

Trex1 Cas9-KO Strategy

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Project Overview

Project Name

Trex1

Project type

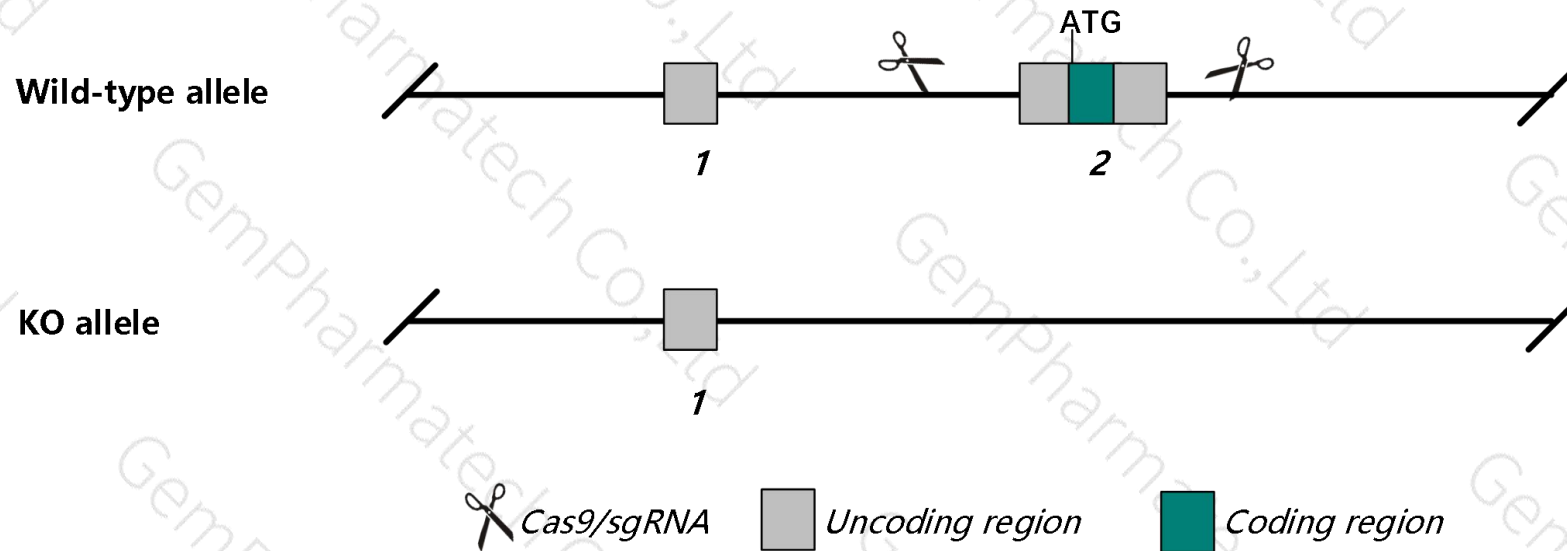
Cas9-KO

Strain background

C57BL/6JGpt

Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Trex1* gene. The schematic diagram is as follows:



- The *Trex1* gene has 2 transcripts. According to the structure of *Trex1* gene, exon2 of *Trex1-201* (ENSMUST00000061973.4) 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 *Trex1* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Nullizygous mice display premature death, cardiomyopathy, myocarditis, atrial thrombosis, and altered spleen morphology. Homozygotes for the D18N allele develop lupus-like disease with systemic inflammation, lymphoid hyperplasia, vasculitis, production of autoantibodies to dsDNA, and renal disease.
- Intron1-2 is small and its effect is unknown.
- The KO region may affect the function of *Shisa5*, *Atrip* gene.
- The *Trex1* gene is located on the Chr9. 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)

Trex1 three prime repair exonuclease 1 [Mus musculus (house mouse)]

Gene ID: 22040, updated on 13-Mar-2020

Summary



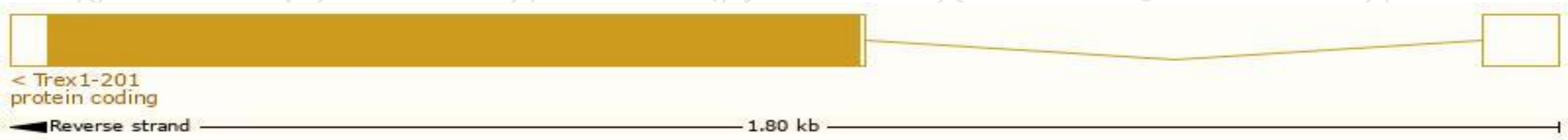
| | |
|---------------------------|---|
| Official Symbol | Trex1 provided by MGI |
| Official Full Name | three prime repair exonuclease 1 provided by MGI |
| Primary source | MGI:MGI:1328317 |
| See related | Ensembl:ENSMUSG00000049734 |
| Gene type | protein coding |
| RefSeq status | VALIDATED |
| Organism | Mus musculus |
| Lineage | Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus |
| Also known as | AU041952 |
| Expression | Ubiquitous expression in spleen adult (RPKM 79.9), mammary gland adult (RPKM 65.5) and 27 other tissues See more |
| Orthologs | human all |

Transcript information (Ensembl)

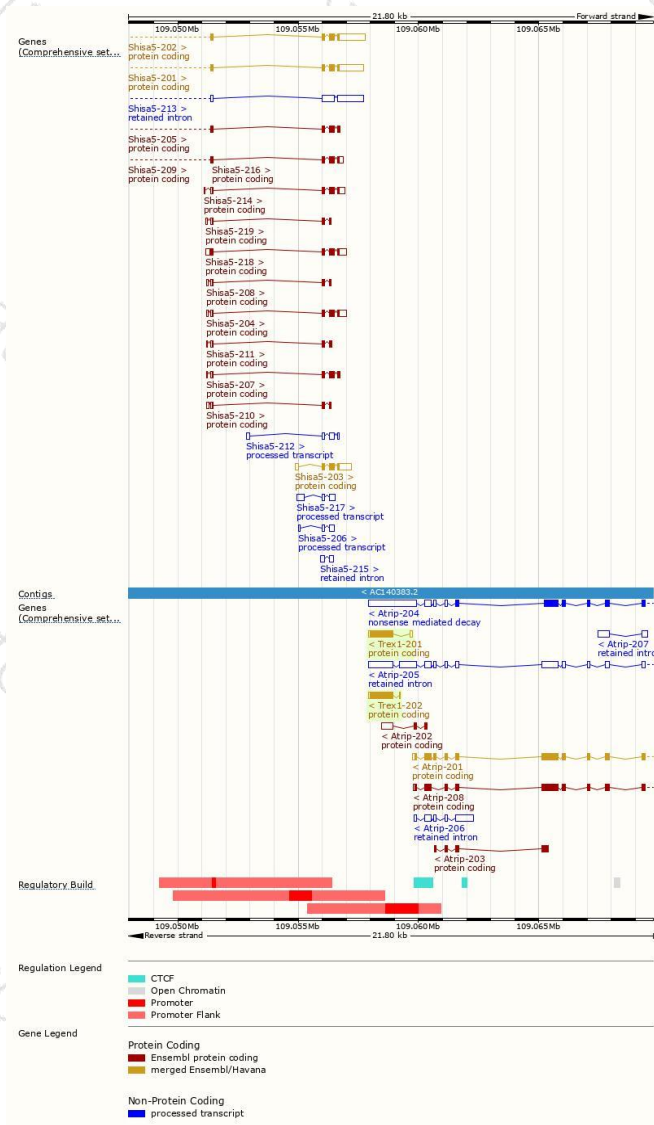
The gene has 2 transcripts,all transcripts are shown below:

| Name | Transcript ID | bp | Protein | Biotype | CCDS | UniProt | Flags |
|-----------|--------------------------------------|------|-----------------------|----------------|---------------------------|------------------------|---|
| Trex1-201 | ENSMUST00000061973.4 | 1084 | 314aa | Protein coding | CCDS23544 | Q91XB0 | TSL:1 GENCODE basic APPRIS is a system to annotate alternatively spliced transcripts based on a range of computational methods to identify the most functionally important transcript(s) of a gene. APPRIS P1 |
| Trex1-202 | ENSMUST00000112053.1 | 1054 | 314aa | Protein coding | CCDS23544 | Q91XB0 | TSL:1 GENCODE basic APPRIS is a system to annotate alternatively spliced transcripts based on a range of computational methods to identify the most functionally important transcript(s) of a gene. APPRIS P1 |

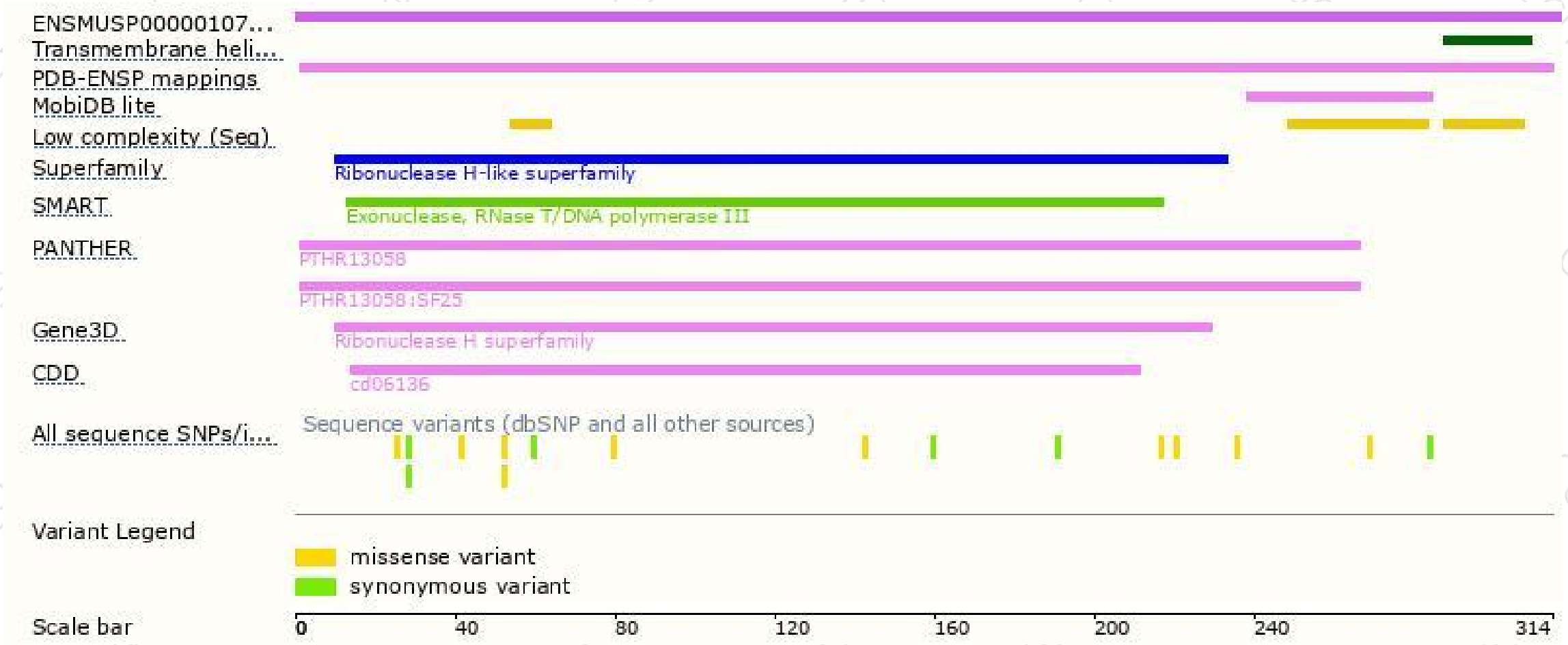
The strategy is based on the design of *Trex1-201* transcript,The transcription is shown below



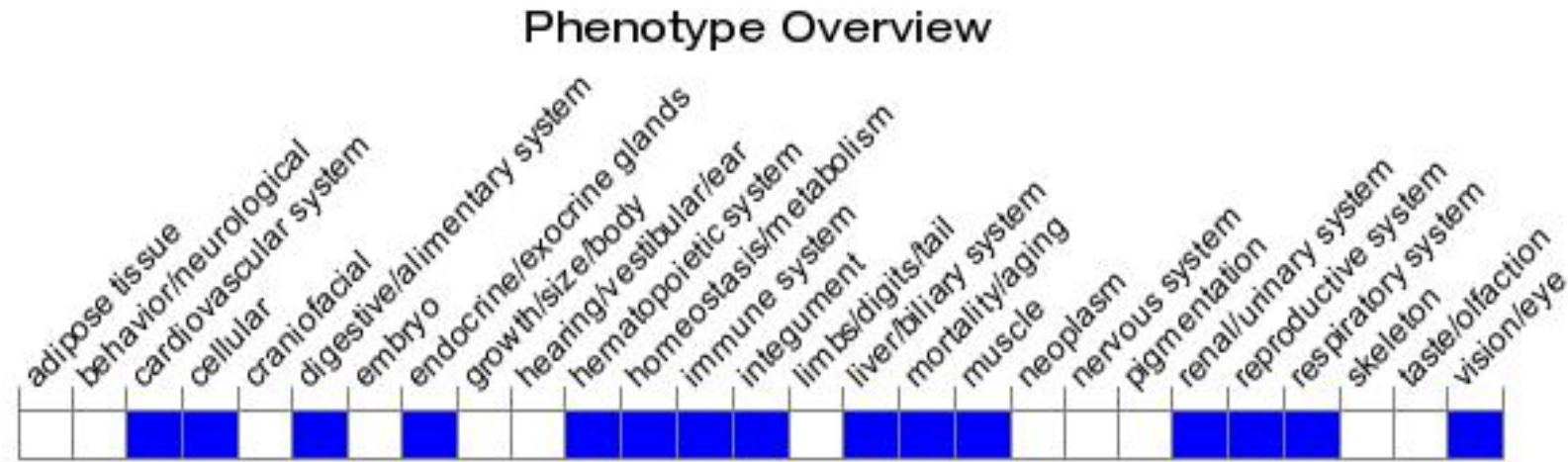
Genomic location distribution



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, Nullizygous mice display premature death, cardiomyopathy, myocarditis, atrial thrombosis, and altered spleen morphology. Homozygotes for the D18N allele develop lupus-like disease with systemic inflammation, lymphoid hyperplasia, vasculitis, production of autoantibodies to dsDNA, and renal disease.

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

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