

Ppp1r3g Cas9-CKO Strategy

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Project Overview

Project Name

Ppp1r3g

Project type

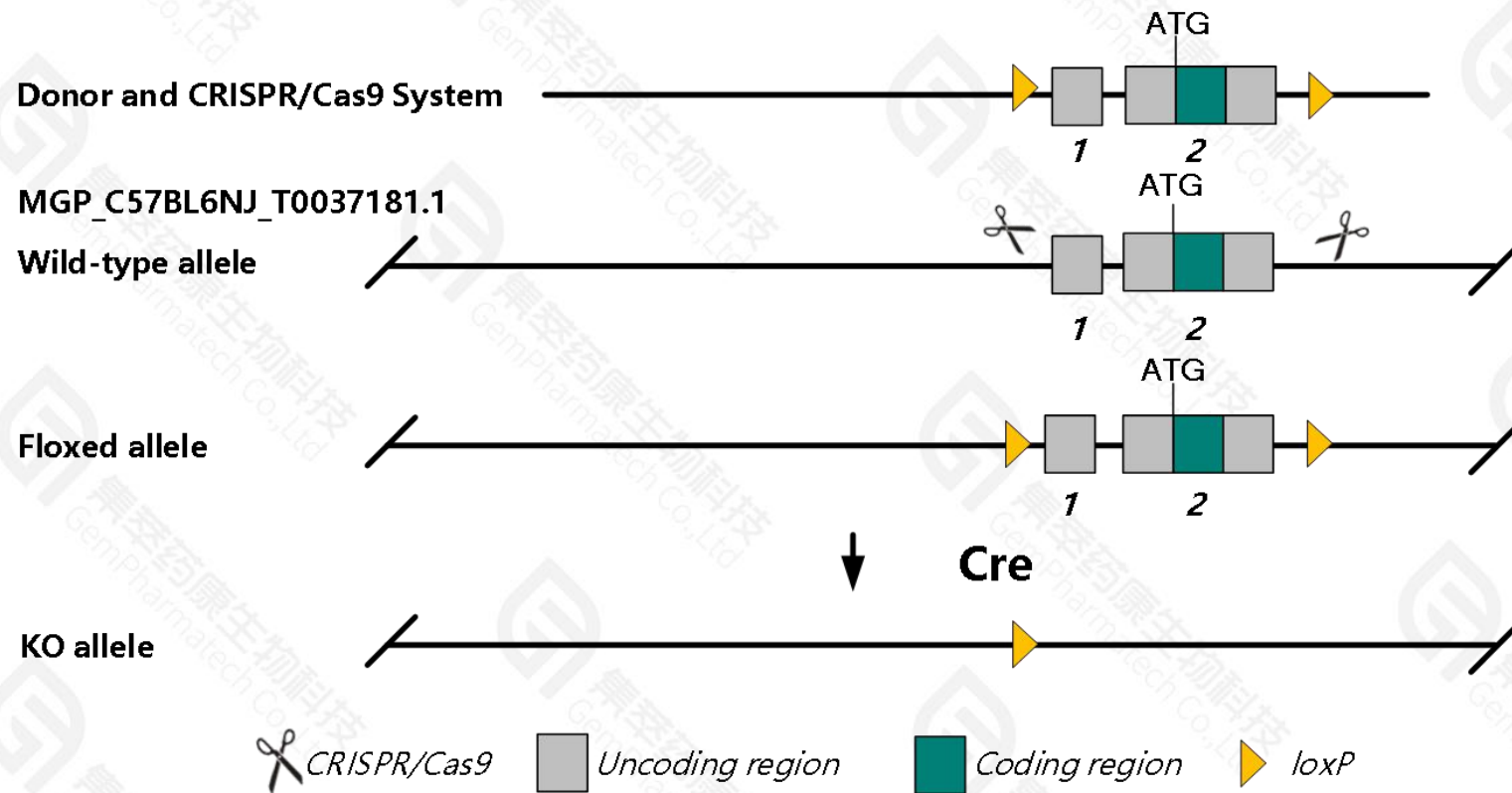
Cas9-CKO

Strain background

C57BL/6NGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Ppp1r3g* gene. The schematic diagram is as follows:



- The *Ppp1r3g* gene has 2 transcripts. According to the structure of *Ppp1r3g* gene, exon1-exon2 of *Ppp1r3g* MGP_C57BL6NJ_T0037181.1 transcript is recommended as the knockout region. The region contains all of the coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Ppp1r3g* gene. The brief process is as follows: gRNA was transcribed in vitro, donor vector was constructed. Cas9, gRNA and Donor were microinjected into the fertilized eggs of C57BL/6NGpt 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/6J Gpt 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 or cell types.

- According to the existing MGI data, under a high-fat diet, mice homozygous for a knock-out allele show decreased susceptibility to diet-induced obesity and hepatic steatosis along with a higher metabolic rate, increased food intake, and decreased glycogen levels in liver and adipose.
- The flox region of the *Ppp1r3g* gene contains the functional region of the *Ppp1r3g* gene. Knockout may affect the function of the *Ppp1r3g* gene.
- Knockout of the *pp1r3g* gene may cause the regulation of the 5'end of *Ppp1r3g*-MGP_C57BL6NJ_T0037181.1
- The *Ppp1r3g* gene is located on the Chr13. 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 the loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

Gene information (NCBI)



Ppp1r3g protein phosphatase 1, regulatory subunit 3G [*Mus musculus* (house mouse)]

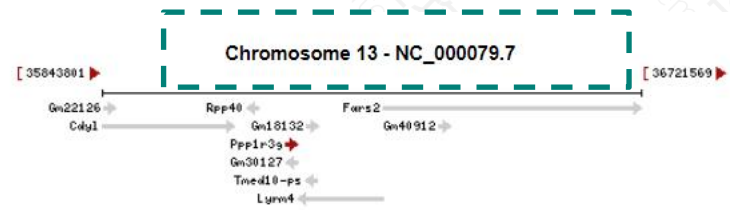
Download Datasets

Gene ID: 76487, updated on 23-Jun-2021

Summary

- Official Symbol** Ppp1r3g provided by MGI
- Official Full Name** protein phosphatase 1, regulatory subunit 3G provided by MGI
- Primary source** MGI: MGI:1923737
- See related** Ensembl: ENSMUSG00000050423
- 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** 1600032L17Rik
- Expression** Biased expression in cortex adult (RPKM 2.4), liver E18 (RPKM 2.2) and 9 other tissues [See more](#)
- Orthologs** [human](#) [all](#)

NEW Try the new [Gene table](#)
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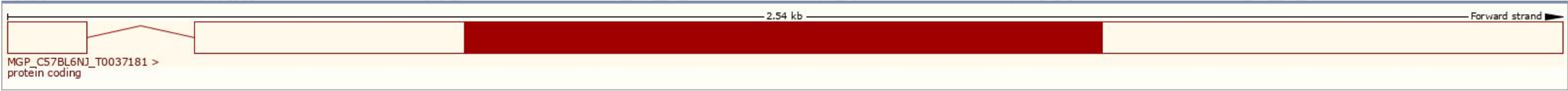


Transcript information (Ensembl)

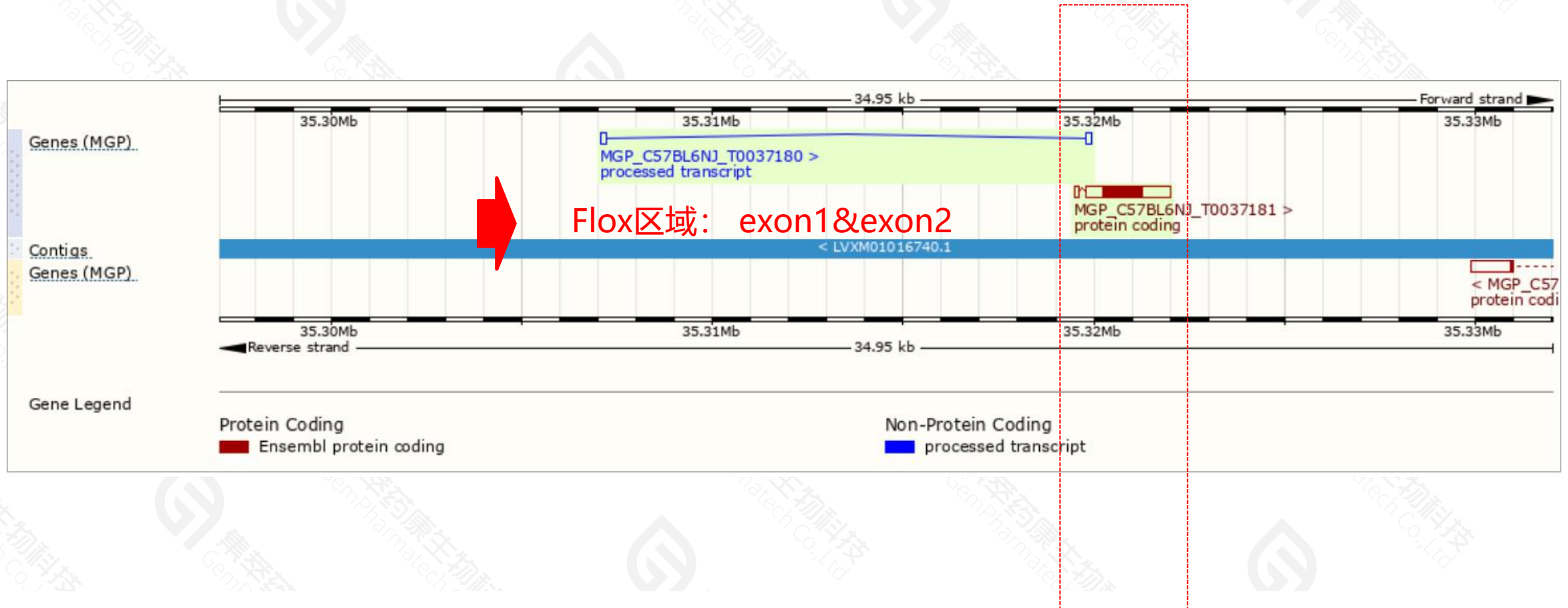
The gene has 2 transcripts, and all transcripts are shown below:

Transcript ID	Name	bp	Protein	Biotype	CCDS	Flags
MGP_C57BL6NJ_T0037181.1	-	2361	347aa	<div><div></div>Protein coding</div>	-	-
MGP_C57BL6NJ_T0037180.1	-	325	No protein	<div><div></div>Processed transcript</div>	CCDS49237	-

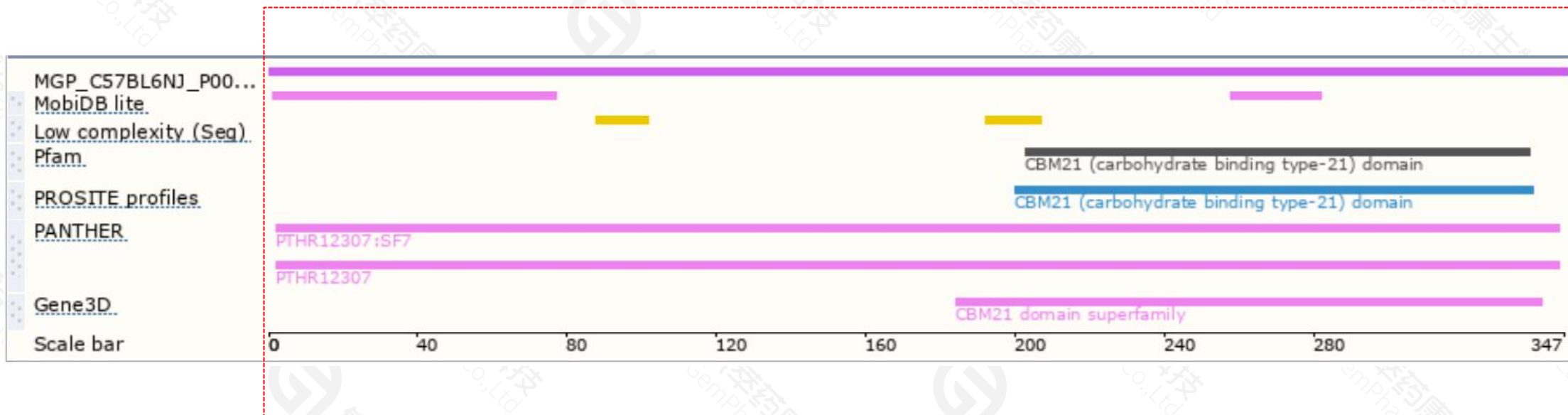
The strategy is based on the design of *Ppp1r3g*-MGP_C57BL6NJ_T0037181.1 transcript, The transcription is shown below



Genomic location distribution

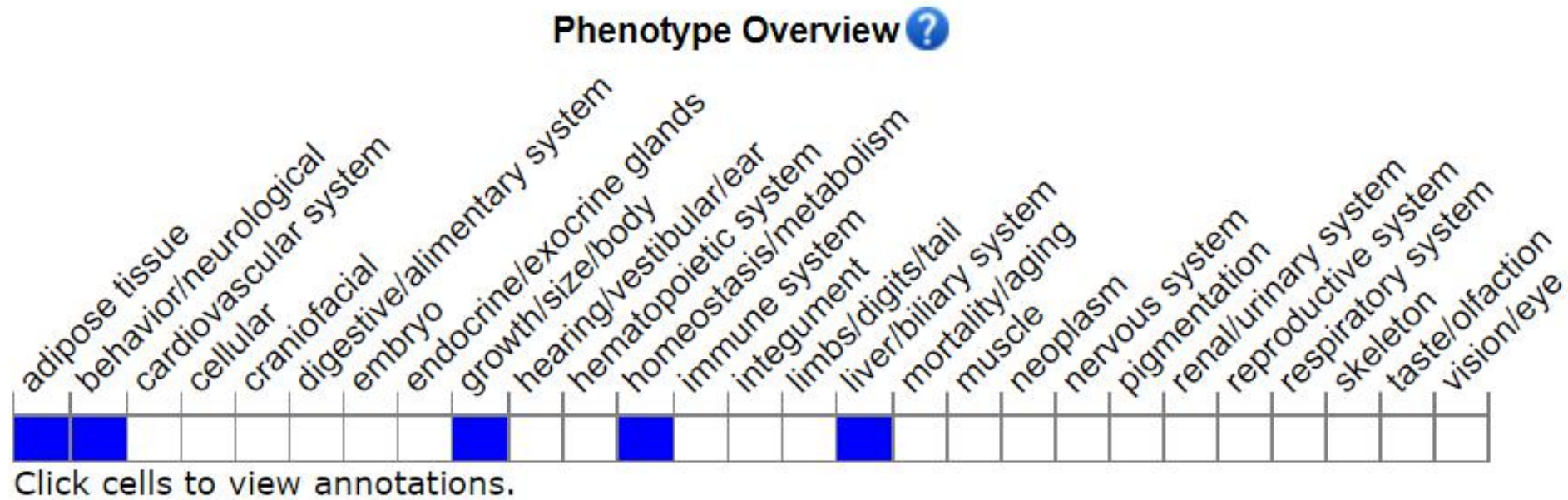


Protein domain



Flox区域: exon1&exon2

Mouse phenotype description(MGI)



Under a high-fat diet, mice homozygous for a knock-out allele show decreased susceptibility to diet-induced obesity and hepatic steatosis along with a higher metabolic rate, increased food intake, and decreased glycogen levels in liver and adipose <http://www.informatics.jax.org/marker/MGI:1923737>

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