

Met Cas9-KO Strategy

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Project Overview

Project Name

Met

Project type

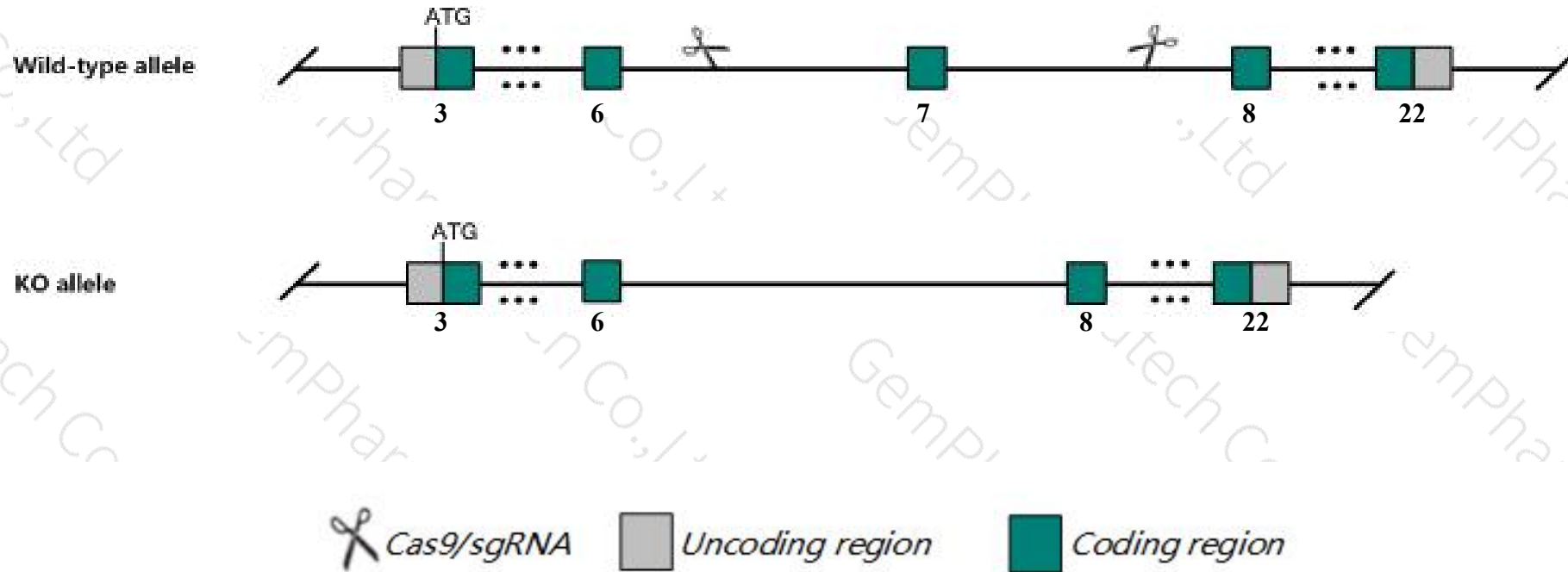
Cas9-KO

Strain background

C57BL/6J

Knockout strategy

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



- The *Met* gene has 7 transcripts. According to the structure of *Met* gene, exon7 of *Met-203* (ENSMUST00000115443.7) transcript is recommended as the knockout region. The region contains 161bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Met* gene. The brief process is as follows: sgRNA was transcribed in vitro. Cas9 and sgRNA were microinjected into the fertilized eggs of C57BL/6J 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/6J mice.

- According to the existing MGI data, homozygous null mutants exhibit impaired embryonic development resulting in death. Abnormalities observed in various mutant lines include muscle agenesis due to impaired migration of myogenic precursors, defects of motor axon migration, and placental and liver defects.
- The *Met* gene is located on the Chr6. 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)

Met met proto-oncogene [Mus musculus (house mouse)]

Gene ID: 17295, updated on 19-Mar-2019

Summary



| | |
|---------------------------|---|
| Official Symbol | Met provided by MGI |
| Official Full Name | met proto-oncogene provided by MGI |
| Primary source | MGI:MGI:96969 |
| See related | Ensembl:ENSMUSG00000009376 |
| 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 | AI838057, HGF, HGFR, Par4, c-Met |
| Expression | Broad expression in kidney adult (RPKM 10.5), bladder adult (RPKM 6.1) and 21 other tissues See more |
| Orthologs | human all |

Transcript information (Ensembl)

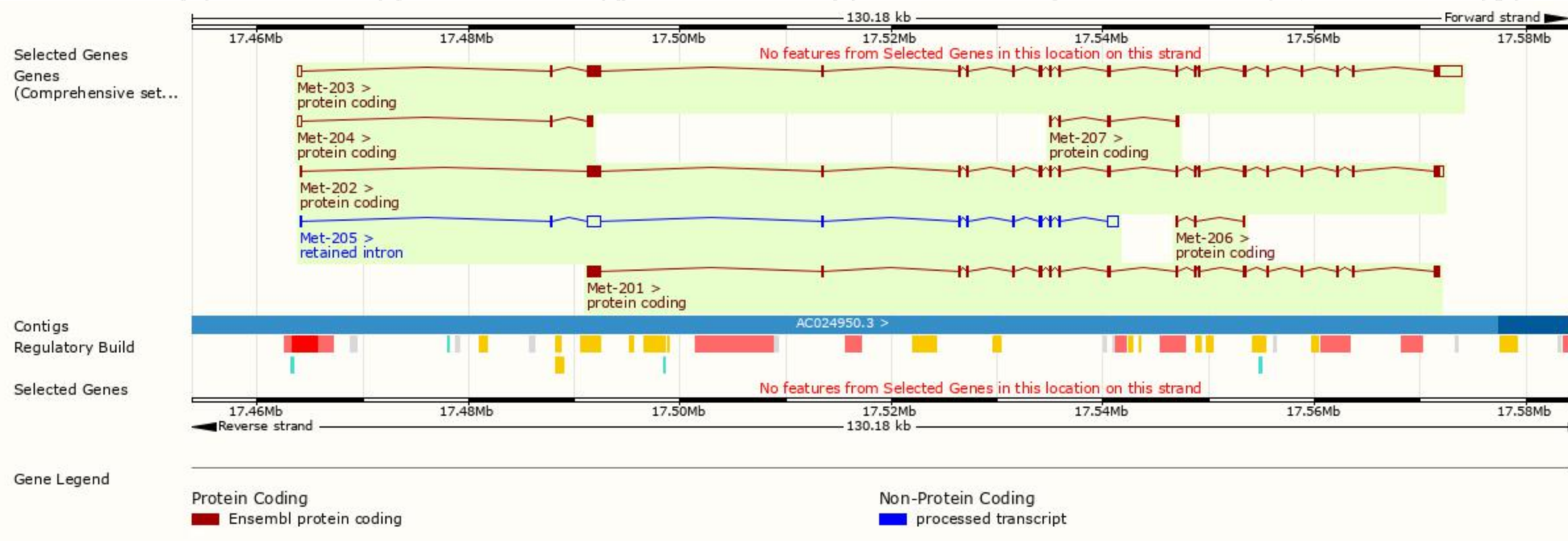
The gene has 7 transcripts,all transcripts are shown below:

| Name | Transcript ID | bp | Protein | Biotype | CCDS | UniProt | Flags |
|---------|---------------------------------------|------|------------------------|-----------------|---------------------------|------------------------|---|
| Met-203 | ENSMUST00000115443.7 | 6809 | 1379aa | Protein coding | CCDS19925 | F8VQL0 | TSL:5 GENCODE basic APPRIS P1 |
| Met-202 | ENSMUST00000115442.7 | 4663 | 1379aa | Protein coding | CCDS19925 | F8VQL0 | TSL:5 GENCODE basic APPRIS P1 |
| Met-201 | ENSMUST00000080469.11 | 4140 | 1379aa | Protein coding | CCDS19925 | F8VQL0 | TSL:1 GENCODE basic APPRIS P1 |
| Met-204 | ENSMUST00000140070.7 | 884 | 169aa | Protein coding | - | D3YVY2 | CDS 3' incomplete TSL:3 |
| Met-207 | ENSMUST00000152802.1 | 683 | 214aa | Protein coding | - | F6X333 | CDS 5' incomplete TSL:5 |
| Met-206 | ENSMUST00000148903.1 | 470 | 157aa | Protein coding | - | F6RCC5 | 5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL:3 |
| Met-205 | ENSMUST00000145473.1 | 3541 | No protein | Retained intron | - | - | TSL:1 |

The strategy is based on the design of *Met-203* transcript,The transcription is shown below



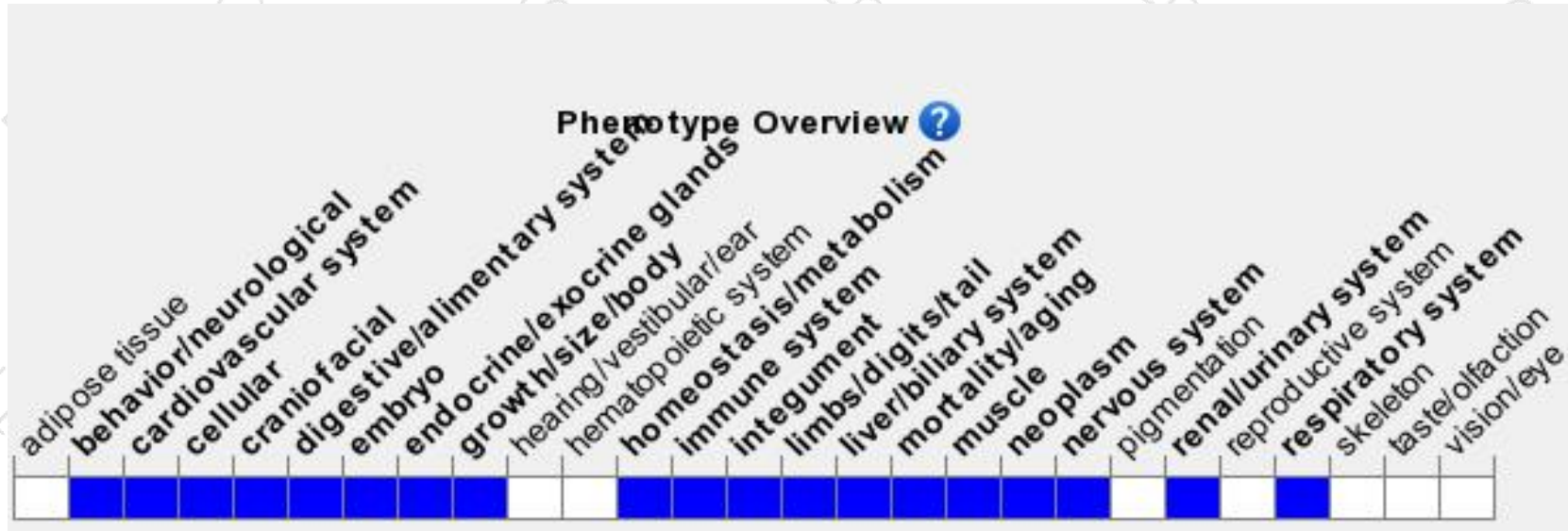
Genomic location distribution



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 null mutants exhibit impaired embryonic development resulting in death.

Abnormalities observed in various mutant lines include muscle agenesis due to impaired migration of myogenic precursors, defects of motor axon migration, and placental and liver defects.

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

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