

***Plin1* Cas9-CKO Strategy**

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Project Overview

Project Name

Plin1

Project type

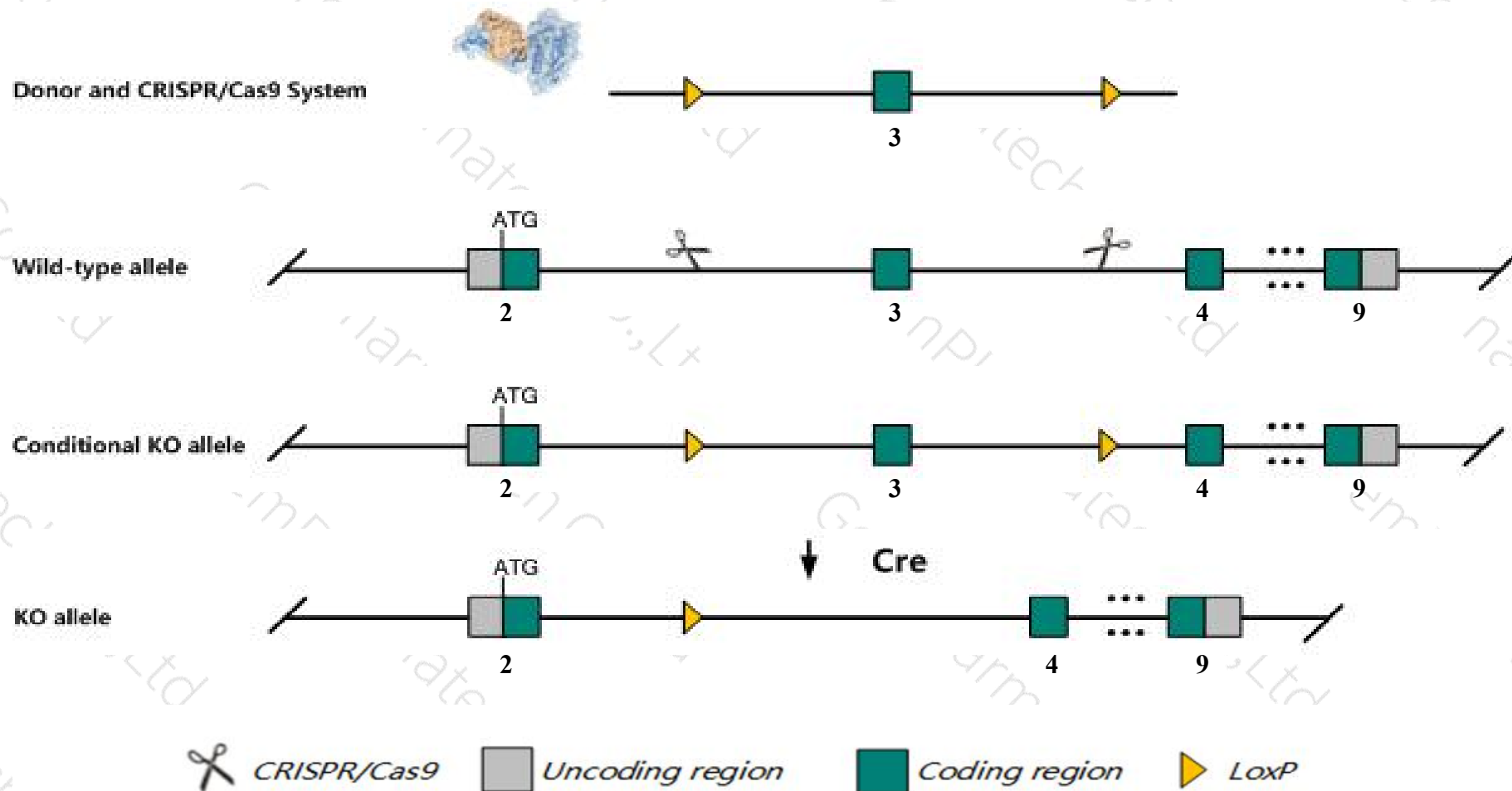
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Plin1* gene. The schematic diagram is as follows:



- The *Plin1* gene has 7 transcripts. According to the structure of *Plin1* gene, exon3 of *Plin1-201* (ENSMUST00000032762.13) transcript is recommended as the knockout region. The region contains 205bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Plin1* 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, Homozygous inactivation of this gene leads to increased lean body mass and altered adipocyte lipolysis, leptin production and susceptibility to diet-induced obesity. Increased oxygen and food consumption, impaired cold adaptation, and altered glucose and blood homeostasis have also been observed.
- The 5' region of transcript *Plin1-204* is incomplete, so the effect on it is unknown.
- The *Plin1* gene is located on the Chr7. 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)

Plin1 perilipin 1 [Mus musculus (house mouse)]

Gene ID: 103968, updated on 5-Mar-2019

Summary



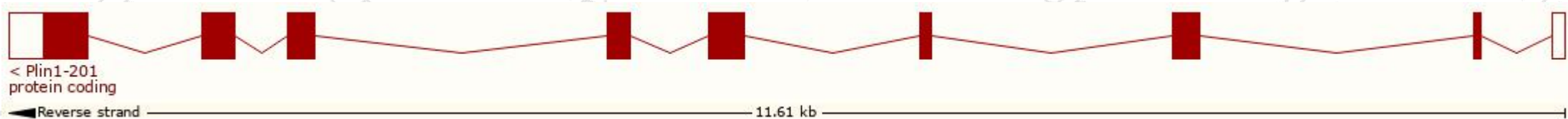
Official Symbol	Plin1 provided by MGI
Official Full Name	perilipin 1 provided by MGI
Primary source	MGI:MGI:1890505
See related	Ensembl:ENSMUSG00000030546
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	6030432J05Rik, Peri, Plin
Expression	Biased expression in subcutaneous fat pad adult (RPKM 403.0), mammary gland adult (RPKM 366.1) and 4 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

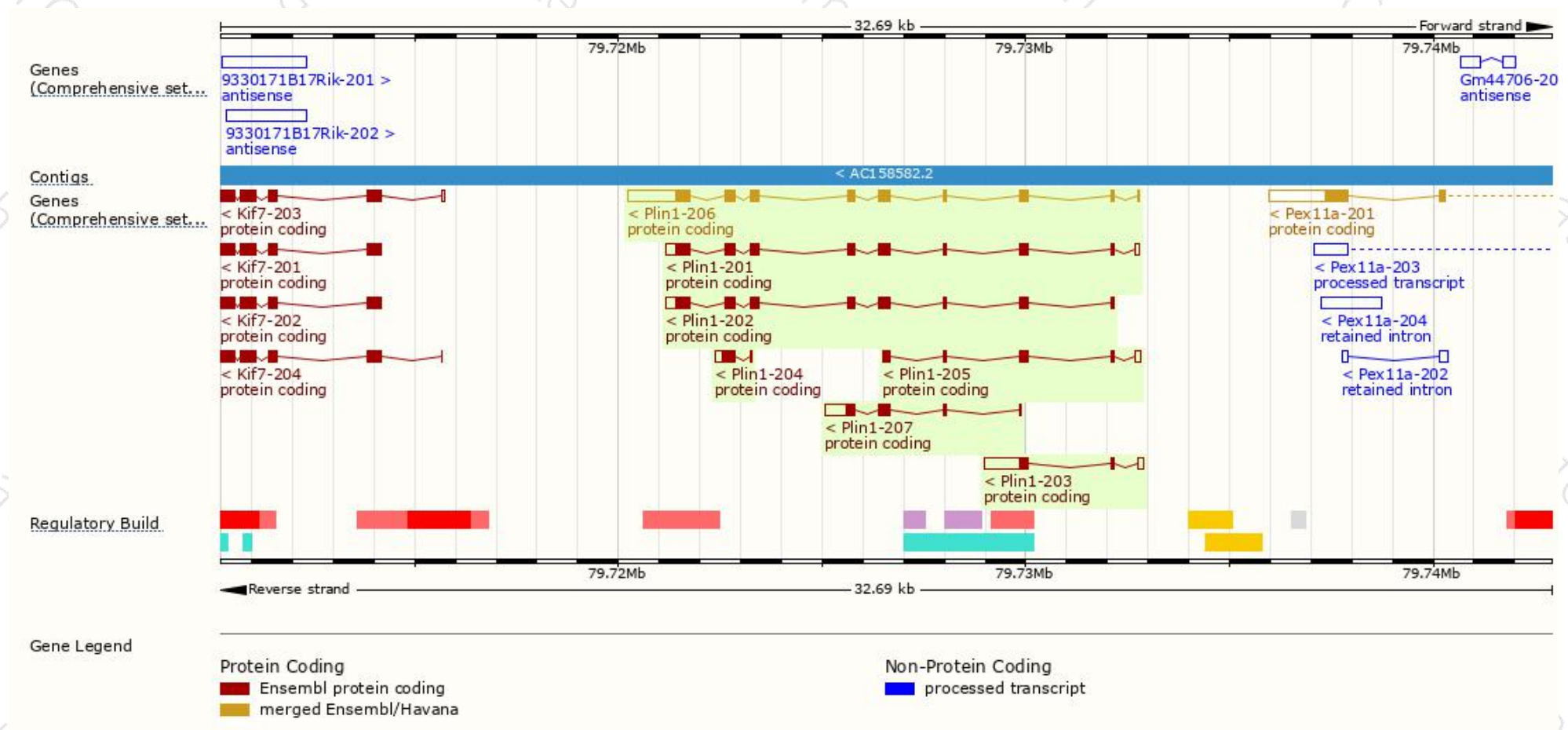
The gene has 7 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Plin1-206	ENSMUST00000205915.1	2817	517aa	Protein coding	CCDS21385	Q8CGN5	TSL:1 GENCODE basic APPRIS P1
Plin1-201	ENSMUST00000032762.13	1916	517aa	Protein coding	CCDS21385	Q8CGN5	TSL:5 GENCODE basic APPRIS P1
Plin1-202	ENSMUST00000178257.2	1841	517aa	Protein coding	CCDS21385	Q8CGN5	TSL:1 GENCODE basic APPRIS P1
Plin1-203	ENSMUST00000205413.1	1294	87aa	Protein coding	-	A0A0U1RNY2	TSL:1 GENCODE basic
Plin1-207	ENSMUST00000206083.1	1082	190aa	Protein coding	-	A0A0U1RPJ3	CDS 5' incomplete TSL:1
Plin1-205	ENSMUST00000205747.1	647	161aa	Protein coding	-	A0A0U1RQ08	CDS 3' incomplete TSL:2
Plin1-204	ENSMUST00000205553.1	511	111aa	Protein coding	-	A0A0U1RNP7	CDS 5' incomplete TSL:3

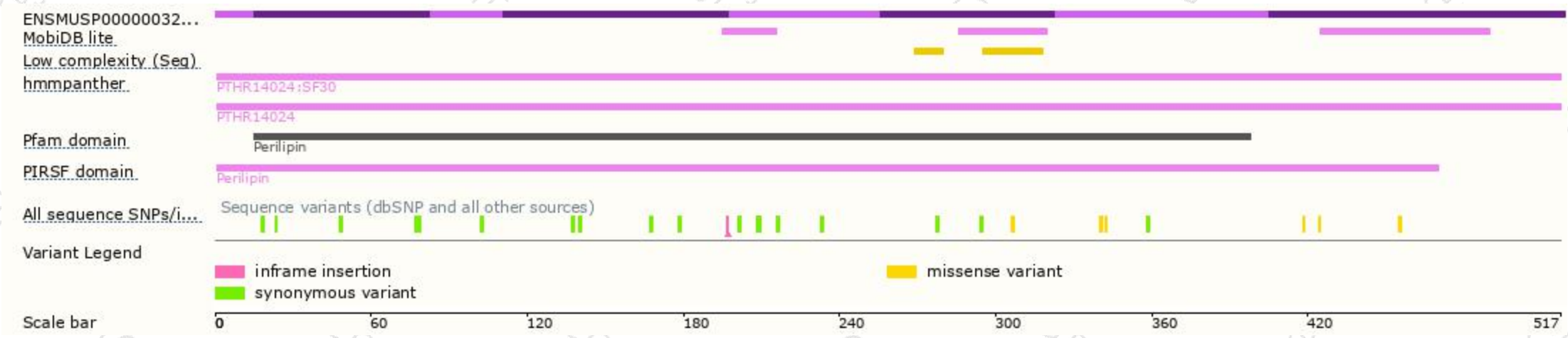
The strategy is based on the design of *Plin1-201* transcript,The transcription is shown below



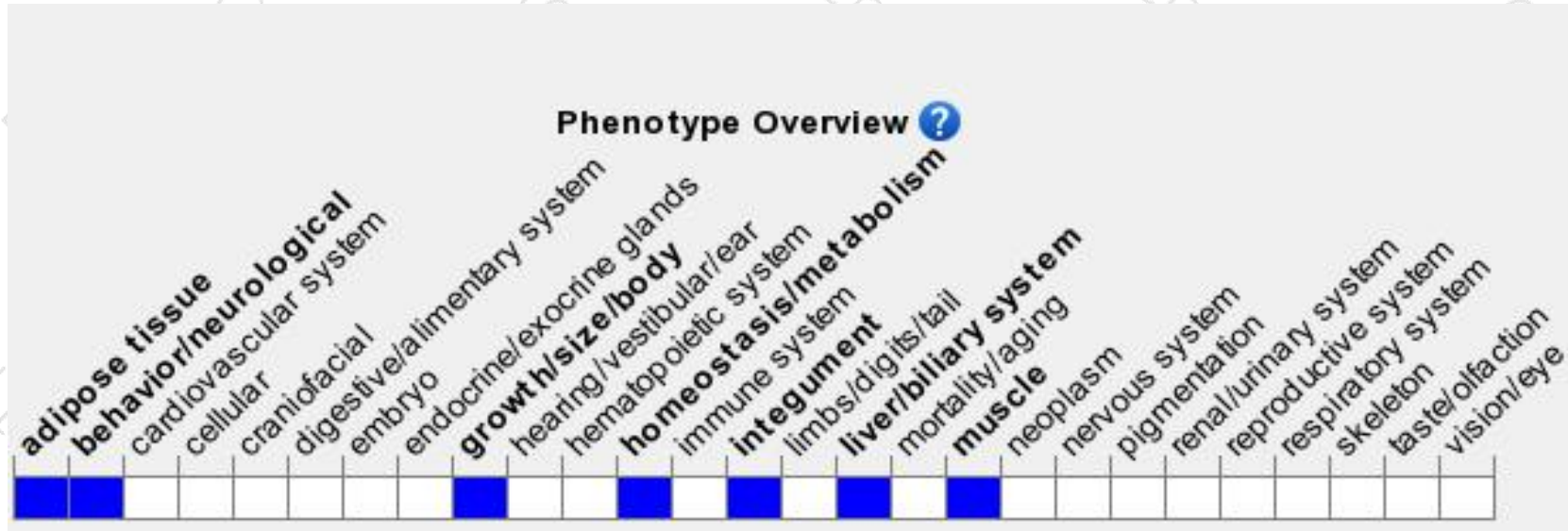
Genomic location distribution



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/>).

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

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