

Mal Cas9-CKO Strategy

Designer:

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Design Date:

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Project Overview



Project Name

Mal

Project type

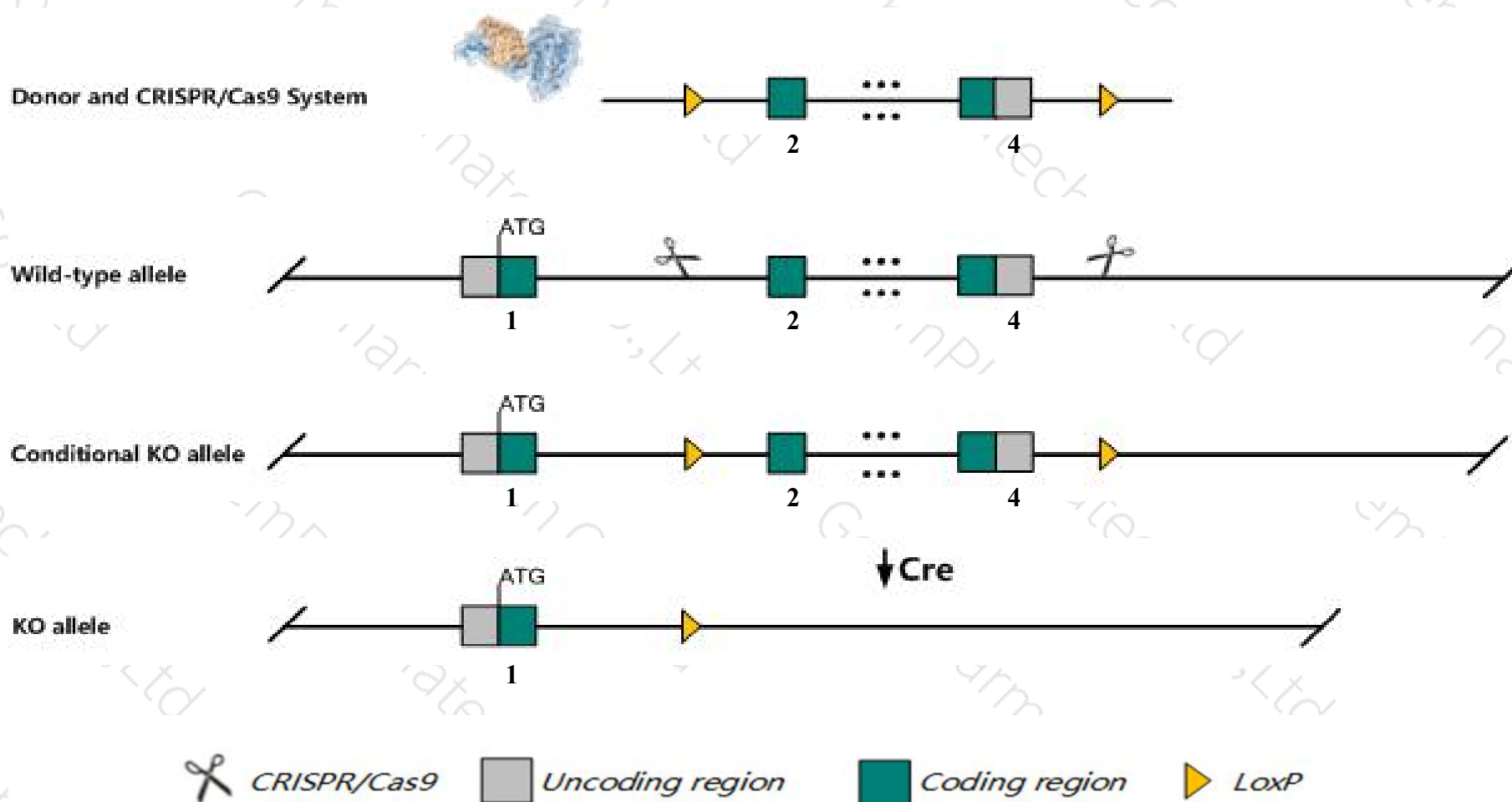
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

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



- The *Mal* gene has 2 transcripts. According to the structure of *Mal* gene, exon2-exon4 of *Mal-202* (ENSMUST00000028854.14) transcript is recommended as the knockout region. The region contains most of the coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Mal* 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.

- According to the existing MGI data, Homozygous null mice display abnormal myelination and optic nerve morphology.
- The *Mal* gene is located on the Chr2. 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)

Mal myelin and lymphocyte protein, T cell differentiation protein [Mus musculus (house mouse)]

Gene ID: 17153, updated on 31-Jan-2019

Summary



Official Symbol Mal provided by [MGI](#)

Official Full Name myelin and lymphocyte protein, T cell differentiation protein provided by [MGI](#)

Primary source [MGI:MGI:892970](#)

See related [Ensembl:ENSMUSG00000027375](#)

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 Mpv17, Vip17

Summary This gene encodes a highly hydrophobic integral membrane protein belonging to the MAL family of proteolipids. The encoded protein has been localized to the endoplasmic reticulum of T-cells and is a candidate linker protein in T-cell signal transduction. In addition, this proteolipid is localized in compact myelin of cells in the nervous system and has been implicated in myelin biogenesis and/or function. The protein plays a role in the formation, stabilization and maintenance of glycosphingolipid-enriched membrane microdomains. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Jan 2010]

Expression Biased expression in bladder adult (RPKM 46.2), stomach adult (RPKM 45.2) and 10 other tissues [See more](#)

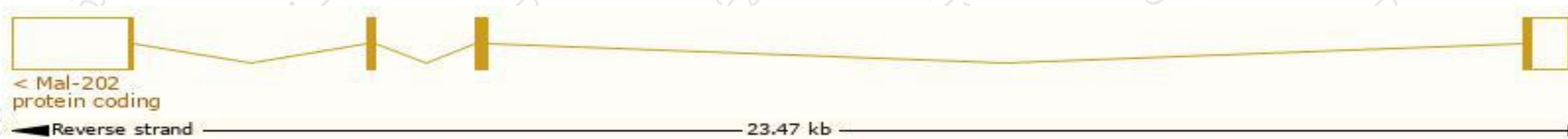
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

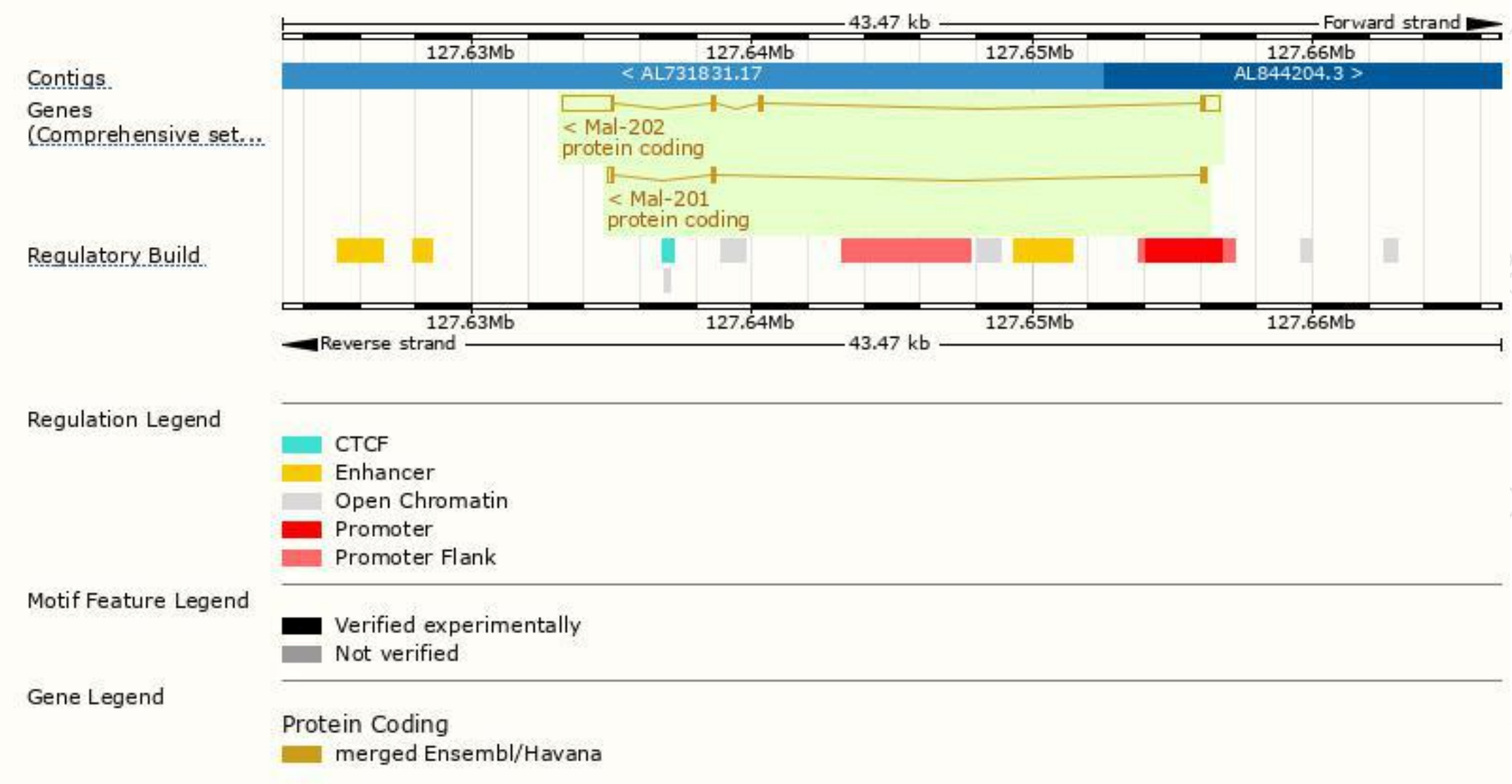
The gene has 2 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Mal-202	ENSMUST00000028854.14	2778	153aa	Protein coding	CCDS16707	O09198	TSL:1 GENCODE basic APPRIS P1
Mal-201	ENSMUST00000028853.6	486	97aa	Protein coding	CCDS50705	O09198	TSL:1 GENCODE basic

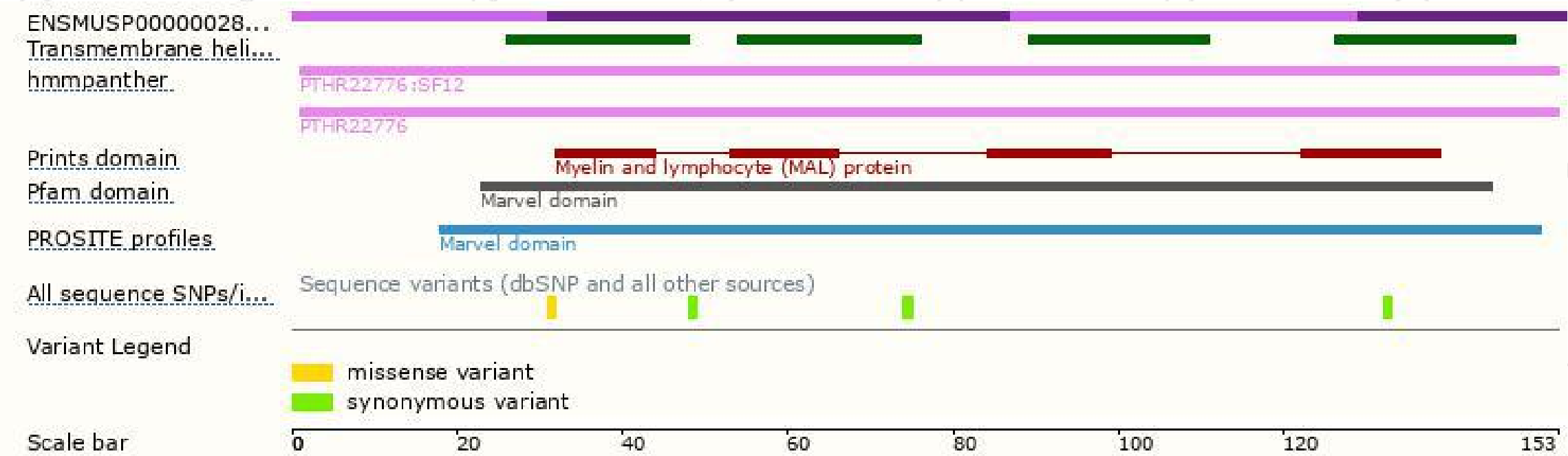
The strategy is based on the design of *Mal-202* transcript, The transcription is shown below



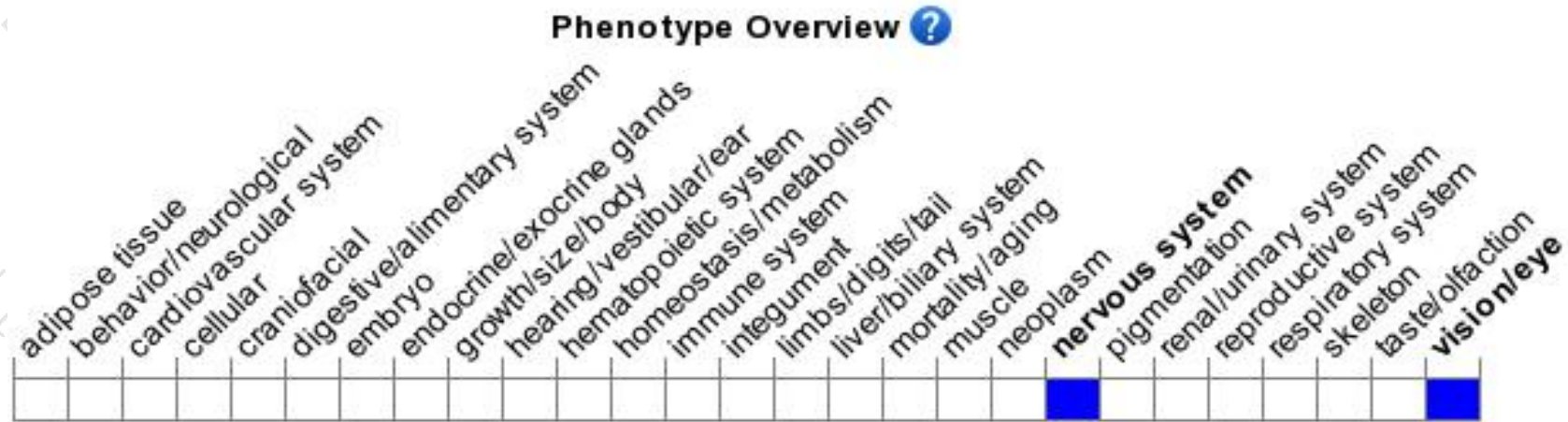
Genomic location distribution



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 display abnormal myelination and optic nerve morphology.

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

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