

***Rps6* Cas9-CKO Strategy**

Designer:

Bingxuan Li

Design Date:

2019-10-17

Project Overview

Project Name

Rps6

Project type

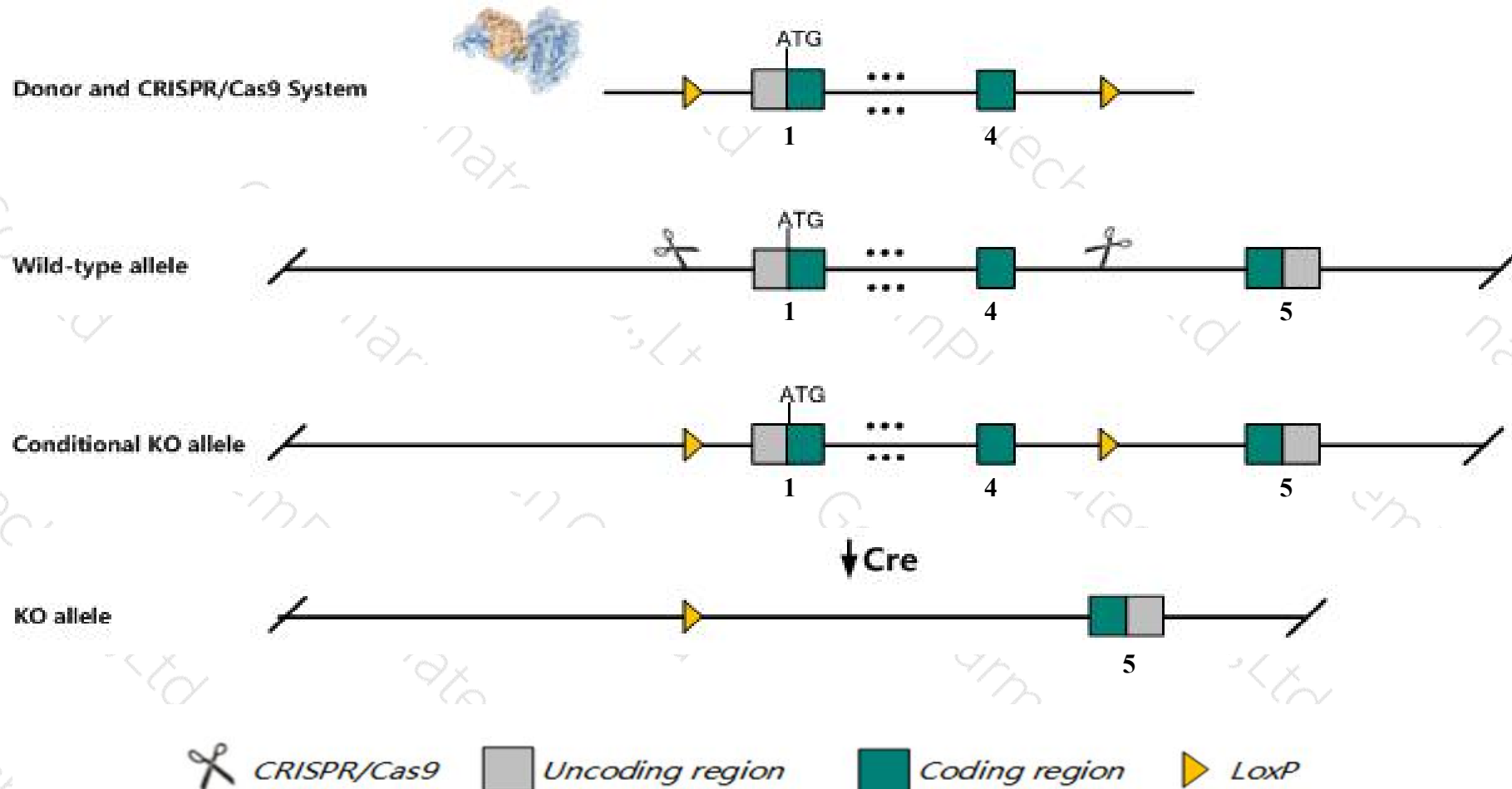
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Rps6* gene. The schematic diagram is as follows:



- The *Rps6* gene has 4 transcripts. According to the structure of *Rps6* gene, exon1-exon4 of *Rps6-201* (ENSMUST00000102814.4) transcript is recommended as the knockout region. The region contains start codon ATG. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Rps6* gene. The brief process is as follows: gRNA was transcribed in vitro, donor was constructed. Cas9, gRNA 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, Mice with an inducible, liver-specific null mutation exhibit failure of liver regeneration and an absence of 40S ribosomes in hepatocytes. Mice with a mutation where serines are unphosphorylatable exhibit hypoinsulinemia, impaired glucose tolerance, and smaller MEFs and beta cells.
- The insertion site of 5-terminal Loxp is in the regulatory region of *Rps6*, which may affect the regulation of *Rps6*.
- The *Rps6* gene is located on the Chr4. 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)

Rps6 ribosomal protein S6 [*Mus musculus* (house mouse)]

Gene ID: 20104, updated on 14-Sep-2019

Summary

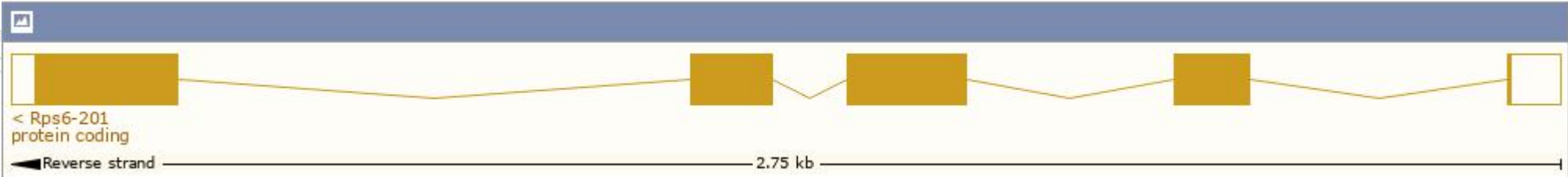
Official Symbol	Rps6 provided by MGI
Official Full Name	ribosomal protein S6 provided by MGI
Primary source	MGI:MGI:98159
See related	Ensembl:ENSMUSG00000028495
Gene type	protein coding
RefSeq status	PROVISIONAL
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	S6R
Expression	Ubiquitous expression in bladder adult (RPKM 484.2), CNS E11.5 (RPKM 432.7) and 25 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

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

Show/hide columns (1 hidden)							Filter	
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags	
Rps6-201	ENSMUST00000102814.4	878	249aa	Protein coding	CCDS18310	P62754 Q5BLK1	TSL:1	GENCODE basic APPRIS P1
Rps6-204	ENSMUST00000136174.7	1128	No protein	lncRNA	-	-	TSL:2	
Rps6-202	ENSMUST00000123229.7	639	No protein	lncRNA	-	-	TSL:2	
Rps6-203	ENSMUST00000130001.1	639	No protein	lncRNA	-	-	TSL:2	

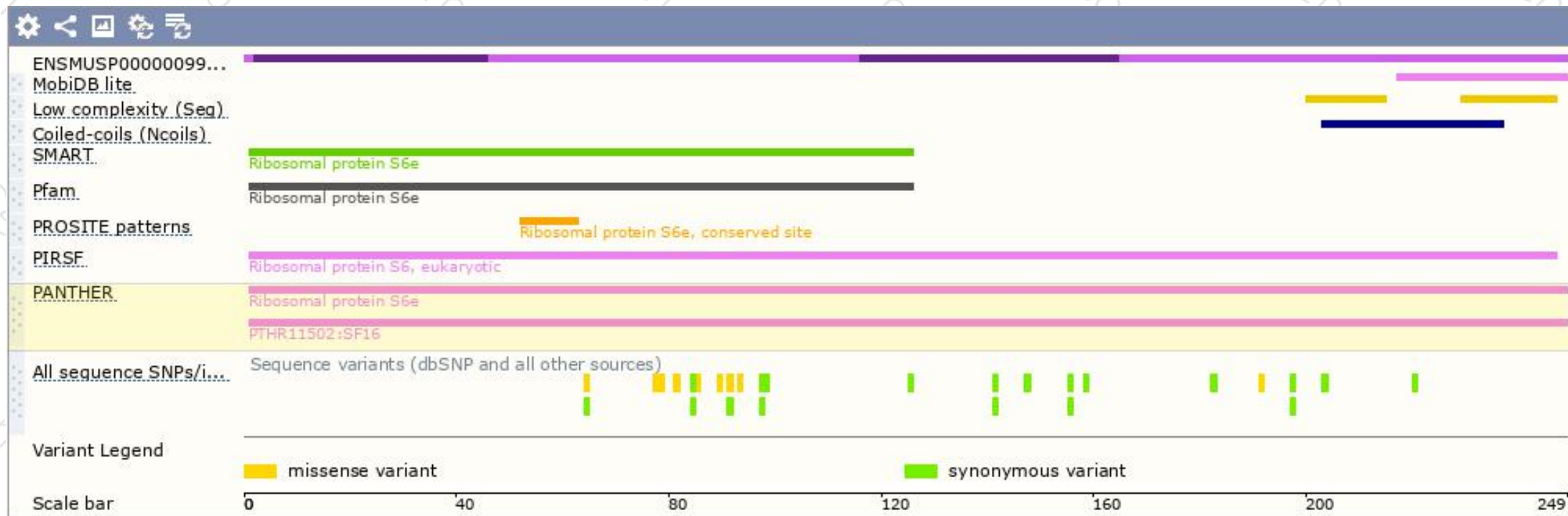
The strategy is based on the design of *Rps6-201* transcript,The transcription is shown below



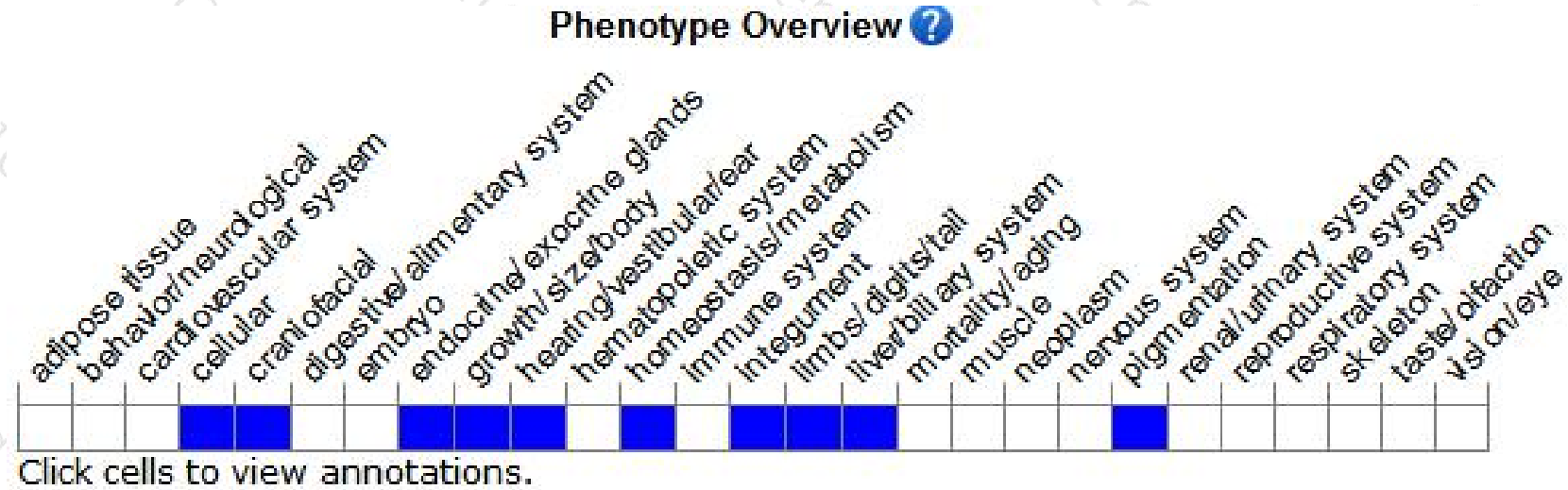
Genomic location distribution



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 with an inducible, liver-specific null mutation exhibit failure of liver regeneration and an absence of 40S ribosomes in hepatocytes. Mice with a mutation where serines are unphosphorylatable exhibit hypoinsulinemia, impaired glucose tolerance, and smaller MEFs and beta cells.

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

Tel: 400-9660890

