



Ocln Cas9-CKO Strategy

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Reviewer:

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Design Date:

2019-11-14

Project Overview

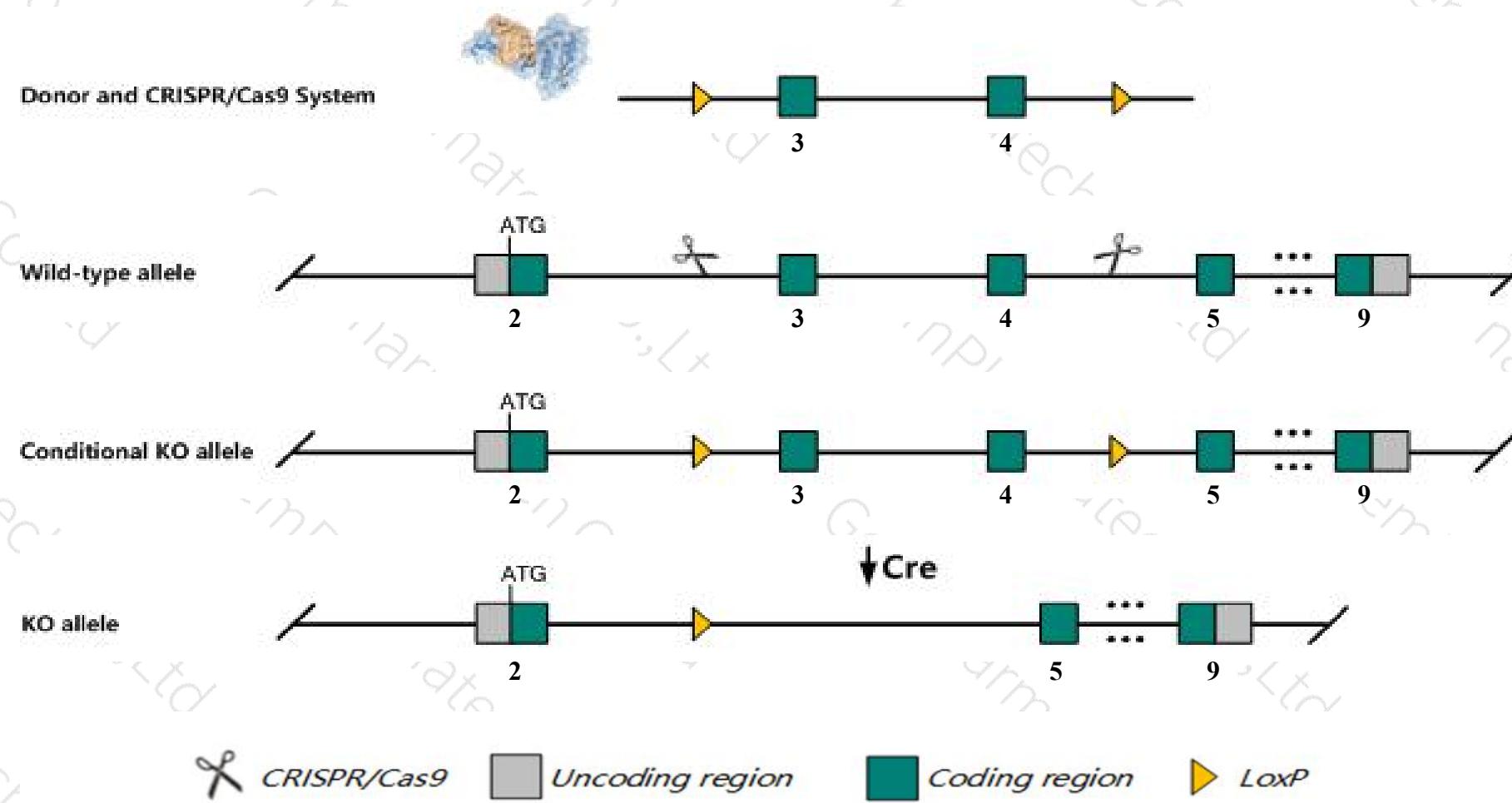
Project Name***Ocln***

Project type**Cas9-CKO**

Strain background**C57BL/6JGpt**

Conditional Knockout strategy

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



Technical routes

- The *Ocln* gene has 5 transcripts. According to the structure of *Ocln* gene, exon3-exon4 of *Ocln*-202 (ENSMUST00000069756.10) transcript is recommended as the knockout region. The region contains 835bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Ocln* 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.



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Notice

- According to the existing MGI data, Homozygous null mice display gastritis, loss of gastric parietal and chief cells, gastric mucus cell hyperplasia, reduced gastric acid secretion, growth retardation, male infertility, seminiferous tubule atrophy, failure to nurse pups, mineral deposits in the brain, and thinning of the compact bone.
- The *Ocln* gene is located on the Chr13. 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)

Ocln occludin [Mus musculus (house mouse)]

Gene ID: 18260, updated on 19-Mar-2019

Summary



Official Symbol	Ocln provided by MGI
Official Full Name	occludin provided by MGI
Primary source	MGI:MGI:106183
See related	Ensembl:ENSMUSG00000021638
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	AI503564, Ocl
Expression	Broad expression in large intestine adult (RPKM 9.2), colon adult (RPKM 5.6) and 22 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

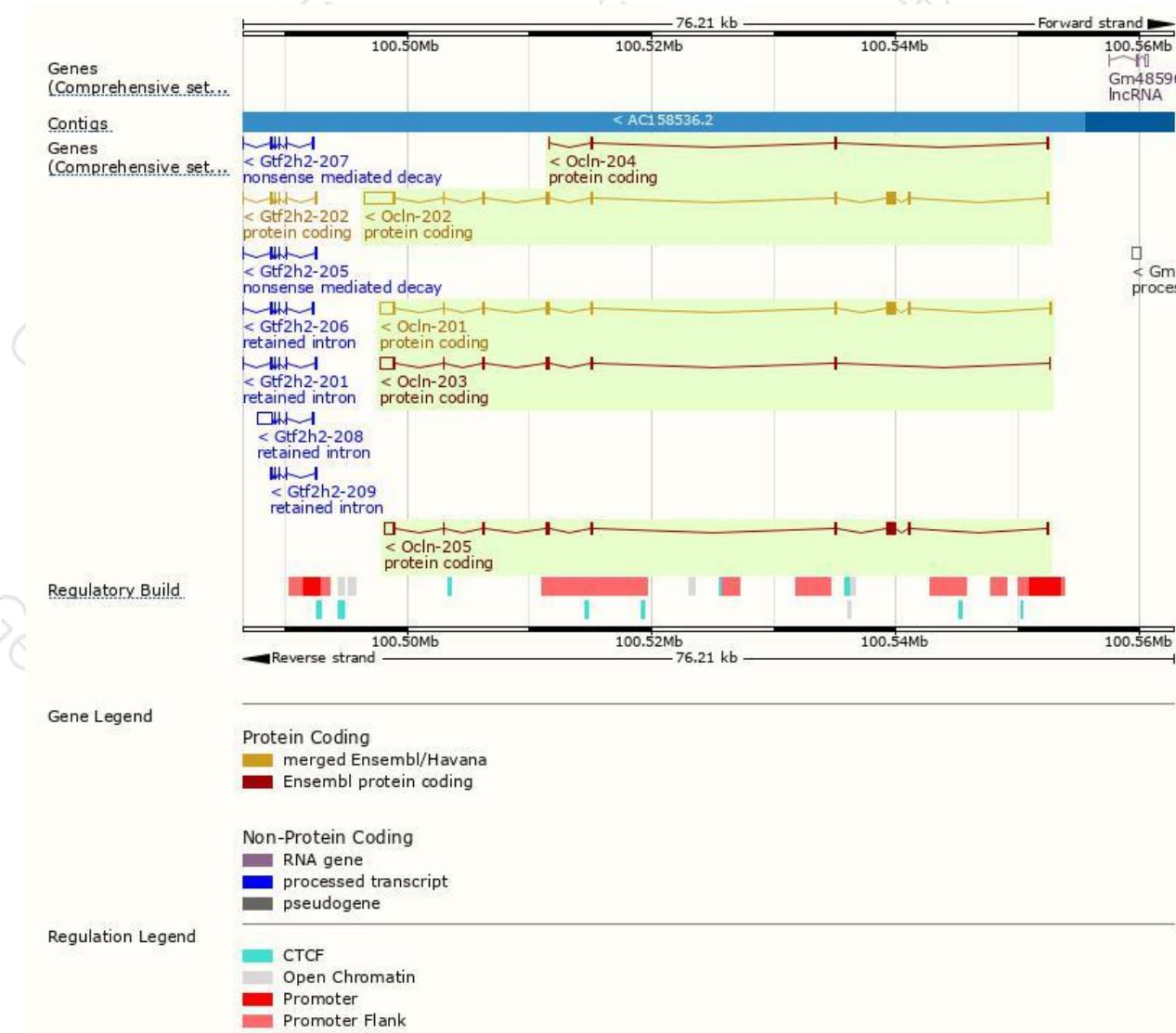
The gene has 5 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Ocln-202	ENSMUST00000069756.10	4052	521aa	Protein coding	CCDS26731	B2RS24 Q61146	TSL:1 GENCODE basic APPRIS P1
Ocln-201	ENSMUST00000022140.11	2839	521aa	Protein coding	CCDS26731	B2RS24 Q61146	TSL:1 GENCODE basic APPRIS P1
Ocln-205	ENSMUST00000160859.7	2443	521aa	Protein coding	CCDS26731	B2RS24 Q61146	TSL:1 GENCODE basic APPRIS P1
Ocln-203	ENSMUST00000159459.7	1950	272aa	Protein coding	-	E0CZ73	TSL:1 GENCODE basic
Ocln-204	ENSMUST00000159515.1	390	107aa	Protein coding	-	E0CZ91	CDS 3' incomplete TSL:5

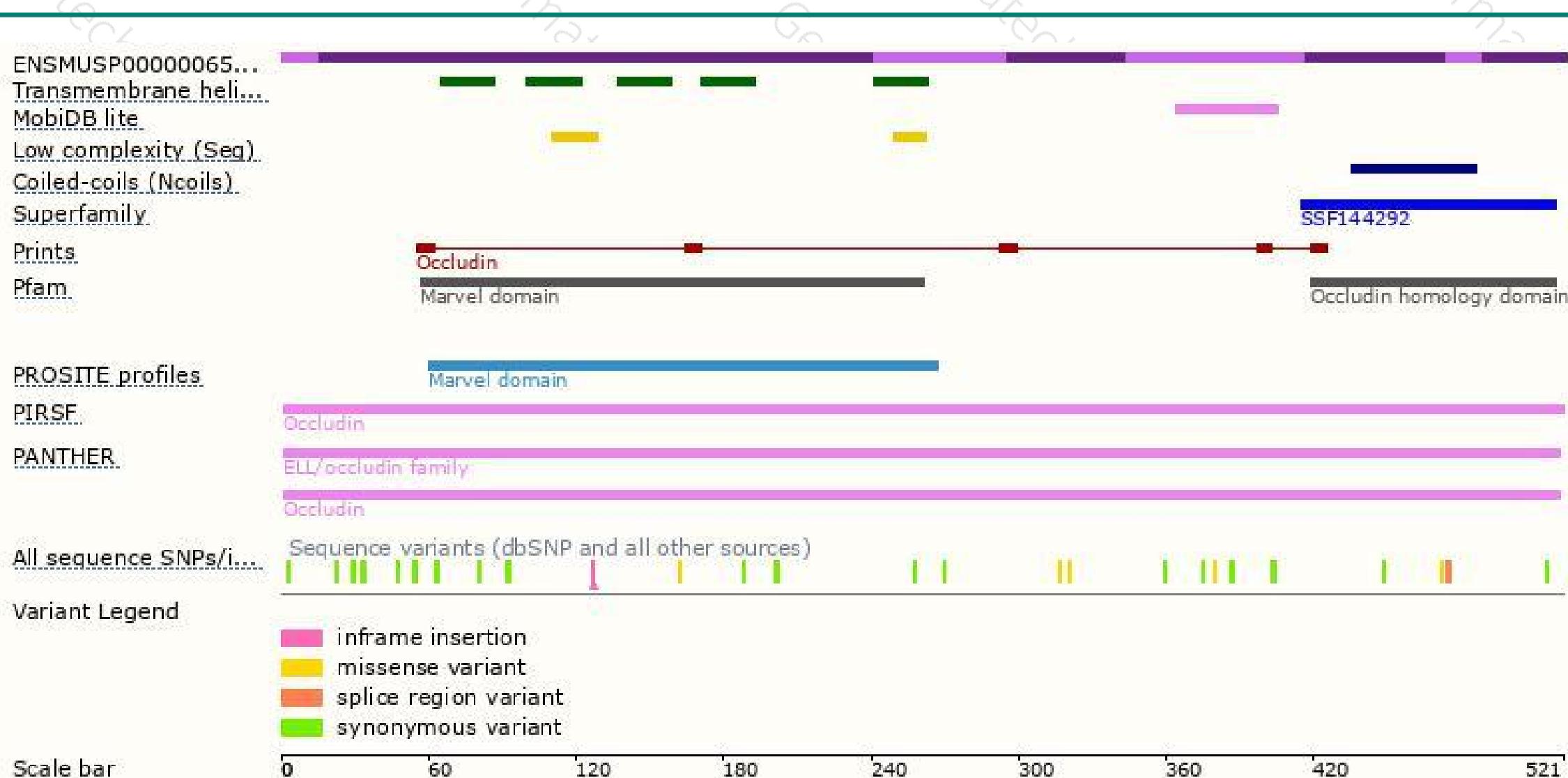
The strategy is based on the design of *Ocln-202* transcript, The transcription is shown below



Genomic location distribution



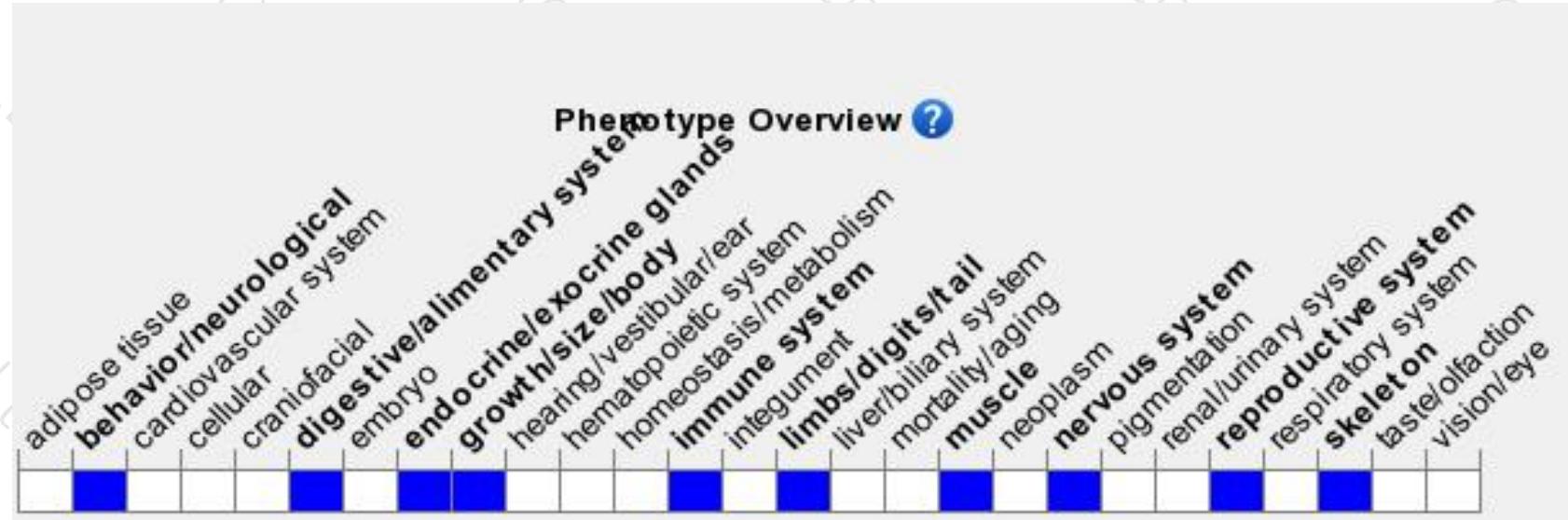
Protein domain





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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 null mice display gastritis, loss of gastric parietal and chief cells, gastric mucus cell hyperplasia, reduced gastric acid secretion, growth retardation, male infertility, seminiferous tubule atrophy, failure to nurse pups, mineral deposits in the brain, and thinning of the compact bone.



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

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