

Slc2a4 Cas9-CKO Strategy

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Project Overview

Project Name

Slc2a4

Project type

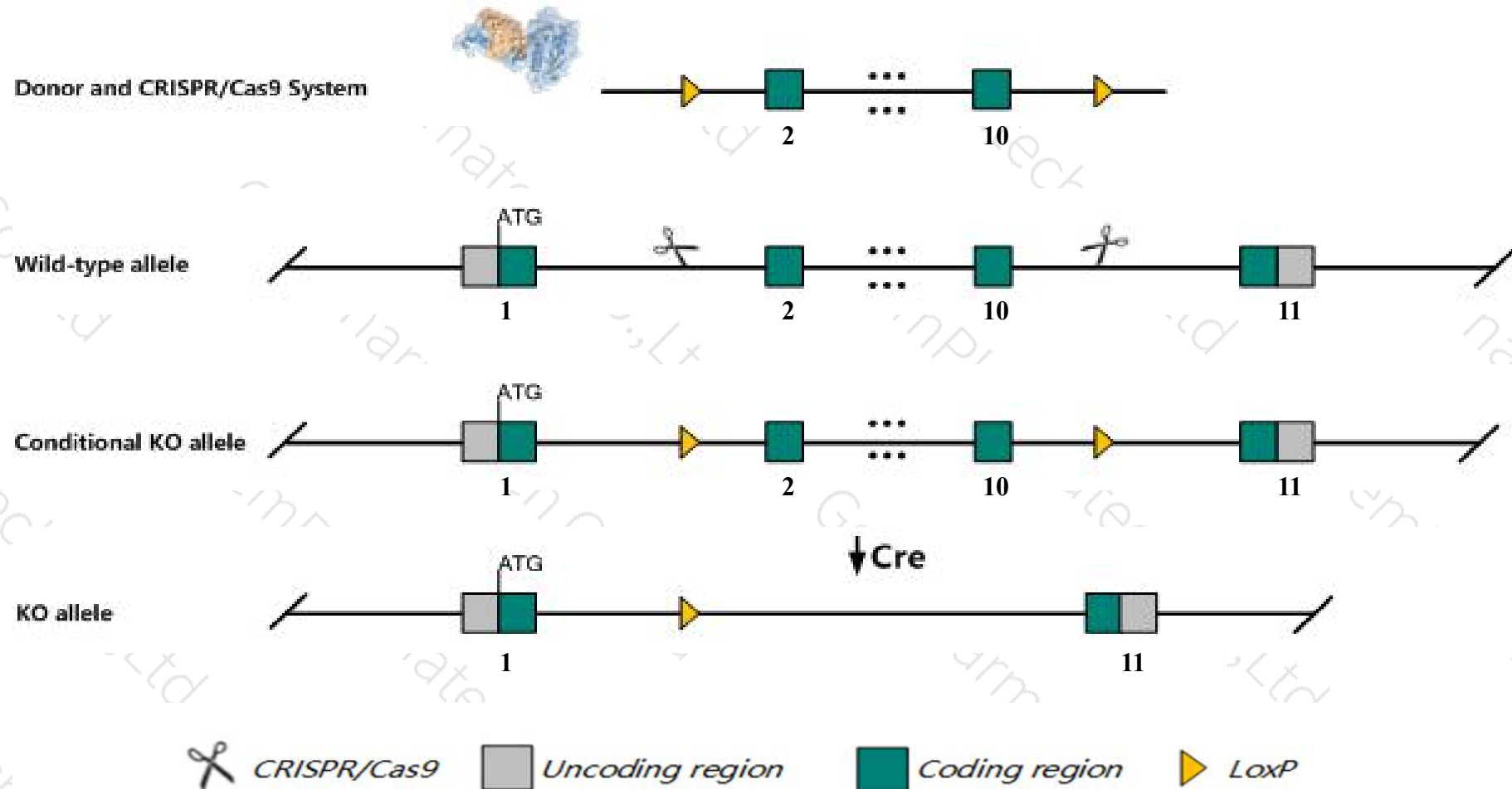
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Slc2a4* gene. The schematic diagram is as follows:



- The *Slc2a4* gene has 8 transcripts. According to the structure of *Slc2a4* gene, exon2-exon10 of *Slc2a4-201* (ENSMUST00000018710.12) 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 *Slc2a4* 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 inactivation of this gene causes impaired glucose metabolism in skeletal muscle and adipose tissue. Mice homozygous for a knock-out allele show premature death associated with cardiac hypertrophy, growth retardation, insulin resistance, reduced adipose tissue deposits, and muscle fatigue.
- The *Slc2a4* 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)

Slc2a4 solute carrier family 2 (facilitated glucose transporter), member 4 [Mus musculus (house mouse)]

Gene ID: 20528, updated on 19-Mar-2019

Summary



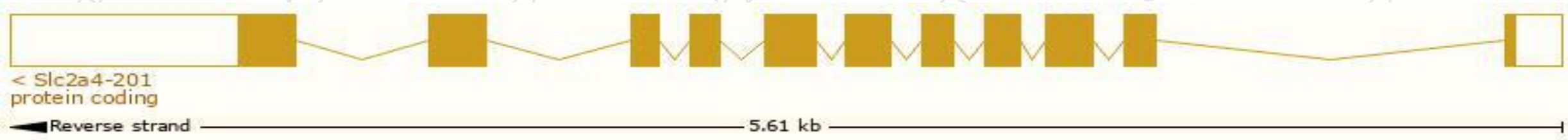
Official Symbol	Slc2a4 provided by MGI
Official Full Name	solute carrier family 2 (facilitated glucose transporter), member 4 provided by MGI
Primary source	MGI:MGI:95758
See related	Ensembl:ENSMUSG00000018566
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	GT2, Glut-4, Glut4, twgy
Expression	Biased expression in heart adult (RPKM 161.8), mammary gland adult (RPKM 97.3) and 12 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

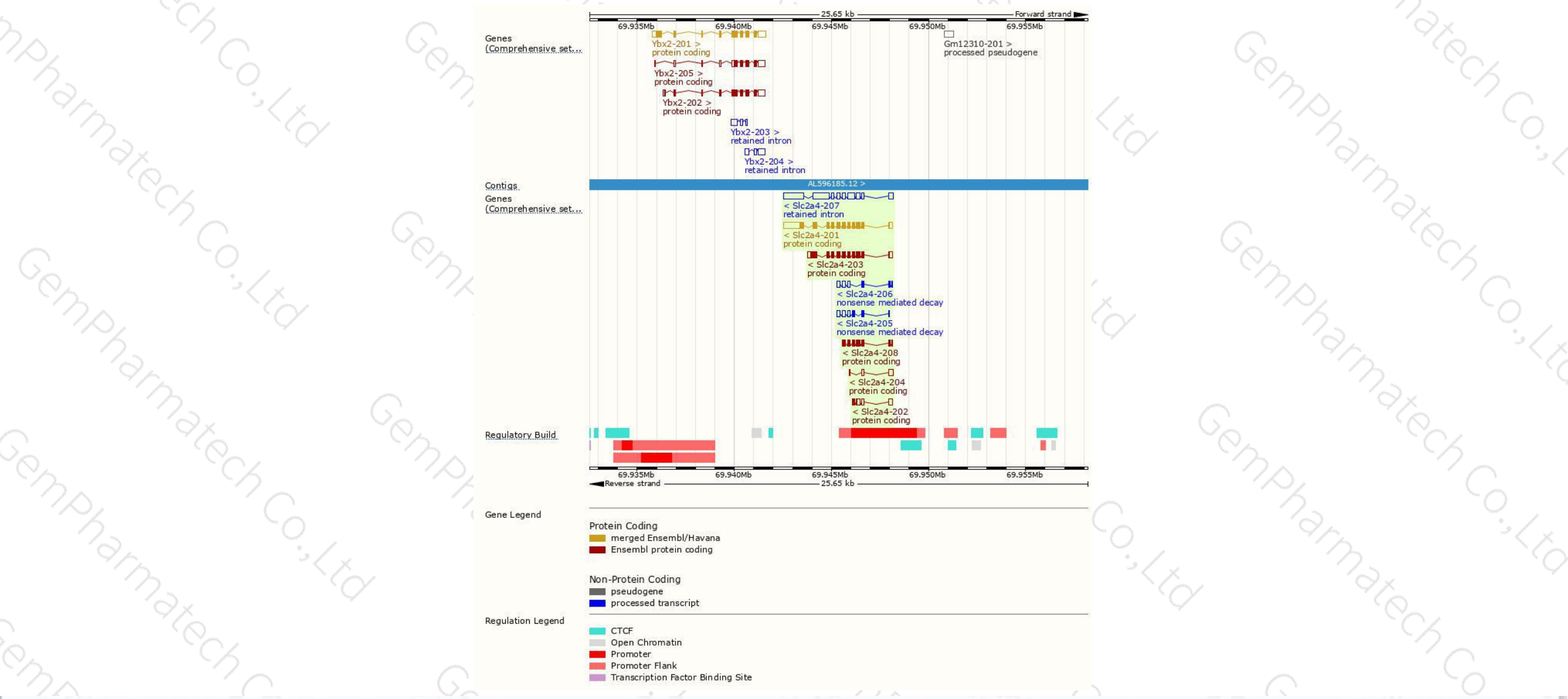
The gene has 8 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Slc2a4-201	ENSMUST00000018710.12	2524	509aa	Protein coding	CCDS36201	P14142	TSL:1 GENCODE basic APPRIS P1
Slc2a4-203	ENSMUST00000141837.8	1776	480aa	Protein coding	-	J3QK17	TSL:1 GENCODE basic
Slc2a4-208	ENSMUST00000179298.2	787	242aa	Protein coding	-	J3QNF4	CDS 3' incomplete TSL:3
Slc2a4-202	ENSMUST00000135437.2	626	57aa	Protein coding	-	J3QP46	CDS 3' incomplete TSL:5
Slc2a4-204	ENSMUST00000142500.7	408	7aa	Protein coding	-	A0A0G2JDD0	CDS 3' incomplete TSL:2
Slc2a4-205	ENSMUST00000152487.7	735	75aa	Nonsense mediated decay	-	J3QMX9	CDS 5' incomplete TSL:5
Slc2a4-206	ENSMUST00000178363.7	679	51aa	Nonsense mediated decay	-	J3QMU2	TSL:5
Slc2a4-207	ENSMUST00000178809.7	3193	No protein	Retained intron	-	-	TSL:2

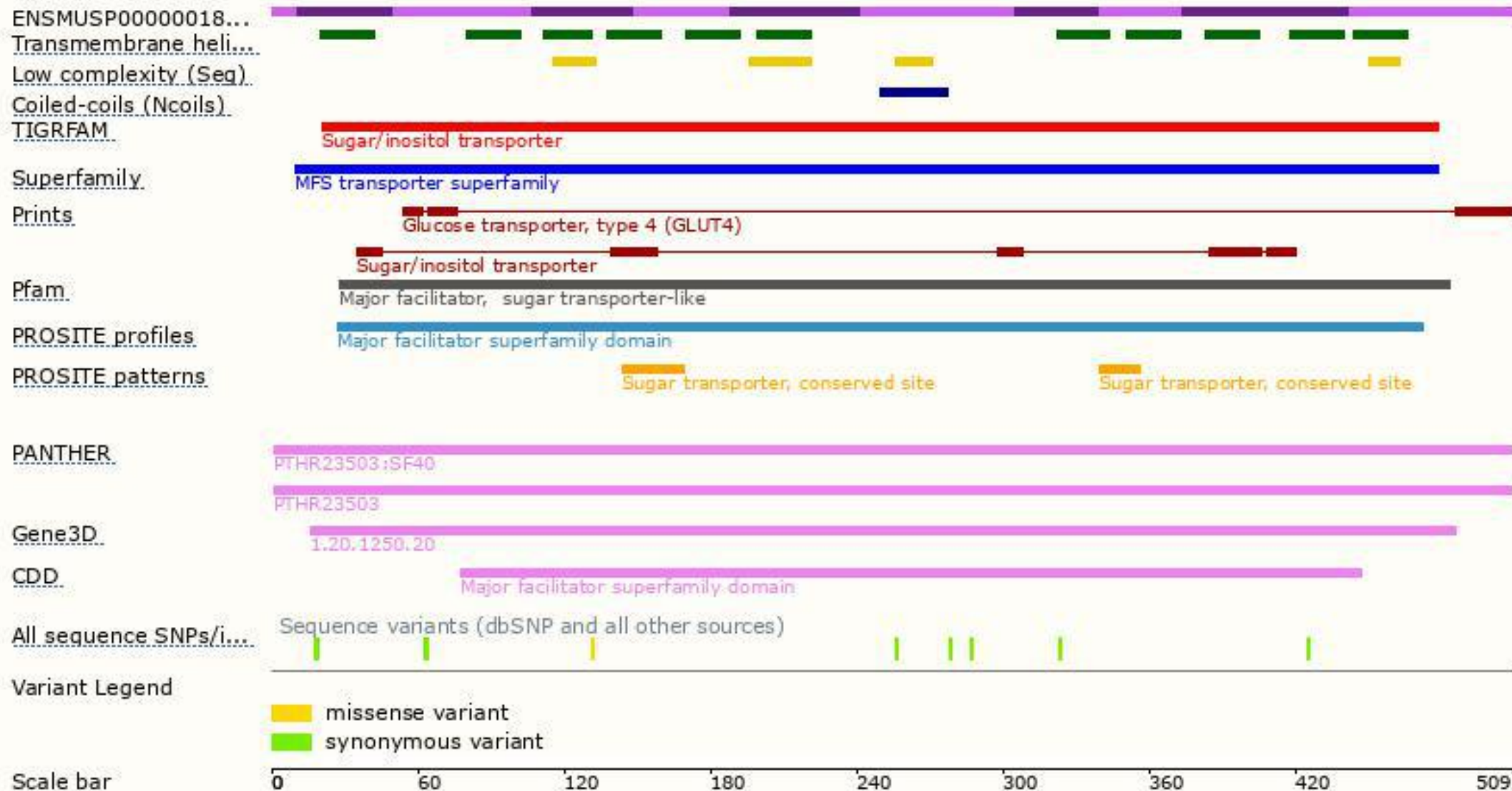
The strategy is based on the design of *Slc2a4-201* transcript,The transcription is shown below



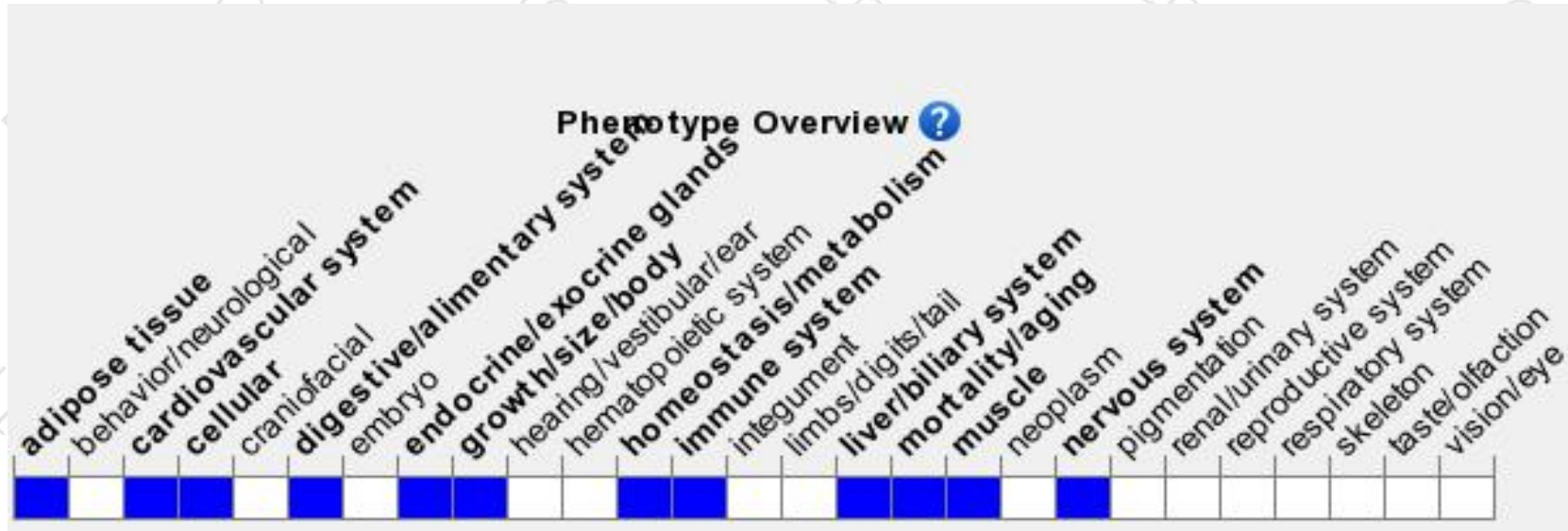
Genomic location distribution



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 inactivation of this gene causes impaired glucose metabolism in skeletal muscle and adipose tissue. Mice homozygous for a knock-out allele show premature death associated with cardiac hypertrophy, growth retardation, insulin resistance, reduced adipose tissue deposits, and muscle fatigue.

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

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