

Als2 Cas9-CKO Strategy

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Reviewer:

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Project Overview

Project Name

Als2

Project type

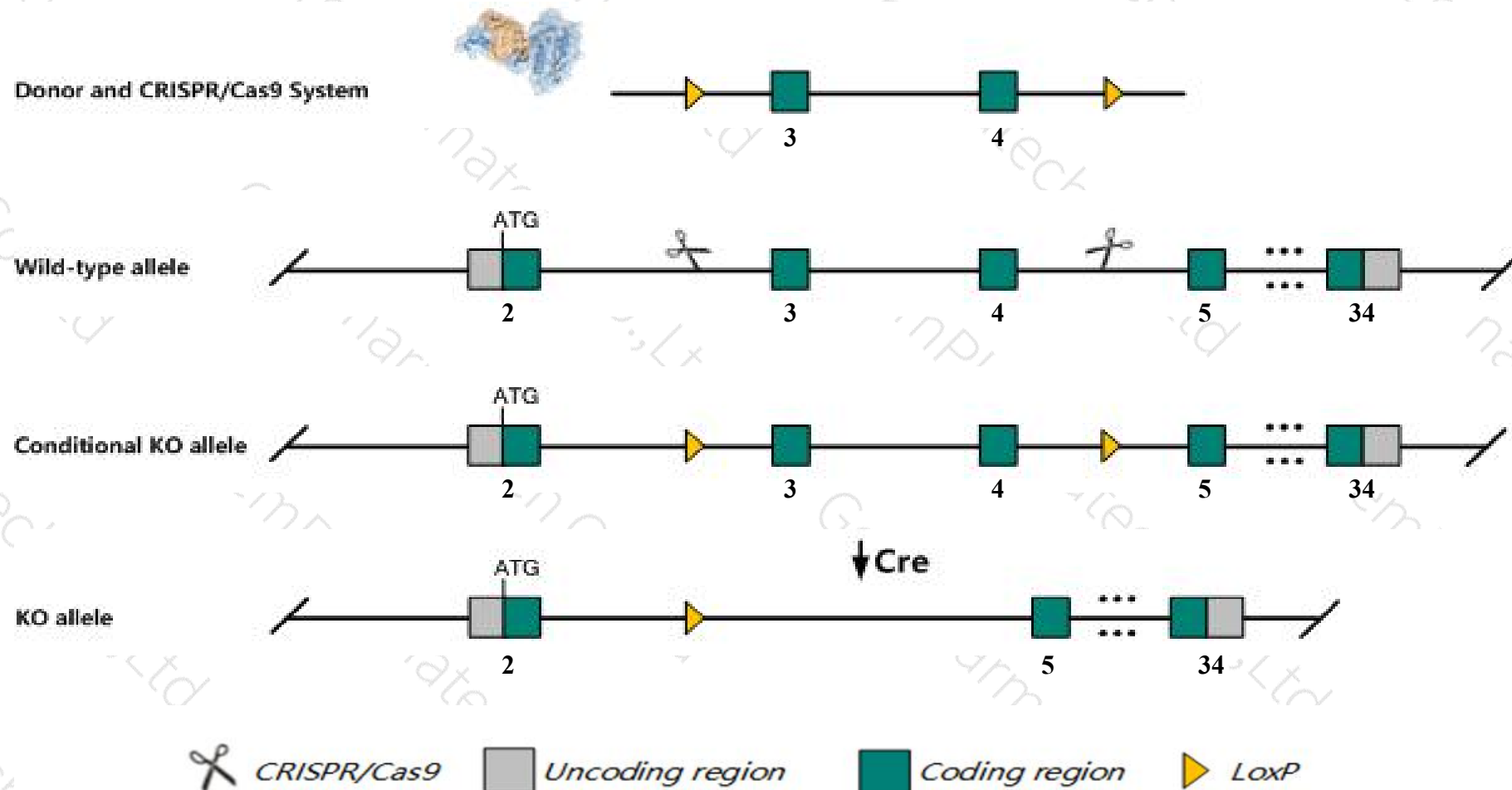
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Als2* gene. The schematic diagram is as follows:



Technical routes

- The *Als2* gene has 5 transcripts. According to the structure of *Als2* gene, exon3-exon4 of *Als2-201* (ENSMUST00000027178.12) transcript is recommended as the knockout region. The region contains 1075bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Als2* 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 mutations in this gene may result in increased body weight, altered endosome trafficking, modest motor behavioral abnormalities, altered anxiety responses, impaired axonal transport, and mild neuropathological deficits including axonal degeneration in the corticospinal tract.
- The *Als2* gene is located on the Chr1. 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)

Als2 alsin Rho guanine nucleotide exchange factor [*Mus musculus* (house mouse)]

Gene ID: 74018, updated on 8-Dec-2019

Summary

Official Symbol Als2 provided by [MGI](#)

Official Full Name alsin Rho guanine nucleotide exchange factor provided by [MGI](#)

Primary source [MGI:MGI:1921268](#)

See related [Ensembl:ENSMUSG000000026024](#)

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 Als1n; Als2cr6; mKIAA1563; 3222402C23Rik; 9430073A21Rik

Expression Ubiquitous expression in cerebellum adult (RPKM 19.3), liver adult (RPKM 7.8) and 25 other tissues [See more](#)

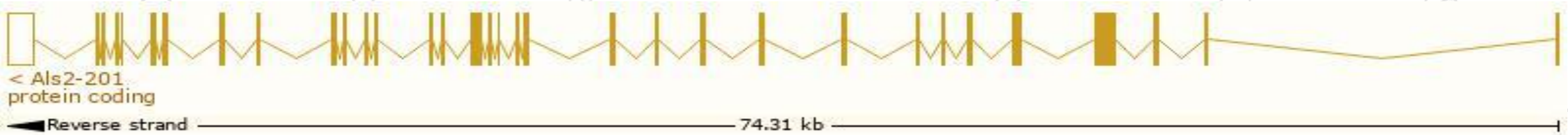
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

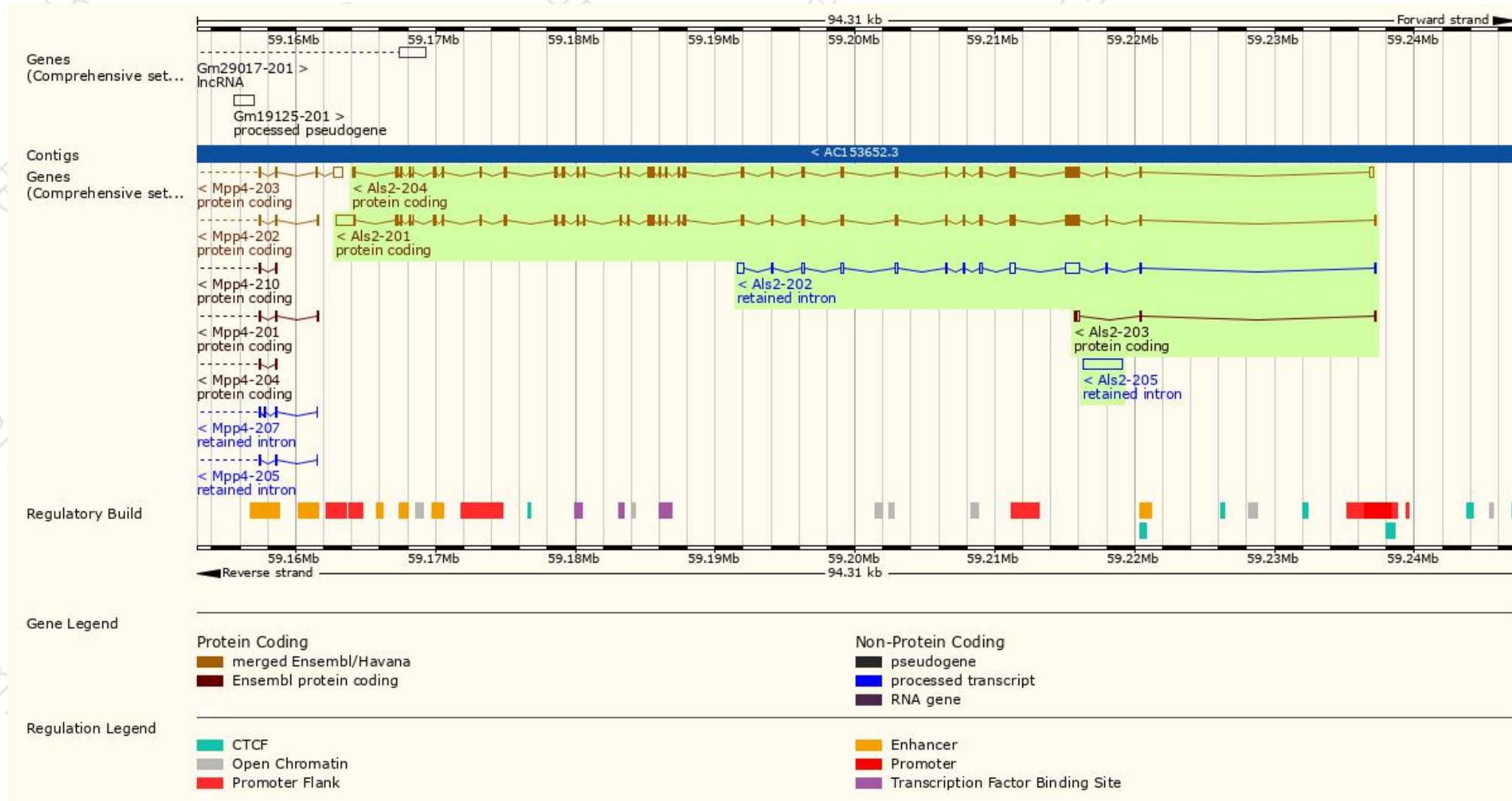
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Translation ID	Biotype	CCDS	UniProt	Flags
Als2-201	ENSMUST00000027178.12	6342	1651aa	ENSMUSP00000027178.6	Protein coding	CCDS35583	Q920R0	TSL:1 GENCODE basic APPRIS P1
Als2-204	ENSMUST00000163058.1	5379	1651aa	ENSMUSP00000125753.1	Protein coding	CCDS35583	Q920R0	TSL:1 GENCODE basic APPRIS P1
Als2-203	ENSMUST00000160945.1	521	69aa	ENSMUSP00000140990.1	Protein coding	-	A0A087WSC7	CDS 3' incomplete TSL:2
Als2-202	ENSMUST00000159166.7	2977	No protein	-	Retained intron	-	-	TSL:1
Als2-205	ENSMUST00000188469.1	2783	No protein	-	Retained intron	-	-	TSL:NA

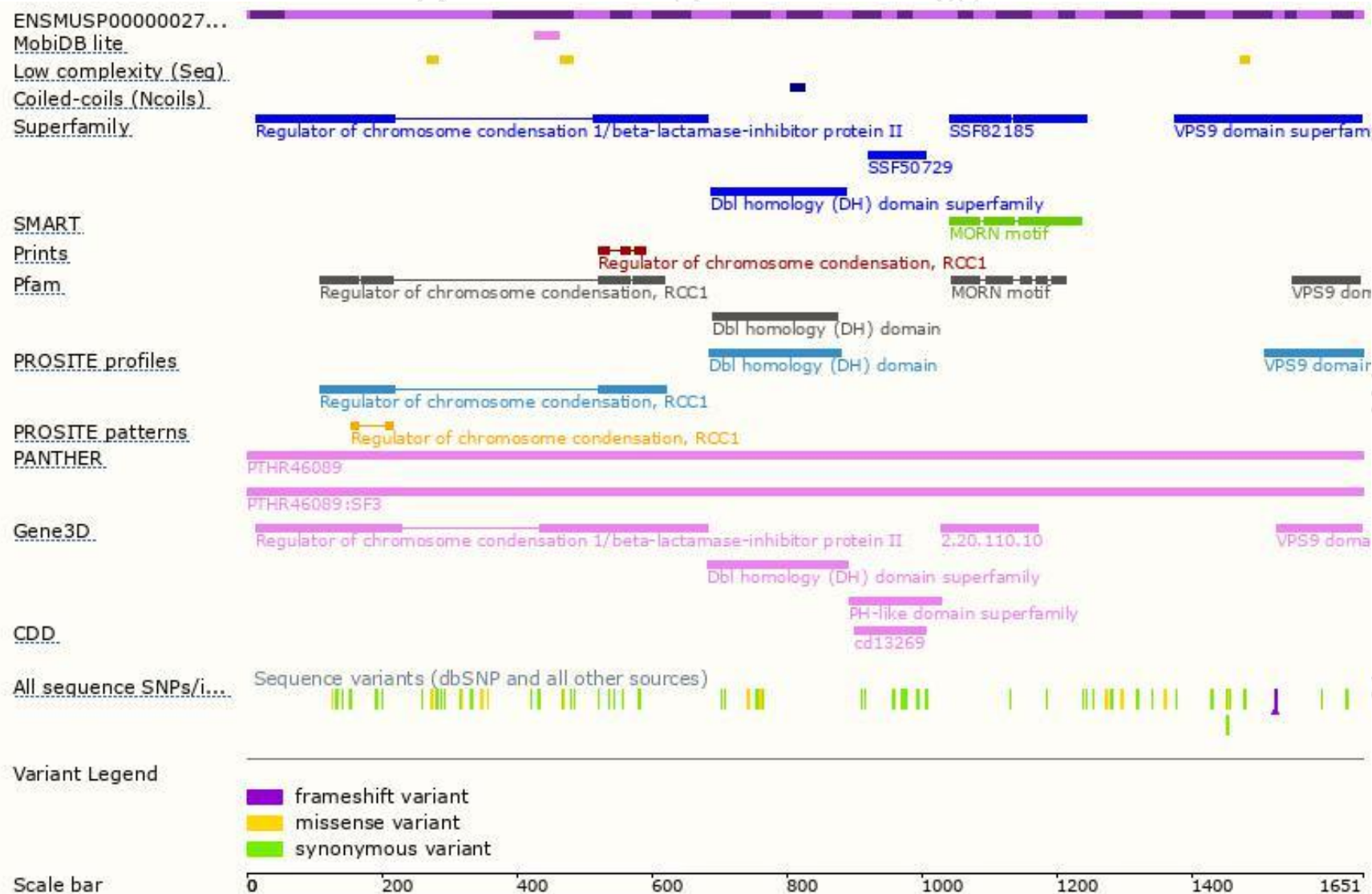
The strategy is based on the design of *Als2-201* transcript,The transcription is shown below



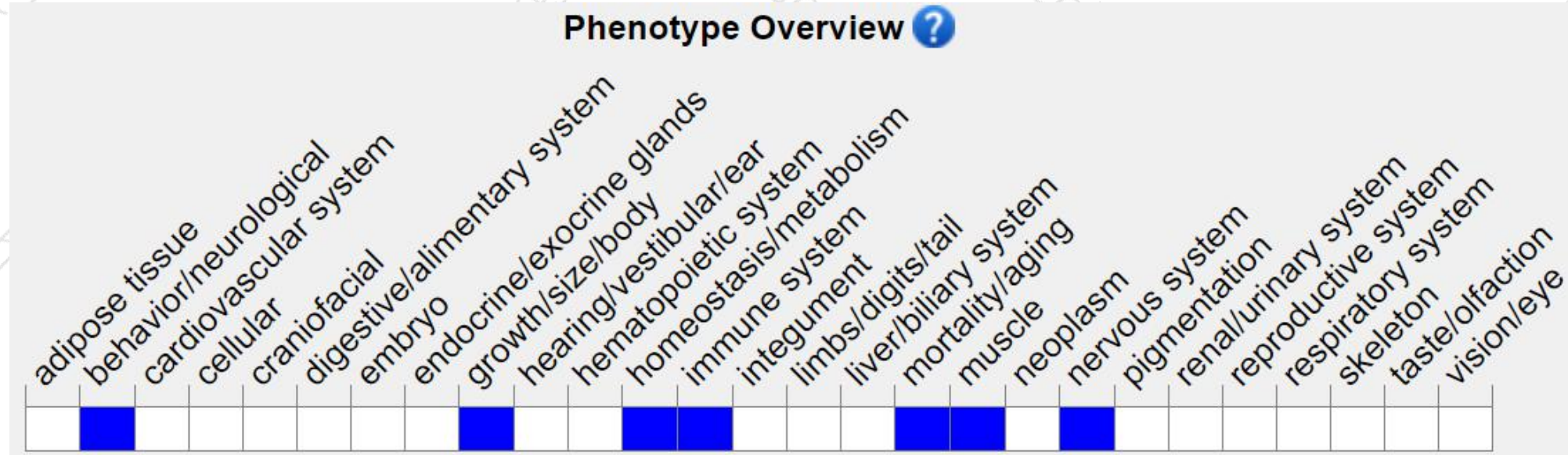
Genomic location distribution



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 mutations in this gene may result in increased body weight, altered endosome trafficking, modest motor behavioral abnormalities, altered anxiety responses, impaired axonal transport, and mild neuropathological deficits including axonal degeneration in the corticospinal tract.

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

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