



Snx10 Cas9-CKO Strategy

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Project Overview

Project Name***Snx10***

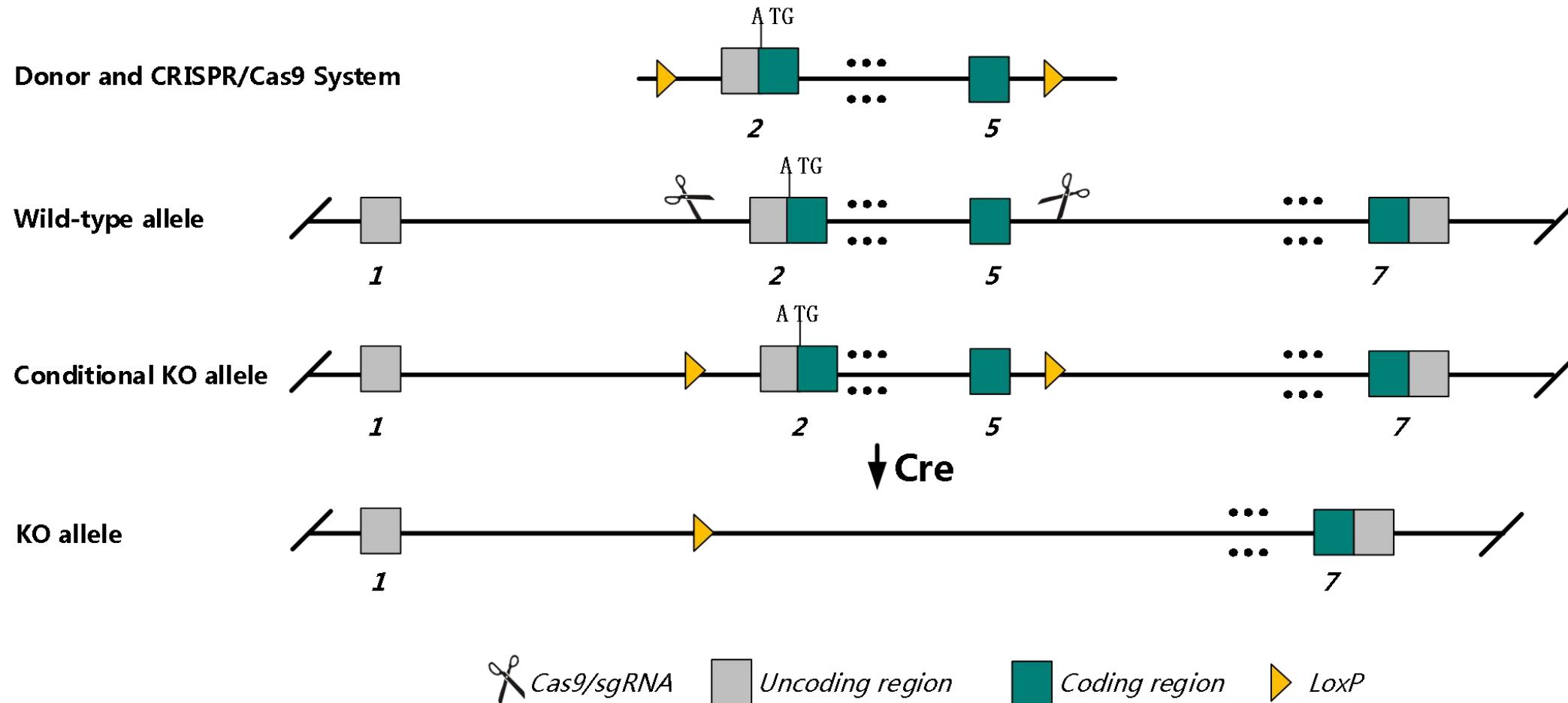
Project type**Cas9-CKO**

Strain background**C57BL/6JGpt**

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Snx10* gene. The schematic diagram is as follows:



Technical routes

- The *Snx10* gene has 7 transcripts. According to the structure of *Snx10* gene, exon2-exon5 of *Snx10-201* (ENSMUST00000049152.14) transcript is recommended as the knockout region. The region contains start codon ATG. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Snx10* 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, Mice homozygous for a hypomorphic allele show postnatal growth retardation, failure of tooth eruption, impaired skeleton development, and osteopetrosis associated with failed osteoclast activity, high stomach pH, low calcium availability, impaired bone mineralization, and premature death.
- The *Snx10* gene is located on the Chr6. 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.



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Gene information (NCBI)

Snx10 sorting nexin 10 [Mus musculus (house mouse)]

Gene ID: 71982, updated on 31-Jan-2019

Summary



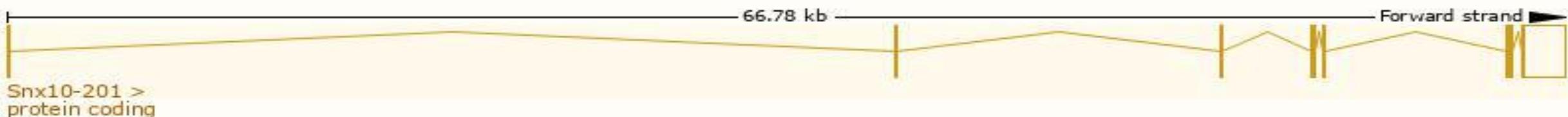
Official Symbol	Snx10 provided by MGI
Official Full Name	sorting nexin 10 provided by MGI
Primary source	MGI:MGI:1919232
See related	Ensembl:ENSMUSG00000038301
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	2410004M09Rik
Expression	Broad expression in frontal lobe adult (RPKM 11.5), cortex adult (RPKM 10.9) and 17 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

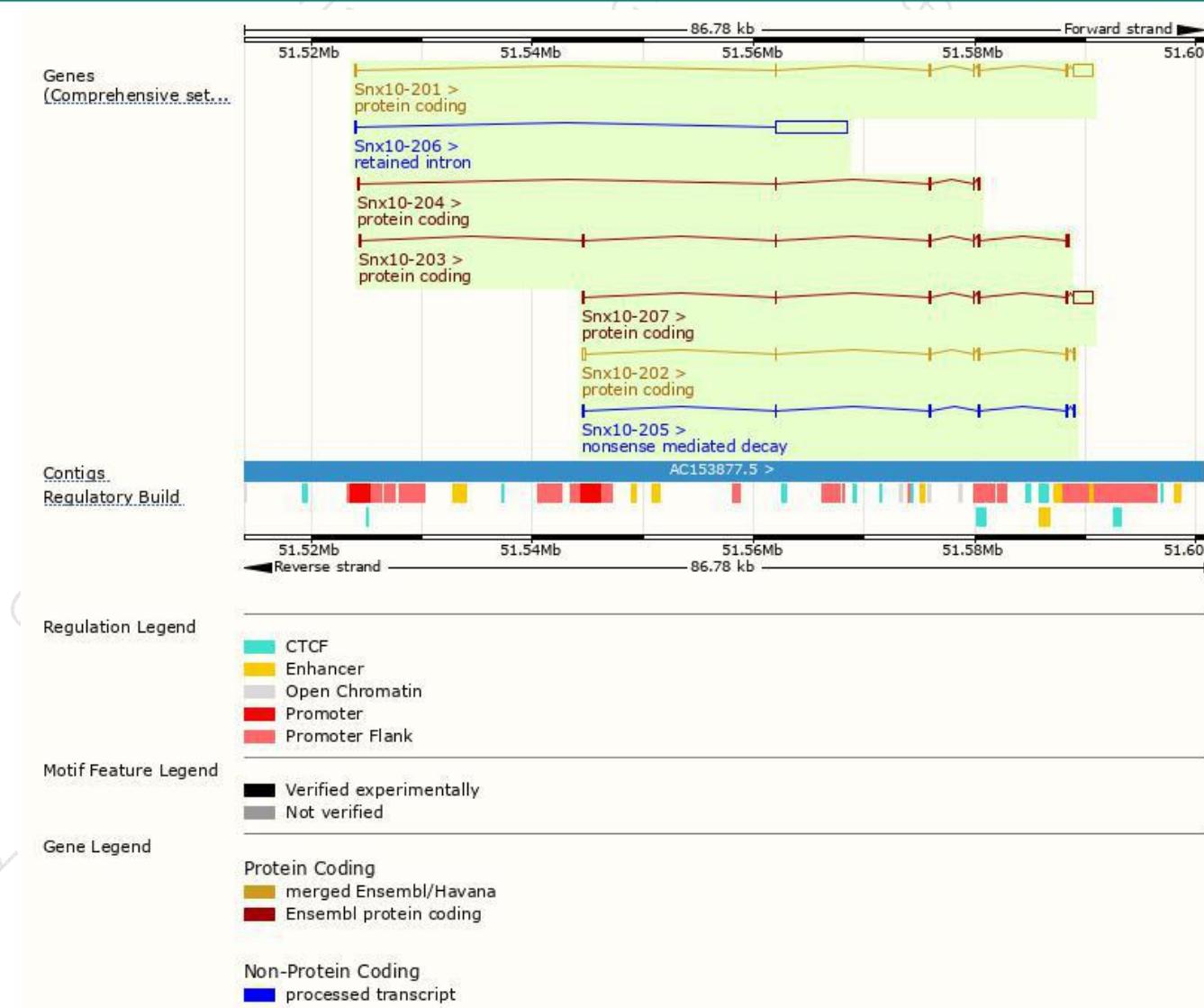
The gene has 7 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Snx10-201	ENSMUST00000049152.14	2480	201aa	Protein coding	CCDS20136	Q4FJX6 Q9CWT3	TSL:1 GENCODE basic APPRIS P1
Snx10-207	ENSMUST00000179365.7	2464	201aa	Protein coding	CCDS20136	Q4FJX6 Q9CWT3	TSL:1 GENCODE basic APPRIS P1
Snx10-202	ENSMUST00000114439.7	968	201aa	Protein coding	CCDS20136	Q4FJX6 Q9CWT3	TSL:1 GENCODE basic APPRIS P1
Snx10-203	ENSMUST00000137212.7	809	177aa	Protein coding	-	D3YW62	CDS 3' incomplete TSL:3
Snx10-204	ENSMUST00000140560.7	433	96aa	Protein coding	-	D3Z4P6	CDS 3' incomplete TSL:5
Snx10-205	ENSMUST00000149024.1	693	44aa	Nonsense mediated decay	-	S4R1A0	TSL:3
Snx10-206	ENSMUST00000155724.2	6512	No protein	Retained intron	-	-	TSL:1

The strategy is based on the design of *Snx10-201* transcript, The transcription is shown below



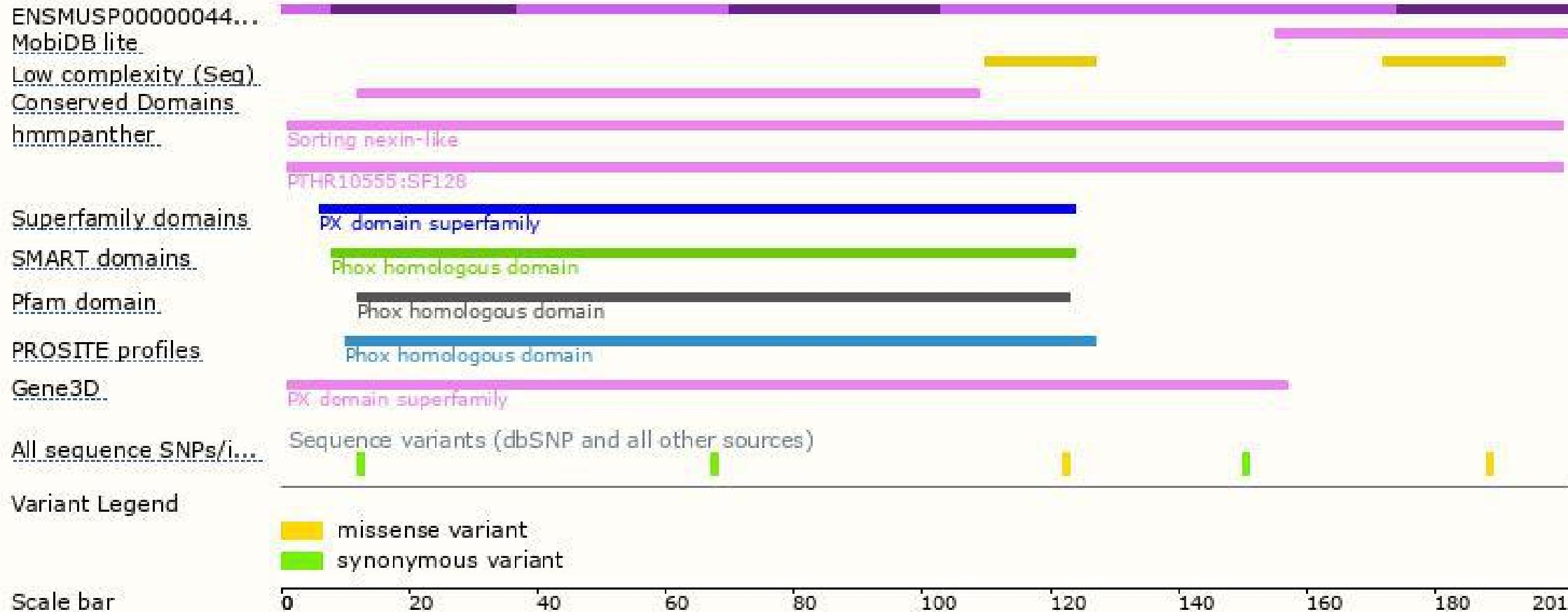
Genomic location distribution





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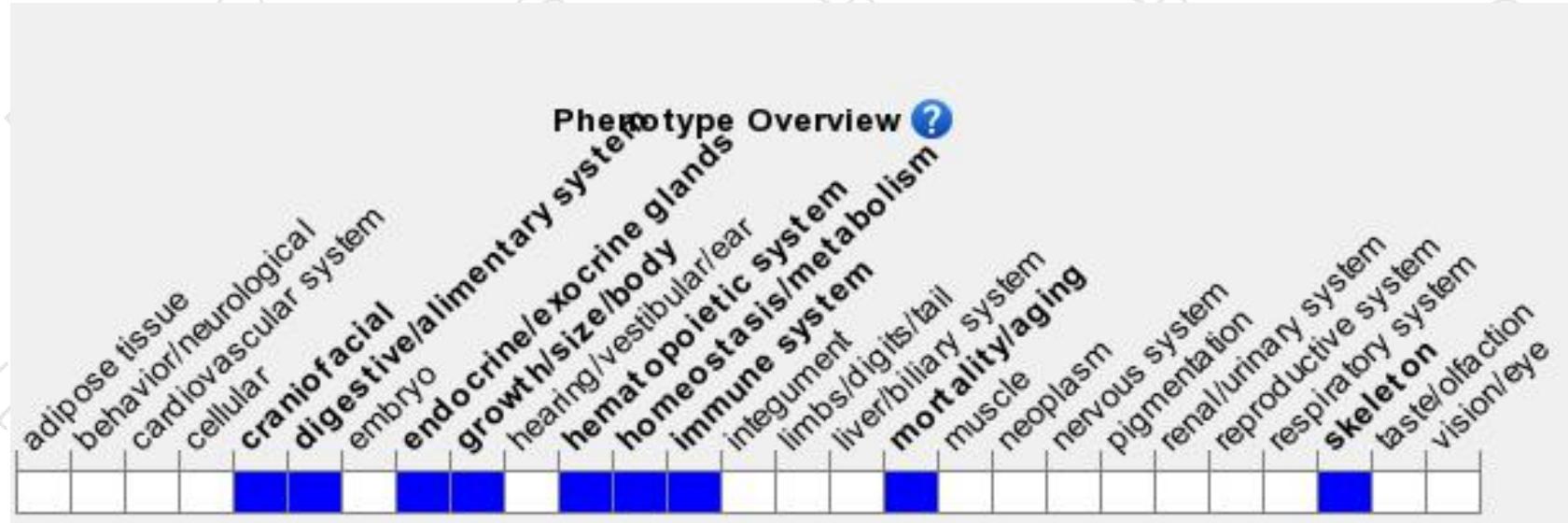
Protein domain





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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, Mice homozygous for a hypomorphic allele show postnatal growth retardation, failure of tooth eruption, impaired skeleton development, and osteopetrosis associated with failed osteoclast activity, high stomach pH, low calcium availability, impaired bone mineralization, and premature death.



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

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