

# ***Slc2a10*** Cas9-KO Strategy

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# Project Overview

**Project Name**

*Slc2a10*

**Project type**

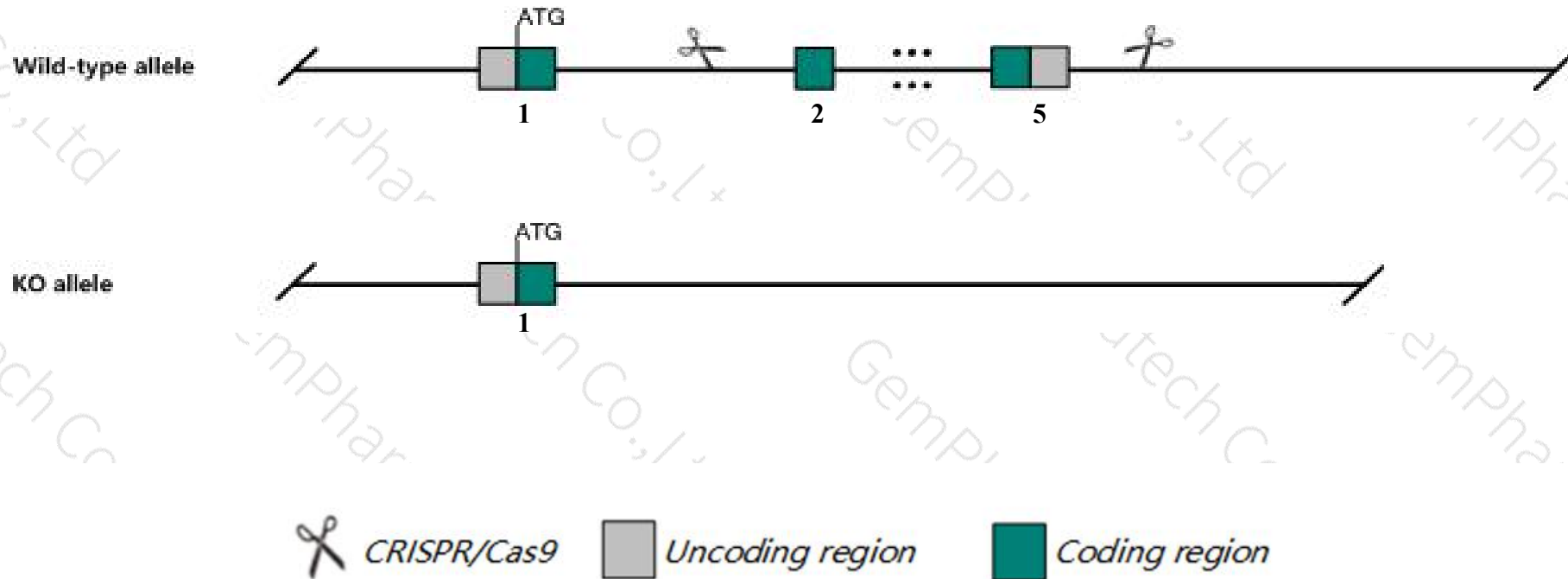
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc2a10* gene. The schematic diagram is as follows:



- The *Slc2a10* gene has 2 transcripts. According to the structure of *Slc2a10* gene, exon2-exon5 of *Slc2a10-201* (ENSMUST00000029196.4) 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 *Slc2a10* gene. The brief process is as follows: CRISPR/Cas9 system w

- According to the existing MGI data, Mice carrying ENU-induced mutations in this gene display thickening and aberrant vessel wall shape of large and medium size arteries, with significantly increased elastic fiber number and size. Cerebral arteries appear normal with no evidence of tortuosity, stenosis/dilatation or aneurysm.
- The *Slc2a10* 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 the gene knockout on gene transcription, RNA splicing and protein translation cannot be predicted at the existing technology level.

# Gene information (NCBI)

## Slc2a10 solute carrier family 2 (facilitated glucose transporter), member 10 [ *Mus musculus* (house mouse) ]

Gene ID: 170441, updated on 21-Jan-2020

### Summary

- Official Symbol** Slc2a10 provided by [MGI](#)
- Official Full Name** solute carrier family 2 (facilitated glucose transporter), member 10 provided by [MGI](#)
- Primary source** [MGI:MGI:2156687](#)
- See related** [Ensembl:ENSMUSG00000027661](#)
- 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** Glut10; AA450473
- Summary** This gene encodes a class III facilitative glucose transporter. Mutations in the related gene in human are associated with arterial tortuosity syndrome. [provided by RefSeq, Dec 2013]
- Expression** Biased expression in stomach adult (RPKM 17.5), colon adult (RPKM 9.3) and 13 other tissues [See more](#)
- Orthologs** [human](#) [all](#)

### Genomic context

**Location:** 2 H3; 2 85.66 cM See Slc2a10 in [Genome Data Viewer](#)

**Exon count:** 5

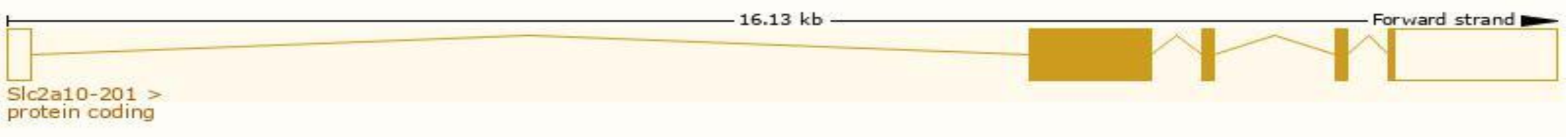
Annotation release	Status	Assembly	Chr	Location
<a href="#">108</a>	current	GRCm38.p6 ( <a href="#">GCF_000001635.26</a> )	2	NC_000068.7 (165503897..165519917)
Build 37.2	previous assembly	MGSCv37 ( <a href="#">GCF_000001635.18</a> )	2	NC_000068.6 (165329478..165345411)

# Transcript information (Ensembl)

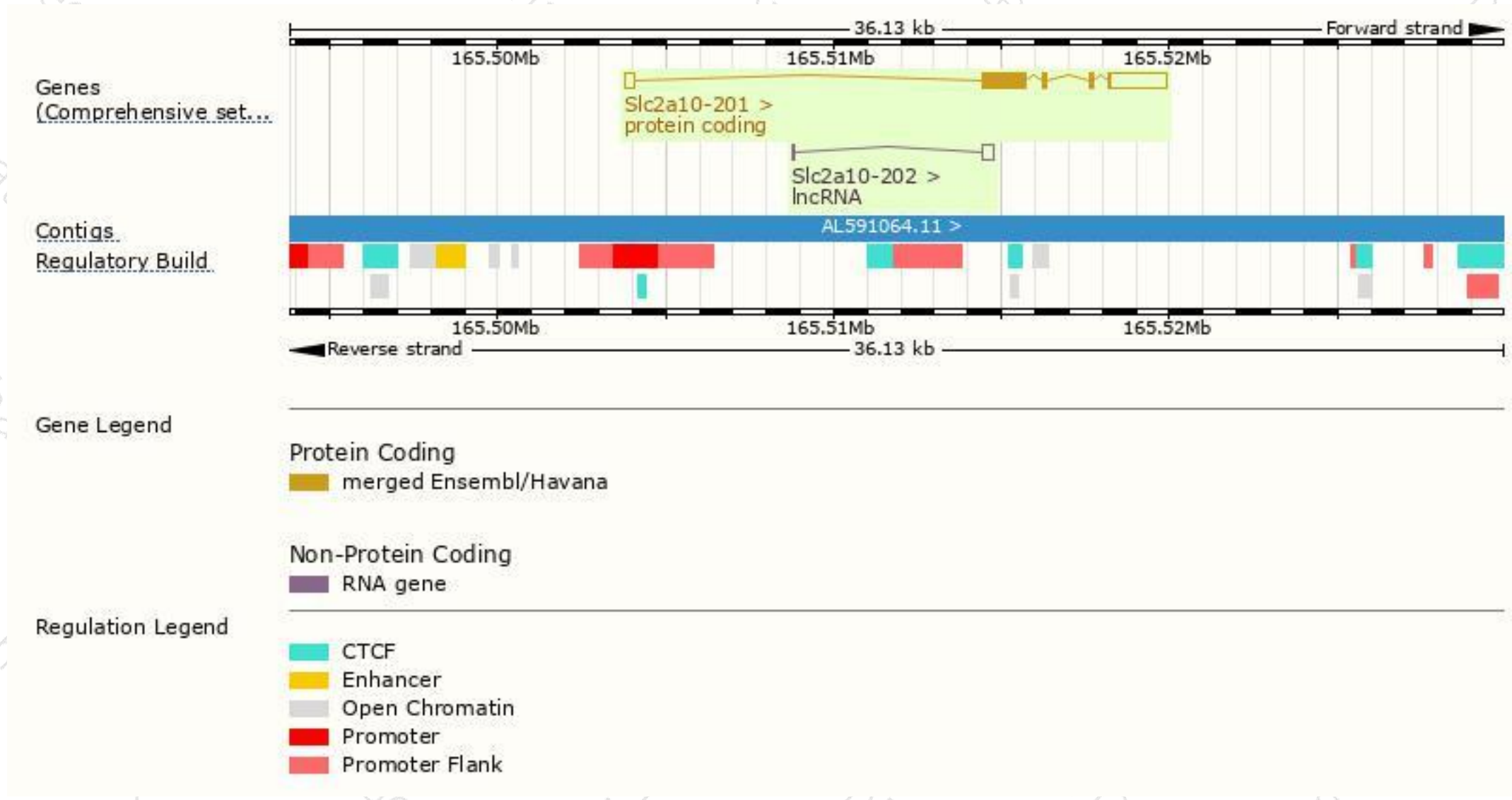
The gene has 2 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc2a10-201	<a href="#">ENSMUST00000029196.4</a>	3544	<a href="#">536aa</a>	Protein coding	<a href="#">CCDS17083</a>	<a href="#">A2A4V1_Q8VHD6</a>	TSL:1 GENCODE basic APPRIS P1
Slc2a10-202	<a href="#">ENSMUST00000148463.1</a>	378	No protein	lncRNA	-	-	TSL:2

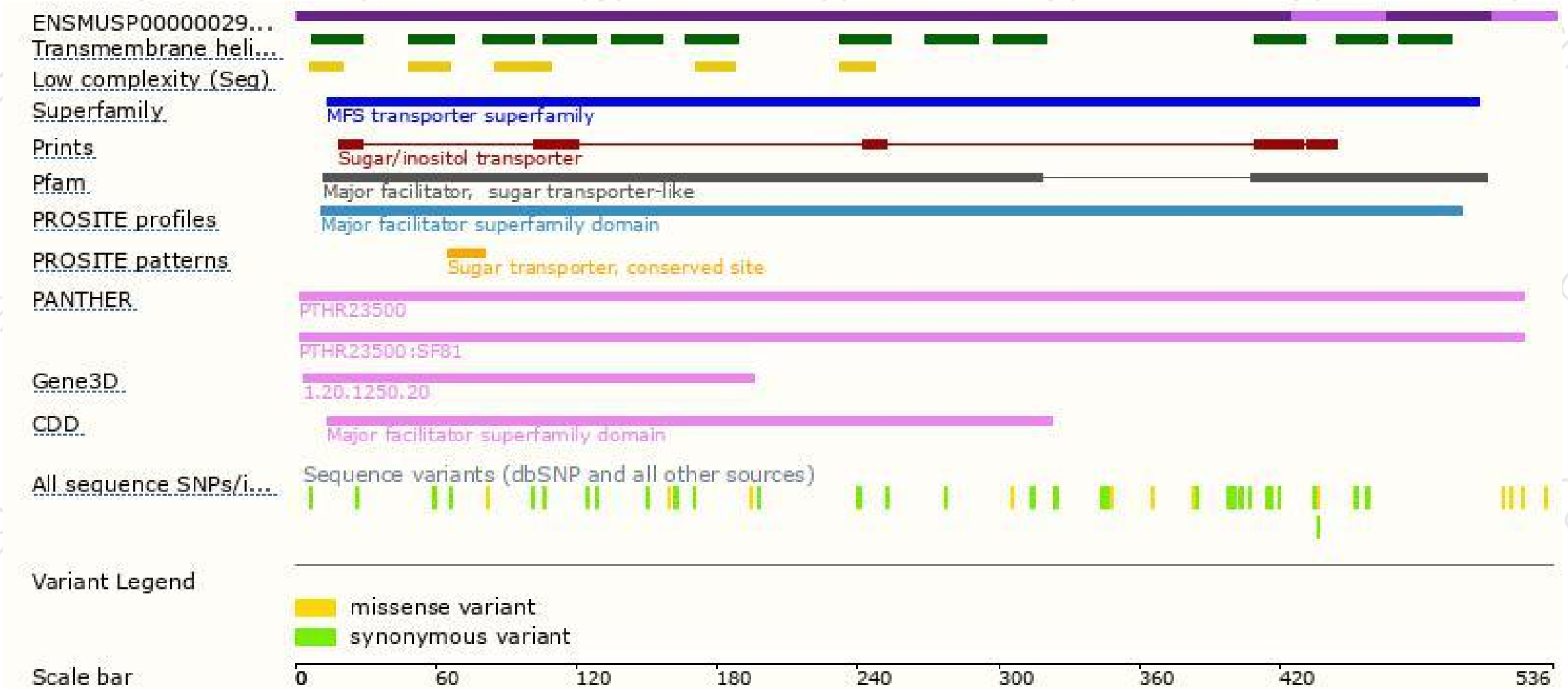
The strategy is based on the design of *Slc2a10-201* transcript,The transcription is shown below



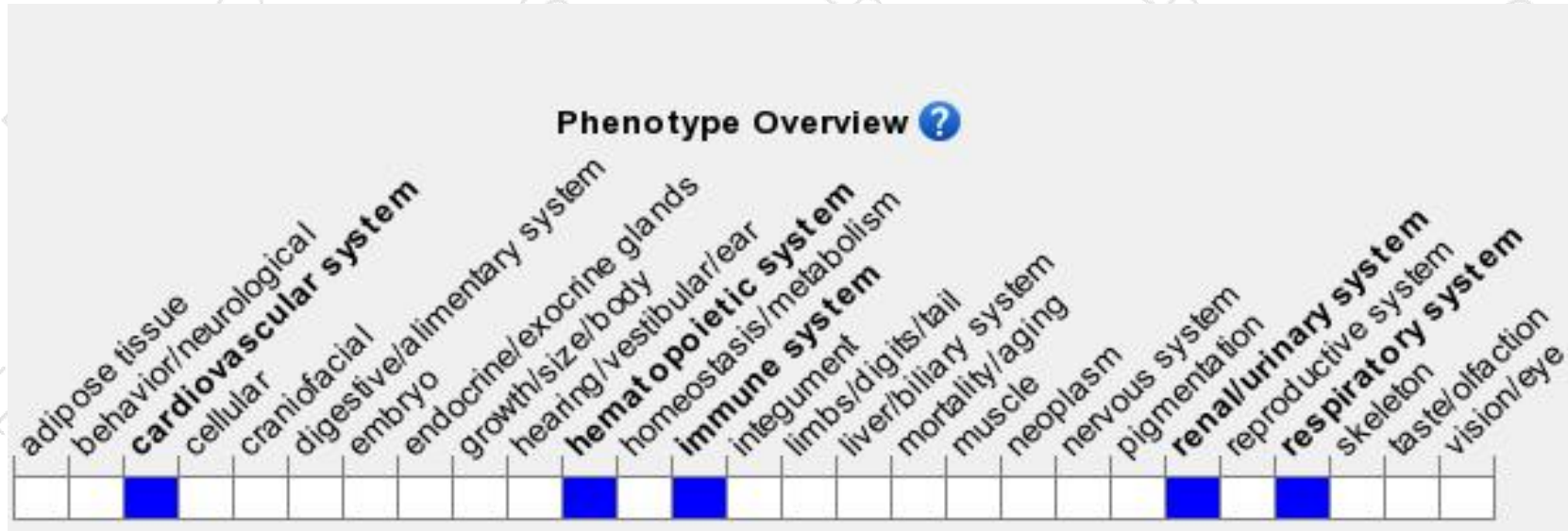
# Genomic location distribution



# 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, Mice carrying ENU-induced mutations in this gene display thickening and aberrant vessel wall shape of large and medium size arteries, with significantly increased elastic fiber number and size.

Cerebral arteries appear normal with no evidence of tortuosity, stenosis/dilatation or aneurysm.

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

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