

***Ncor1* Cas9-CKO Strategy**

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Project Overview

Project Name

Ncor1

Project type

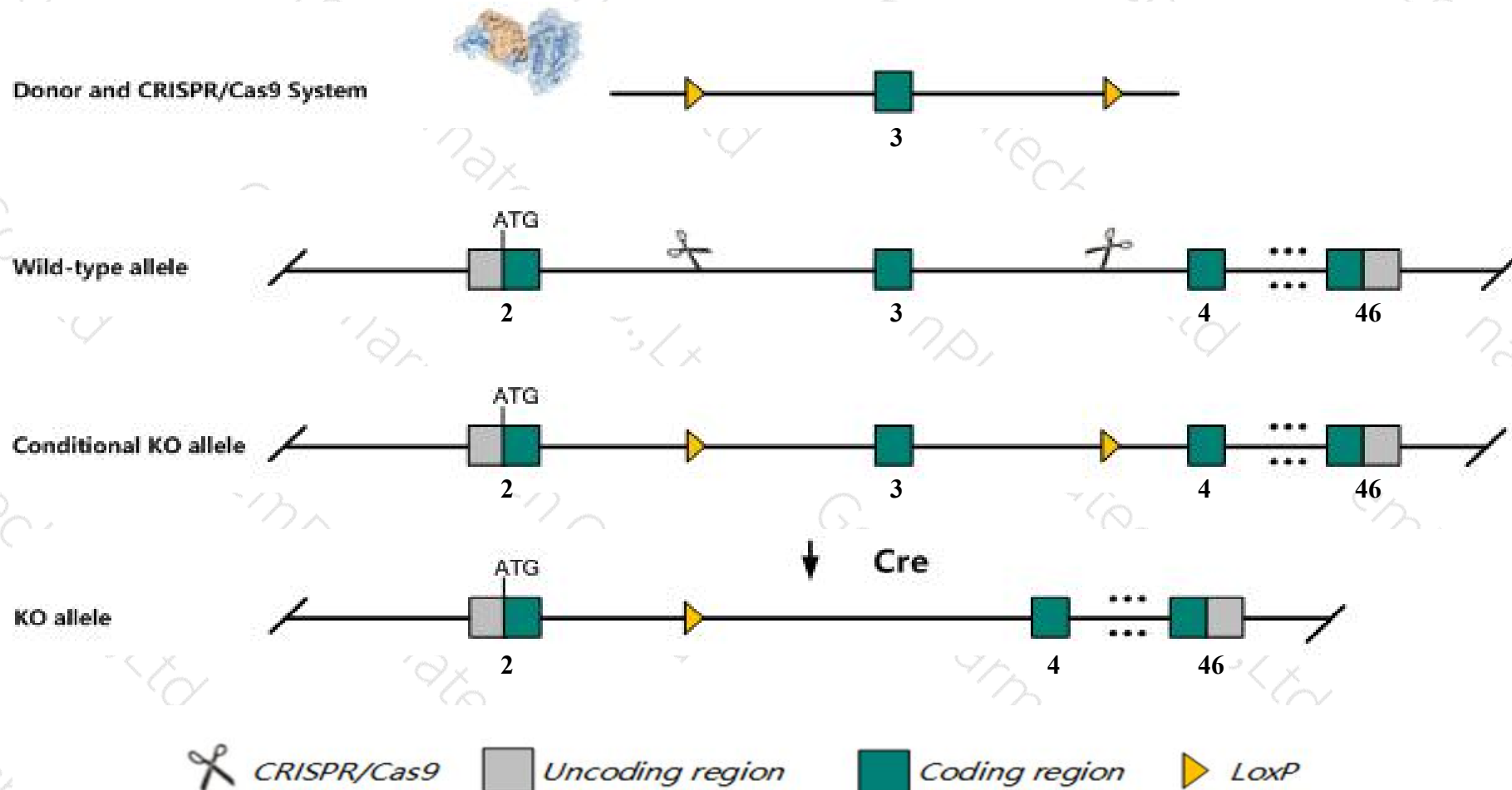
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Ncor1* gene. The schematic diagram is as follows:



Technical routes

- The *Ncor1* gene has 25 transcripts. According to the structure of *Ncor1* gene, exon3 of *Ncor1-201* (ENSMUST00000018645.12) transcript is recommended as the knockout region. The region contains 137bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Ncor1* 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, Mice homozygous for a targeted mutation in this gene exhibit embryonic lethality with erythrocytic, thymocytic and central nervous system development abnormalities. Mice homozygous for a hypomorphic allele exhibit increased thyroid hormone sensitivity under hypothyroid conditions.
- The *Ncor1* gene is located on the Chr11. 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)

Ncor1 nuclear receptor co-repressor 1 [Mus musculus (house mouse)]

Gene ID: 20185, updated on 9-Apr-2019

Summary



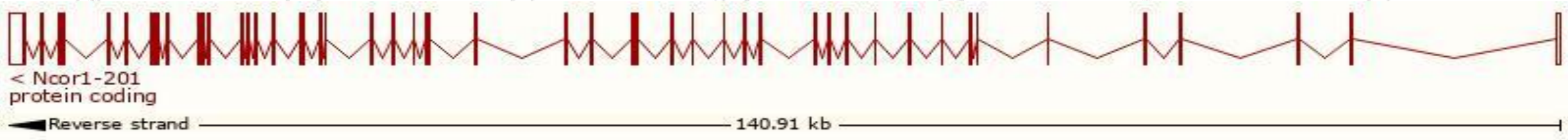
Official Symbol	Ncor1 provided by MGI
Official Full Name	nuclear receptor co-repressor 1 provided by MGI
Primary source	MGI:MGI:1349717
See related	Ensembl:ENSMUSG00000018501
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	5730405M06Rik, A230020K14Rik, N-CoR, RIP13, Rxrip13, mKIAA1047
Expression	Ubiquitous expression in thymus adult (RPKM 15.4), CNS E14 (RPKM 13.2) and 28 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

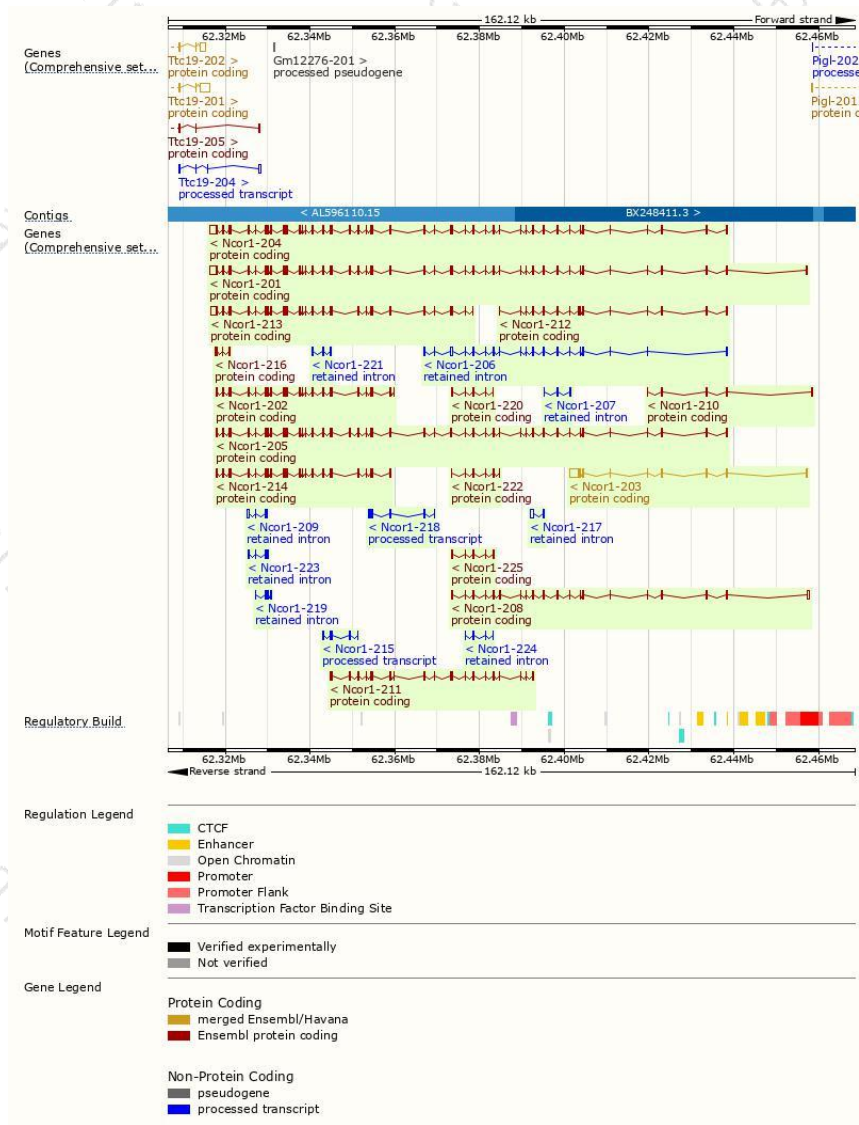
The gene has 25 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Ncor1-201	ENSMUST00000018645.12	9037	2454aa	Protein coding	CCDS56781	Q5RIM6	TSL5 GENCODE basic APPRIS P2
Ncor1-204	ENSMUST000000101066.9	8852	2454aa	Protein coding	CCDS56781	Q5RIM6	TSL5 GENCODE basic APPRIS P2
Ncor1-203	ENSMUST00000069456.10	3171	291aa	Protein coding	CCDS56782	Q8BK32	TSL1 GENCODE basic
Ncor1-205	ENSMUST000000101067.9	7243	2386aa	Protein coding	-	E9Q2B2	TSL5 GENCODE basic APPRIS ALT2
Ncor1-213	ENSMUST000000155712.8	6328	1724aa	Protein coding	-	Q8CHB6	CDS 5' incomplete TSL5
Ncor1-202	ENSMUST00000037575.14	4246	1399aa	Protein coding	-	E9Q9Y2	TSL1 GENCODE basic
Ncor1-214	ENSMUST000000156740.8	4215	1389aa	Protein coding	-	F7C134	CDS 5' incomplete TSL5
Ncor1-211	ENSMUST000000151498.8	2979	993aa	Protein coding	-	Q3UV08	5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL1
Ncor1-208	ENSMUST000000127471.8	2615	791aa	Protein coding	-	E9Q8K6	CDS 3' incomplete TSL5
Ncor1-212	ENSMUST000000155486.7	1695	541aa	Protein coding	-	Q5RIN0	CDS 3' incomplete TSL5
Ncor1-225	ENSMUST000000162236.7	781	261aa	Protein coding	-	F6SFD2	5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL3
Ncor1-222	ENSMUST000000161699.7	762	254aa	Protein coding	-	F6TWR1	5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL3
Ncor1-216	ENSMUST000000159315.1	716	157aa	Protein coding	-	F6TRR6	CDS 5' incomplete TSL2
Ncor1-220	ENSMUST000000161286.7	649	216aa	Protein coding	-	F6U338	5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL5
Ncor1-210	ENSMUST000000141447.1	646	163aa	Protein coding	-	E0CYX5	CDS 3' incomplete TSL5
Ncor1-218	ENSMUST000000161116.2	723	No protein	Processed transcript	-	-	TSL5
Ncor1-215	ENSMUST000000159224.4	563	No protein	Processed transcript	-	-	TSL5
Ncor1-206	ENSMUST000000101066.8	2815	No protein	Retained intron	-	-	TSL5
Ncor1-221	ENSMUST000000161432.1	800	No protein	Retained intron	-	-	TSL2
Ncor1-209	ENSMUST000000131911.7	793	No protein	Retained intron	-	-	TSL2
Ncor1-217	ENSMUST000000160171.1	690	No protein	Retained intron	-	-	TSL5
Ncor1-223	ENSMUST000000161767.7	667	No protein	Retained intron	-	-	TSL2
Ncor1-219	ENSMUST000000161281.1	643	No protein	Retained intron	-	-	TSL2
Ncor1-207	ENSMUST000000124027.2	566	No protein	Retained intron	-	-	TSL5
Ncor1-224	ENSMUST000000162077.1	452	No protein	Retained intron	-	-	TSL3

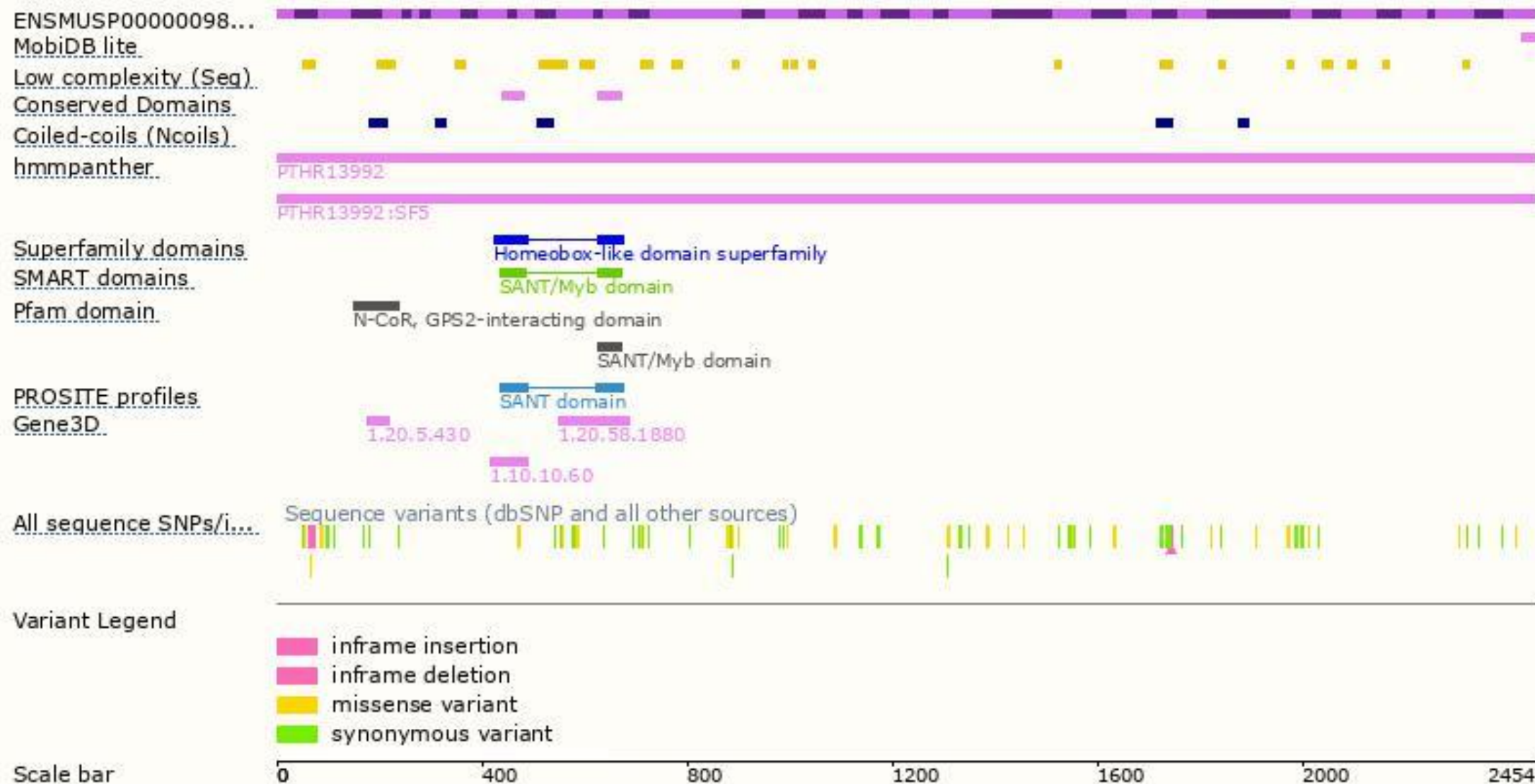
The strategy is based on the design of *Ncor1-201* transcript,The transcription is shown below



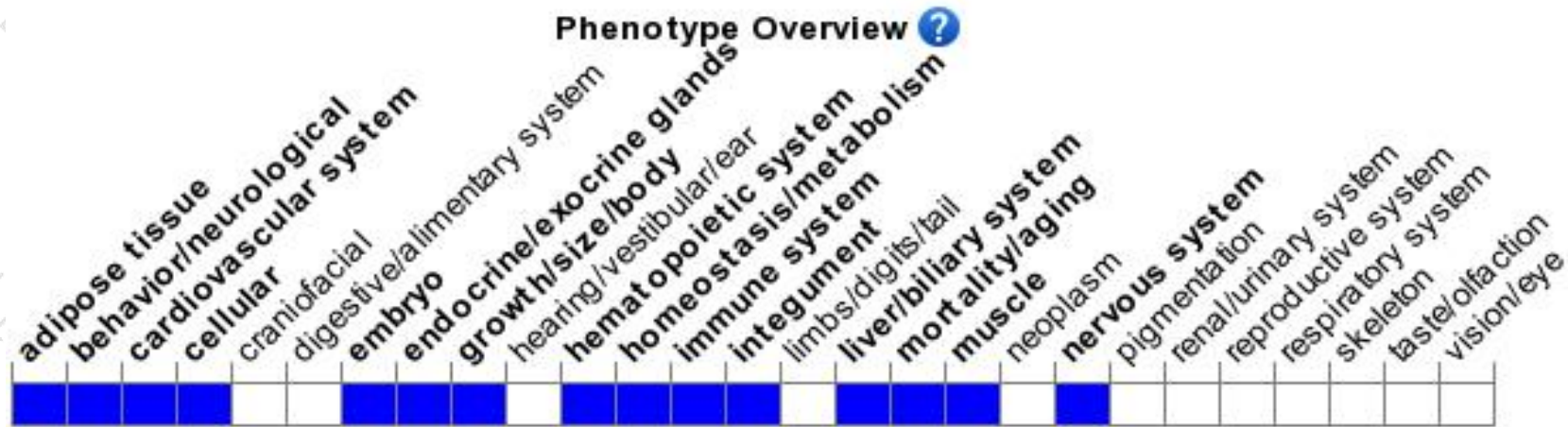
Genomic location distribution



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 homozygous for a targeted mutation in this gene exhibit embryonic lethality with erythrocytic, thymocytic and central nervous system development abnormalities. Mice homozygous for a hypomorphic allele exhibit increased thyroid hormone sensitivity under hypothyroid conditions.

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

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