

Prokr2 Cas9-CKO Strategy

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Design Date: 2019-8-5

Project Overview

Project Name

Prokr2

Project type

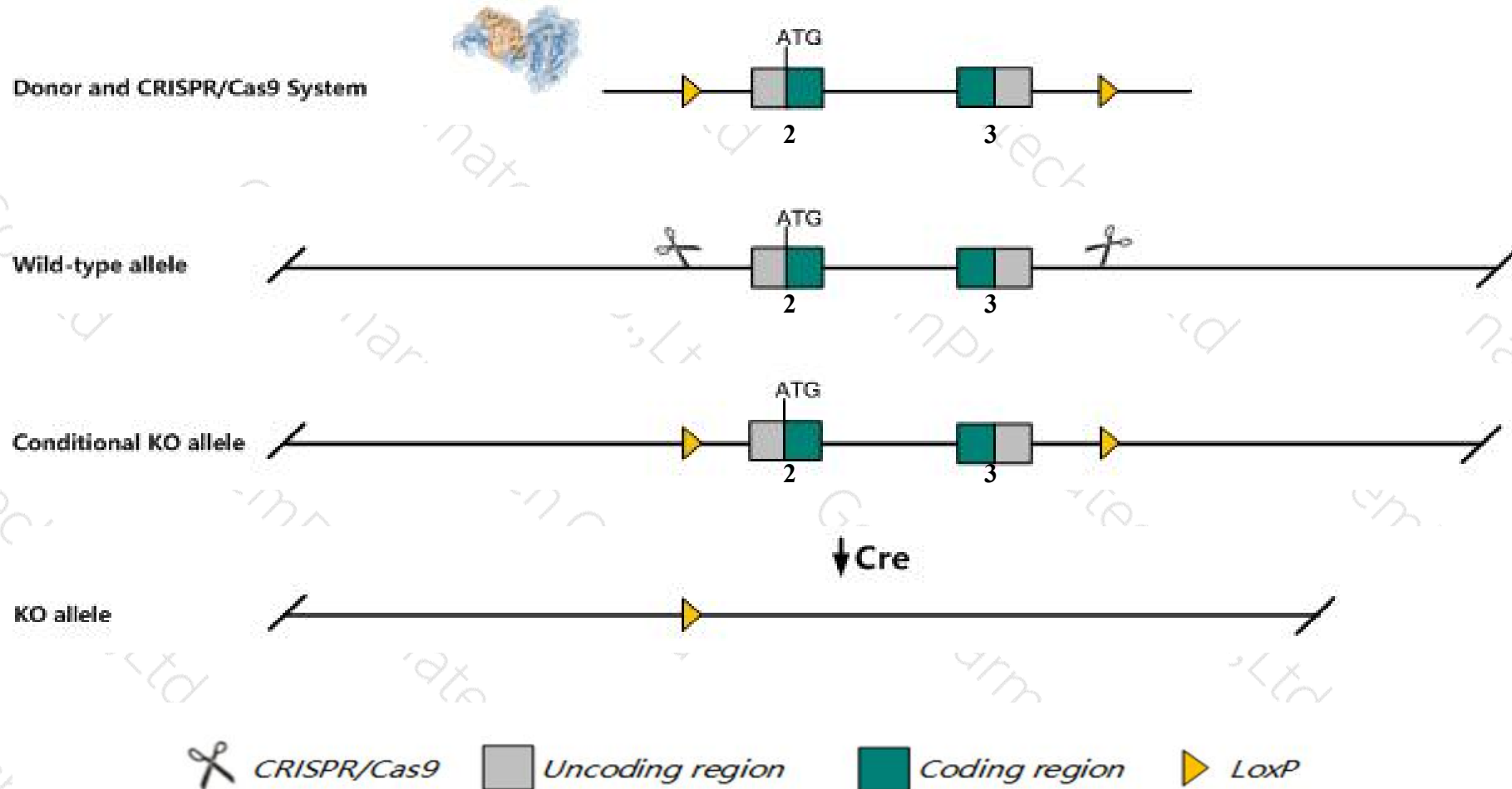
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Prokr2* gene. The schematic diagram is as follows:



- The *Prokr2* gene has 5 transcripts. According to the structure of *Prokr2* gene, exon2-exon3 of *Prokr2-201* (ENSMUST00000049997.13) 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 *Prokr2* 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, Homozygotes for a null allele show 50% neonatal lethality, olfactory bulb malformation, and reproductive system atrophy related to a lack of hypothalamic gonadotropin-releasing hormone synthesizing neurons. Homozygotes for another null allele show impaired circadian behavior and thermoregulation.
- The *Prokr2* 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)

Prokr2 prokineticin receptor 2 [Mus musculus (house mouse)]

Gene ID: 246313, updated on 31-Jan-2019

Summary



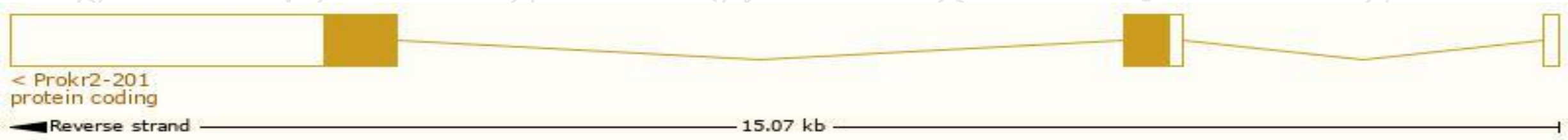
Official Symbol	Prokr2 provided by MGI
Official Full Name	prokineticin receptor 2 provided by MGI
Primary source	MGI:MGI:2181363
See related	Ensembl:ENSMUSG00000050558
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	B830005M06Rik, EG-VEGRF2, Gpcr73l1, Gpr73l1, PKR2
Expression	Biased expression in frontal lobe adult (RPKM 1.4), CNS E18 (RPKM 1.3) and 7 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

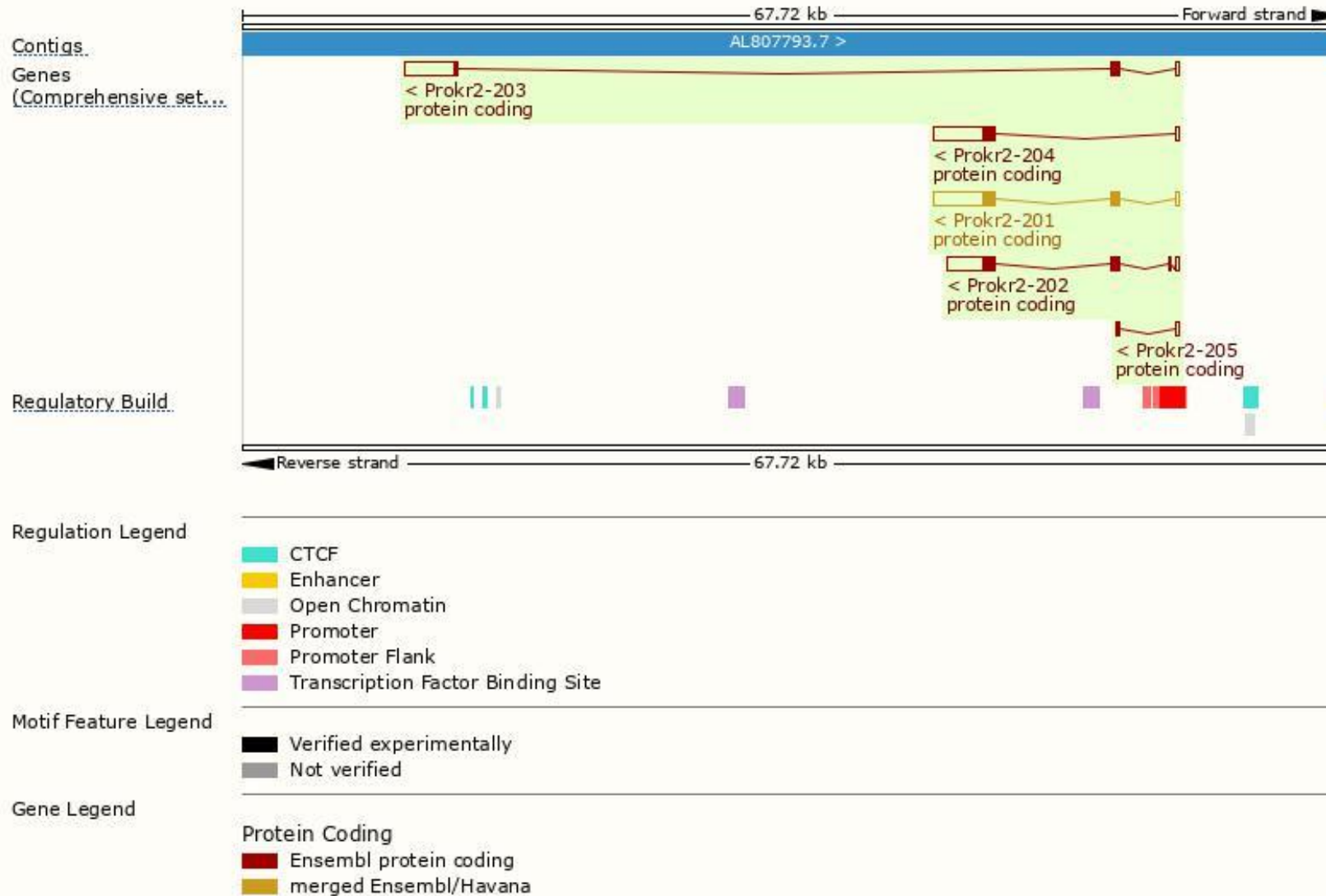
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Prokr2-201	ENSMUST00000049997.13	4486	381aa	Protein coding	CCDS16773	Q8K458	TSL:1 GENCODE basic APPRIS P1
Prokr2-202	ENSMUST00000110156.1	3829	381aa	Protein coding	CCDS16773	Q8K458	TSL:5 GENCODE basic APPRIS P1
Prokr2-203	ENSMUST00000110157.8	4005	198aa	Protein coding	-	A2AMQ7	TSL:1 GENCODE basic
Prokr2-204	ENSMUST00000142766.1	3932	220aa	Protein coding	-	E0CY28	TSL:1 GENCODE basic
Prokr2-205	ENSMUST00000145995.1	345	17aa	Protein coding	-	A2AMQ9	CDS 3' incomplete TSL:3

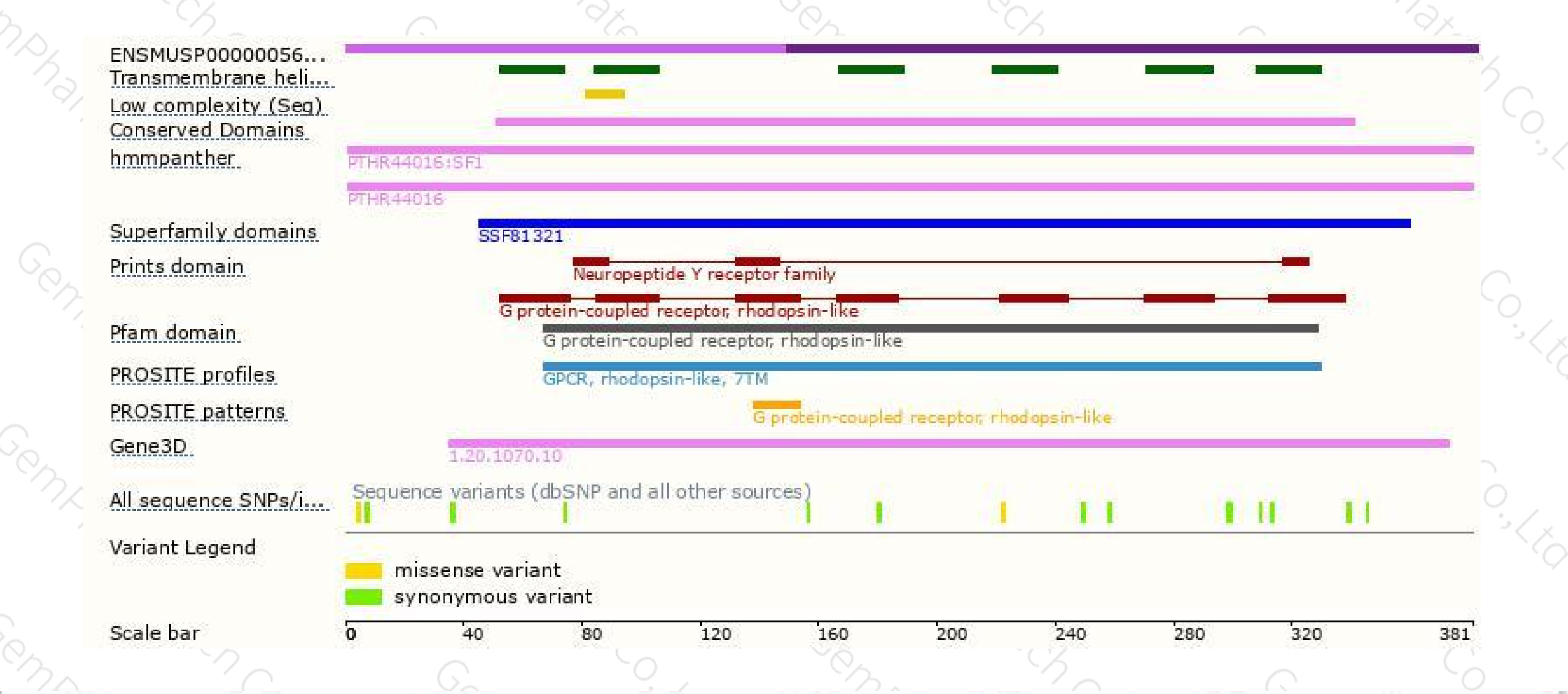
The strategy is based on the design of *Prokr2-201* transcript,The transcription is shown below



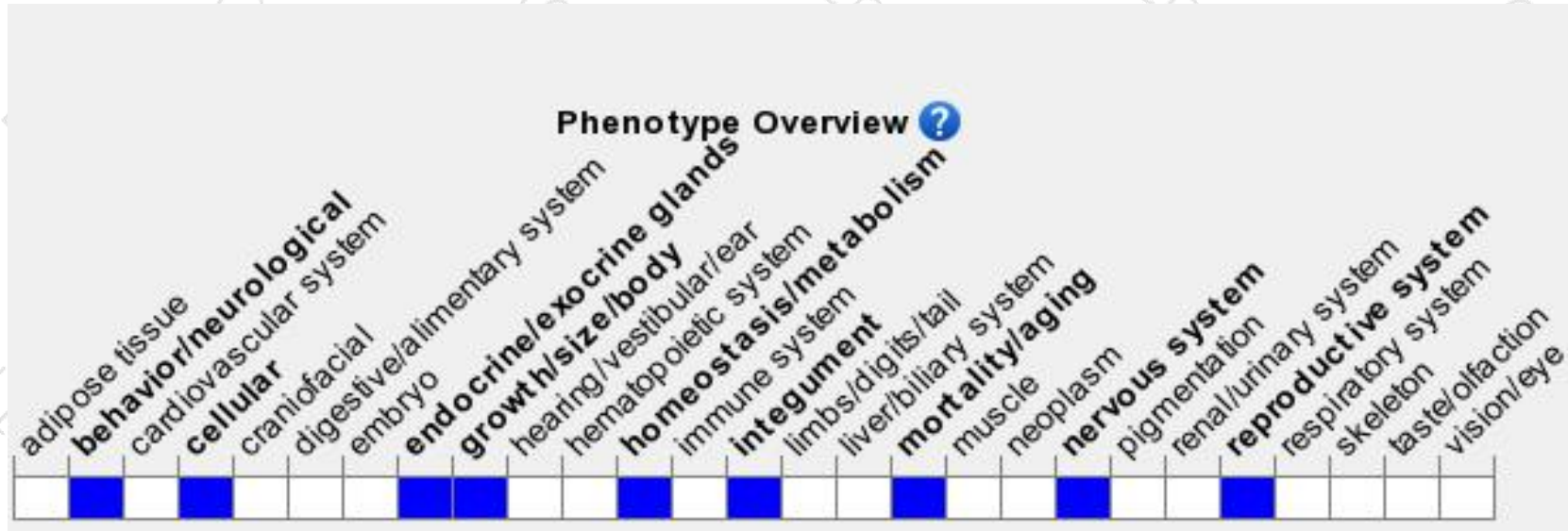
Genomic location distribution



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, Homozygotes for a null allele show 50% neonatal lethality, olfactory bulb malformation, and reproductive system atrophy related to a lack of hypothalamic gonadotropin-releasing hormone synthesizing neurons. Homozygotes for another null allele show impaired circadian behavior and thermoregulation.

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

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