Lpar1 Cas9-CKO Strategy

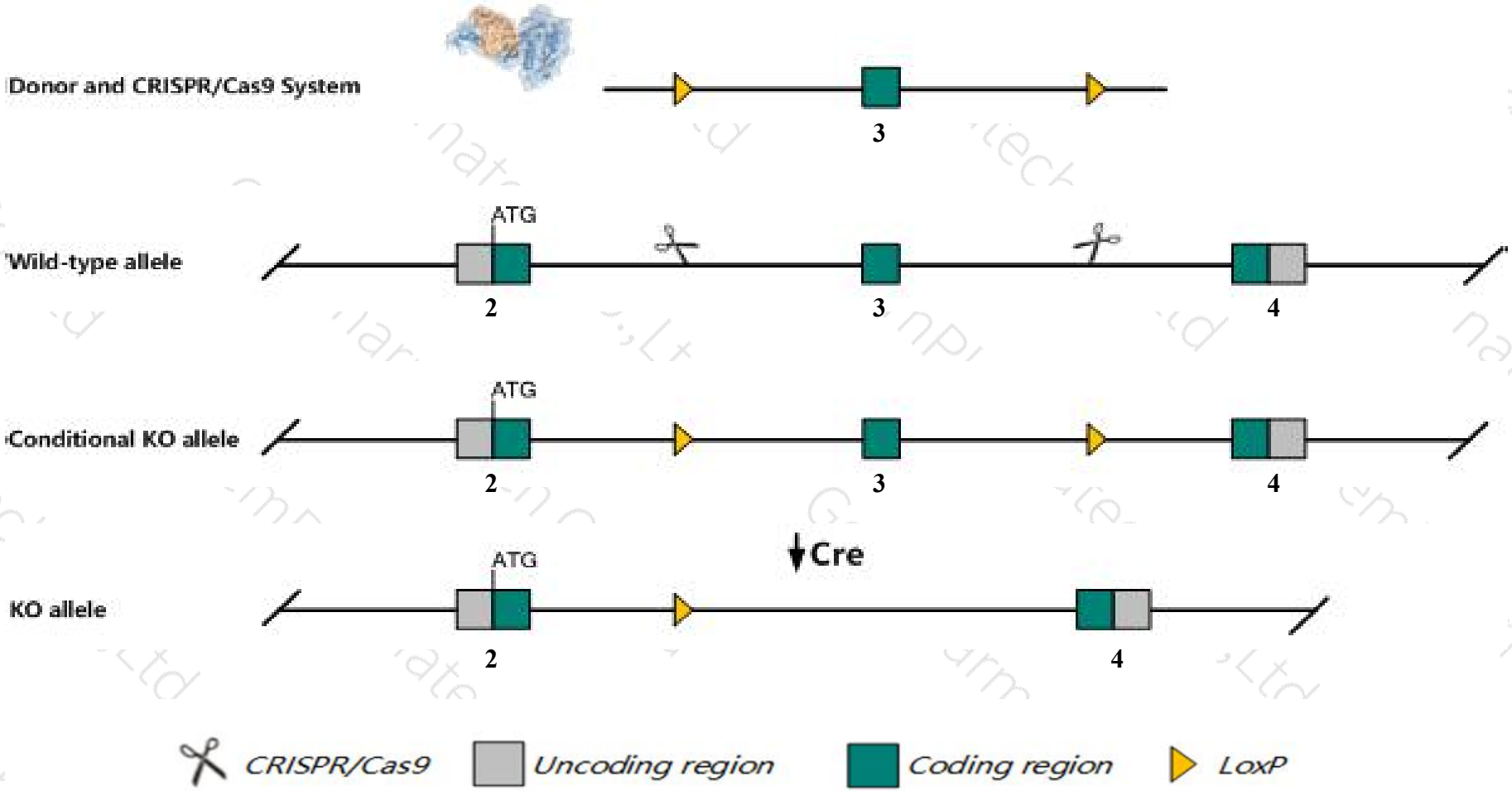
Designer: Lingyan Wu
Reviewer: Jiayuan Yao
Design Date: 2019-7-22

Project Overview

Project Name	***Lpar1***
Project type	**Cas9-CKO**
Strain background	**C57BL/6JGpt**

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Lpar1* gene. The schematic diagram is as follows:



Technical routes

- The *Lpar1* gene has 8 transcripts. According to the structure of *Lpar1* gene, exon3 of *Lpar1-203* (ENSMUST00000107571.7) transcript is recommended as the knockout region.The region contains 748bp coding sequence. Knock out the region will result in disruption of protein function.

- In this project we use CRISPR/Cas9 technology to modify *Lpar1* 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,Nullizygous mutations cause partial peri- and postnatal lethality, growth defects, craniofacial anomalies, and wide set eyes. Additional phenotypes include altered brain 5-HT and amino acids, reduced prepulse inhibition, impaired suckling, and increased apoptosis in sciatic nerve Schwann cells.

- The *Lpar1* 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）

集萃药康 GemPharmatech

Lpar1 lysophosphatidic acid receptor 1 [Mus musculus (house mouse)]

Gene ID: 14745, updated on 31-Jan-2019

Summary

Official Symbol	Lpar1 provided by MGI
Official Full Name	lysophosphatidic acid receptor 1 provided by MGI
Primary source	MGI:MGI:108429
See related	Ensembl:ENSMUSG00000038668
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	AI326300, Edg2, Gpcr26, Kdt2, lpA1, vzg-1
Expression	Ubiquitous expression in limb E14.5 (RPKM 20.9), colon adult (RPKM 17.1) and 24 other tissues See more
Orthologs	human all

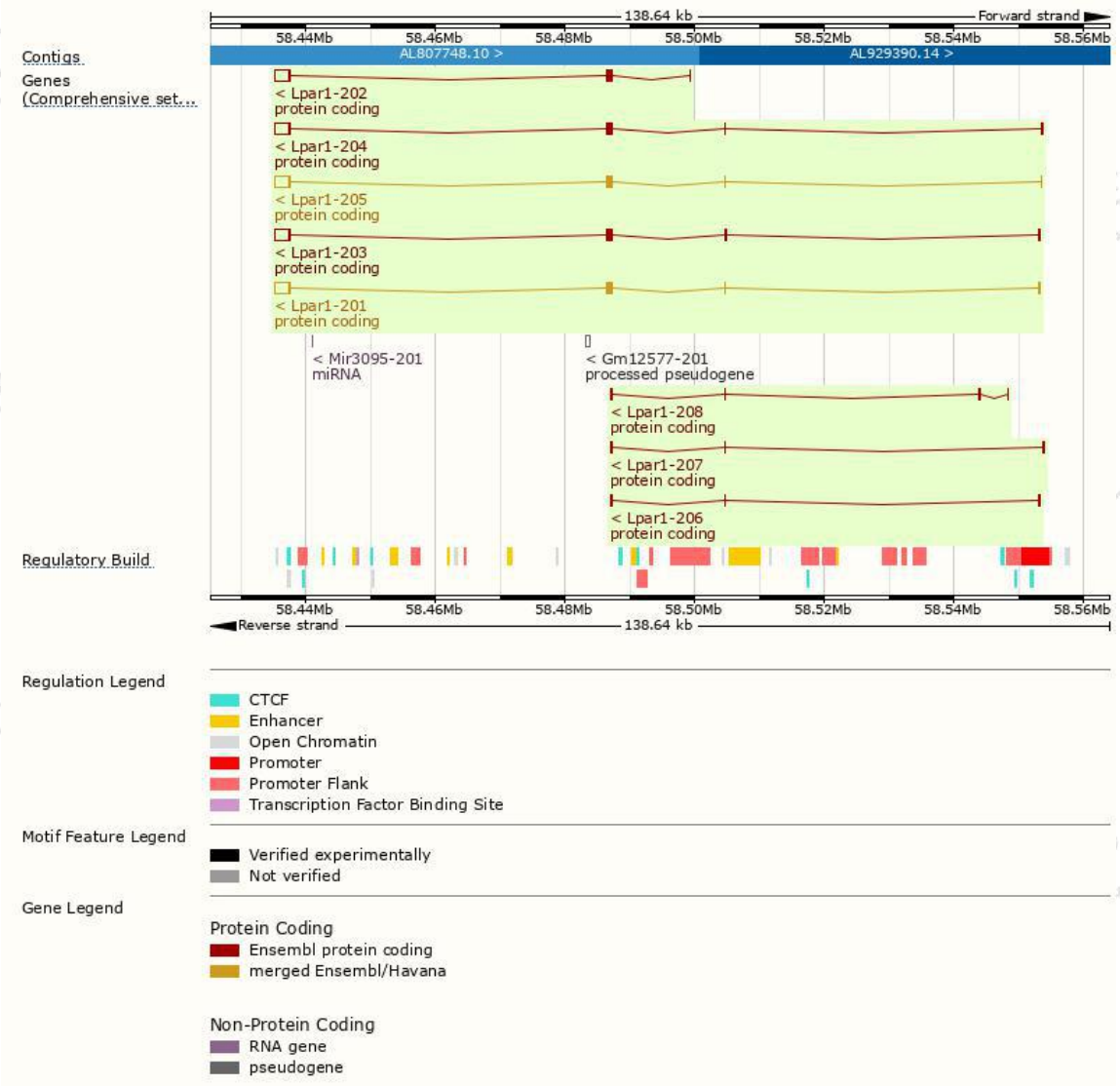
Transcript information（Ensembl）

The gene has 8 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Lpar1-203	ENSMUST00000107571.7	3543	364aa	Protein coding	CCDS18212	P61793 Q544V2	TSL:5 GENCODE basic APPRIS P1
Lpar1-201	ENSMUST00000055018.10	3512	364aa	Protein coding	CCDS18212	P61793 Q544V2	TSL:1 GENCODE basic APPRIS P1
Lpar1-204	ENSMUST00000107574.7	3437	364aa	Protein coding	CCDS18212	P61793 Q544V2	TSL:5 GENCODE basic APPRIS P1
Lpar1-205	ENSMUST00000107575.8	3421	364aa	Protein coding	CCDS18212	P61793 Q544V2	TSL:1 GENCODE basic APPRIS P1
Lpar1-202	ENSMUST00000107570.1	3244	346aa	Protein coding	CCDS71391	P61793	TSL:1 GENCODE basic
Lpar1-208	ENSMUST00000155170.7	514	75aa	Protein coding	-	A2AMI9	CDS 3' incomplete TSL:5
Lpar1-207	ENSMUST00000147354.7	454	56aa	Protein coding	-	A2AMJ0	CDS 3' incomplete TSL:2
Lpar1-206	ENSMUST00000145361.1	335	45aa	Protein coding	-	A2AMJ1	CDS 3' incomplete TSL:2

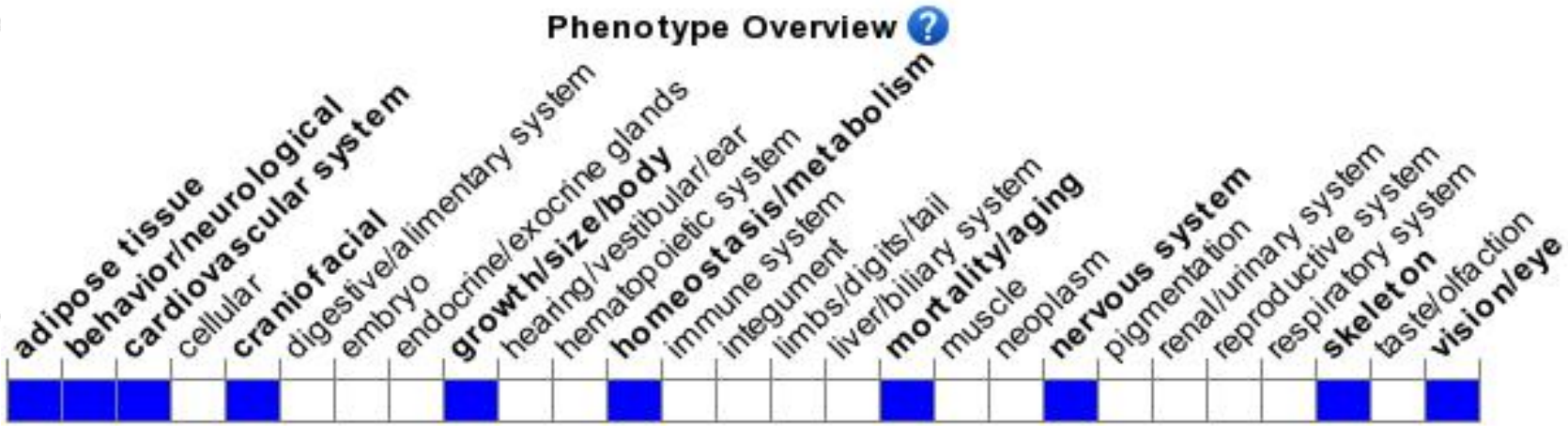
The strategy is based on the design of *Lpar1-203* transcript,The transcription is shown below

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 mutations cause partial peri- and postnatal lethality, growth defects, craniofacial anomalies, and wide set eyes. Additional phenotypes include altered brain 5-HT and amino acids, reduced prepulse inhibition, impaired suckling, and increased apoptosis in sciatic nerve Schwann cells.

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