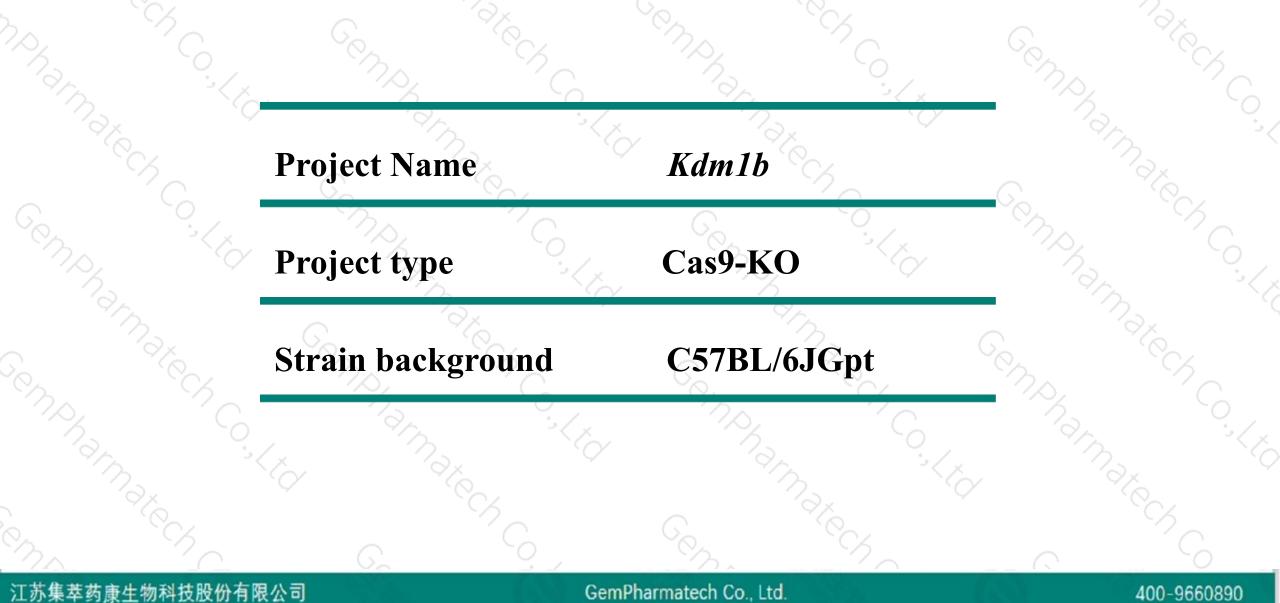


# Kdm1b Cas9-KO Strategy

Designer: Xueting Zhang Reviewer:Yanhua Shen Date:2020-02-24

## **Project Overview**

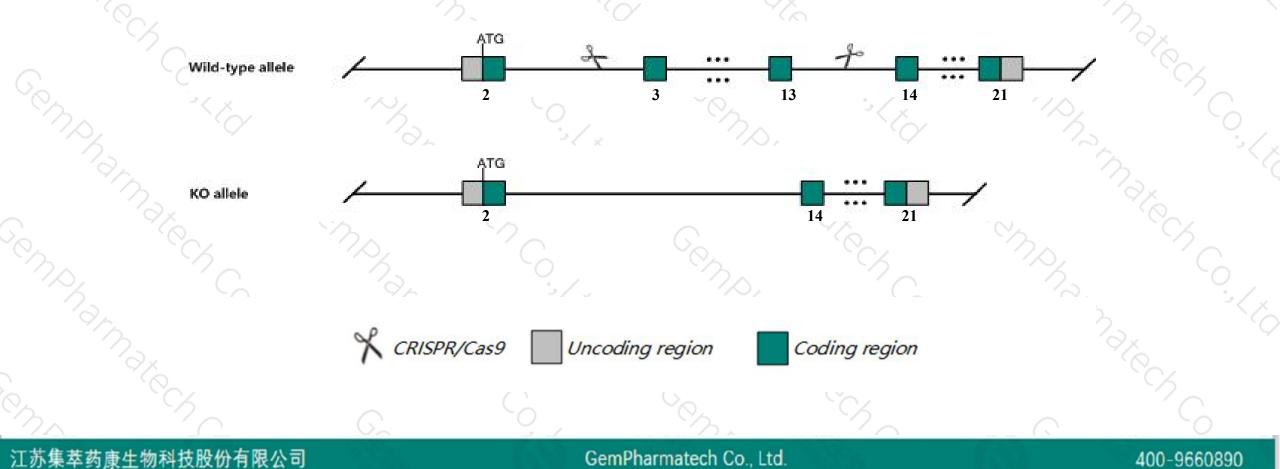




# **Knockout** strategy



This model will use CRISPR/Cas9 technology to edit the *Kdm1b* gene. The schematic diagram is as follows:





- The Kdm1b gene has 5 transcripts. According to the structure of Kdm1b gene, exon3-exon13 of Kdm1b-201 (ENSMUST00000037025.15) transcript is recommended as the knockout region. The region contains 1462bp coding sequence. Knock out the region will result in disruption of protein function.
- > In this project we use CRISPR/Cas9 technology to modify Kdm1b gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Homozygous null mice of both sexes are viable, grossly normal and male mice are fertile; however, heterozygous progeny of homozygous null mothers display severe placental defects, embryonic growth impairment, neural tube defects and pericardial edema, and do not survive past E10.5.
  The knockout region is near to the N-terminal of *Tpmt* gene, this strategy may influence the regulatory function of the N-terminal of *Tpmt* gene.
- The *Kdm1b* gene is located on the Chr13. 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.

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# Gene information (NCBI)



Kdm1b lysine (K)-specific demethylase 1B [ Mus musculus (house mouse) ]

Gene ID: 218214, updated on 10-Oct-2019

#### Summary

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Official Symbol	Kdm1b provided by MGI
Official Full Name	lysine (K)-specific demethylase 1B provided by MGI
Primary source	MGI:MGI:2145261
See related	Ensembl:ENSMUSG00000038080
Gene type	protein coding
<b>RefSeq status</b>	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	Aof1; Al482520; 4632428N09Rik
Expression	Ubiquitous expression in bladder adult (RPKM 25.1), liver E14 (RPKM 13.2) and 27 other tissues See more
Orthologs	human all

#### Genomic context

☆ ?

Location: 13; 13 A5

Exon count: 23

See Kdm1b in Genome Data Viewer

Annotation release	Status	Assembly	Chr	Location
<u>108</u>	current	GRCm38.p6 (GCF_000001635.26)	13	NC_000079.6 (4704337347085279)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	13	NC_000079.5 (4713890847179982)

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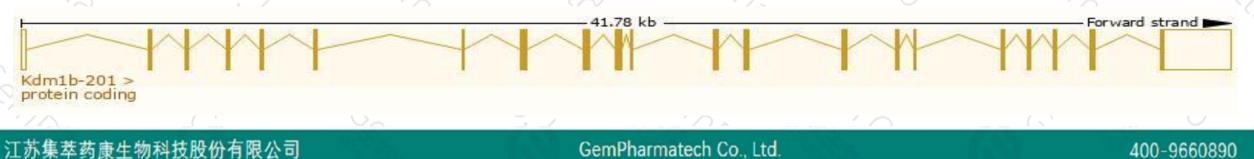
## **Transcript information (Ensembl)**



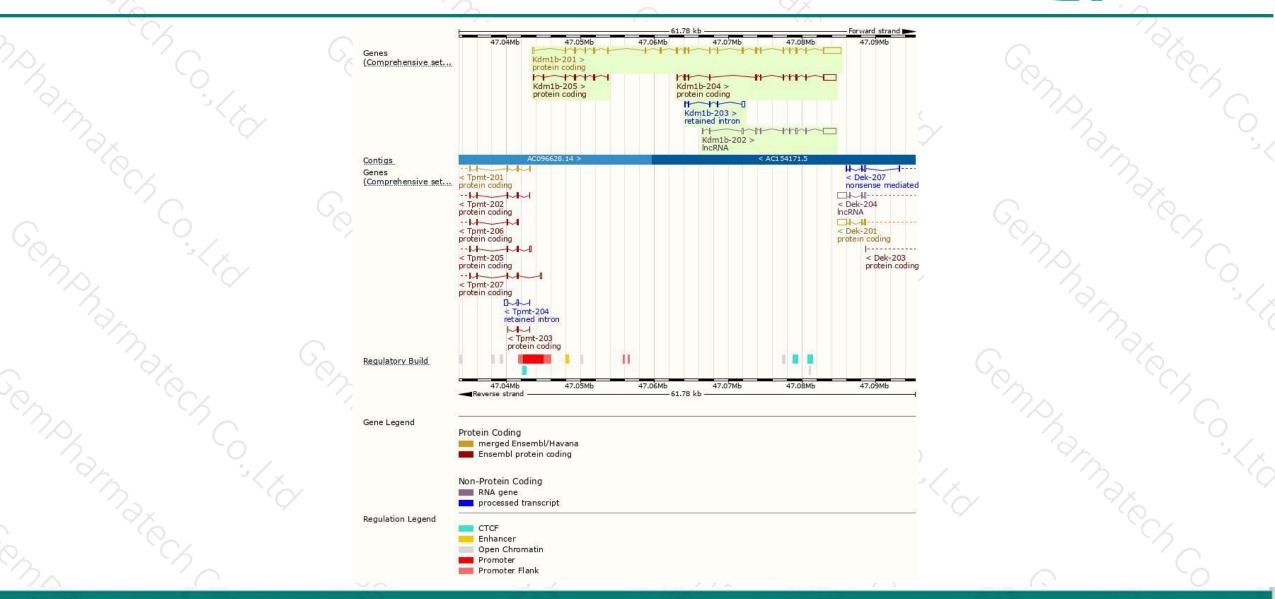
### The gene has 5 transcripts, all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Kdm1b-201	ENSMUST0000037025.15	4987	<u>826aa</u>	Protein coding	CCDS26489	Q8CIG3	TSL:1 GENCODE basic APPRIS P1
Kdm1b-204	ENSMUST00000143518.2	2969	<u>443aa</u>	Protein coding	. <del>1</del> 8	F6V3V2	CDS 5' incomplete TSL:1
Kdm1b-205	ENSMUST00000143868.1	736	<u>176aa</u>	Protein coding	10	D3Z353	CDS 3' incomplete TSL:3
Kdm1b-203	ENSMUST00000131120.7	870	No protein	Retained intron	20		TSL:3
Kdm1b-202	ENSMUST00000128977.2	2876	No protein	IncRNA	5.6		TSL:1

The strategy is based on the design of Kdm1b-201 transcript, The transcription is shown below



### **Genomic location distribution**



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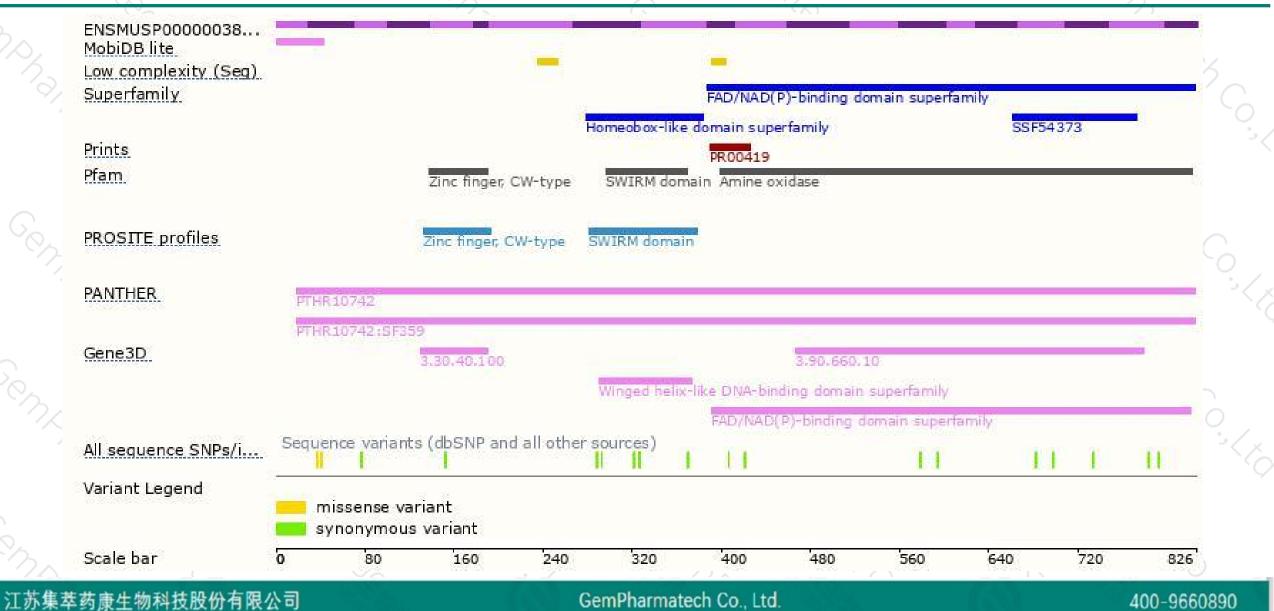
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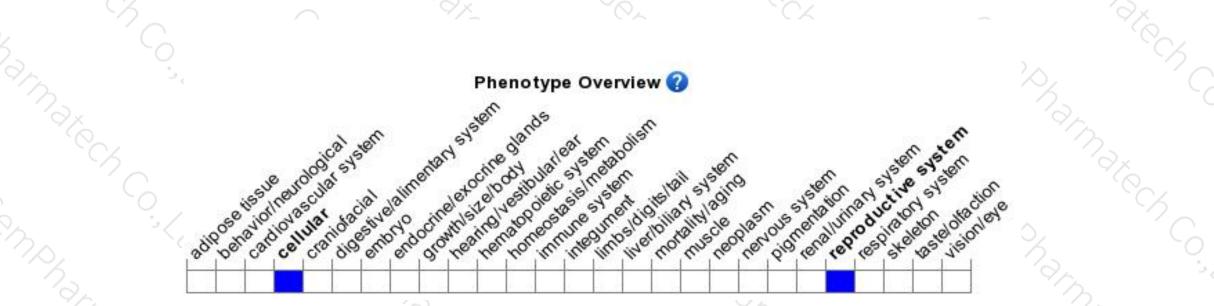
### **Protein domain**





### 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 mice of both sexes are viable, grossly normal and male mice are fertile; however, heterozygous progeny of homozygous null mothers display severe placental defects, embryonic growth impairment, neural tube defects and pericardial edema, and do not survive past E10.5.

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If you have any questions, you are welcome to inquire. Tel: 400-9660890



