Ephb1 Cas9-CKO Strategy

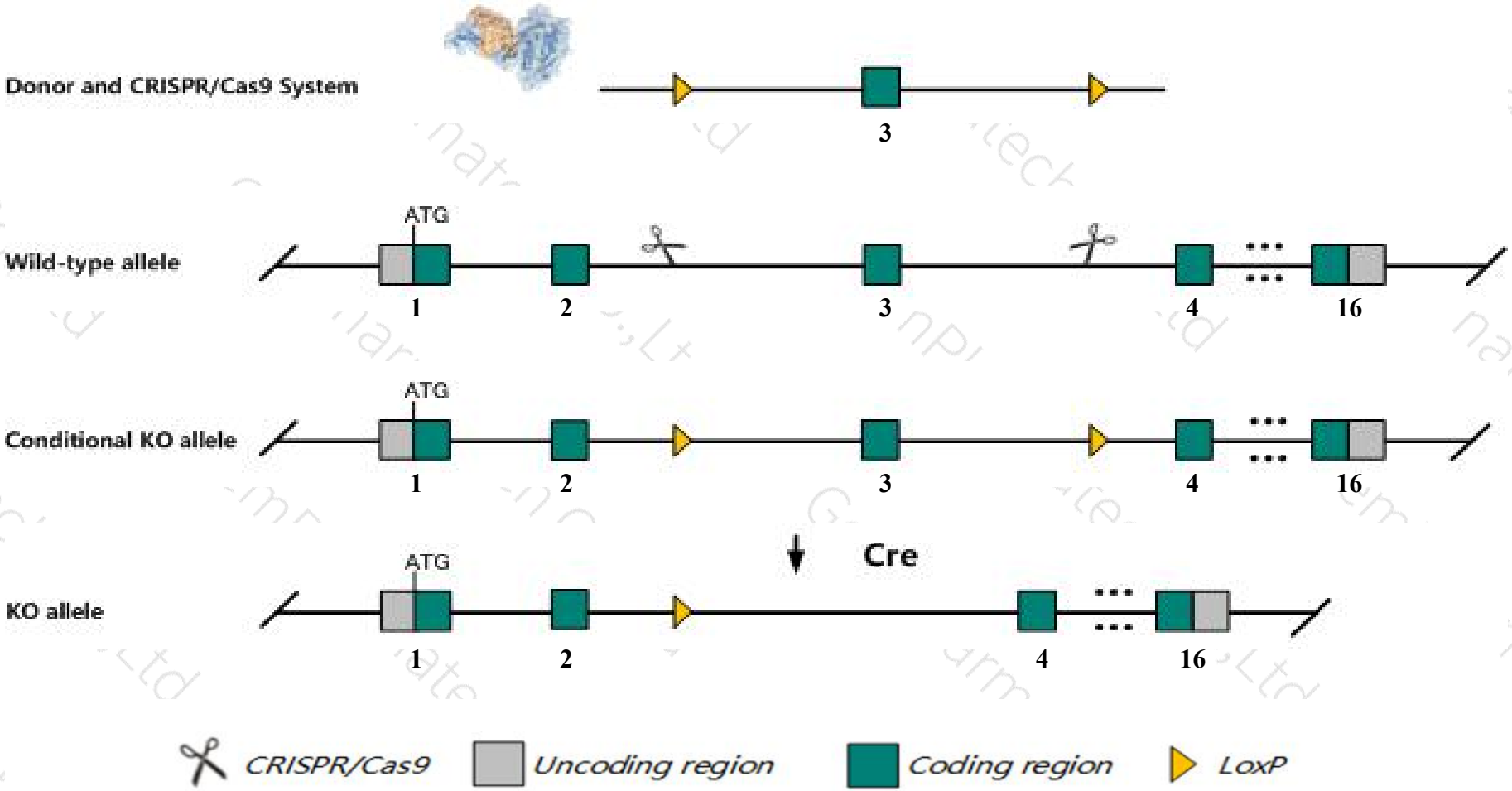
Designer: Xiaojing Li
Design Date:2019-11-29
Reviewer:JiaYu

Project Overview

Project Name	***Ephb1***
Project type	**Cas9-CKO**
Strain background	**C57BL/6JGpt**

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Ephb1* gene. The schematic diagram is as follows:



Technical routes

- The *Ephb1* gene has 6 transcripts. According to the structure of *Ephb1* gene, exon3 of *Ephb1-201* (ENSMUST00000035129.13) transcript is recommended as the knockout region.The region contains 682bp coding sequence. Knock out the region will result in disruption of protein function.

- In this project we use CRISPR/Cas9 technology to modify *Ephb1* 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 homozygous for disruptions of this gene display marked reductions of the ipsilateral optic tract. Homozygotes for one null allele show reduced corticospinal tract and abnormal anterior commissure axon crossing.

➢ The *Ephb1* gene is located on the Chr9. 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）

集萃药康 GemPharmatech

Ephb1 Eph receptor B1 [*Mus musculus* (house mouse)]

Gene ID: 270190, updated on 10-Oct-2019

Summary

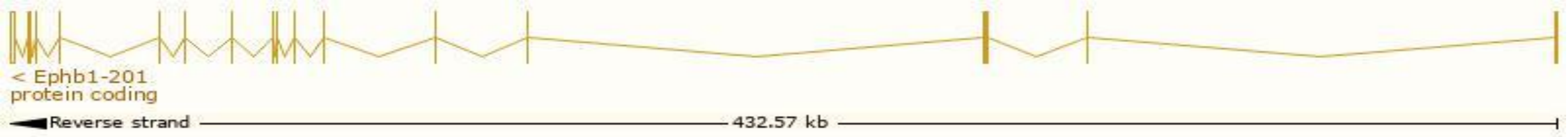
Official Symbol	Ephb1 provided by MGI
Official Full Name	Eph receptor B1 provided by MGI
Primary source	MGI:MGI:1096337
See related	Ensembl:ENSMUSG00000032537
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Elk; Net; Cek6; Elkh; Hek6; AW488255; 9330129L11; C130099E04Rik
Expression	Biased expression in whole brain E14.5 (RPKM 16.5), CNS E18 (RPKM 12.9) and 7 other tissues See more
Orthologs	human all

Transcript information（Ensembl）

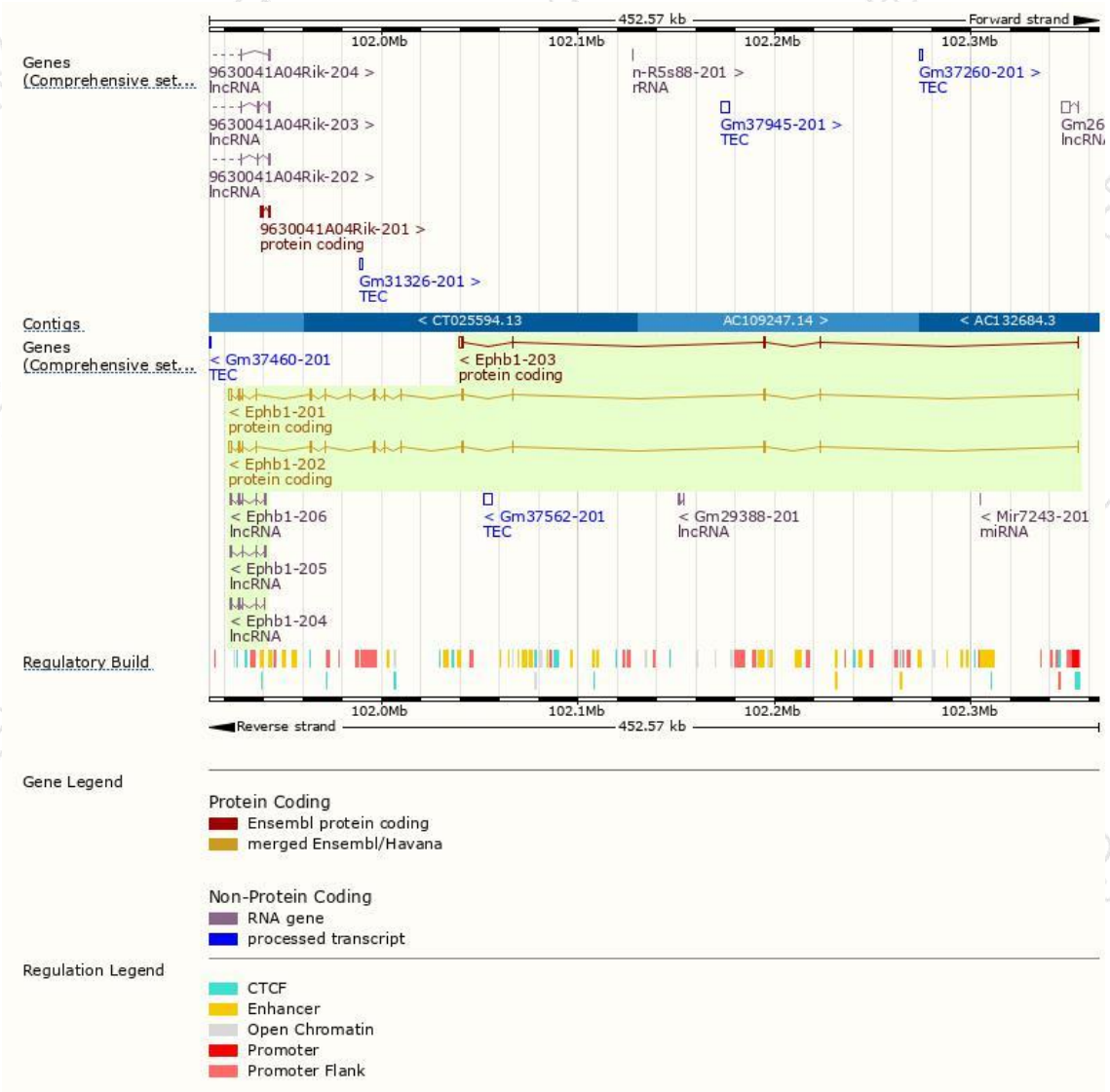
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Ephb1-201	ENSMUST00000035129.13	4667	984aa	Protein coding	CCDS40742	Q8CBF3	TSL:1 GENCODE basic APPRIS P1
Ephb1-202	ENSMUST00000085169.11	4101	943aa	Protein coding	CCDS52901	Q8CBF3	TSL:1 GENCODE basic
Ephb1-203	ENSMUST00000149800.2	3471	456aa	Protein coding	-	Q8CA63	TSL:1 GENCODE basic
Ephb1-204	ENSMUST00000215514.1	1366	No protein	lncRNA	-	-	TSL:5
Ephb1-205	ENSMUST00000217014.1	1263	No protein	lncRNA	-	-	TSL:5
Ephb1-206	ENSMUST00000217184.1	1419	No protein	lncRNA	-	-	TSL:5

The strategy is based on the design of *Ephb1-201* transcript,The transcription is shown below

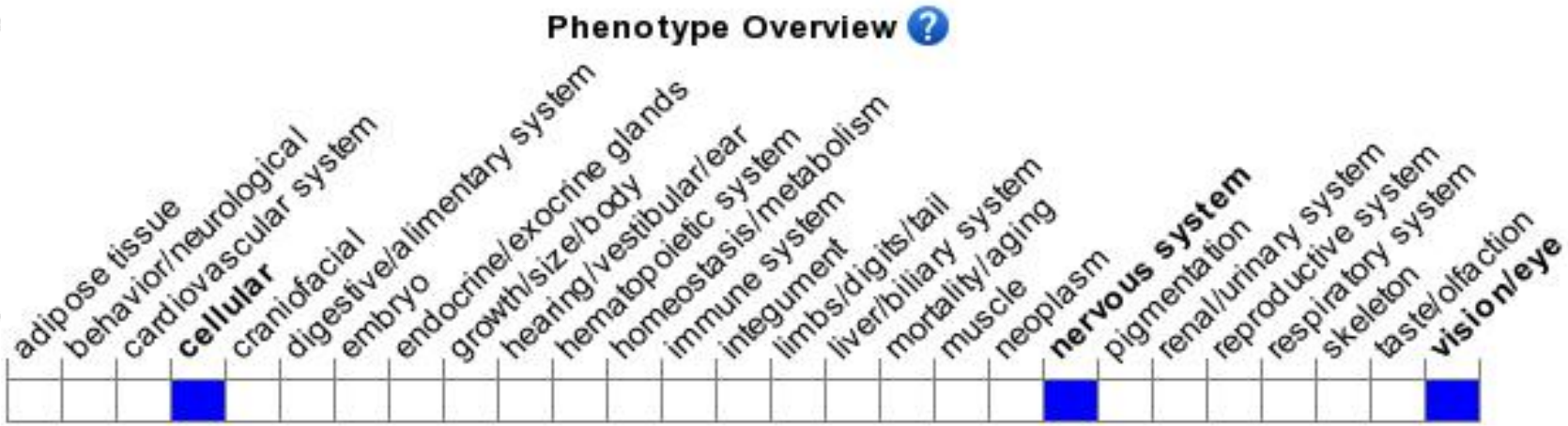


Genomic location distribution



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 disruptions of this gene display marked reductions of the ipsilateral optic tract. Homozygotes for one null allele show reduced corticospinal tract and abnormal anterior commissure axon crossing.

集萃药康
GemPharmatech

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

Tel: 400-9660890

集萃药康生物科技
GemPharmatech Co.,Ltd