

# *Gpr55* Cas9-CKO Strategy

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# Project Overview

**Project Name**

***Gpr55***

**Project type**

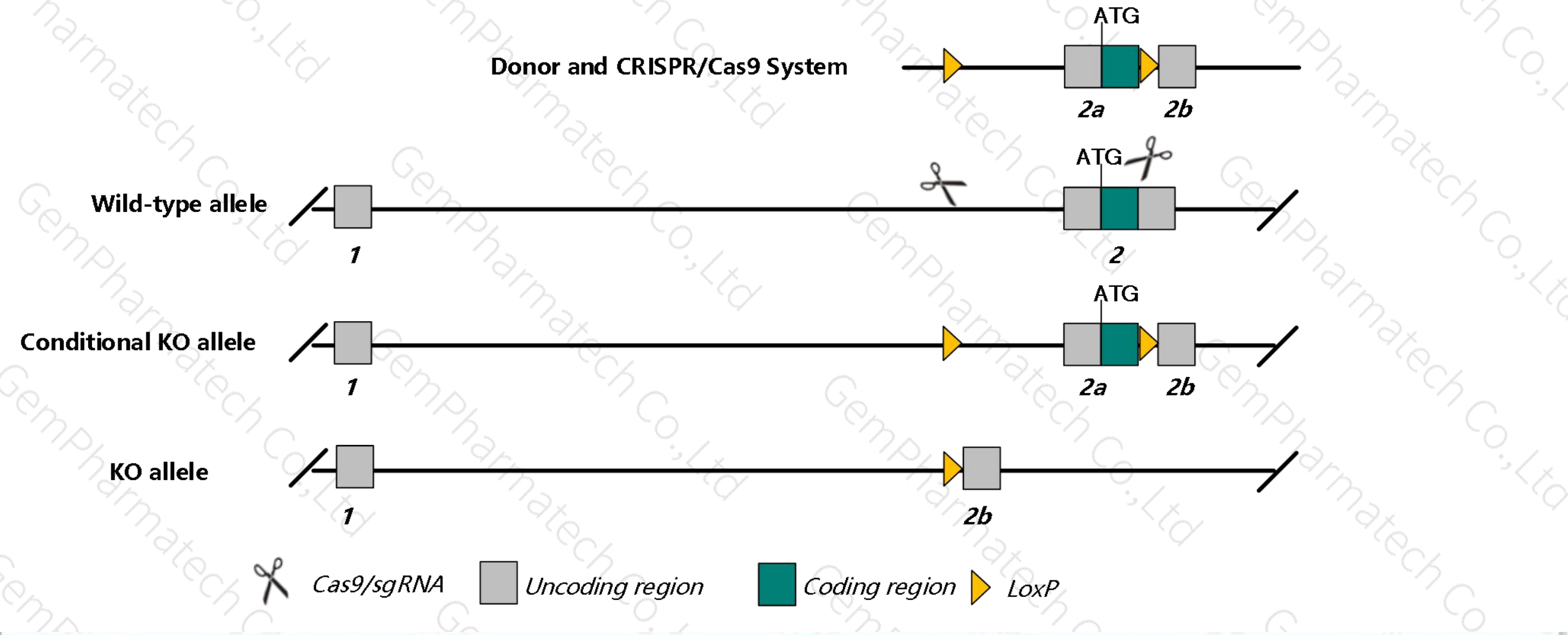
**Cas9-CKO**

**Strain background**

**C57BL/6JGpt**

# Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Gpr55* gene. The schematic diagram is as follows:



# Technical routes

- The *Gpr55* gene has 1 transcript. According to the structure of *Gpr55* gene, exon2 of *Gpr55-201* (ENSMUST00000086975.5) 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 *Gpr55* 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 for a knock-out allele exhibit increased bone volume due to impaired osteoclast function in male mice. Female mice exhibit a milder phenotype.
- The floxed region is near to the N-terminal of *Gm28884* gene and C-terminal of *4933407L21Rik* gene, this strategy may influence the regulatory function of the N-terminal of *Gm28884* gene and C-terminal of *4933407L21Rik* gene.
- There will be 1 to 2 amino acid synonymous mutation in exon2 of *Gpr55* gene in this strategy.
- The *Gpr55* gene is located on the Chr1. 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)

## Gpr55 G protein-coupled receptor 55 [ *Mus musculus* (house mouse) ]

Gene ID: 227326, updated on 8-Oct-2019

### Summary

Official Symbol	Gpr55 provided by <a href="#">MGI</a>
Official Full Name	G protein-coupled receptor 55 provided by <a href="#">MGI</a>
Primary source	<a href="#">MGI:MGI:2685064</a>
See related	<a href="#">Ensembl:ENSMUSG00000049608</a>
Gene type	protein coding
RefSeq status	VALIDATED
Organism	<a href="#">Mus musculus</a>
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	CTFL; Gm218; Lpir1
Expression	Broad expression in testis adult (RPKM 1.4), small intestine adult (RPKM 1.0) and 15 other tissues <a href="#">See more</a>
Orthologs	<a href="#">human</a> <a href="#">all</a>

### Genomic context

Location: 1; 1 C5

Exon count: 9

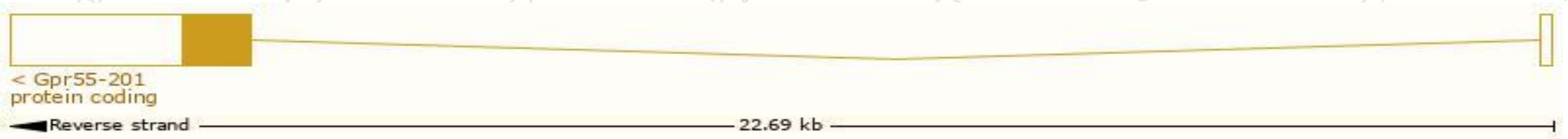
See Gpr55 in [Genome Data Viewer](#)

# Transcript information (Ensembl)

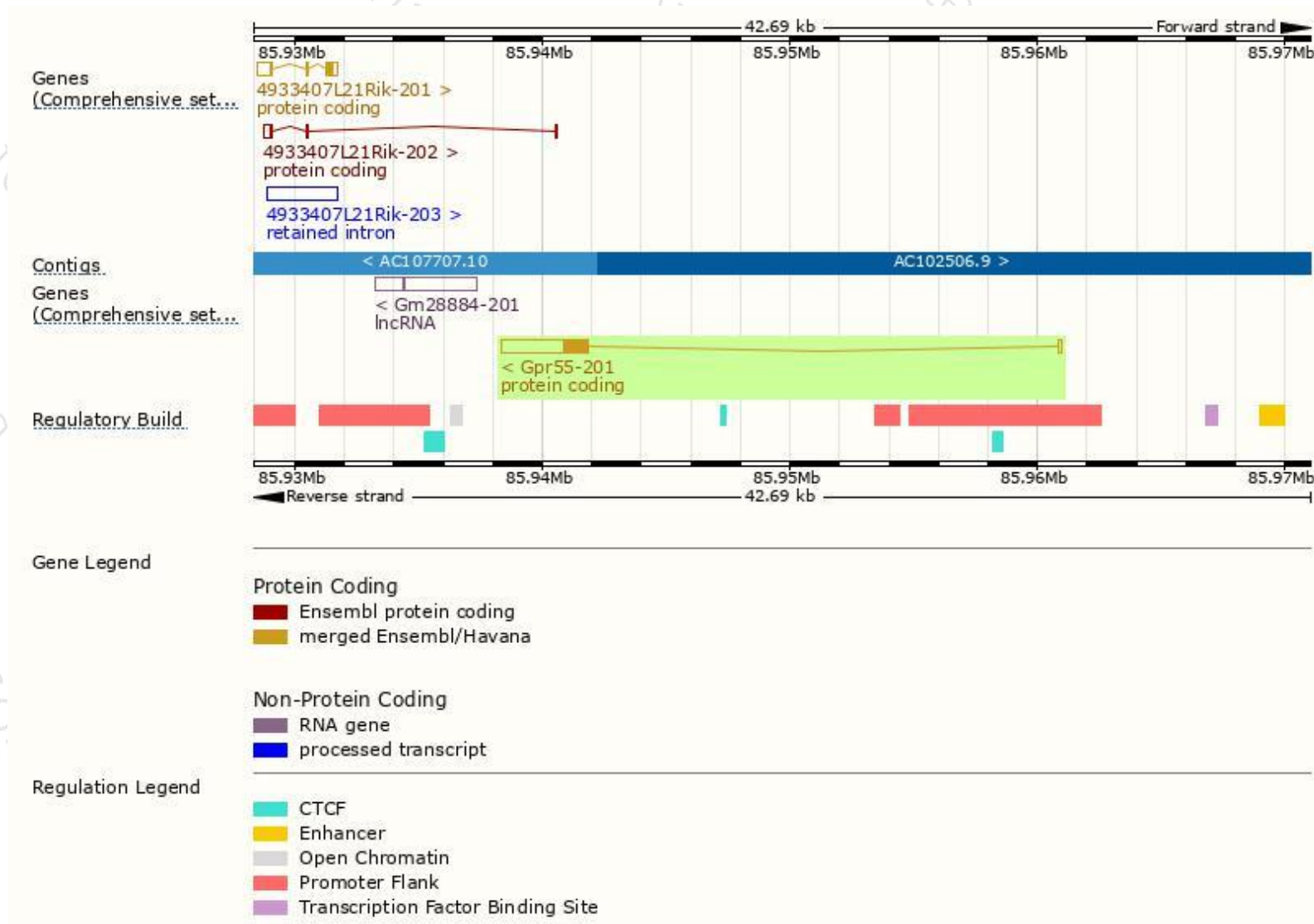
The gene has 1 transcript, and the transcript is shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Gpr55-201	<a href="#">ENSMUST00000086975.5</a>	3721	<a href="#">327aa</a>	Protein coding	<a href="#">CCDS15115</a>	<a href="#">Q3UJF0</a>	TSL:1 GENCODE basic APPRIS P1

The strategy is based on the design of *Gpr55-201* transcript, The transcription is shown below



# Genomic location distribution

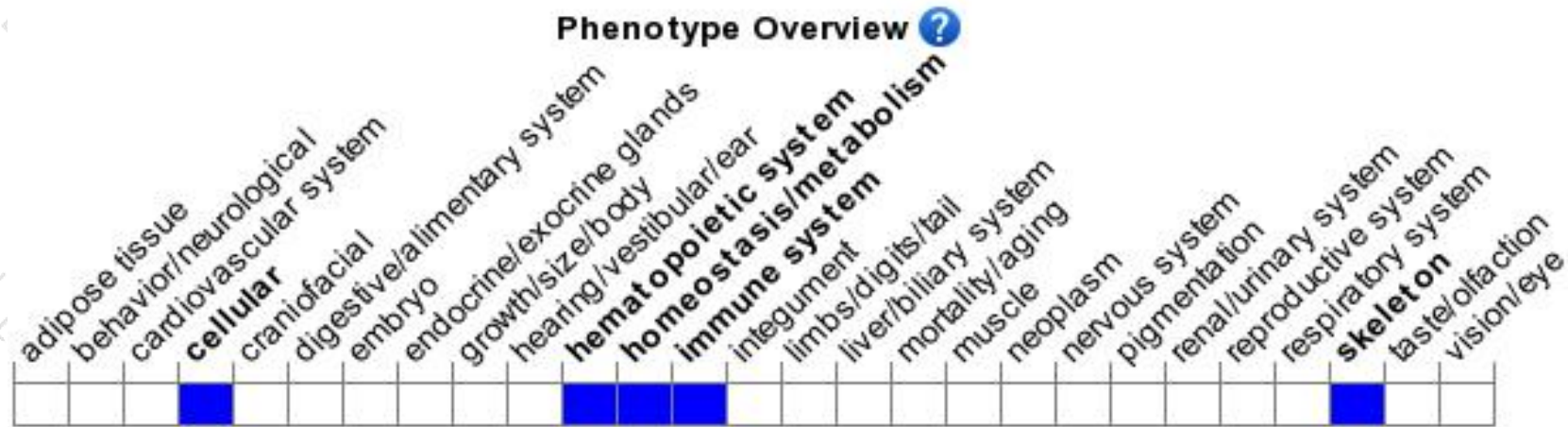




# Protein domain



# 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 for a knock-out allele exhibit increased bone volume due to impaired osteoclast function in male mice. Female mice exhibit a milder phenotype.

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

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