

Neurog3 Cas9-CKO Strategy

Designer: Xueting Zhang

Reviewer: Yanhua Shen

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Project Overview

Project Name

Neurog3

Project type

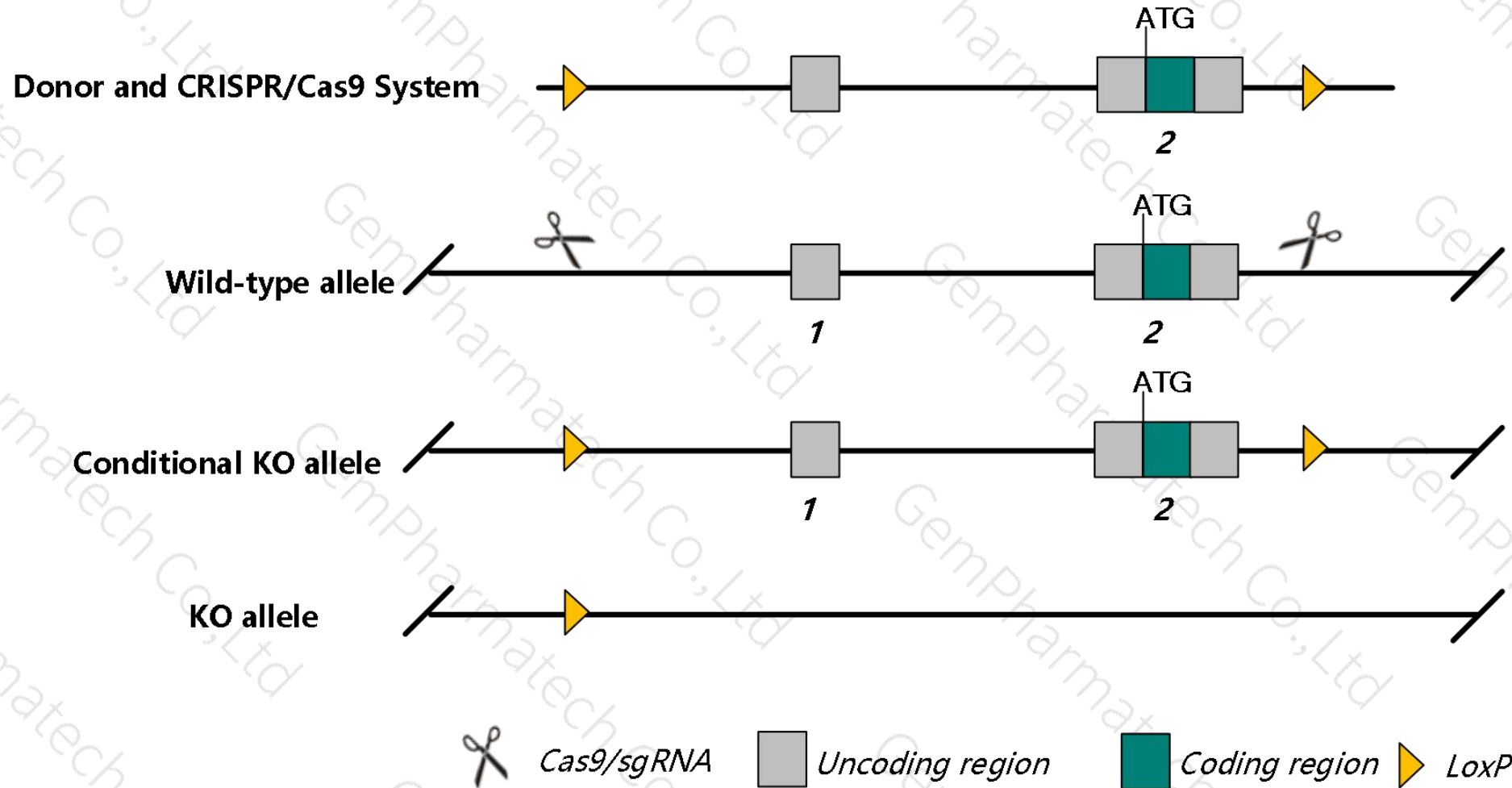
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Neurog3* gene. The schematic diagram is as follows:



- The *Neurog3* gene has 3 transcripts. According to the structure of *Neurog3* gene, exon1-exon2 of *Neurog3-201* (ENSMUST00000050103.1) transcript is recommended as the knockout region. The region contains all 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 *Neurog3* gene. The brief process is as follows: gRNA was transcribed in vitro, donor was constructed. Cas9, gRNA 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, omozygotes for targeted null mutations are deficient in endocrine cells of the glandular stomach and intestinal epithelium, and lack glucagon- and insulin-producing cells of the pancreas. Mutants die postnatally from diabetes.
- *Neurog3* gene is located inside of *Fam241b* gene, partial sequence of *Fam241b* gene will be deleted together in this strategy.
- The *Neurog3* gene is located on the Chr10. 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)

Neurog3 neurogenin 3 [*Mus musculus* (house mouse)]

Gene ID: 11925, updated on 26-Nov-2019

Summary

Official Symbol Neurog3 provided by [MGI](#)
Official Full Name neurogenin 3 provided by [MGI](#)
Primary source [MGI:MGI:893591](#)
See related [Ensembl:ENSMUSG00000044312](#)
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 ngn3; Atoh5; Math4B; bHLHa7
Expression Biased expression in duodenum adult (RPKM 2.0), large intestine adult (RPKM 1.3) and 5 other tissues [See more](#)
Orthologs [human](#) [all](#)

Genomic context

Location: 10; 10 B4

See Neurog3 in [Genome Data Viewer](#)

Exon count: 2

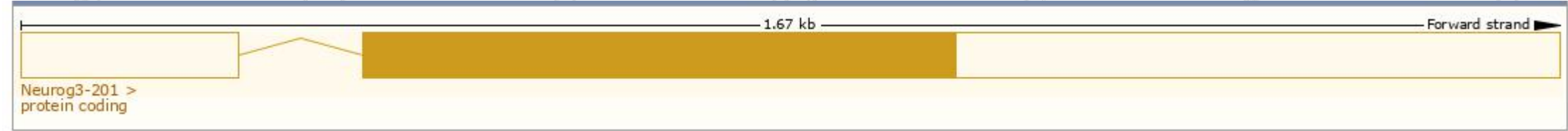
Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	10	NC_000076.6 (62133090..62134763)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	10	NC_000076.5 (61595838..61597511)

Transcript information (Ensembl)

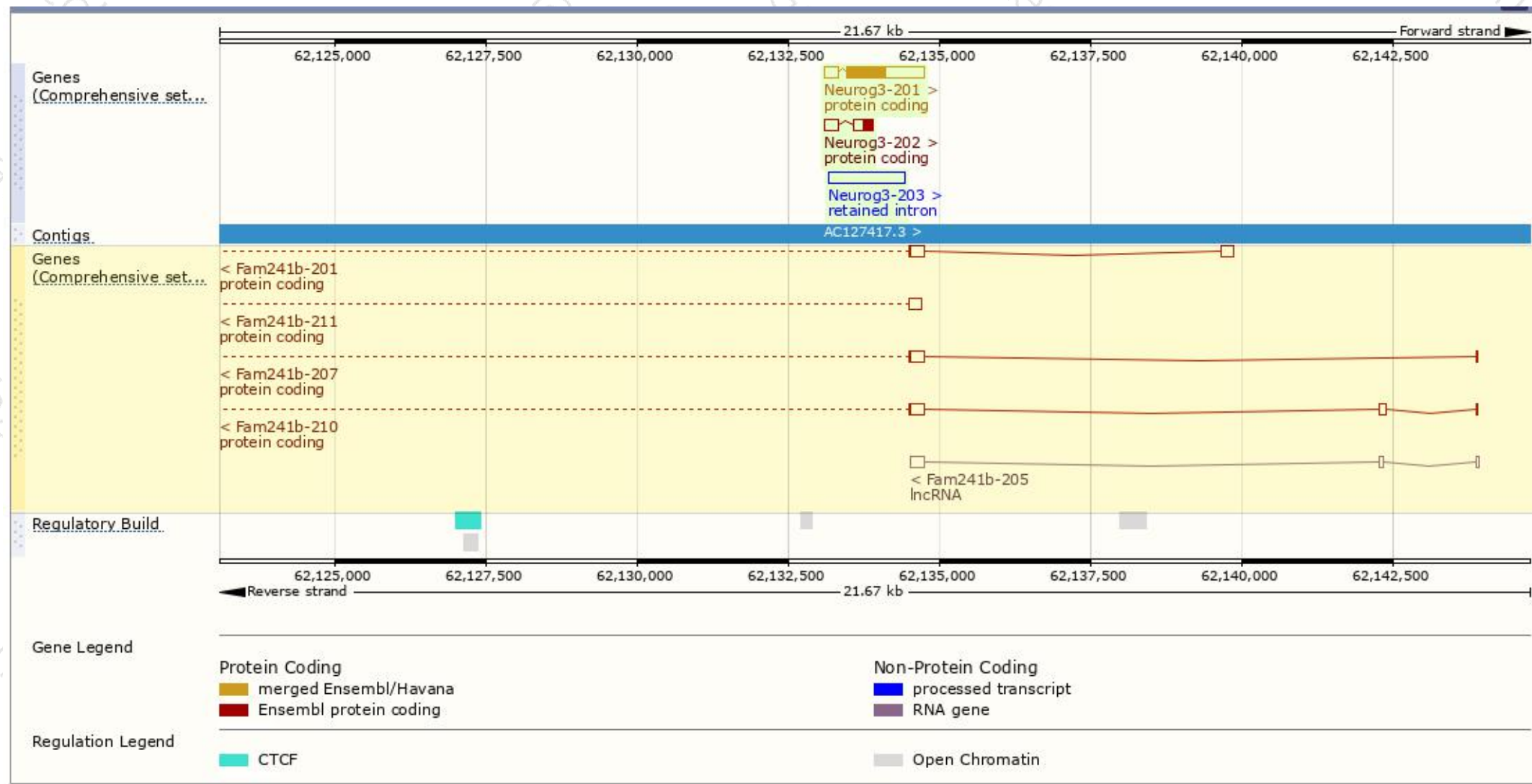
The gene has 3 transcripts, and all the transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Neurog3-201	ENSMUST00000050103.1	1540	214aa	Protein coding	CCDS23887	P70661 Q548G3	TSL:1 GENCODE basic APPRIS P1
Neurog3-202	ENSMUST00000218121.1	553	50aa	Protein coding	-	A0A1W2P770	CDS 3' incomplete TSL:5
Neurog3-203	ENSMUST00000218216.1	1259	No protein	Retained intron	-	-	TSL:NA

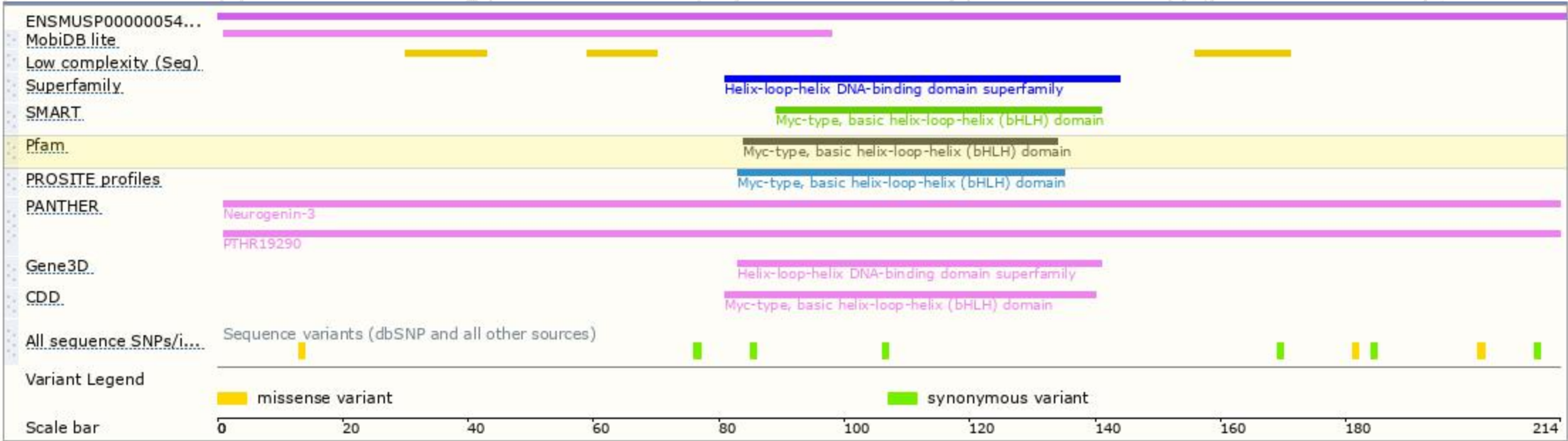
The strategy is based on the design of *Neurog3-201* transcript, The transcription is shown below



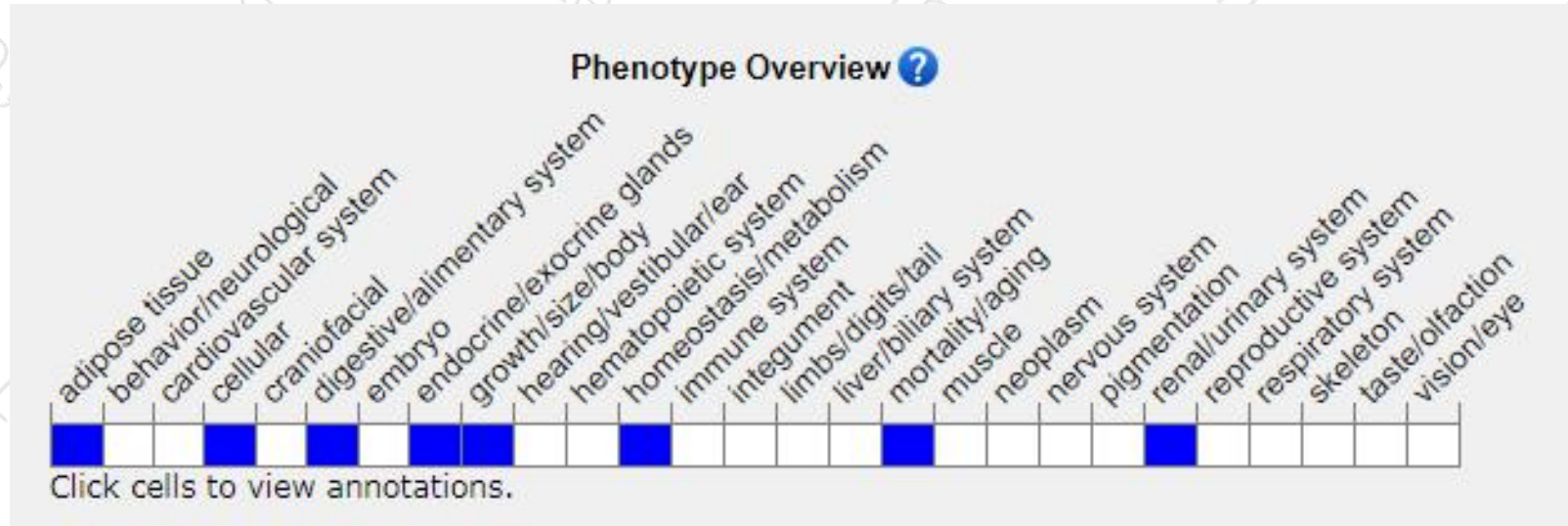
Genomic location distribution



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, omozygotes for targeted null mutations are deficient in endocrine cells of the glandular stomach and intestinal epithelium, and lack glucagon- and insulin-producing cells of the pancreas. Mutants die postnatally from diabetes.

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

Tel: 400-9660890

