

# *Dusp6* Cas9-CKO Strategy

**Designer:**

**Daohua Xu**

**Design Date:**

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# Project Overview

**Project Name**

*Dusp6*

**Project type**

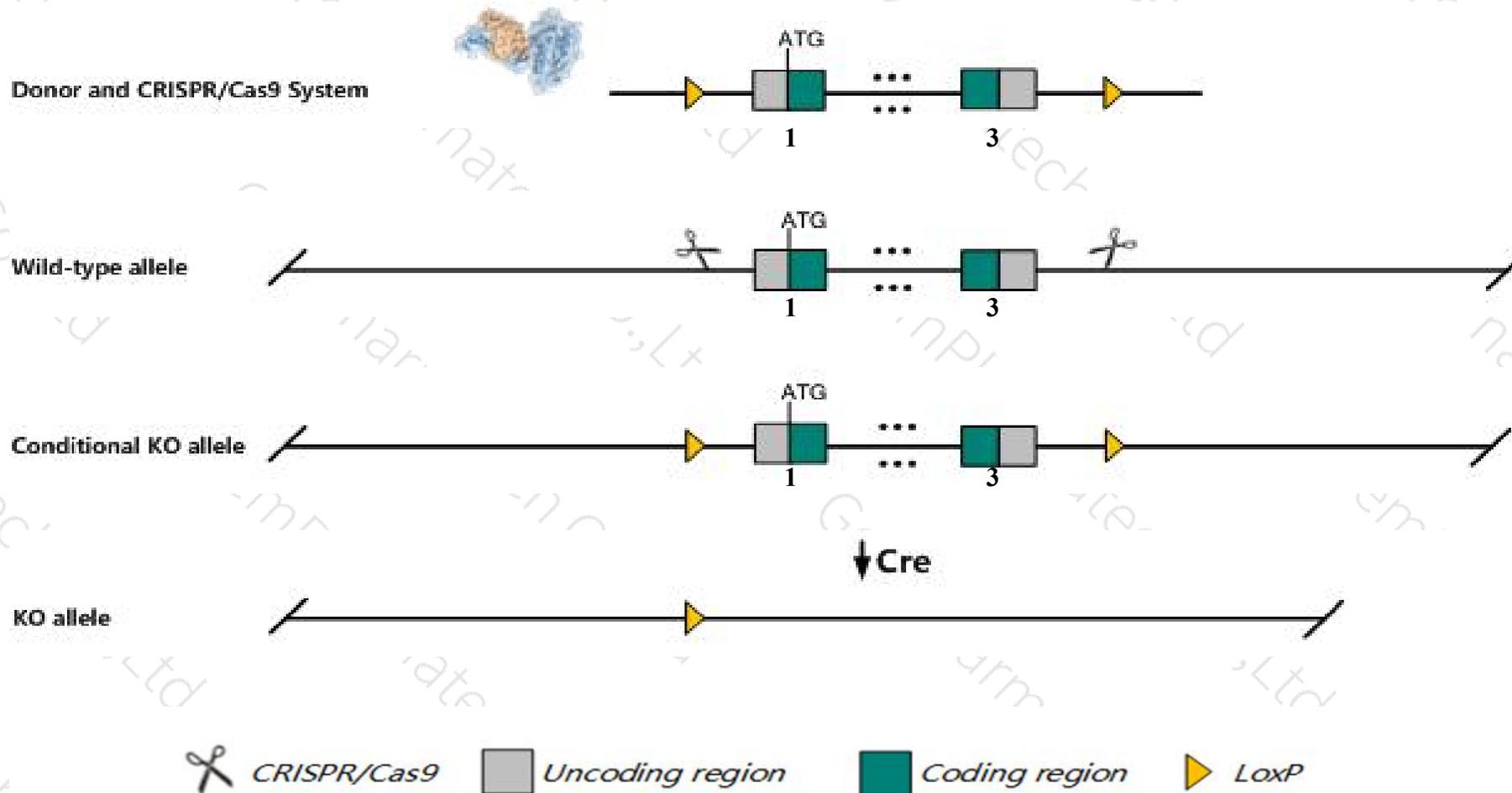
**Cas9-CKO**

**Strain background**

**C57BL/6JGpt**

# Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Dusp6* gene. The schematic diagram is as follows:



- The *Dusp6* gene has 4 transcripts. According to the structure of *Dusp6* gene, exon1-exon3 of *Dusp6-201* (ENSMUST00000020118.4) 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 *Dusp6* 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, Mice homozygous or heterozygous for a null mutation display partial penetrance of postnatal lethality, reduced body weight, and abnormal growth plate morphology.
- The strategy affects the 5-terminal regulation of Gm48089 and affects the 3-terminal regulation of Gm34921.
- The *Dusp6* gene is located on the Chr10. 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)

## Dusp6 dual specificity phosphatase 6 [Mus musculus (house mouse)]

Gene ID: 67603, updated on 5-Mar-2019

### Summary



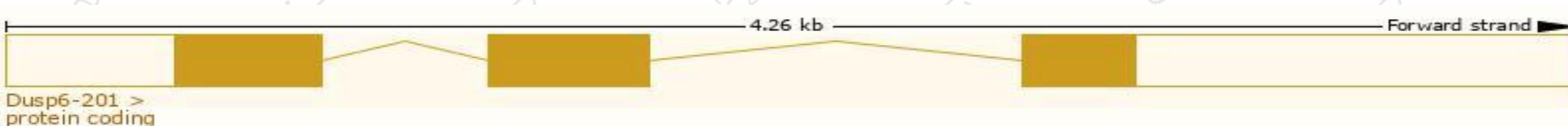
<b>Official Symbol</b>	Dusp6 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	dual specificity phosphatase 6 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:1914853</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000019960</a>
<b>Gene type</b>	protein coding
<b>RefSeq status</b>	VALIDATED
<b>Organism</b>	<a href="#">Mus musculus</a>
<b>Lineage</b>	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
<b>Also known as</b>	1300019I03Rik, MKP-3, MKP3, PYST1
<b>Expression</b>	Ubiquitous expression in lung adult (RPKM 46.1), adrenal adult (RPKM 45.5) and 28 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

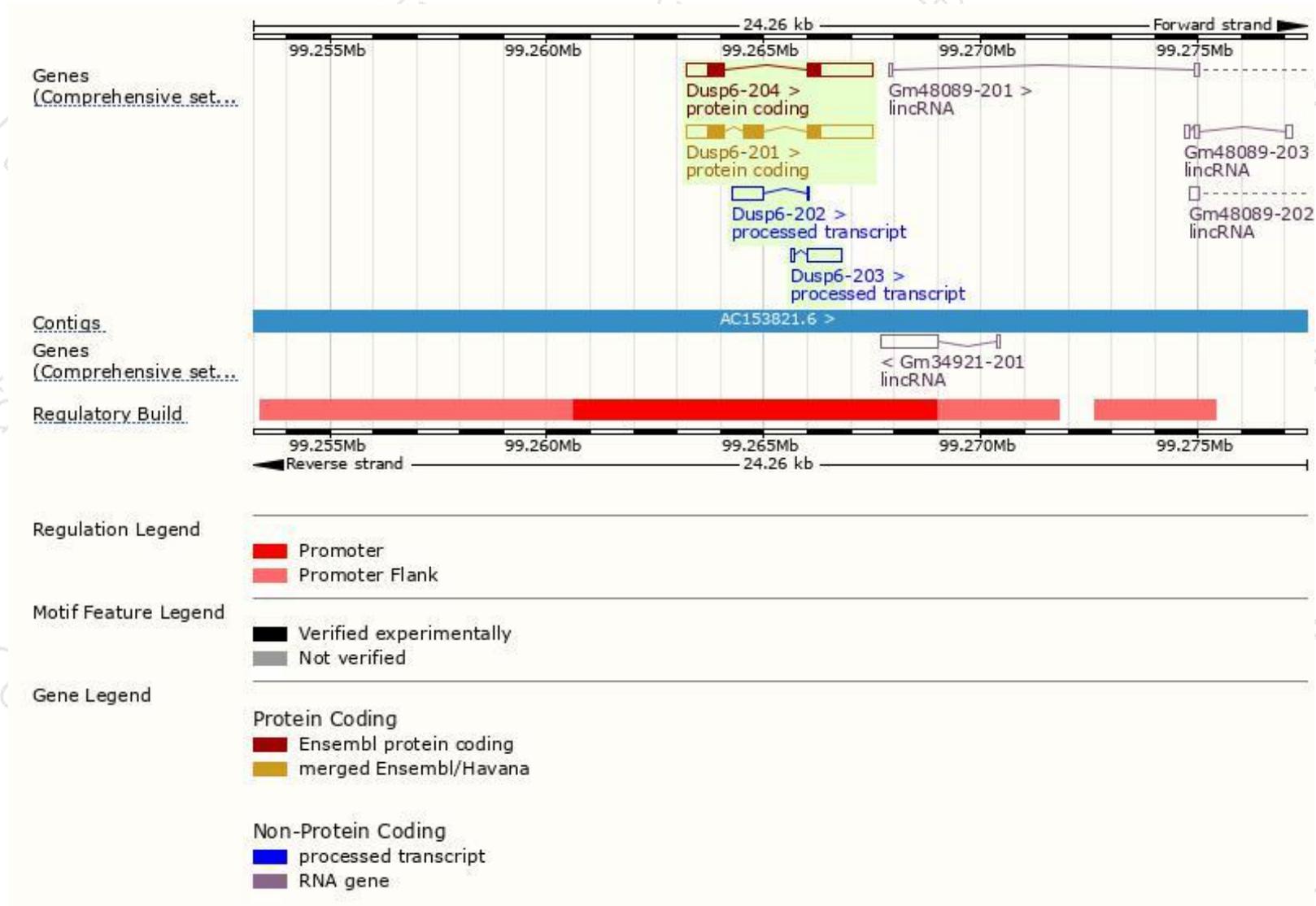
The gene has 4 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Dusp6-201	<a href="#">ENSMUST00000020118.4</a>	2796	<a href="#">381aa</a>	Protein coding	<a href="#">CCDS24147</a>	<a href="#">Q9DBB1</a>	TSL:1 GENCODE basic APPRIS P1
Dusp6-204	<a href="#">ENSMUST00000220291.1</a>	2359	<a href="#">235aa</a>	Protein coding	-	<a href="#">A0A1W2P715</a>	TSL:5 GENCODE basic
Dusp6-203	<a href="#">ENSMUST00000220218.1</a>	876	No protein	Processed transcript	-	-	TSL:2
Dusp6-202	<a href="#">ENSMUST00000219988.1</a>	758	No protein	Processed transcript	-	-	TSL:2

The strategy is based on the design of *Dusp6-201* transcript, The transcription is shown below



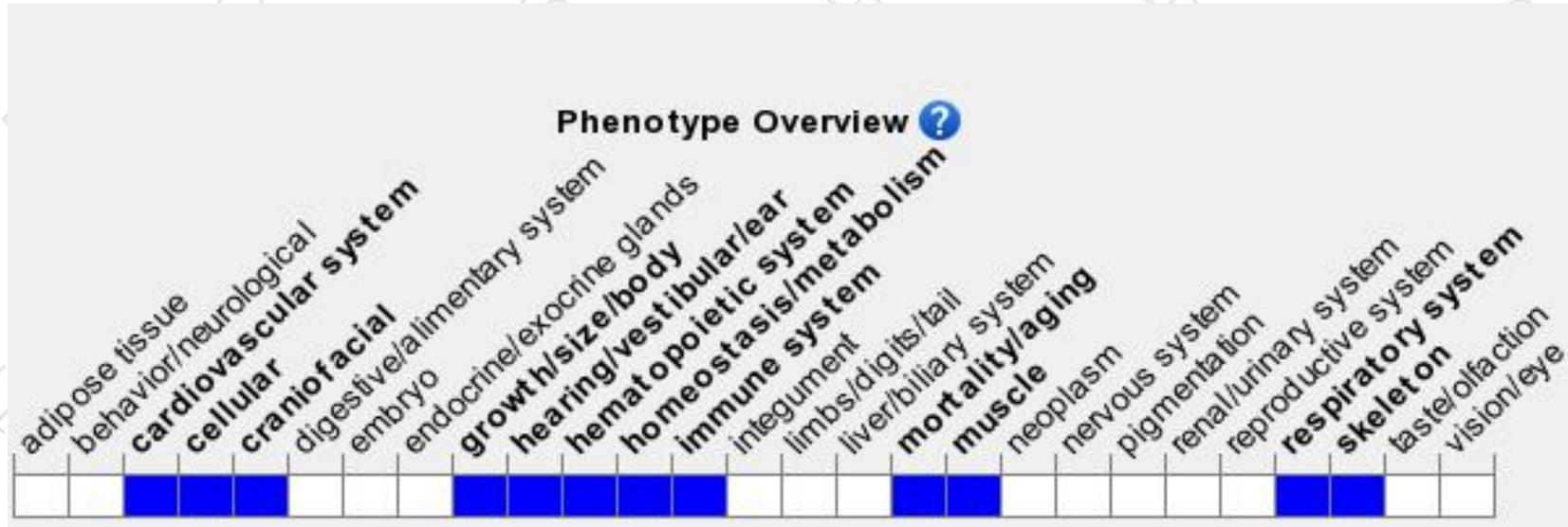
# Genomic location distribution



# 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 or heterozygous for a null mutation display partial penetrance of postnatal lethality, reduced body weight, and abnormal growth plate morphology.

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

Tel: 400-9660890

