-203	ENSMUST00000113694.7	629	145aa	Protein coding	-	Q9D9B2	TSL:1 GENCODE basic
Stk36-206	ENSMUST00000148456.7	3345	1048aa	Protein coding	-	E9Q341	CDS 3' incomplete TSL:1
Stk36-202	ENSMUST00000087186.10	4065	1188aa	Protein coding	-	Q69ZM6	TSL:1 GENCODE basic
Stk36-201	ENSMUST00000087183.10	5245	1316aa	Protein coding	CCDS35619	Q69ZM6	TSL:5 GENCODE basic APPRIS P1
Stk36-207	ENSMUST00000155473.7	2874	No protein	Processed transcript	-	-	TSL:1
Stk36-205	ENSMUST00000145673.7	5489	No protein	Retained intron	-	-	TSL:1
Stk36-204	ENSMUST00000123154.1	3016	No protein	Retained intron	-	-	TSL:1
Stk36-208	ENSMUST00000157007.7	646	No protein	Retained intron	-	-	TSL:5

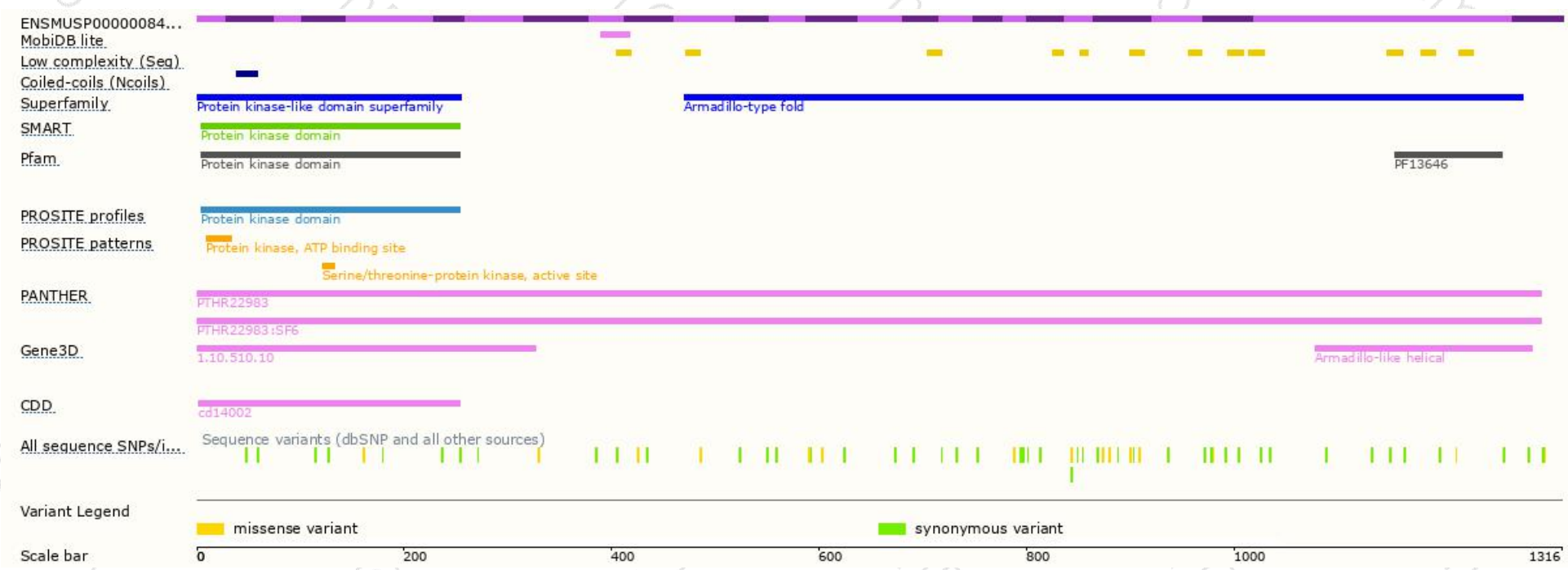
The strategy is based on the design of *Stk36-201* transcript,the transcription is shown below:



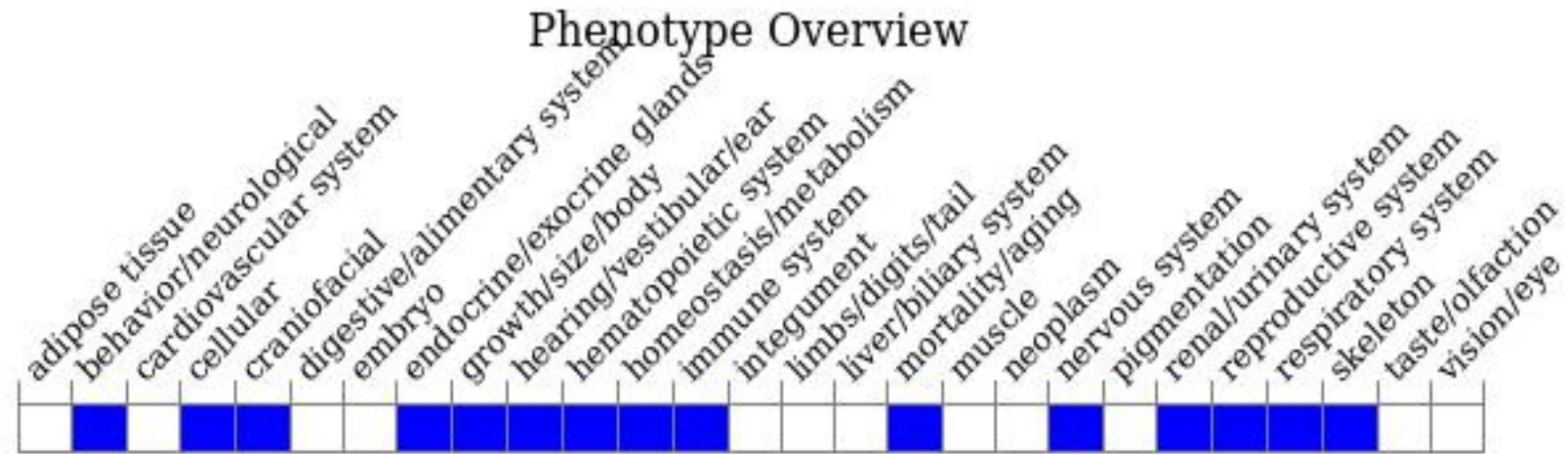
Genomic location distribution



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, nullizygous mutations cause postnatal growth defects and lethality. Homozygotes for a null allele show hydrocephaly, cranial defects, otitis media and sterility. Homozygotes for another null allele show additional defects in lung and renal development, thymus and spleen atrophy, rhinitis and ataxia.

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

Tel: 400-9660890

