

# Cckbr Cas9-KO Strategy

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**Reviewer:** 

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## **Project Overview**



**Project Name** 

Project type Cas9-KO

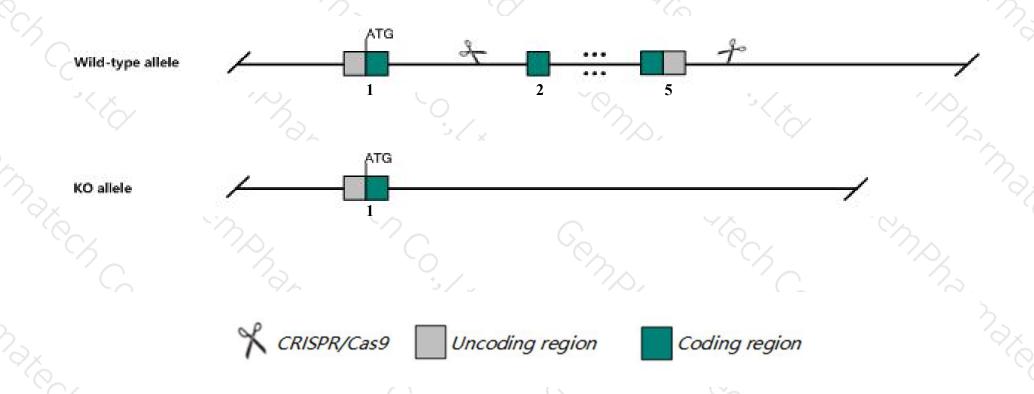
Strain background C57BL/6JGpt

Cckbr

## **Knockout strategy**



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



## **Technical routes**



- ➤ The *Cckbr* gene has 2 transcripts. According to the structure of *Cckbr* gene, exon2-exon5 of *Cckbr-201* (ENSMUST00000033189.5) transcript is recommended as the knockout region. The region contains 1211bp coding sequence Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Cckbr* gene. The brief process is as follows: CRISPR/Cas9 system

### **Notice**



- ➤ According to the existing MGI data, Nullizygous mice show gastic mucoca defects, high gastic pH and hypergastrenemia. Homozygotes for a null allele also exhibit higher energy intake and expenditure, less susceptibility to endotoxin shock, altered pain and mechanical sensitivity, and behavioral changes to isolation and addictive drugs.
- The *Cckbr* gene is located on the Chr7. 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 the gene knockout on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

## Gene information (NCBI)



#### Cckbr cholecystokinin B receptor [Mus musculus (house mouse)]

Gene ID: 12426, updated on 3-Feb-2019

#### Summary

☆ ?

Official Symbol Cckbr provided by MGI

Official Full Name cholecystokinin B receptor provided by MGI

Primary source MGI:MGI:99479

See related Ensembl: ENSMUSG00000030898

Gene type protein coding
RefSeq status REVIEWED
Organism Mus musculus

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha;

Muroidea; Muridae; Murinae; Mus; Mus

Also known as CCK-BR, CCK2-R, CCK2R, CCKR-2

Summary This gene encodes a multipass transmembrane receptor protein expressed in the central nervous system and gastrointestinal tract.

Cholecystokinin and gastrin bind to the encoded protein to stimulate gastric acid secretion and mucosal growth in the gastrointestinal tract, and anxiety, pain sensation and memory in the brain. Mice lacking the encoded protein exhibit an increase in the basal gastric pH and gastrin levels in the bloodstream as well as mild hypocalcemia, secondary hyperparathyroidism and increased bone resorption. [provided

by RefSeq, Apr 2015]

Expression Biased expression in cortex adult (RPKM 23.1), frontal lobe adult (RPKM 18.4) and 3 other tissuesSee more

Orthologs <u>human</u> all

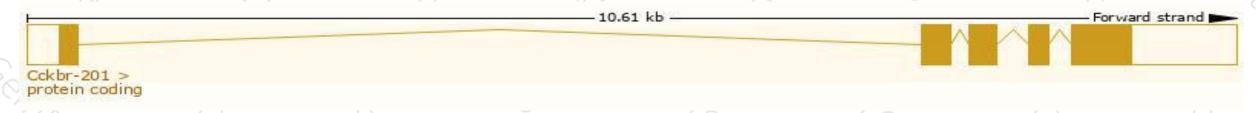
## Transcript information (Ensembl)



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

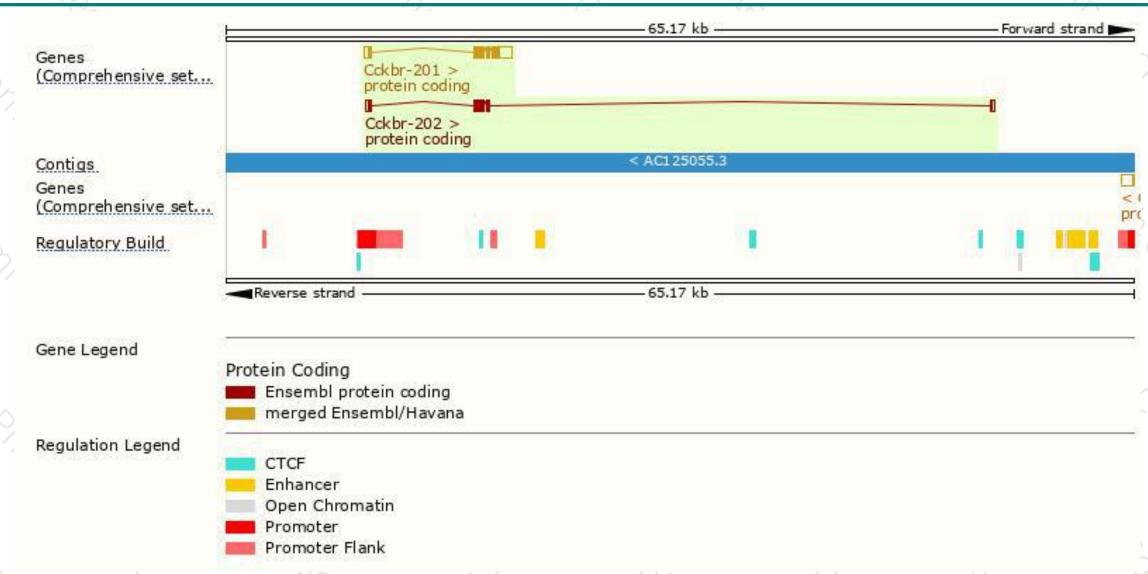
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Cckbr-201	ENSMUST00000033189.5	2573	<u>453aa</u>	Protein coding	CCDS21651	P56481 Q3ZB46	TSL:1 GENCODE basic APPRIS P1
Cckbr-202	ENSMUST00000181339.7	1326	314aa	Protein coding	<del>.</del>	Q8BYG7	TSL:1 GENCODE basic

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



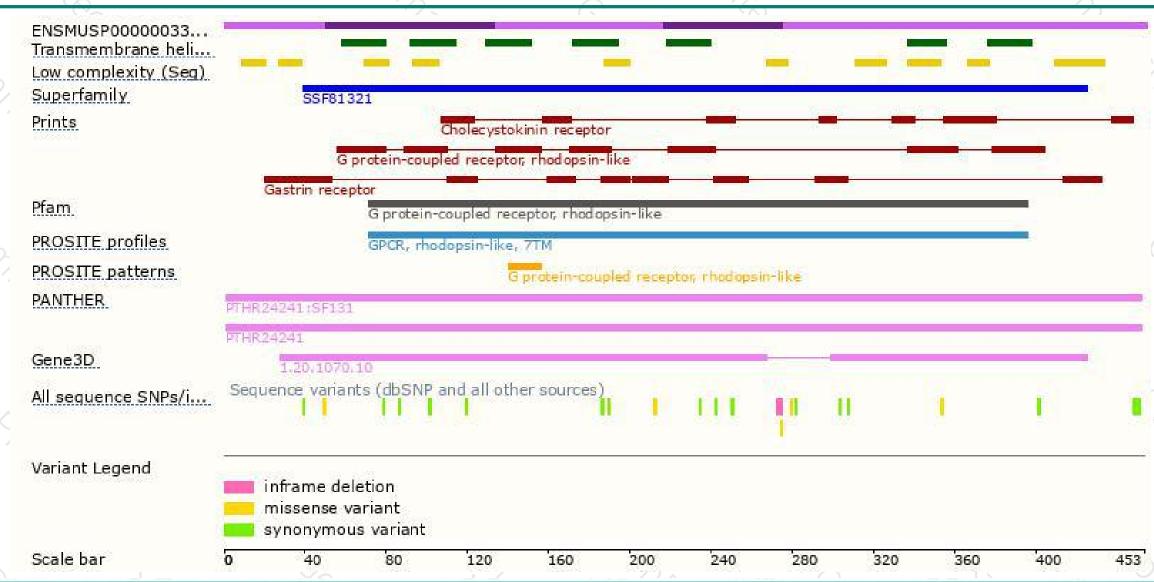
### Genomic location distribution





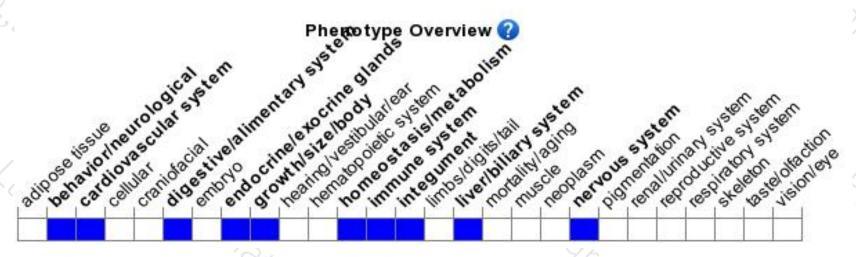
### Protein domain





## 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, Nullizygous mice show gastic mucoca defects, high gastic pH and hypergastrenemia. Homozygotes for a null allele also exhibit higher energy intake and expenditure, less susceptibility to endotoxin shock, altered pain and mechanical sensitivity, and behavioral changes to isolation and addictive drugs.



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





