

# ***Jund*** Cas9-CKO Strategy

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**Reviewer: Shilei Zhu**

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# Project Overview

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**Project Name**

***Jund***

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**Project type**

**Cas9-CKO**

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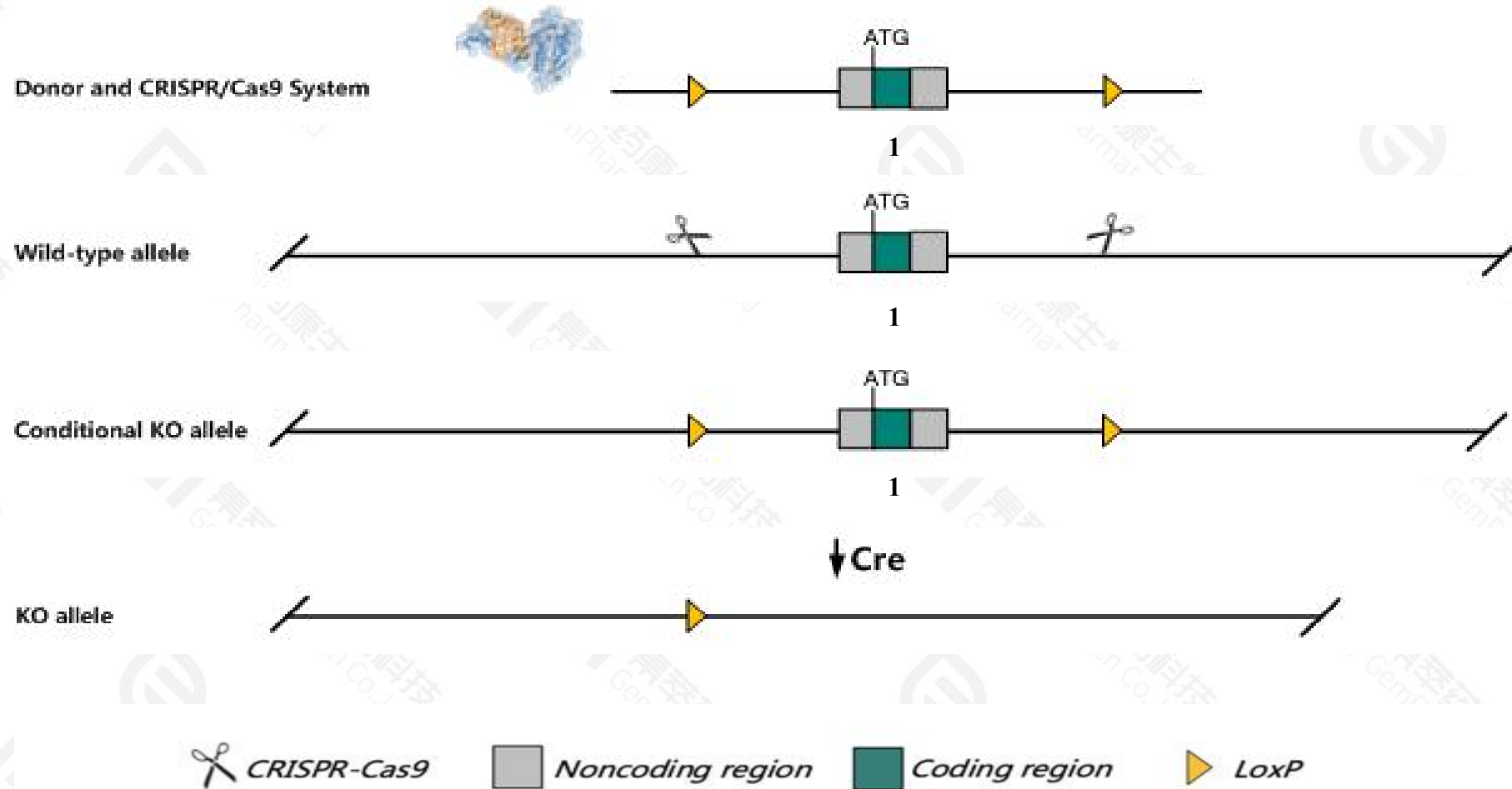
**Strain background**

**C57BL/6JGpt**

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# Conditional Knockout strategy

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



- The *Jund* gene has 1 transcript. According to the structure of *Jund* gene, exon1 of *Jund-201*(ENSMUST00000095267.8) 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 *Jund* 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 show reduced growth, sensitivity to LPS-induced hepatitis, and male infertility due to hormonal imbalance and impaired spermatogenesis. Targeted cells and mice are sensitive to p53-dependent stress and TNF-induced apoptosis, and show aberrant T cell proliferation and Th2 differentiation.
- *Gm11175* gene will be deleted.
- The flox region is about ~2.5kb away from the N-terminal of *Iqcn* gene, this strategy may influence the regulatory function of the N-terminal of *Iqcn* gene.
- The *Jund* gene is located on the Chr8. 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)

## Jund jun D proto-oncogene [Mus musculus (house mouse)]

Gene ID: 16478, updated on 13-Mar-2020

### Summary

**Official Symbol** Jund provided by [MGI](#)

**Official Full Name** jun D proto-oncogene provided by [MGI](#)

**Primary source** [MGI:MGI:96648](#)

**See related** [Ensembl:ENSMUSG00000071076](#)

**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** Jund1

**Summary** The protein encoded by this intronless gene is a member of the JUN family, and a functional component of the AP1 transcription factor complex. This protein has been proposed to protect cells from p53-dependent senescence and apoptosis. Alternative translation initiation site usage results in the production of different isoforms (PMID:12105216). [provided by RefSeq, Nov 2013]

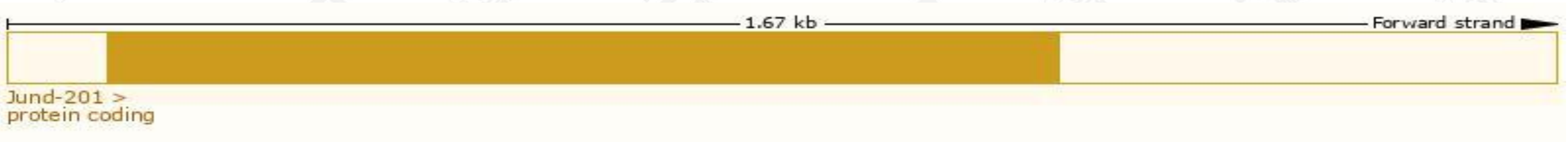
**Orthologs** [human](#) [all](#)

# Transcript information (Ensembl)

The gene has 1 transcript,and the transcript is shown below:

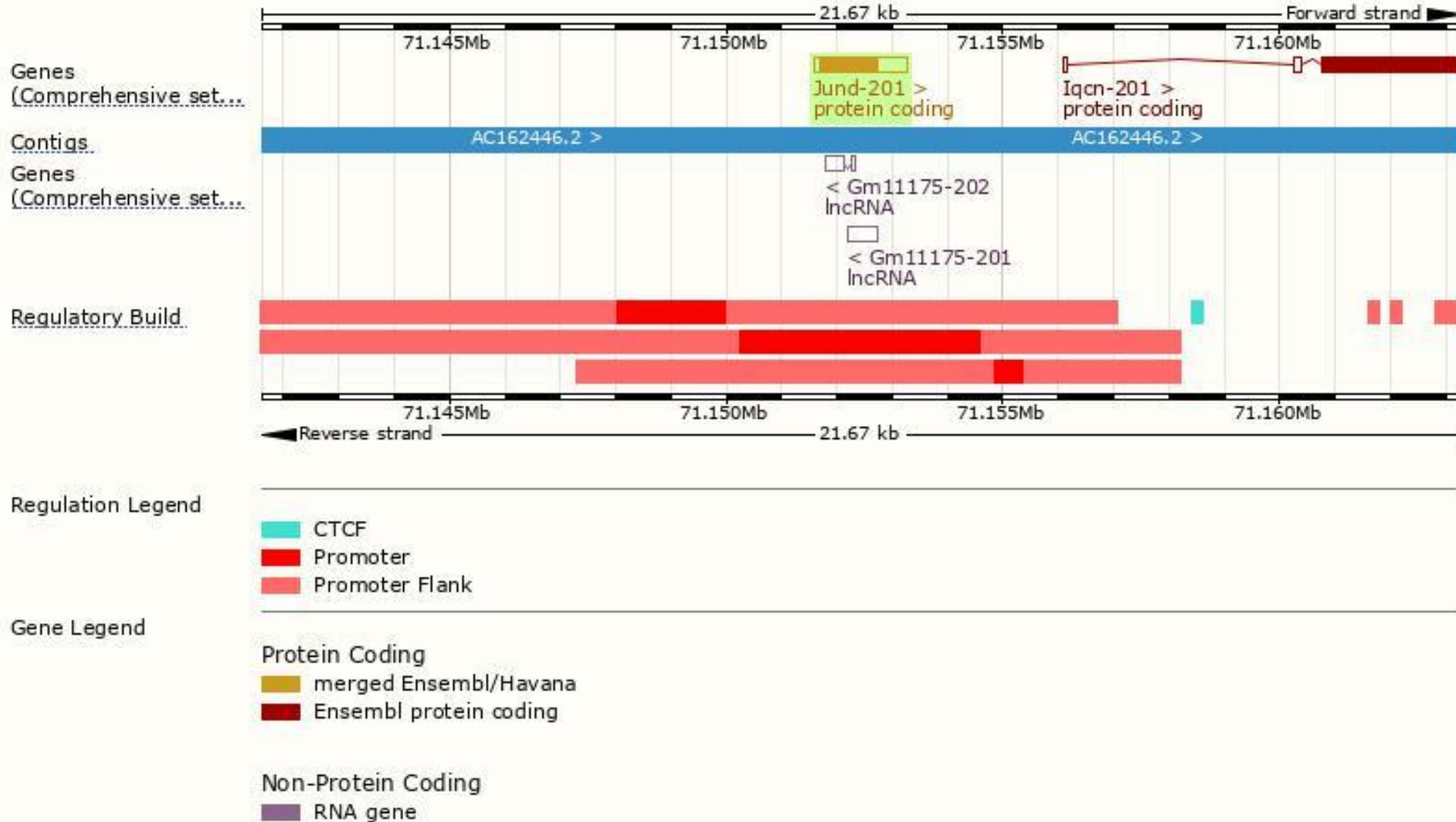
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Jund-201	<a href="#">ENSMUST00000095267.5</a>	1668	<a href="#">341aa</a>	Protein coding	<a href="#">CCDS40375</a>	<a href="#">P15066</a>	TSL:NA GENCODE basic APPRIS is a system to annotate alternatively spliced transcripts based on a range of computational methods to identify the most functionally important transcript(s) of a gene. APPRIS P1

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





# Genomic location distribution

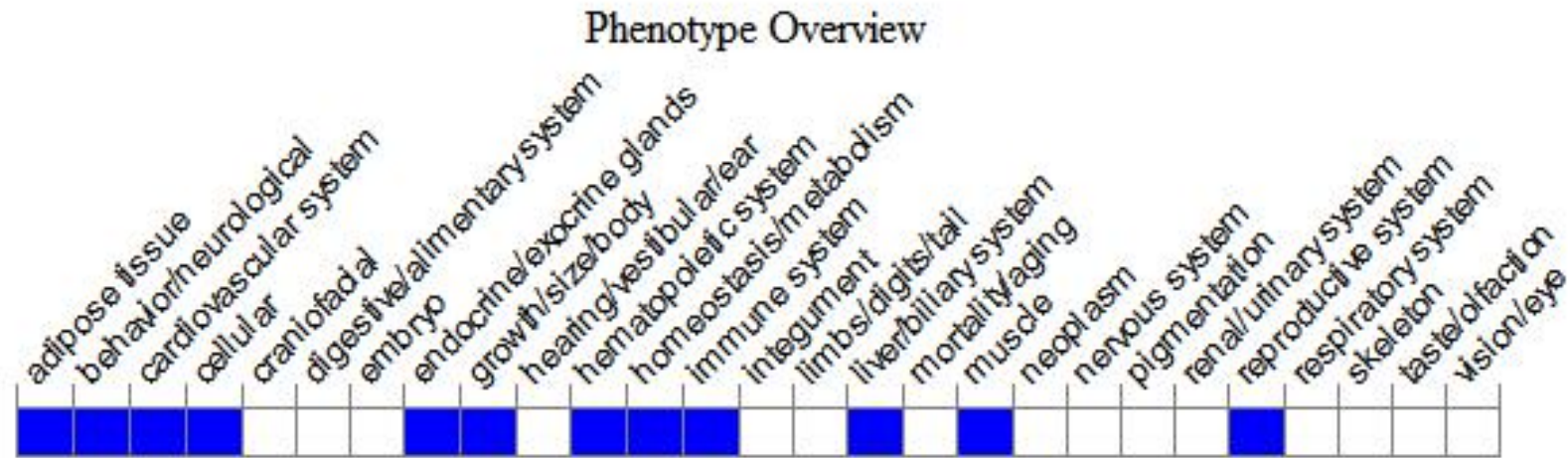




# Protein domain



# 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 show reduced growth, sensitivity to LPS-induced hepatitis, and male infertility due to hormonal imbalance and impaired spermatogenesis. Targeted cells and mice are sensitive to p53-dependent stress and TNF-induced apoptosis, and show aberrant T cell proliferation and Th2 differentiation.

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

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