

Pten Cas9-KO Strategy

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Project Overview

Project Name

Pten

Project type

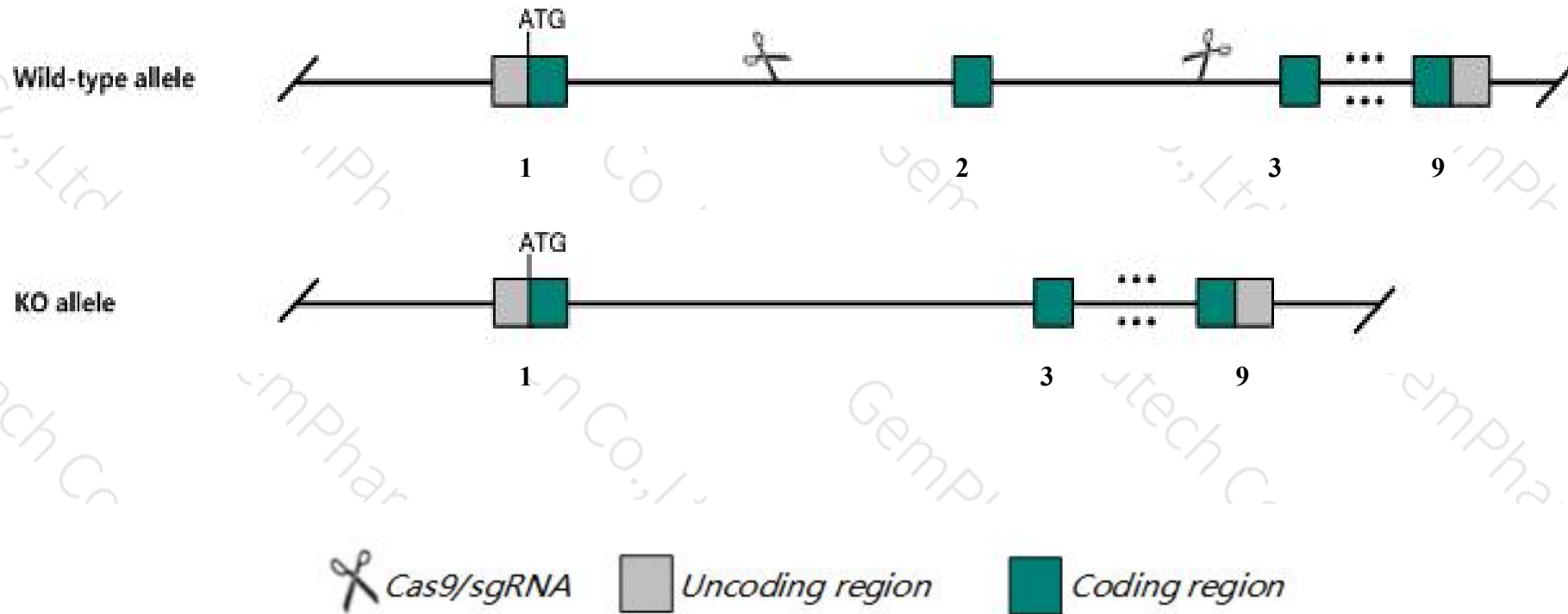
Cas9-KO

Strain background

C57BL/6J

Knockout strategy

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



- The *Pten* gene has 3 transcripts. According to the structure of *Pten* gene, exon2 of *Pten-201* (ENSMUST00000013807.7) transcript is recommended as the knockout region. The region contains 85bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Pten* gene. The brief process is as follows: sgRNA was transcribed in vitro. Cas9 and sgRNA were microinjected into the fertilized eggs of C57BL/6J 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/6J mice.

- According to the existing MGI data, Homozygous null mutants die by E9.5 with abnormally patterned enlarged brains and defective placentas. Heterozygotes develop a range of neoplasms. Conditional mutants demonstrate effects on basic processes of proliferation, differentiation and apoptosis.
- The *Pten* gene is located on the Chr19. 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 gene transcription and translation processes, all risks cannot be predicted under existing information.

Gene information (NCBI)

Pten phosphatase and tensin homolog [Mus musculus (house mouse)]

Gene ID: 19211, updated on 9-Apr-2019

Summary



Official Symbol Pten provided by [MGI](#)

Official Full Name phosphatase and tensin homolog provided by [MGI](#)

Primary source [MGI:MGI:109583](#)

See related [Ensembl:ENSMUSG00000013663](#)

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 2310035O07Rik, A130070J02Rik, A1463227, B430203M17Rik, MMAC1, PTENbeta, TEP1

Summary This gene encodes a phosphatase with dual activity against phospholipids and proteins, and acts as a tumor-suppressor. The protein contains four structural domains, a PIP2-binding domain, a catalytic tensin-type phosphatase domain, a C2 tensin-type domain and a PDZ-binding domain. The protein belongs to the protein tyrosine phosphatase family. Deletion of this gene in mice contribute to tumorigenesis in multiple tissues. [provided by RefSeq, Sep 2015]

Expression Ubiquitous expression in frontal lobe adult (RPKM 14.6), cortex adult (RPKM 12.9) and 28 other tissues [See more](#)

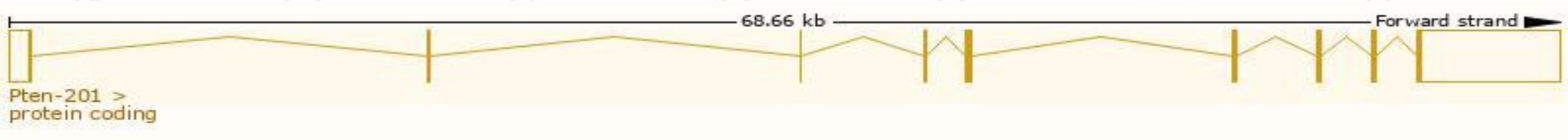
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

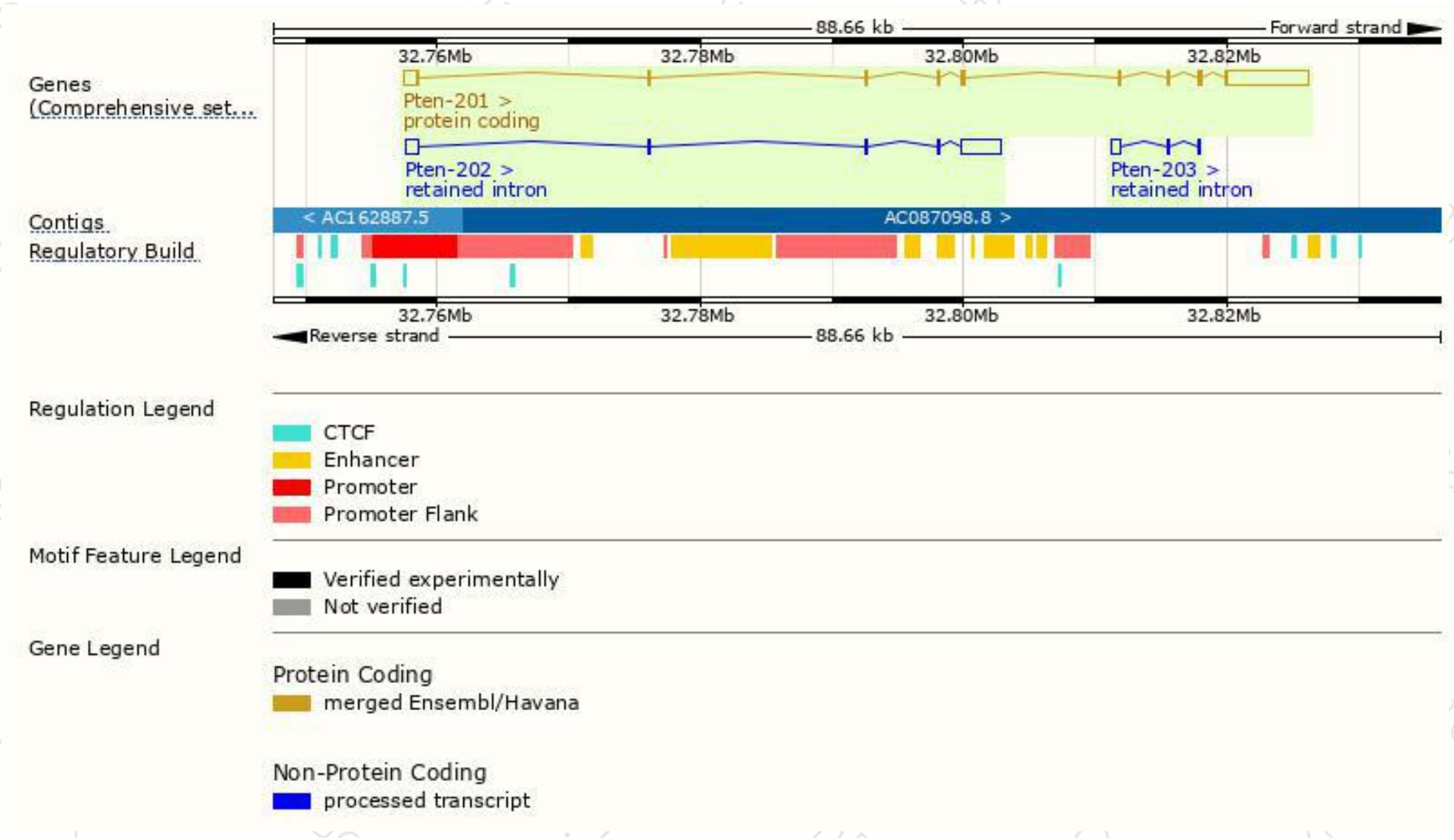
The gene has 3 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Pten-201	ENSMUST00000013807.7	8292	403aa	Protein coding	CCDS29753	O08586	TSL:1 GENCODE basic APPRIS P1
Pten-202	ENSMUST00000140014.1	4086	No protein	Retained intron	-	-	TSL:1
Pten-203	ENSMUST00000154806.1	922	No protein	Retained intron	-	-	TSL:2

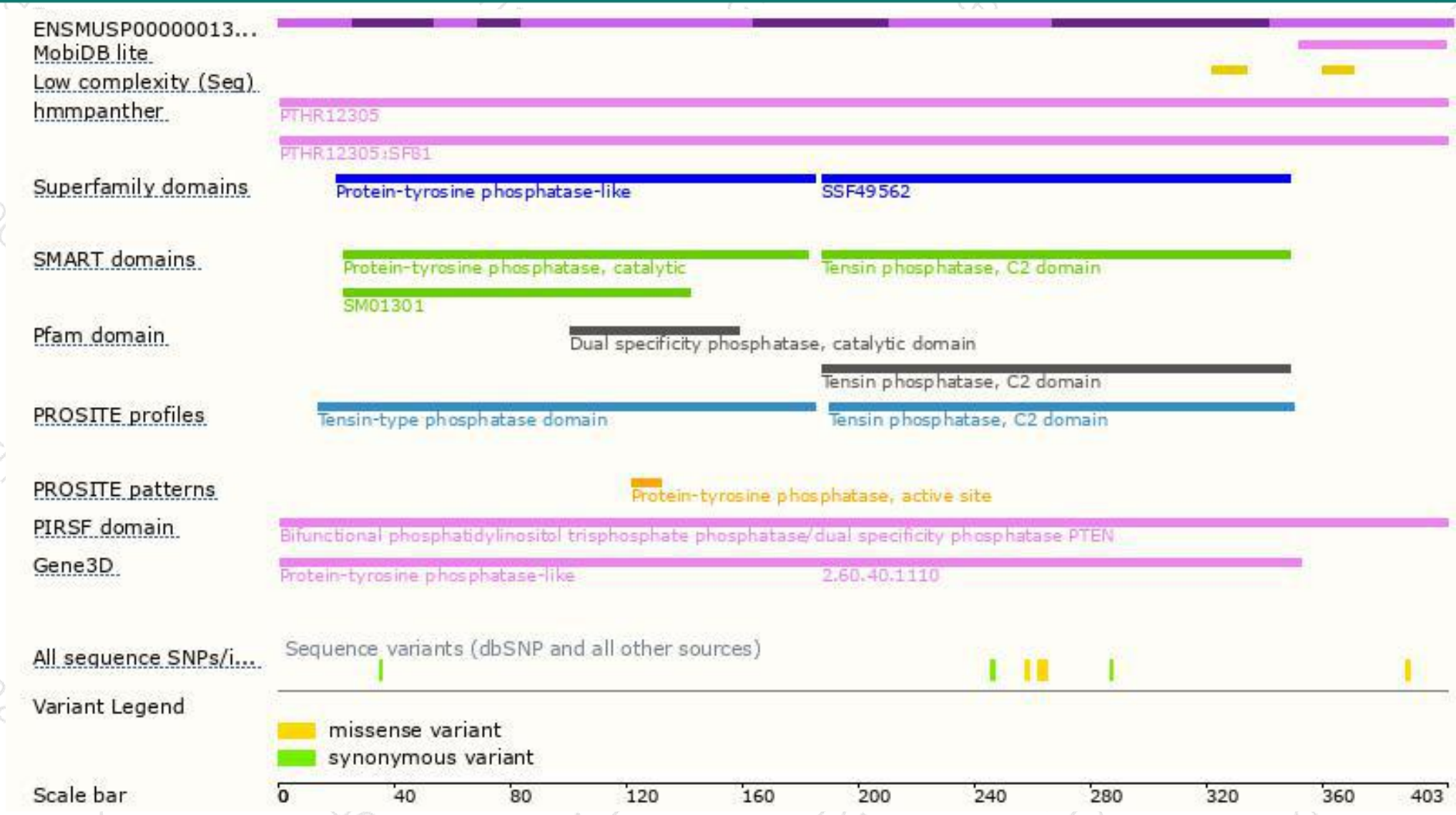
The strategy is based on the design of *Pten-201* transcript,The transcription is shown below



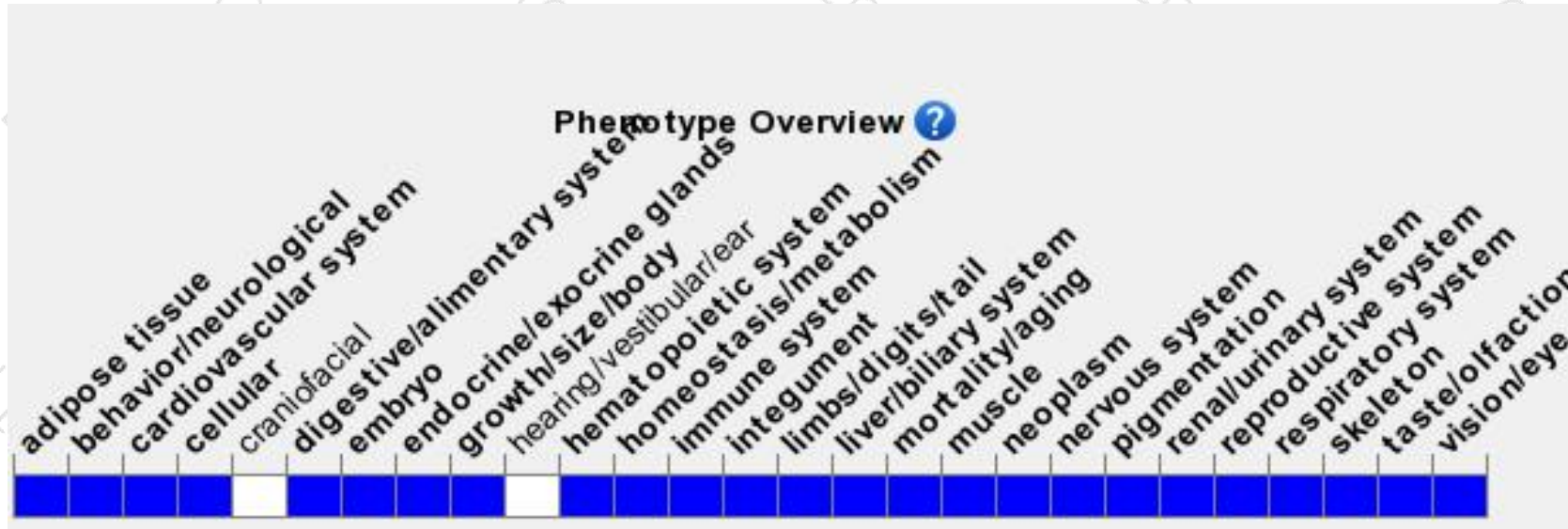
Genomic location distribution



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, Homozygous null mutants die by E9.5 with abnormally patterned enlarged brains and defective placentas. Heterozygotes develop a range of neoplasms. Conditional mutants demonstrate effects on basic processes of proliferation, differentiation and apoptosis.

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

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