

Vipr1 Cas9-CKO Strategy

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Project Overview

Project Name

Vipr1

Project type

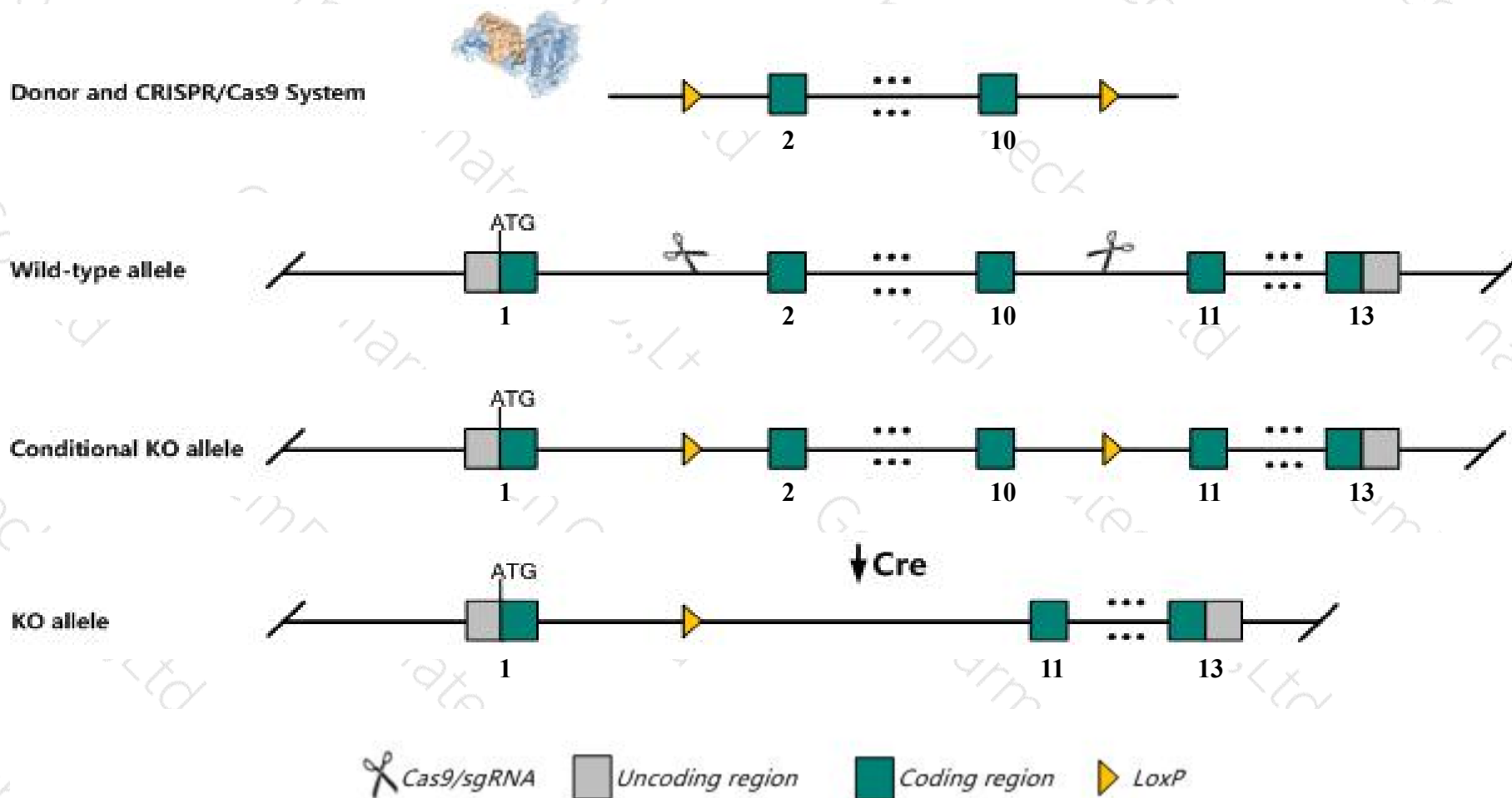
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Vipr1* gene. The schematic diagram is as follows:



Technical routes

- The *Vipr1* gene has 5 transcripts. According to the structure of *Vipr1* gene, exon2-exon10 of *Vipr1-201* (ENSMUST00000035115.4) transcript is recommended as the knockout region. The region contains 938bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Vipr1* gene. The brief process is as follows: sgRNA was transcribed in vitro, donor vector was constructed. Cas9, sgRNA 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 was 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.

- According to the existing MGI data, Mice homozygous for a knock-out allele exhibit prenatal lethality associated with severe neonatal growth failure, enlarged cecum, intestinal hemorrhage, and enterocyte hyperproliferation in addition to disorganized islets and impaired glucose homeostasis in surviving mice.
- Transcript *Vipr1-204* may not be affected.
- The *Vipr1* gene is located on the Chr9. 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)

Vipr1 vasoactive intestinal peptide receptor 1 [Mus musculus (house mouse)]

Gene ID: 22354, updated on 19-Mar-2019

Summary



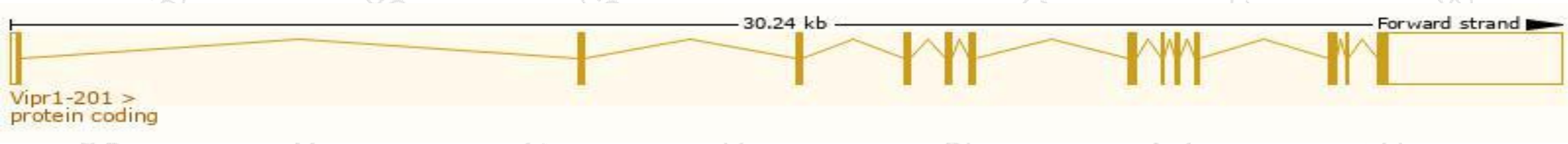
Official Symbol	Vipr1 provided by MGI
Official Full Name	vasoactive intestinal peptide receptor 1 provided by MGI
Primary source	MGI:MGI:109272
See related	Ensembl:ENSMUSG00000032528
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	AV071699, VIP-R1, VPAC1
Expression	Biased expression in colon adult (RPKM 47.0), small intestine adult (RPKM 34.2) and 8 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

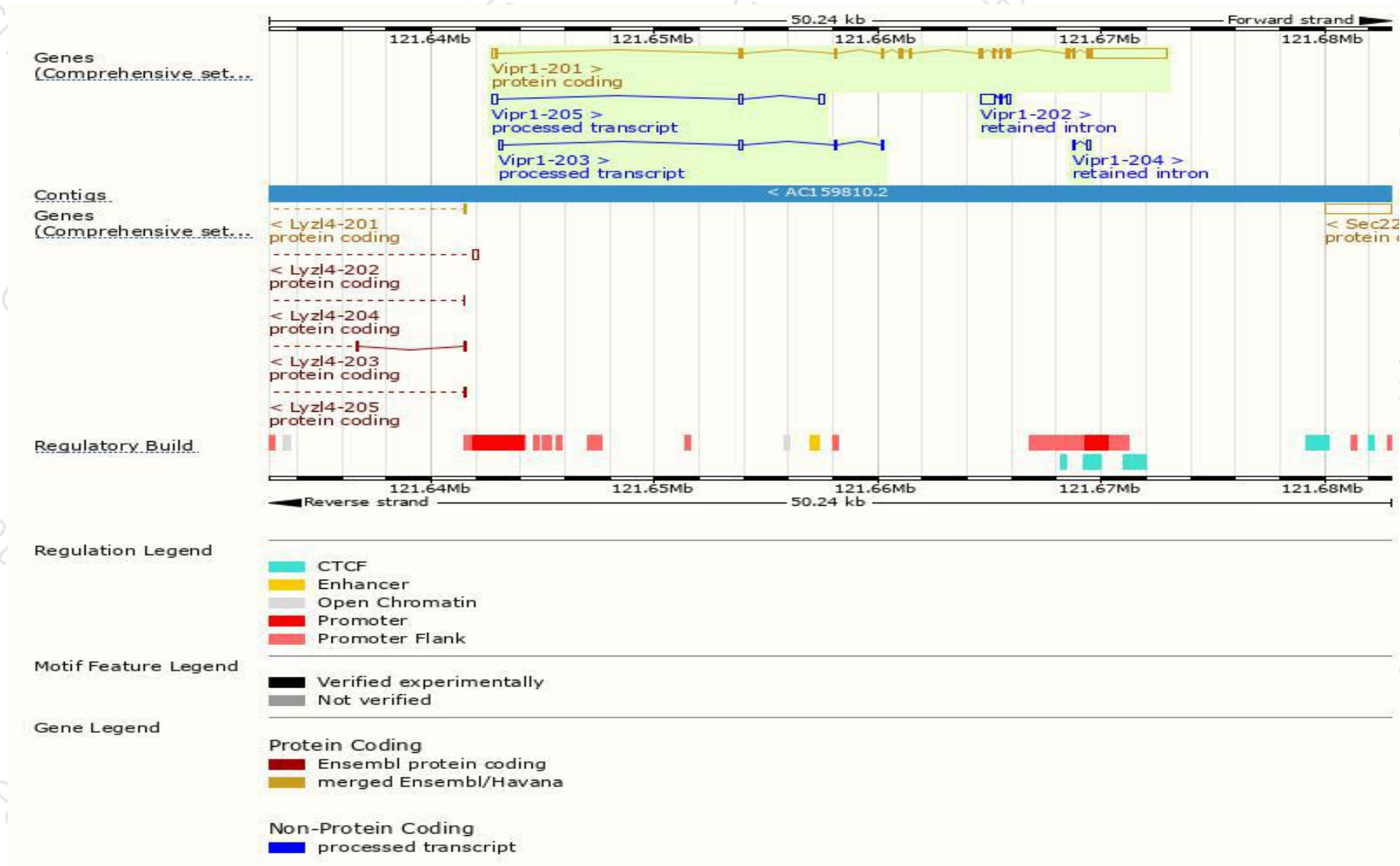
The gene has 5 transcripts,all transcripts are shown below:

Show/hide columns								Filter	
Name ▲	Transcript ID ▲	bp ▲	Protein ▲	Translation ID ▲	Biotype ▲	CCDS ▲	UniProt ▲	Flags ▲	
Vipr1-201	ENSMUST00000035115.4	4902	459aa	ENSMUSP00000035115.4	Protein coding	CCDS23633	P97751	TSL:1	GENCODE basic APPRIS P1
Vipr1-202	ENSMUST00000129394.1	763	No protein	-	Retained intron	-	-	TSL:3	
Vipr1-203	ENSMUST00000139189.1	412	No protein	-	lncRNA	-	-	TSL:5	
Vipr1-204	ENSMUST00000149959.1	224	No protein	-	Retained intron	-	-	TSL:5	
Vipr1-205	ENSMUST00000213272.1	508	No protein	-	lncRNA	-	-	TSL:5	

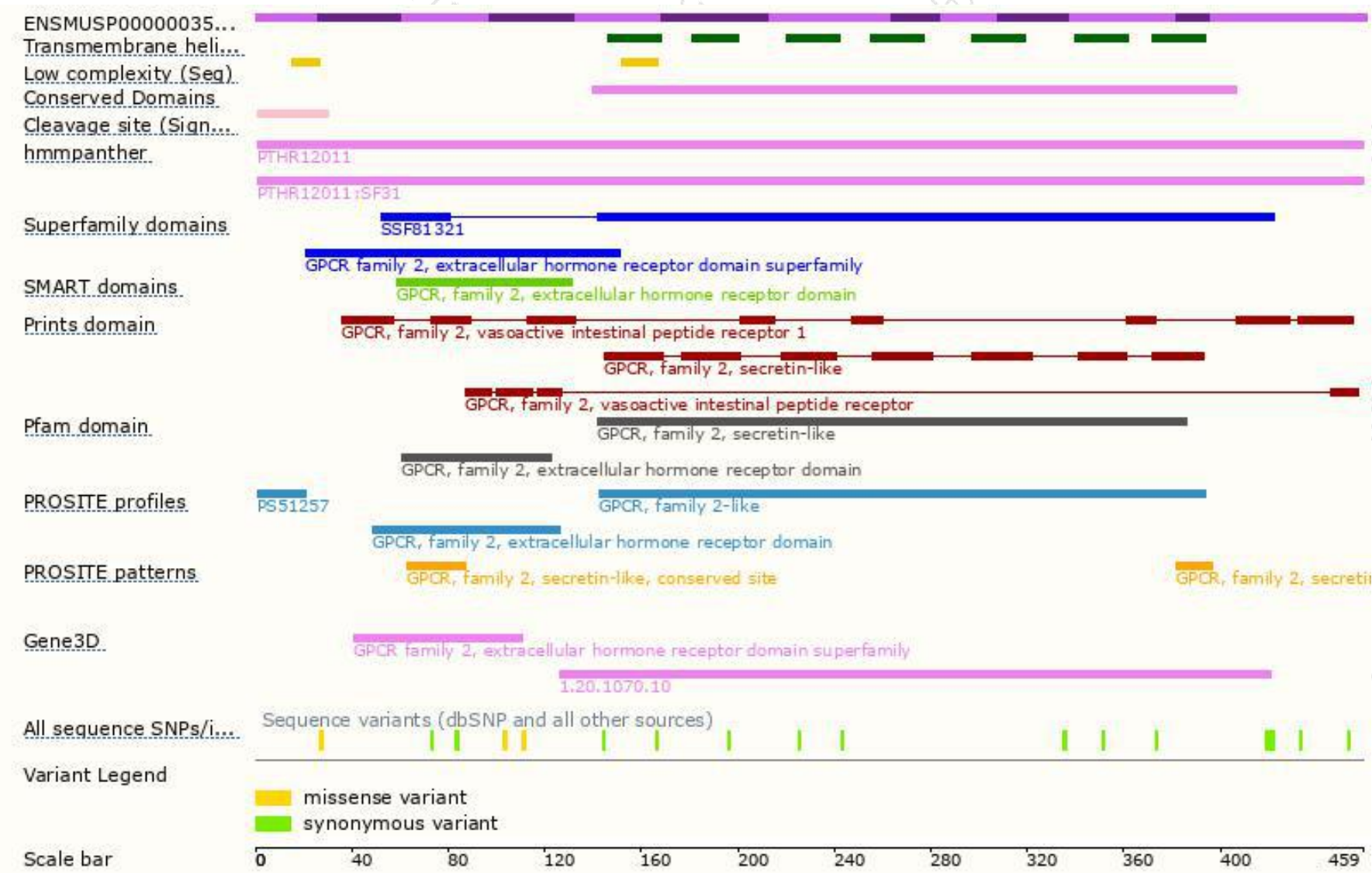
The strategy is based on the design of *Vipr1-201* transcript,The transcription is shown below



Genomic location distribution

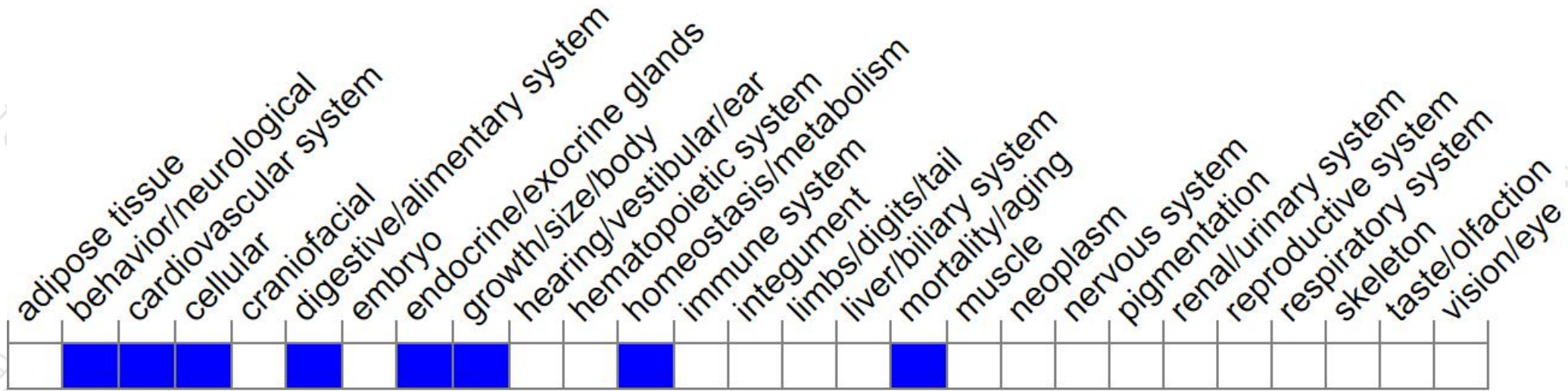


Protein domain



Mouse phenotype description(MGI)

Phenotype Overview ?



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 knock-out allele exhibit prenatal lethality associated with severe neonatal growth failure, enlarged cecum, intestinal hemorrhage, and enterocyte hyperproliferation in addition to disorganized islets and impaired glucose homeostasis in surviving mice.

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

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