

Hsp90b1 Cas9-CKO Strategy

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Project Overview

Project Name

Hsp90b1

Project type

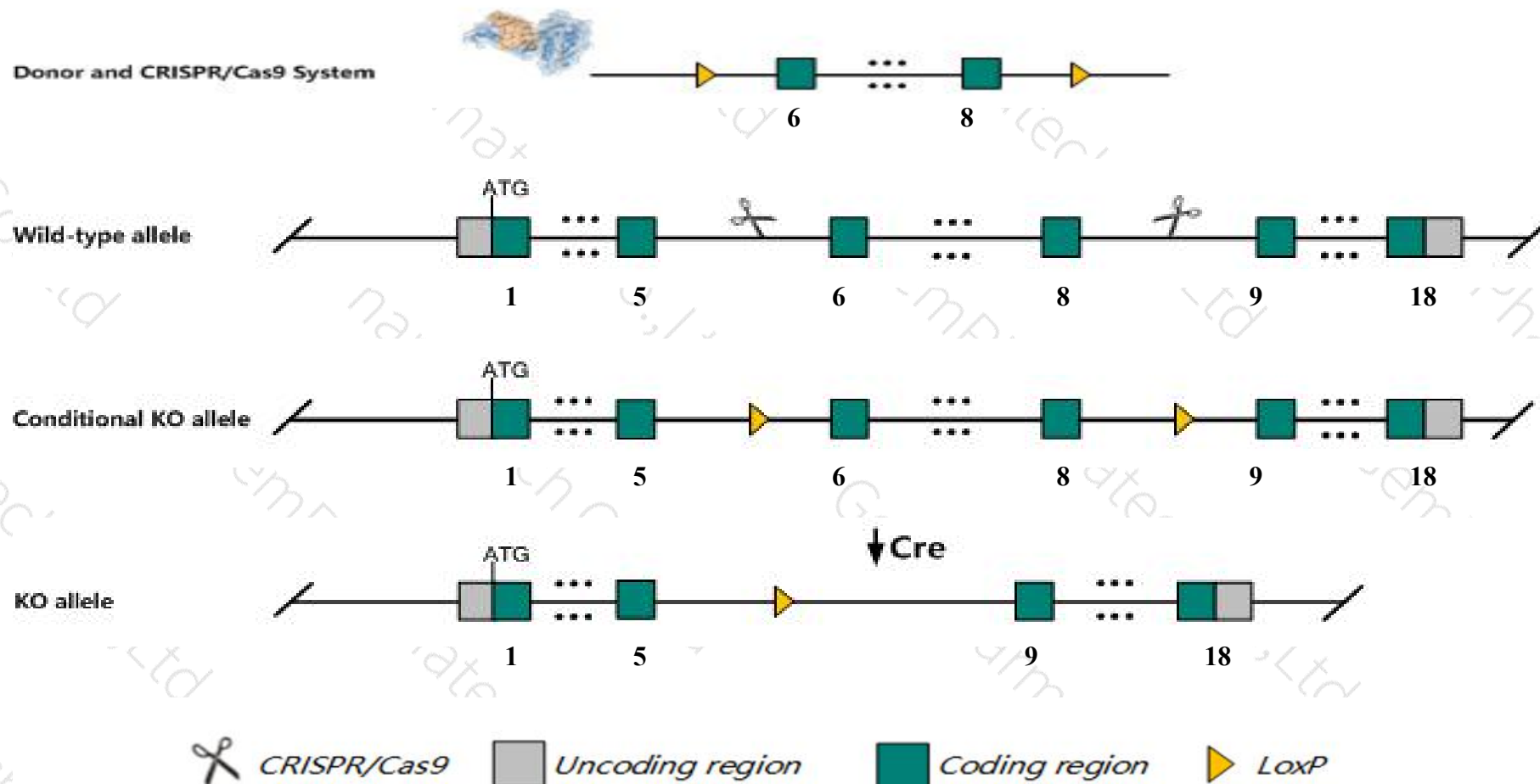
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Hsp90b1* gene. The schematic diagram is as follows:



- The *Hsp90b1* gene has 5 transcripts. According to the structure of *Hsp90b1* gene, exon6-exon8 of *Hsp90b1-201* (ENSMUST00000020238.13) transcript is recommended as the knockout region. The region contains 349bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Hsp90b1* 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, Mice homozygous for a null mutation display embryonic lethality before somite formation with failure of primitive streak formation, absence of the chorion and amnion, and failure of mesoderm formation.
- Because it is too close to the *Ttc41* gene, there are more gene residues during production.
- The *Hsp90b1* 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 gene transcription and translation processes, all risks cannot be predicted under existing information.

Gene information (NCBI)

Hsp90b1 heat shock protein 90, beta (Grp94), member 1 [Mus musculus (house mouse)]

Gene ID: 22027, updated on 19-Mar-2019

Summary



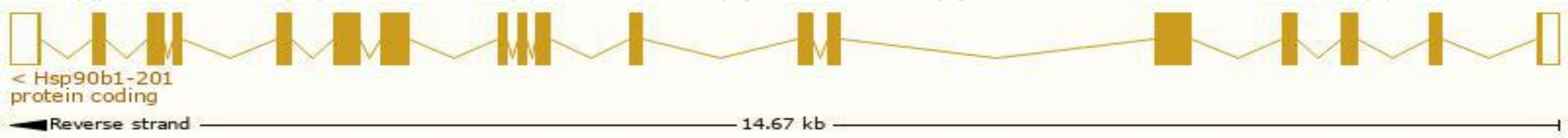
Official Symbol	Hsp90b1 provided by MGI
Official Full Name	heat shock protein 90, beta (Grp94), member 1 provided by MGI
Primary source	MGI:MGI:98817
See related	Ensembl:ENSMUSG00000020048
Gene type	protein coding
RefSeq status	PROVISIONAL
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	ERp99, GRP94, TA-3, Targ2, Tra-1, Tra1, endoplasmin, gp96
Expression	Broad expression in placenta adult (RPKM 303.0), CNS E11.5 (RPKM 168.9) and 20 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

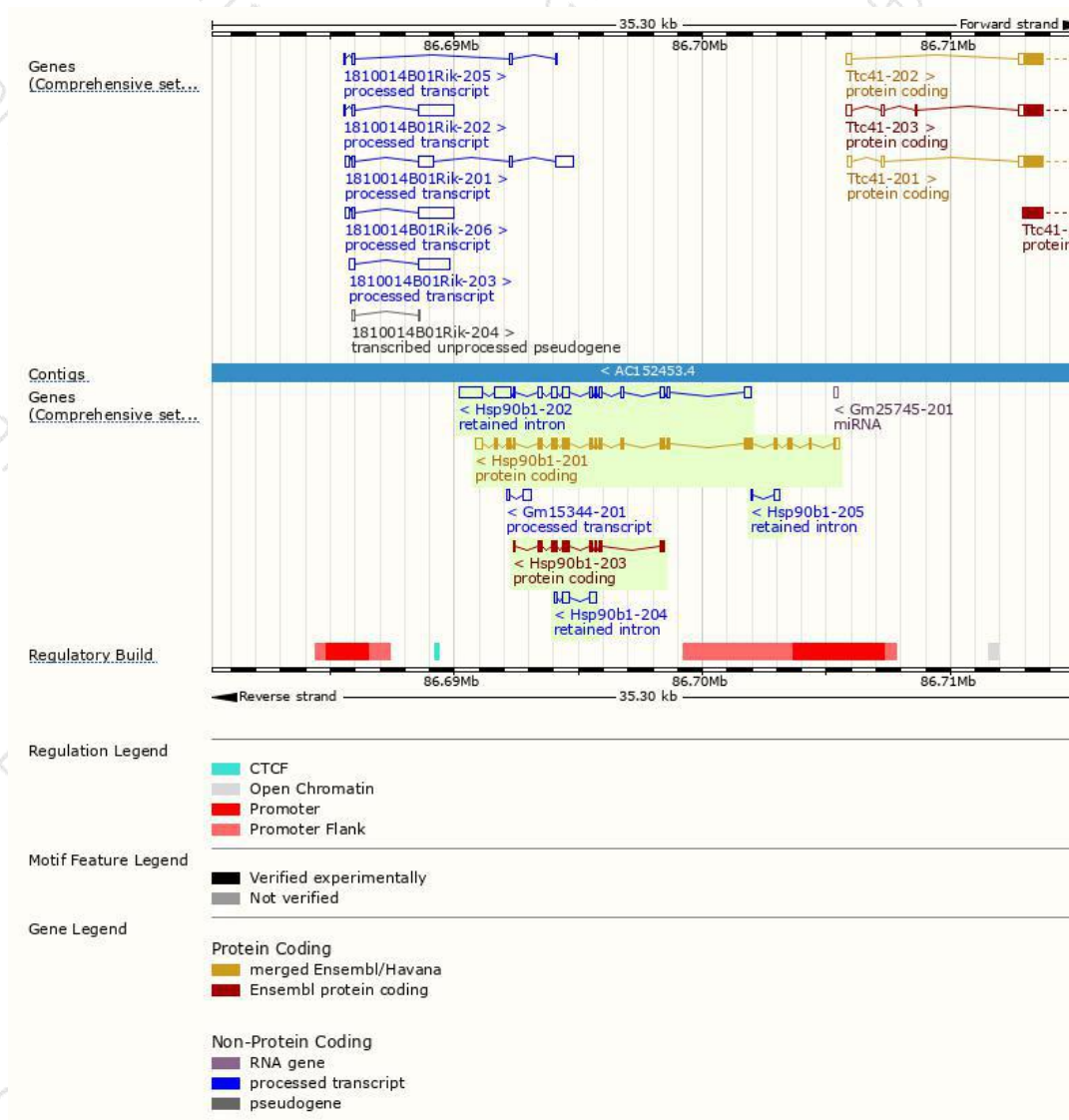
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Hsp90b1-201	ENSMUST00000020238.13	2825	802aa	Protein coding	CCDS36019	P08113 Q3UAD6	TSL:1 GENCODE basic APPRIS P1
Hsp90b1-203	ENSMUST00000129413.1	1119	373aa	Protein coding	-	F7C312	5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL:5
Hsp90b1-202	ENSMUST00000129178.7	3222	No protein	Retained intron	-	-	TSL:1
Hsp90b1-204	ENSMUST00000134515.1	654	No protein	Retained intron	-	-	TSL:1
Hsp90b1-205	ENSMUST00000146897.1	268	No protein	Retained intron	-	-	TSL:2

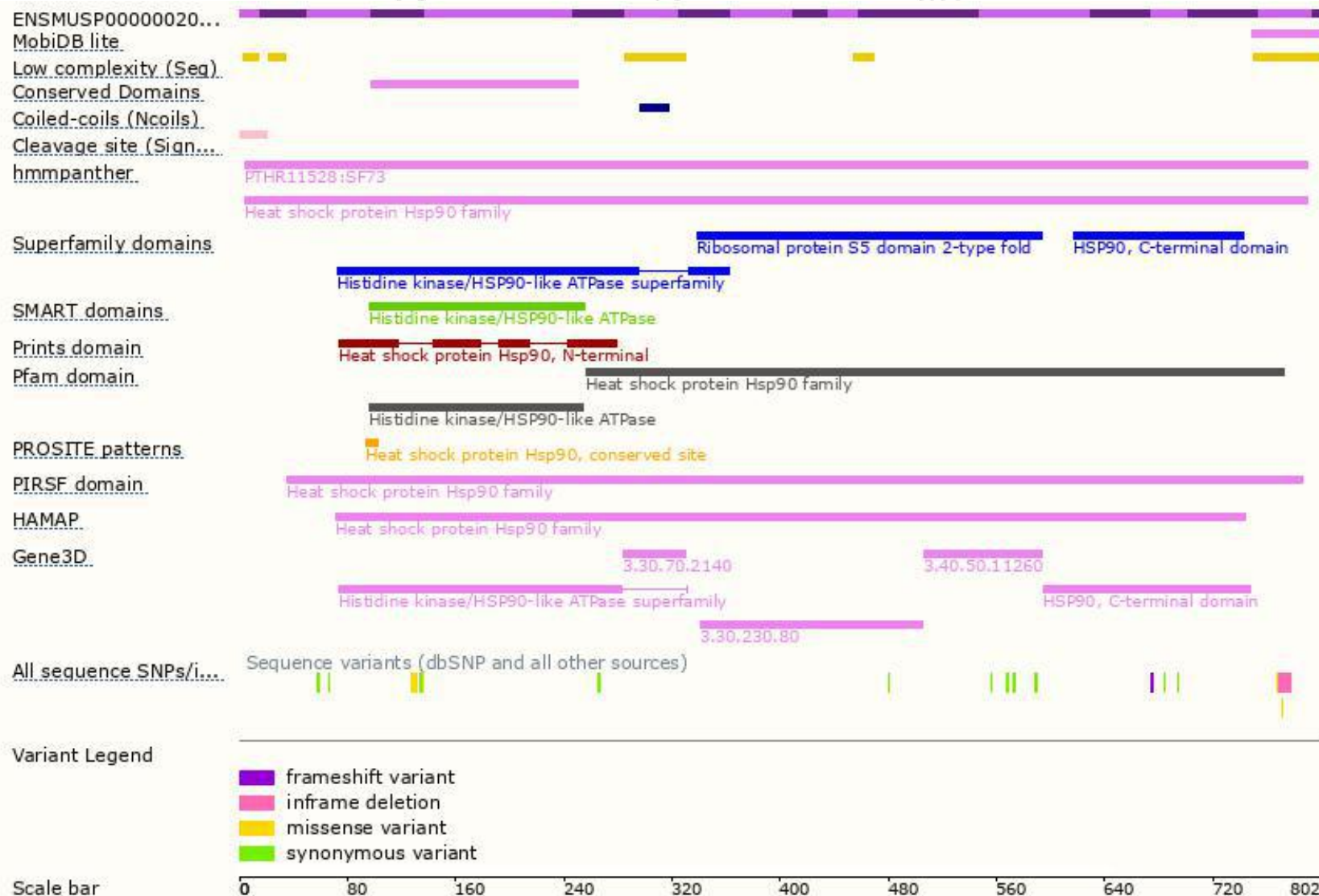
The strategy is based on the design of *Hsp90b1-201* transcript,The transcription is shown below



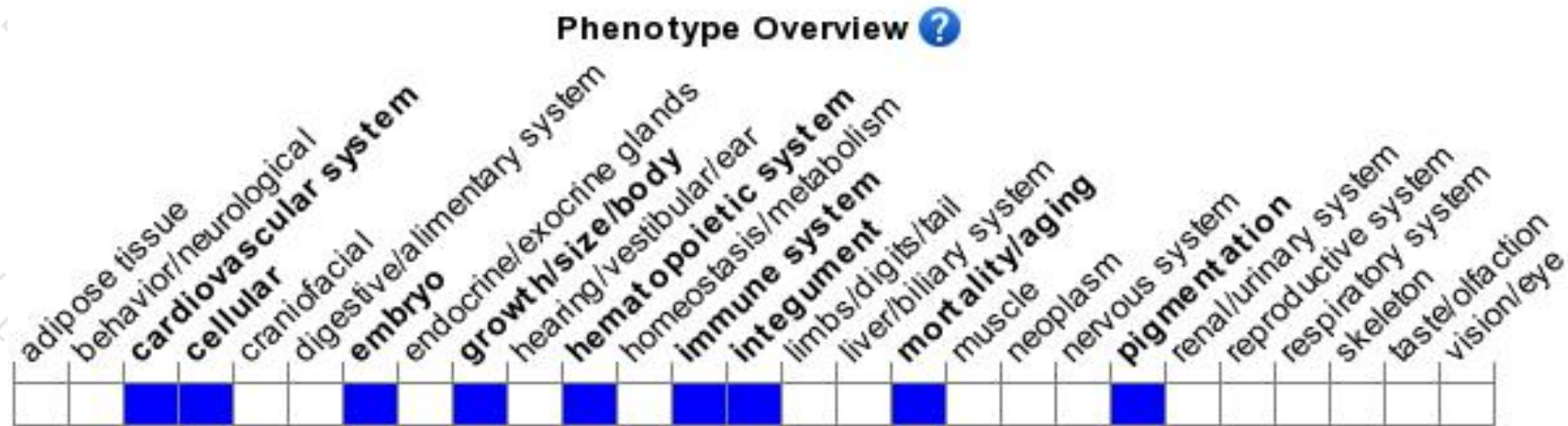
Genomic location distribution



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 homozygous for a null mutation display embryonic lethality before somite formation with failure of primitive streak formation, absence of the chorion and amnion, and failure of mesoderm formation.

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

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