

Selenon Cas9-KO Strategy

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Project Overview

Project Name

Selenon

Project type

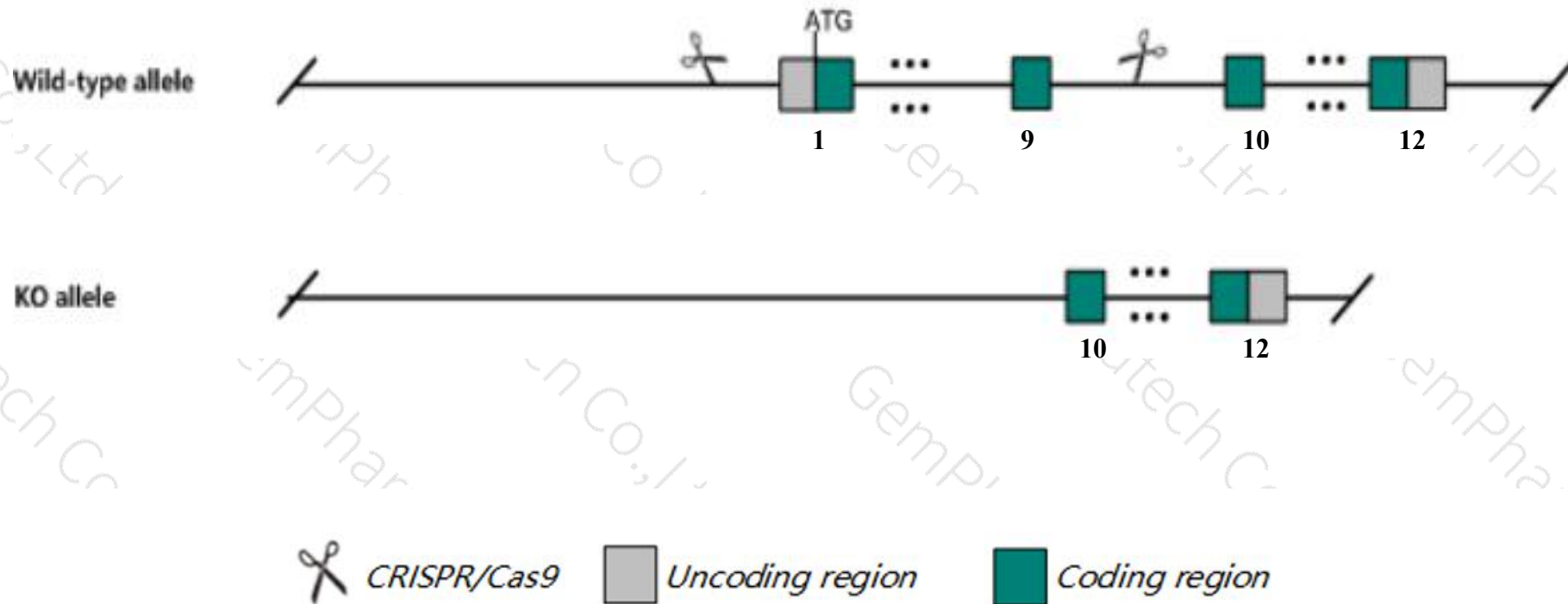
Cas9-KO

Strain background

C57BL/6JGpt

Knockout strategy

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



- The *Selenon* gene has 2 transcripts. According to the structure of *Selenon* gene, exon1-exon9 of *Selenon-201* (ENSMUST00000060435.6) transcript is recommended as the knockout region. The region contains start codon ATG. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Selenon* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Mice homozygous for a knock-out allele exhibit satellite cell loss and impaired muscle regeneration. Mice homozygous for a different knock-out allele exhibit subtle core lesions in skeletal muscle after induced oxidative stress and abnormal lung development.
- The *Selenon* gene is located on the Chr4. 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)

Selenon selenoprotein N [*Mus musculus* (house mouse)]

Gene ID: 74777, updated on 13-Mar-2020

Summary

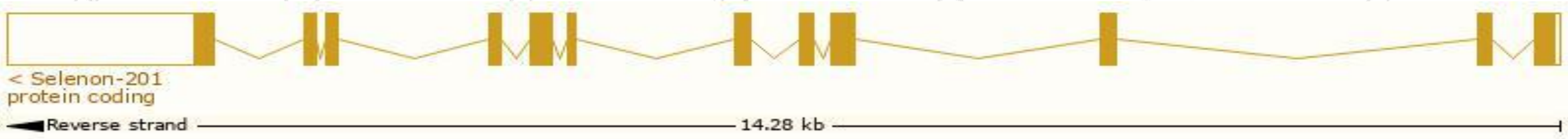
Official Symbol	Selenon provided by MGI
Official Full Name	selenoprotein N provided by MGI
Primary source	MGI:MGI:2151208
See related	Ensembl:ENSMUSG00000050989
Gene type	protein coding
RefSeq status	REVIEWED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	SeIN; Sepn1; AI414492; 1110019I12Rik
Summary	<p>This gene encodes a glycoprotein that is localized in the endoplasmic reticulum. It plays an important role in cell protection against oxidative stress, and in the regulation of redox-related calcium homeostasis. Mutations in the orthologous gene in human are associated with early onset muscle disorders, referred to as SEPNI-related myopathy. Knockout mice deleted for this gene exhibit abnormal lung development. This protein is a selenoprotein, containing the rare amino acid selenocysteine (Sec). Sec is encoded by the UGA codon, which normally signals translation termination. The 3' UTRs of selenoprotein mRNAs contain a conserved stem-loop structure, designated the Sec insertion sequence (SECIS) element, that is necessary for the recognition of UGA as a Sec codon, rather than as a stop signal. A second stop-codon redefinition element (SRE) adjacent to the UGA codon has been identified in this gene (PMID:15791204). SRE is a phylogenetically conserved stem-loop structure that stimulates readthrough at the UGA codon, and augments the Sec insertion efficiency by SECIS. [provided by RefSeq, Dec 2016]</p>
Expression	Ubiquitous expression in limb E14.5 (RPKM 26.2), ovary adult (RPKM 24.0) and 26 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

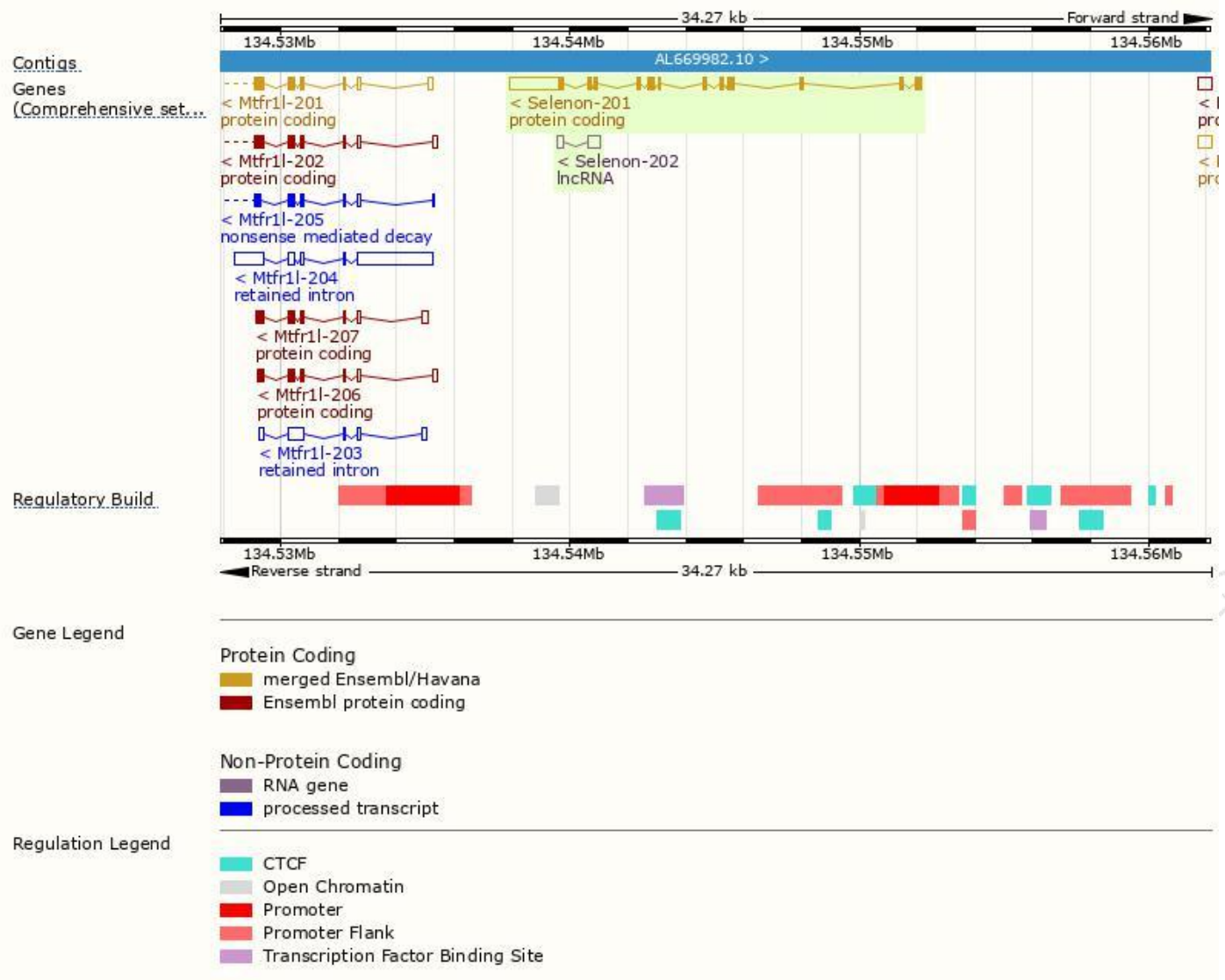
The gene has 2 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Selenon-201	ENSMUST00000060435.6	3445	557aa	Protein coding	CCDS38917	D3Z2R5	TSL:5 Gencode basic APPRIS P1
Selenon-202	ENSMUST00000127585.1	600	No protein	Processed transcript	-	-	TSL:1

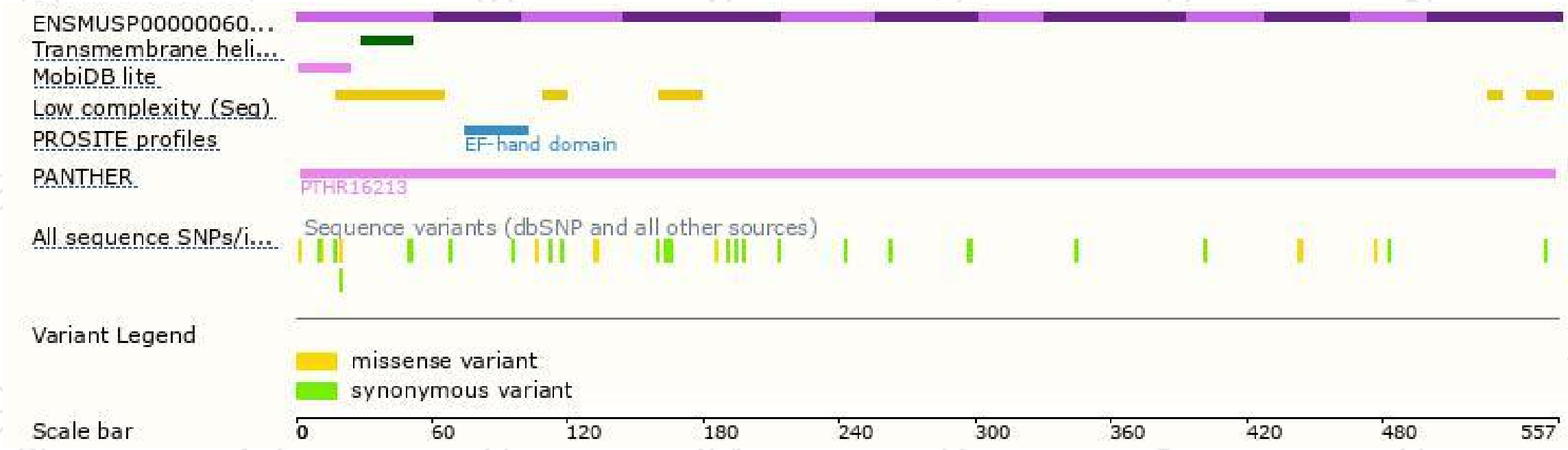
The strategy is based on the design of *Selenon-201* transcript,The transcription is shown below



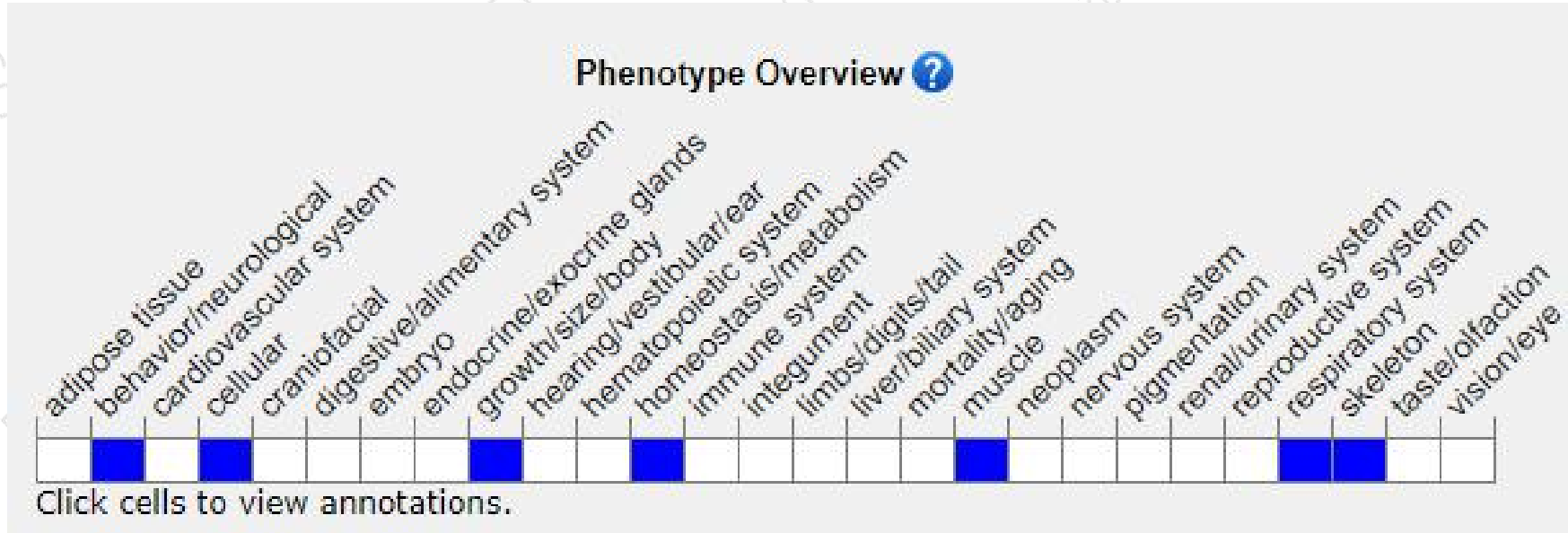
Genomic location distribution



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 knock-out allele exhibit satellite cell loss and impaired muscle regeneration. Mice homozygous for a different knock-out allele exhibit subtle core lesions in skeletal muscle after induced oxidative stress and abnormal lung development.

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

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