

# *Agtr2* Cas9-CKO Strategy

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# Project Overview

**Project Name**

*Agtr2*

**Project type**

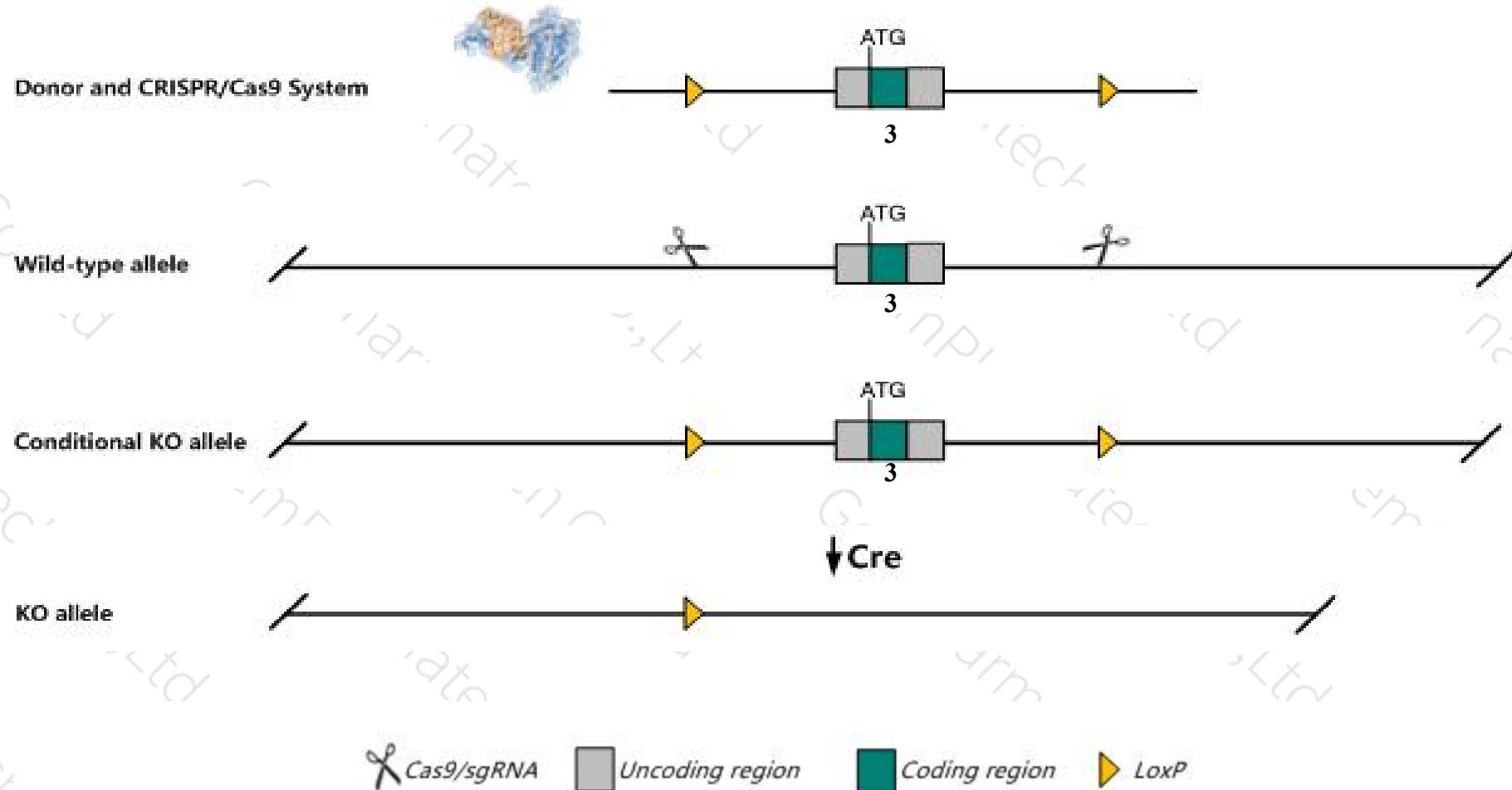
**Cas9-CKO**

**Strain background**

**C57BL/6JGpt**

# Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Agtr2* gene. The schematic diagram is as follows:



- The *Agtr2* gene has 2 transcripts. According to the structure of *Agtr2* gene, exon3 of *Agtr2-201* (ENSMUST00000089188.8) 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 *Agtr2* gene. The brief process is as follows: sgRNA was transcribed in vitro, donor vector was constructed. Cas9, sgRNA 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 was 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 may exhibit abnormal response to angiotensin II, induced pancreatitis, and myocardial infarction; cardiovascular morphology and physiology; renal and urinary morphology and physiology; and glucose and lipid homeostasis.
- The *Agtr2* gene is located on the ChrX. 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)

## Agtr2 angiotensin II receptor, type 2 [ *Mus musculus* (house mouse) ]

Gene ID: 11609, updated on 23-Apr-2019

### Summary

**Official Symbol** Agtr2 provided by [MGI](#)  
**Official Full Name** angiotensin II receptor, type 2 provided by [MGI](#)  
**Primary source** [MGI:MGI:87966](#)  
**See related** [Ensembl:ENSMUSG00000068122](#)  
**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** AI316812; AW107640  
**Expression** Biased expression in limb E14.5 (RPKM 70.4) and CNS E14 (RPKM 3.9) [See more](#)  
**Orthologs** [human](#) [all](#)

### Genomic context

Location: X A2; X 16.71 cM

See Agtr2 in [Genome Data Viewer](#)

Exon count: 3

Annotation release	Status	Assembly	Chr	Location
<a href="#">106</a>	current	GRCm38.p4 ( <a href="#">GCF_000001635.24</a> )	X	NC_000086.7 (21484549..21488833)
Build 37.2	previous assembly	MGSCv37 ( <a href="#">GCF_000001635.18</a> )	X	NC_000086.6 (21061752..21065957)

# Transcript information (Ensembl)

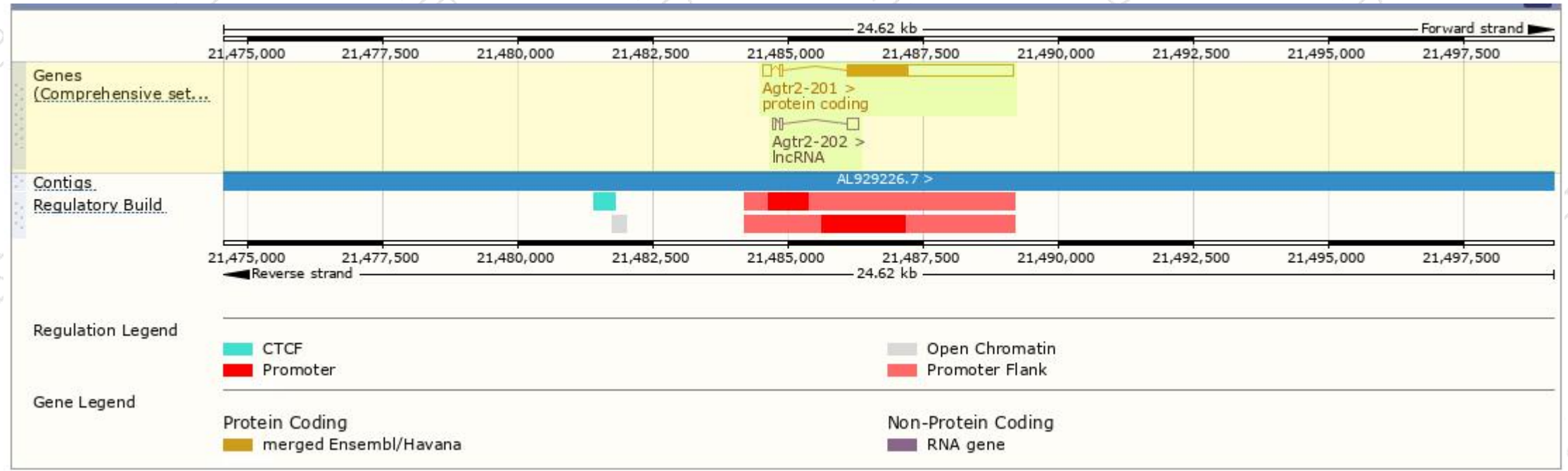
The gene has 2 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Agtr2-201	<a href="#">ENSMUST00000089188.8</a>	3283	<a href="#">363aa</a>	Protein coding	<a href="#">CCDS40889</a>	<a href="#">P35374</a>	TSL:1 GENCODE basic APPRIS P1
Agtr2-202	<a href="#">ENSMUST00000131150.1</a>	314	No protein	lncRNA	-	-	TSL:3

The strategy is based on the design of *Agtr2-201* transcript,The transcription is shown below

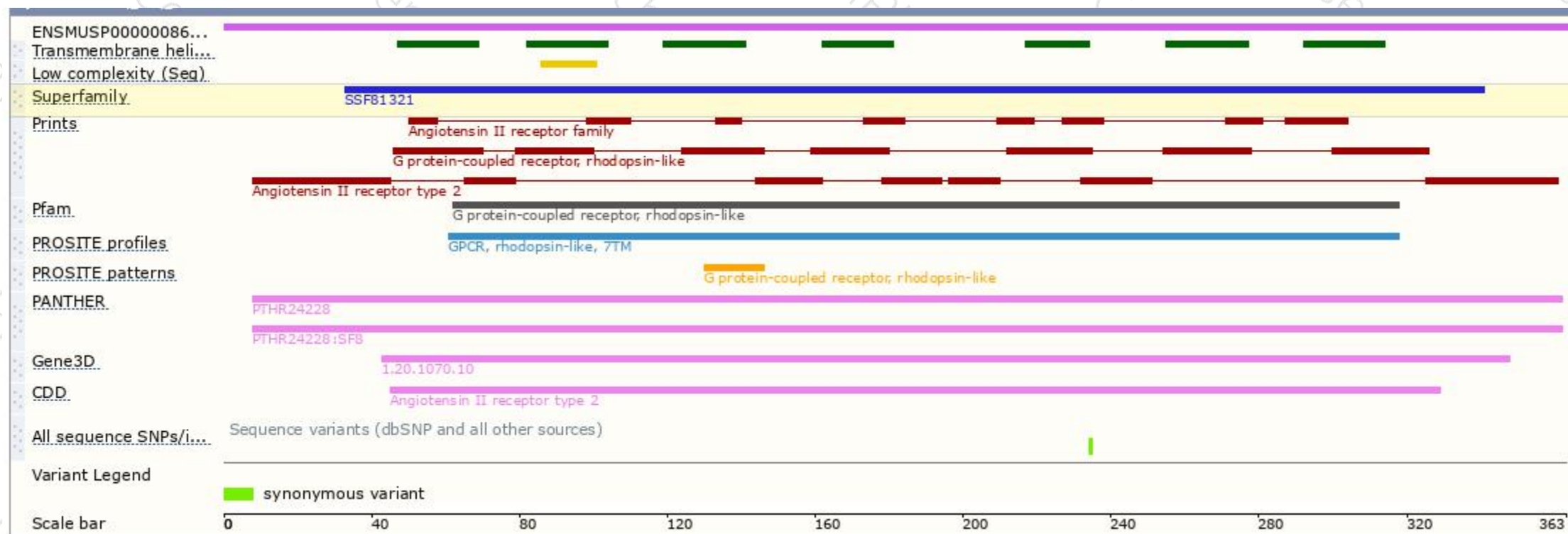


# Genomic location distribution

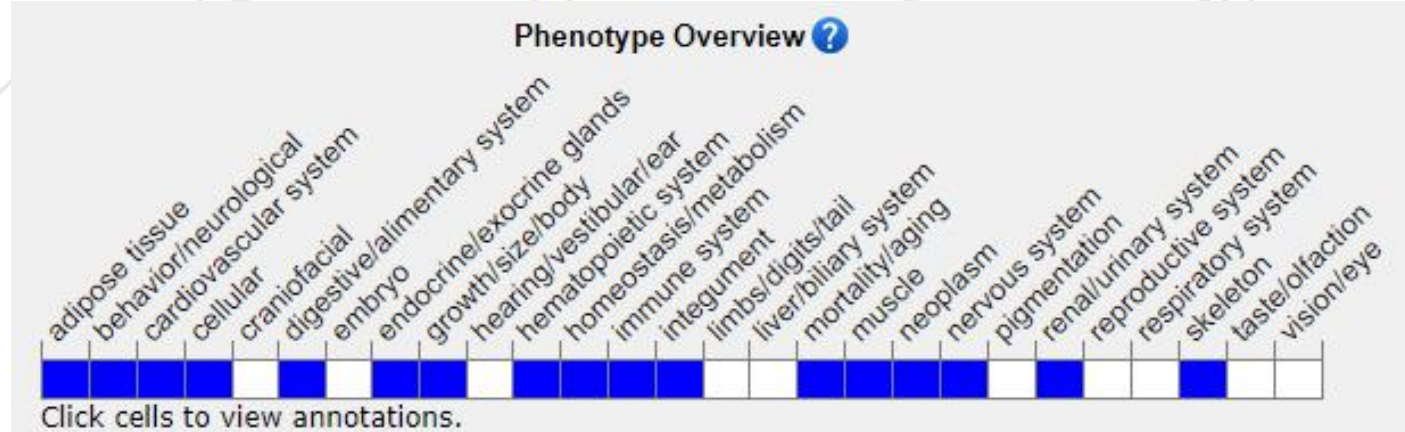




# Protein domain



# Mouse phenotype description(MGI )



*Phenotypes affected by the gene are marked in blue. Data quoted from MGI database(<http://www.informatics.jax.org/>).*

Mice homozygous for a knock-out allele may exhibit abnormal response to angiotensin II, induced pancreatitis, and myocardial infarction; cardiovascular morphology and physiology; renal and urinary morphology and physiology; and glucose and lipid homeostasis.

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

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