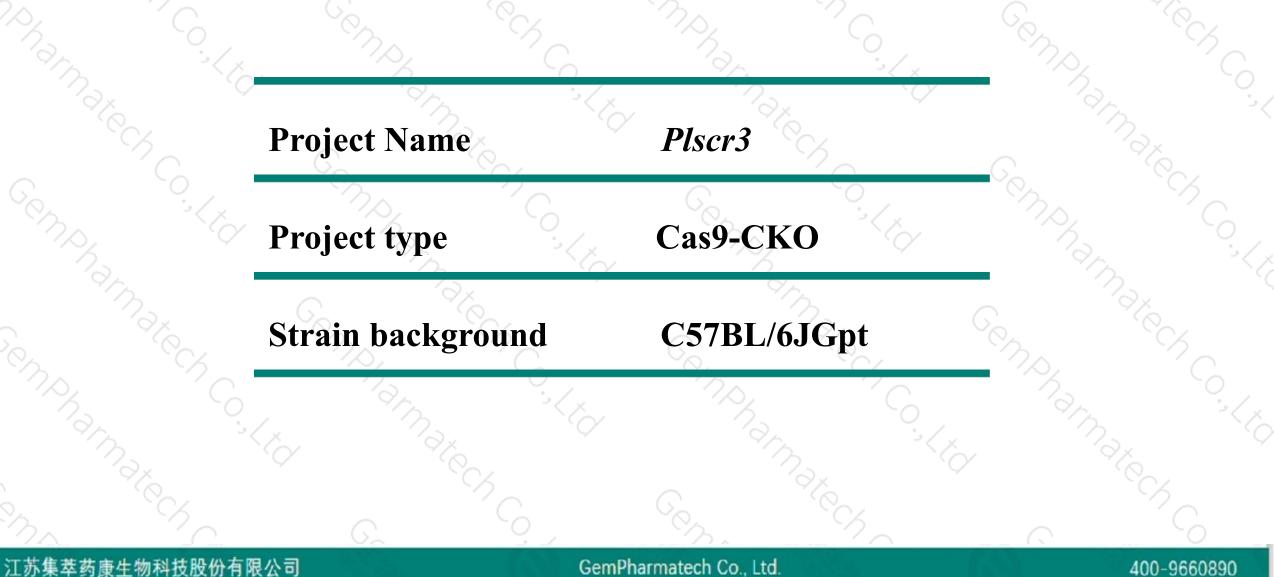


# **Plscr3** Cas9-CKO Strategy

Designer: Reviewer: Design Date: JiaYu Xiaojing Li 2020-2-26

# **Project Overview**





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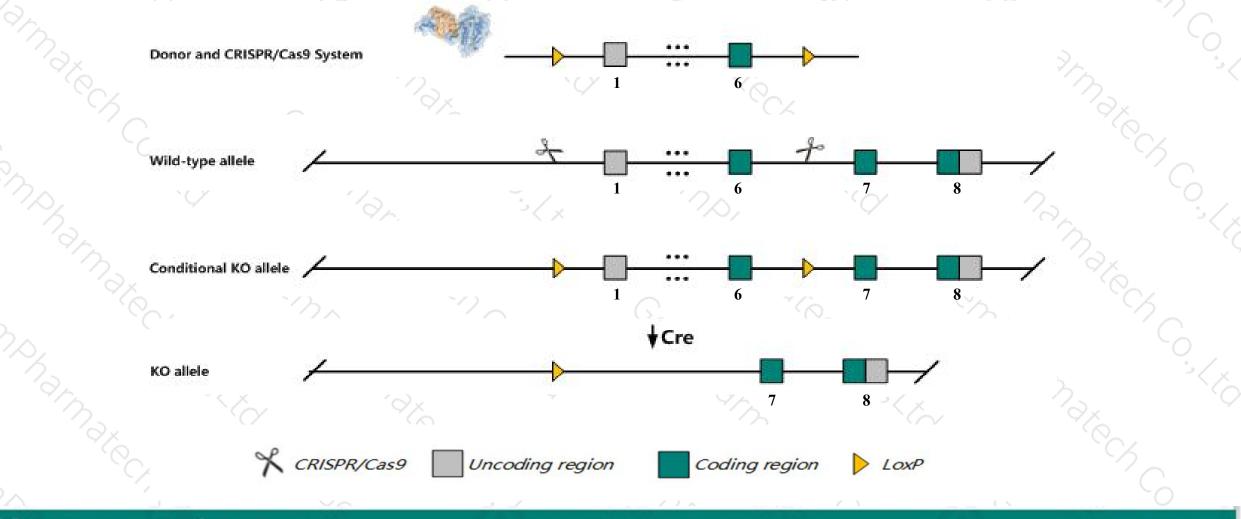
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# **Conditional Knockout strategy**



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This model will use CRISPR/Cas9 technology to edit the *Plscr3* gene. The schematic diagram is as follows:



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The Plscr3 gene has 4 transcripts. According to the structure of Plscr3 gene, exon1-exon6 of Plscr3-203 (ENSMUST00000108633.8) transcript is recommended as the knockout region. The region contains most 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 *Plscr3* 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, Homozygous null mice display lipid-engorged adipocytes, increased abdominal fat stores, mild hyperglycemia, dyslipidemia, impaired glucose tolerance, insulin resistance, altered plasma adiponectin and leptin levels, and impaired insulin-stimulated glucose uptake by adipocytes.
- The *Plscr3* gene is located on the Chr11. 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)



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### PIscr3 phospholipid scramblase 3 [Mus musculus (house mouse)]

Gene ID: 70310, updated on 31-Jan-2019

#### Summary

Official Symbol Plscr3 provided by MGI Official Full Name phospholipid scramblase 3 provided by MGI Primary source MGI:MGI:1917560 Ensembl:ENSMUSG00000019461 See related Gene type protein coding RefSeq status VALIDATED Organism Mus musculus Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Lineage Muroidea; Muridae; Murinae; Mus; Mus Also known as 2210403O21Rik, 2610037N06Rik, ESTM3, X83310 Expression Ubiquitous expression in lung adult (RPKM 35.8), limb E14.5 (RPKM 32.0) and 28 other tissuesSee more Orthologs human all

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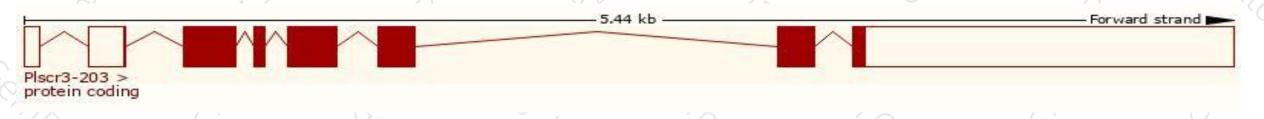
# **Transcript information (Ensembl)**



The gene has 4 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Piscr3-203	ENSMUST00000108633.8	2785	<u>296aa</u>	Protein coding	CCDS24916	Q5F283 Q9JIZ9	TSL:1 GENCODE basic APPRIS P1
Piscr3-201	ENSMUST00000019605.3	1927	<u>296aa</u>	Protein coding	CCDS24916	Q5F283 Q9JIZ9	TSL:1 GENCODE basic APPRIS P1
Piscr3-202	ENSMUST00000108632.7	1835	<u>296aa</u>	Protein coding	CCDS24916	Q5F283 Q9JIZ9	TSL:1 GENCODE basic APPRIS P1
Plscr3-204	ENSMUST00000152566.7	908	<u>224aa</u>	Protein coding	Nigil.	Q5F284	CDS 3' incomplete TSL:3

The strategy is based on the design of *Plscr3-203* transcript, The transcription is shown below

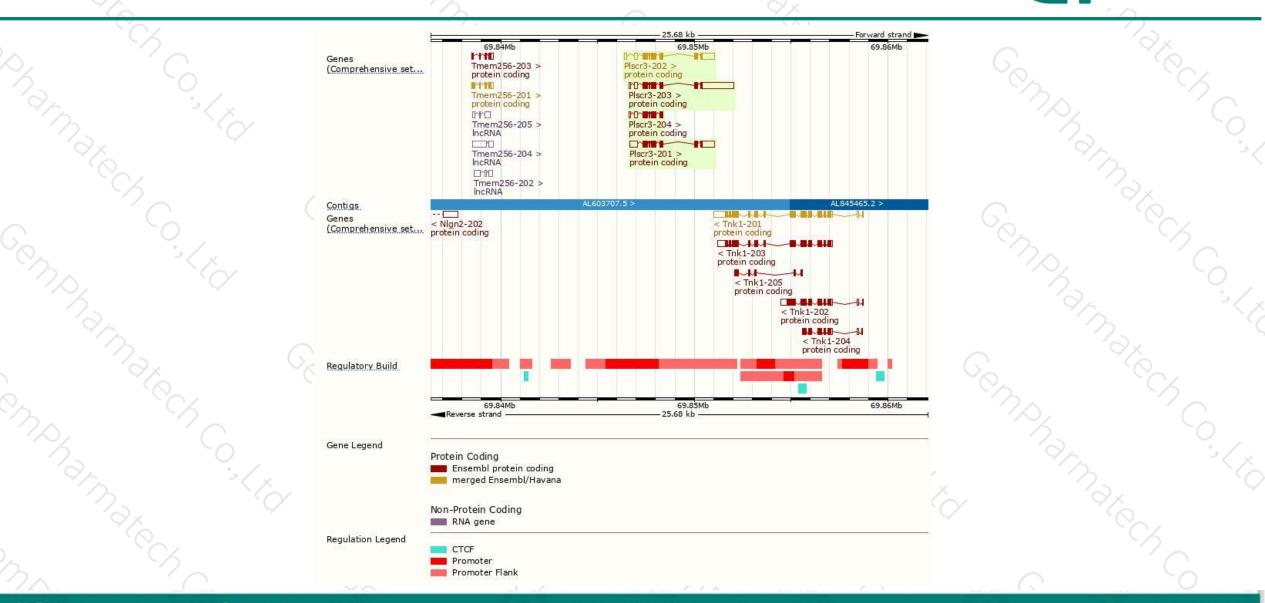


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# **Genomic location distribution**



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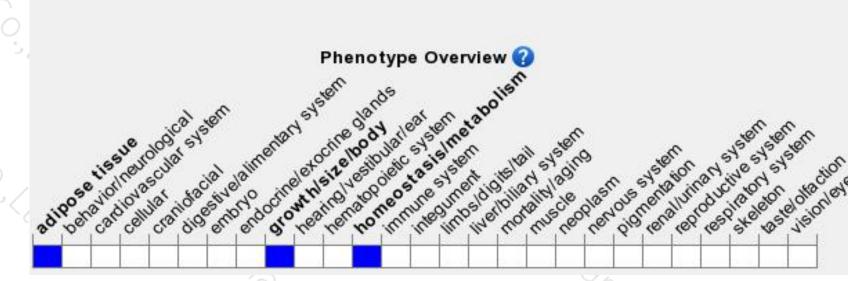
# **Protein domain**





# 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, Homozygous null mice display lipid-engorged adipocytes, increased abdominal fat stores, mild hyperglycemia, dyslipidemia, impaired glucose tolerance, insulin resistance, altered plasma adiponectin and leptin levels, and impaired insulin-stimulated glucose uptake by adipocytes.

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If you have any questions, you are welcome to inquire. Tel: 400-9660890



