

$Snx13$ Cas9-CKO Strategy

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Project Overview

Project Name

$Snx13$

Project type

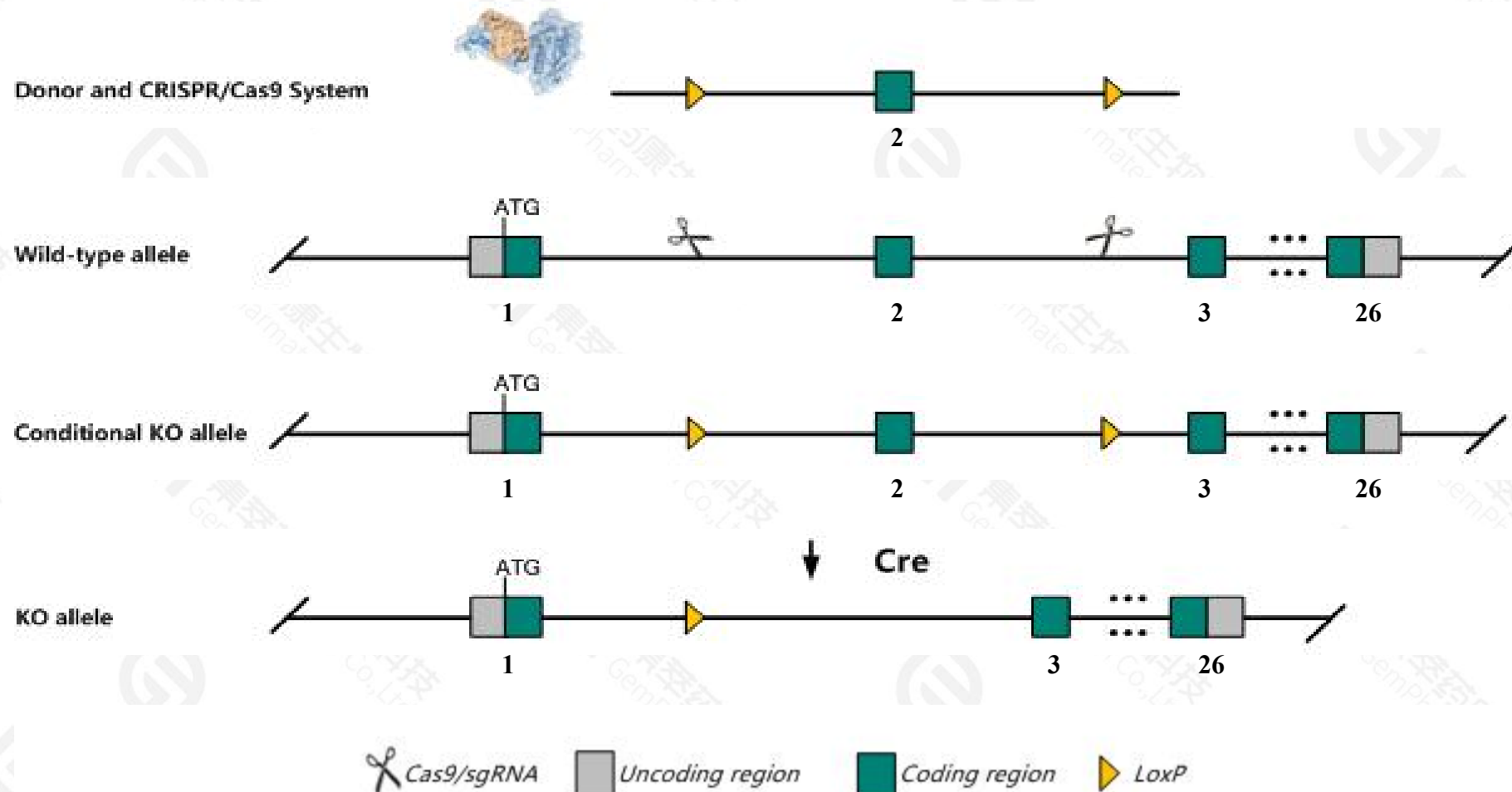
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Snx13* gene. The schematic diagram is as follows:



Technical routes

- The *Snx13* gene has 7 transcripts. According to the structure of *Snx13* gene, exon2 of *Snx13*-201(ENSMUST00000048519.17) transcript is recommended as the knockout region. The region contains 113bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Snx13* gene. The brief process is as follows: sgRNA was transcribed in vitro, donor 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, homozygous null mice are growth retarded and die at midgestation with defects in neural tube closure, vasculogenesis and placental development. Mutant visceral yolk sac endoderm cells exhibit altered endocytic compartments, abundant autophagic vacuoles and mislocalization of endocytic markers.
- The *Snx13* gene is located on the Chr12. 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)

Snx13 sorting nexin 13 [Mus musculus (house mouse)]

Gene ID: 217463, updated on 17-Dec-2020

Summary



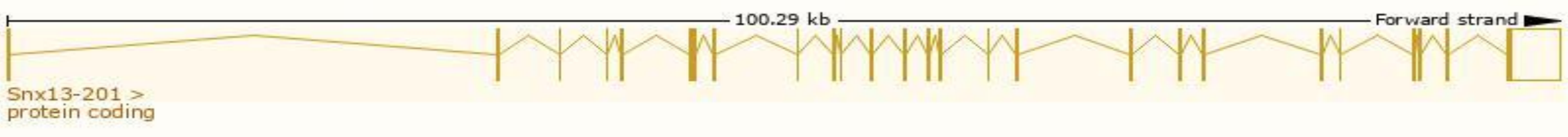
Official Symbol	Snx13 provided by MGI
Official Full Name	sorting nexin 13 provided by MGI
Primary source	MGI:MGI:2661416
See related	Ensembl:ENSMUSG00000020590
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	RGS-, Rgs-px1, mKIAA0713
Expression	Ubiquitous expression in cerebellum adult (RPKM 6.5), frontal lobe adult (RPKM 6.1) and 28 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

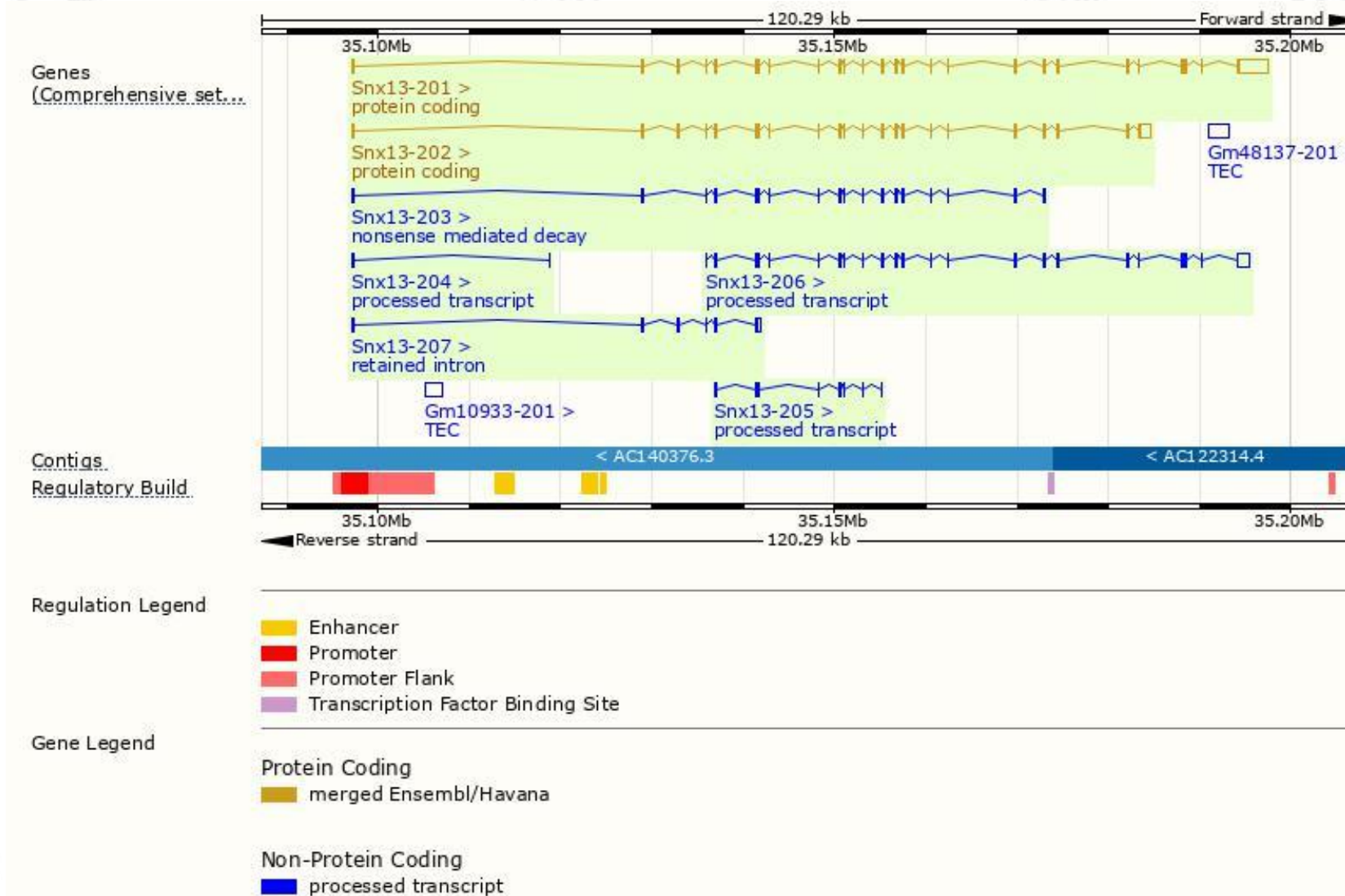
The gene has 7 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Snx13-201	ENSMUST00000048519.17	6231	958aa	Protein coding	CCDS56837		TSL:1 , GENCODE basic , APPRIS P1 ,
Snx13-202	ENSMUST00000163677.3	3681	778aa	Protein coding	-		TSL:1 , GENCODE basic ,
Snx13-203	ENSMUST00000221272.2	2027	44aa	Nonsense mediated decay	-		TSL:1 ,
Snx13-206	ENSMUST00000221876.2	3676	No protein	Processed transcript	-		TSL:1 ,
Snx13-205	ENSMUST00000221870.2	781	No protein	Processed transcript	-		TSL:3 ,
Snx13-204	ENSMUST00000221831.2	361	No protein	Processed transcript	-		TSL:3 ,
Snx13-207	ENSMUST00000222101.2	1145	No protein	Retained intron	-		TSL:1 ,

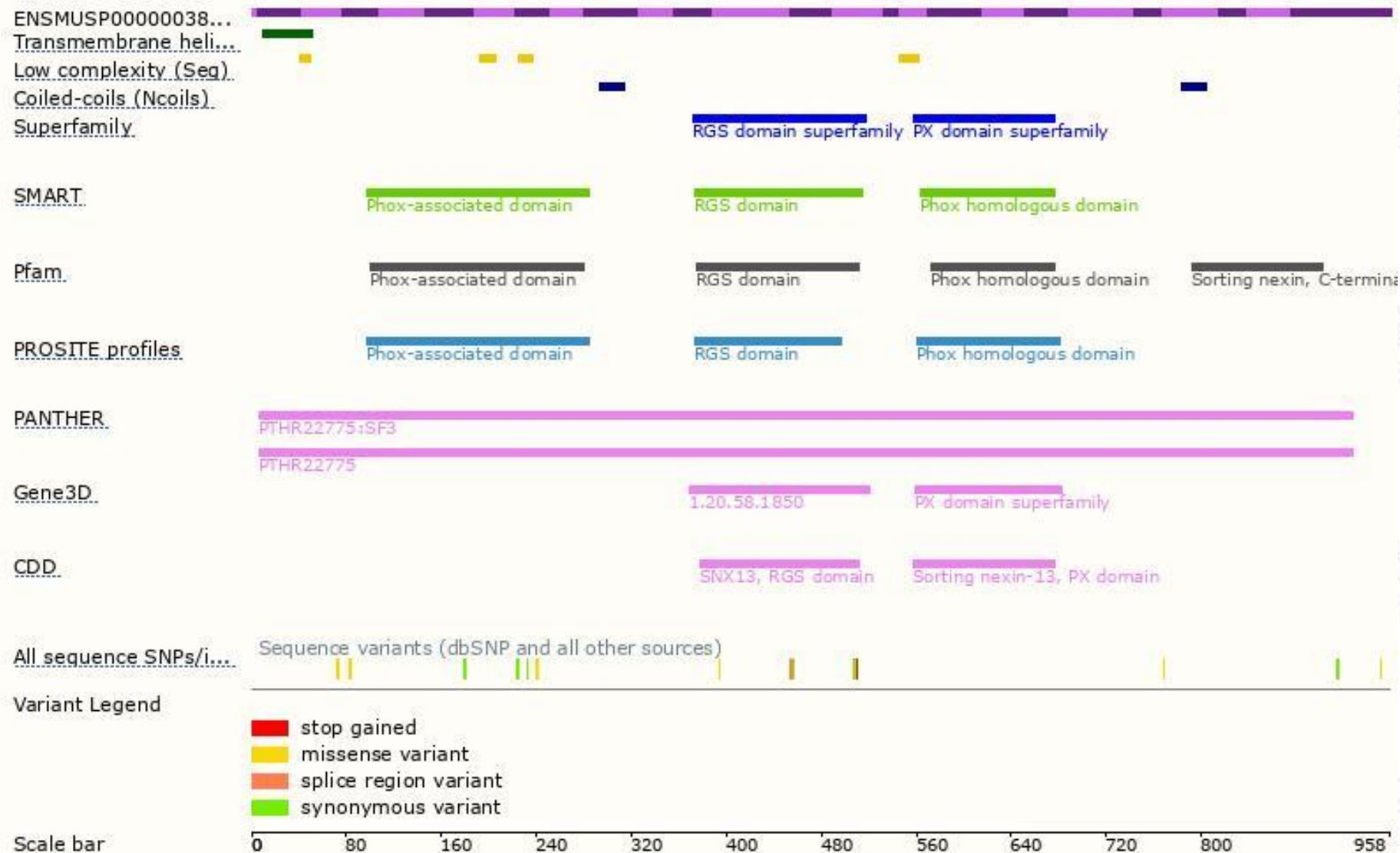
The strategy is based on the design of *Snx13-201* transcript,the transcription is shown below:



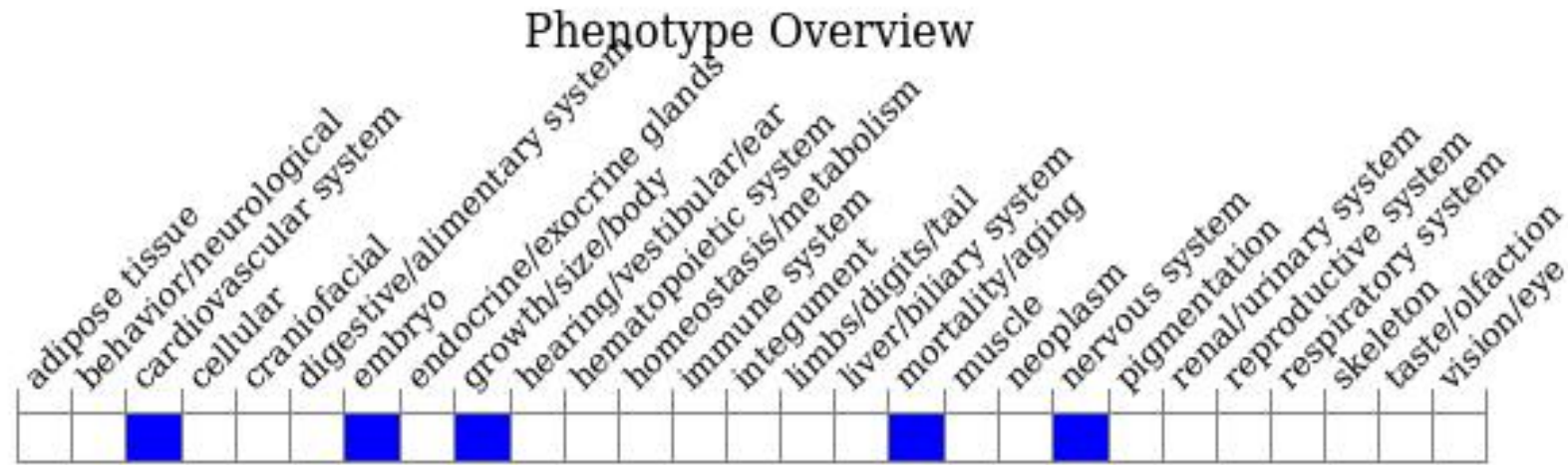
Genomic location distribution



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 mice are growth retarded and die at midgestation with defects in neural tube closure, vasculogenesis and placental development. Mutant visceral yolk sac endoderm cells exhibit altered endocytic compartments, abundant autophagic vacuoles and mislocalization of endocytic markers.

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

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