

# Bicc1 Cas9-CKO Strategy

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Reviewer: Huimin Su

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# **Project Overview**



**Project Name** 

Bicc1

**Project type** 

Cas9-CKO

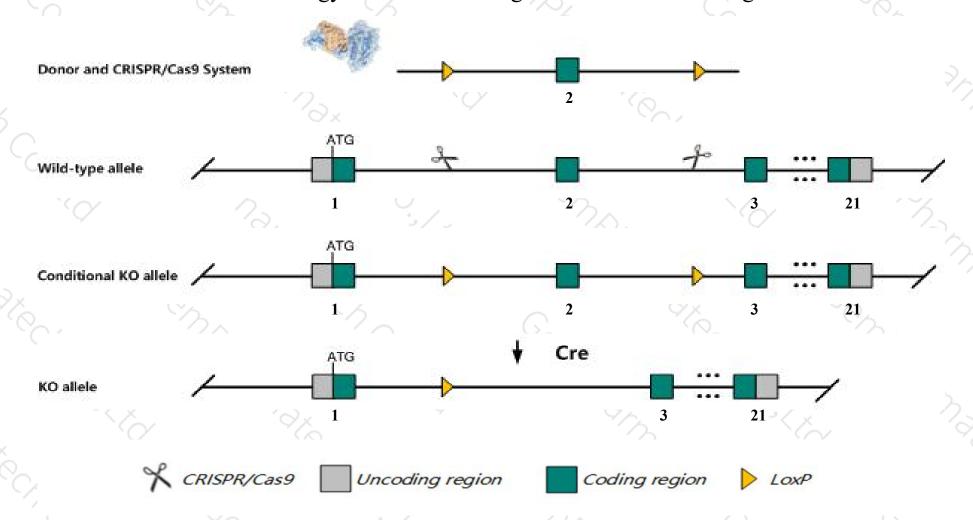
Strain background

C57BL/6JGpt

## Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Bicc1* gene. The schematic diagram is as follows:



### Technical routes



- The *Bicc1* gene has 4 transcripts. According to the structure of *Bicc1* gene, exon2 of *Bicc1-203*(ENSMUST00000143791.7) transcript is recommended as the knockout region. The region contains 47bp coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Bicc1* 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.

### **Notice**



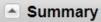
- > According to the existing MGI data, homozygous inactivation of this gene causes heteroxia, impaired nodal flow, ventricular septal defects, partial prenatal lethality and postnatal death due to renal failure. Chemically induced mutants develop kidney cysts and may show bulging abdomens, bile duct anomalies and cardiovascular defects.
- > The *Bicc1* 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)



#### Bicc1 BicC family RNA binding protein 1 [ Mus musculus (house mouse) ]

Gene ID: 83675, updated on 21-Jul-2020



☆ ?

Official Symbol Bicc1 provided by MGI

Official Full Name BicC family RNA binding protein 1 provided by MGI

Primary source MGI:MGI:1933388

See related Ensembl: ENSMUSG00000014329

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 bpk; jcpk; Bic-C

Expression Broad expression in kidney adult (RPKM 21.7), ovary adult (RPKM 11.9) and 18 other tissues See more

Orthologs <u>human</u> all

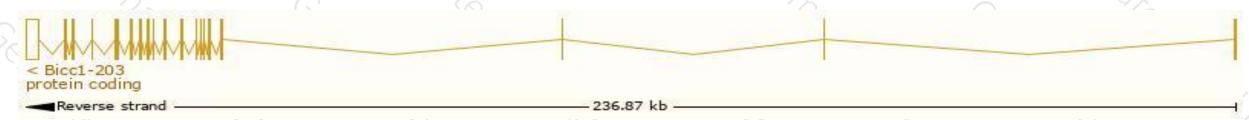
# Transcript information (Ensembl)



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

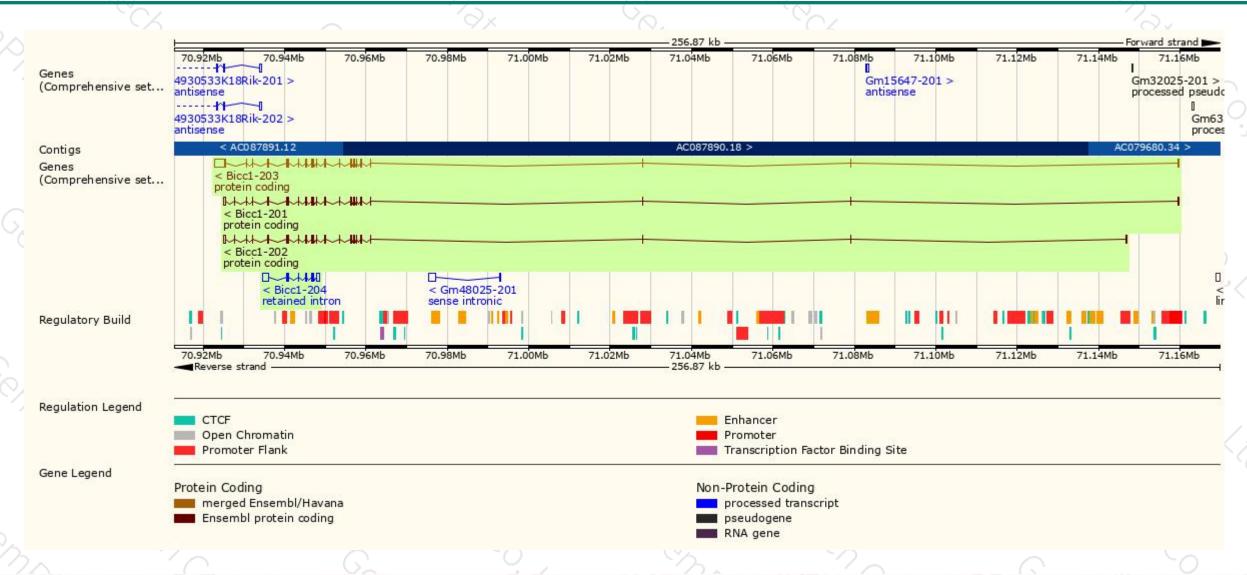
Name	Transcript ID	bp 🛊	Protein	Biotype A	CCDS 🍦	UniProt 🍦	Flags			
Bicc1-204	ENSMUST00000144740.1	3053	No protein	Retained intron			TSL:1			
Bicc1-203	ENSMUST00000143791.7	5431	<u>977aa</u>	Protein coding	CCDS23915 ₽	Q99MQ1@	TSL:1 GENCODE basic APPR		APPRIS P1	
Bicc1-201	ENSMUST00000014473.5	3231	<u>951aa</u>	Protein coding	CCDS83710 ₽	<u>G3X8S6</u> ₺	TSL:1 GENCODE basic		basic	
Bicc1-202	ENSMUST00000131445.7	3005	869aa	Protein coding	120	Q99MQ1 ₽		TSL:1	GENCODE	basic

The strategy is based on the design of *Bicc1-203* transcript, the transcription is shown below:



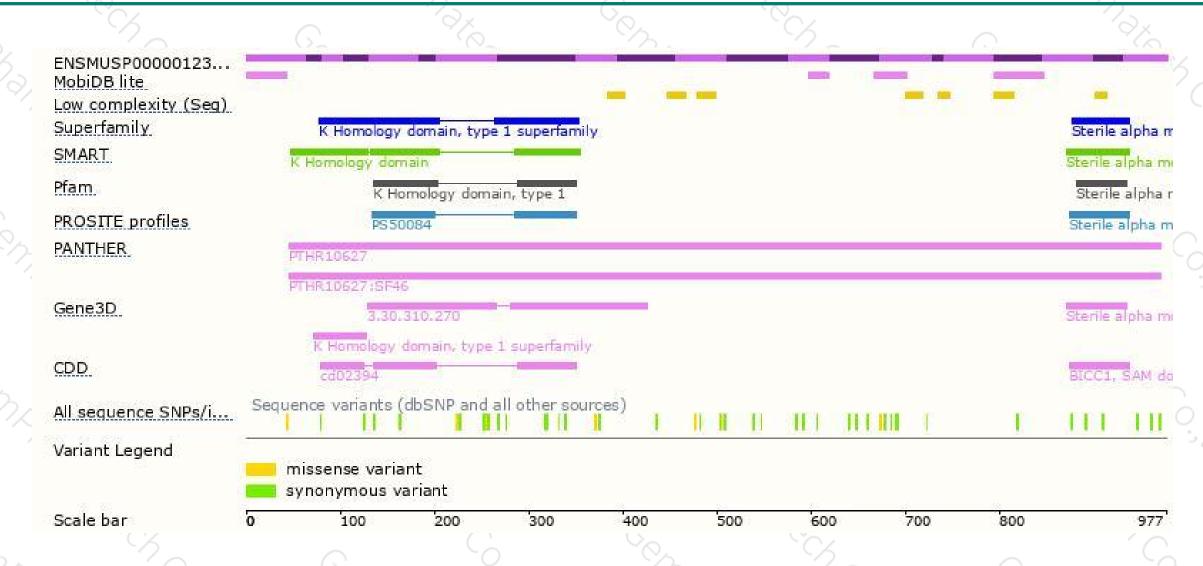
### Genomic location distribution





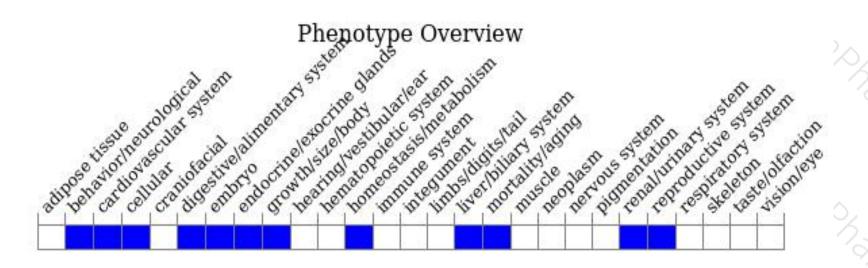
### Protein domain





# 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, homozygous inactivation of this gene causes heteroxia, impaired nodal flow, ventricular septal defects, partial prenatal lethality and postnatal death due to renal failure. Chemically induced mutants develop kidney cysts and may show bulging abdomens, bile duct anomalies and cardiovascular defects.



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





