

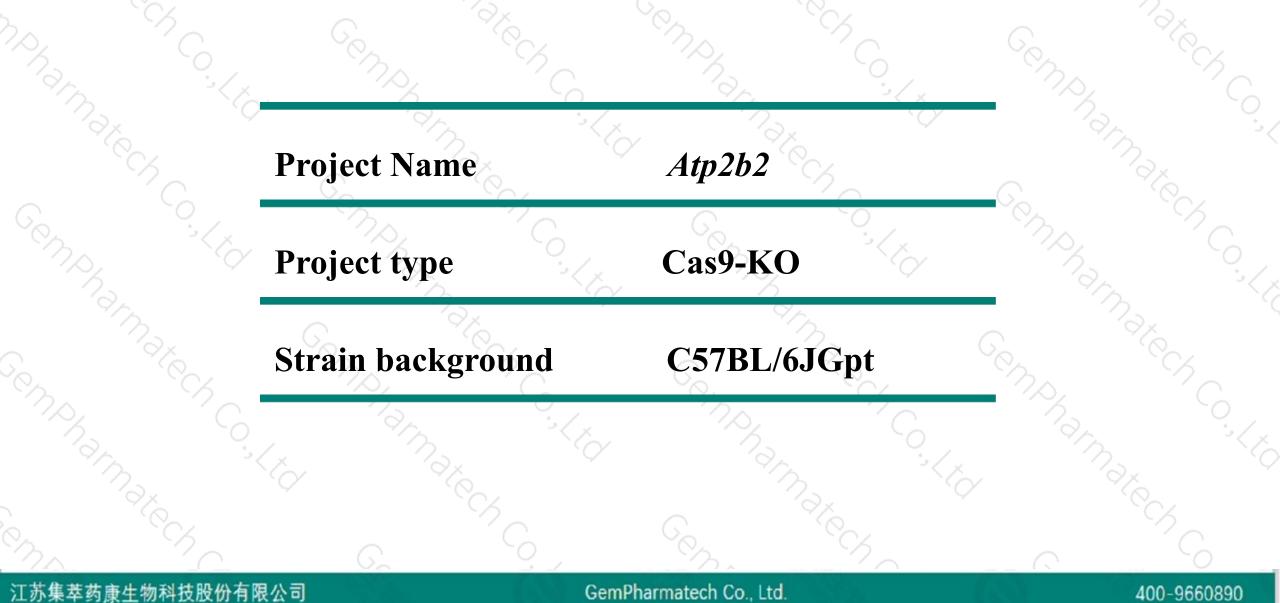
# Atp2b2 Cas9-KO Strategy

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# **Project Overview**

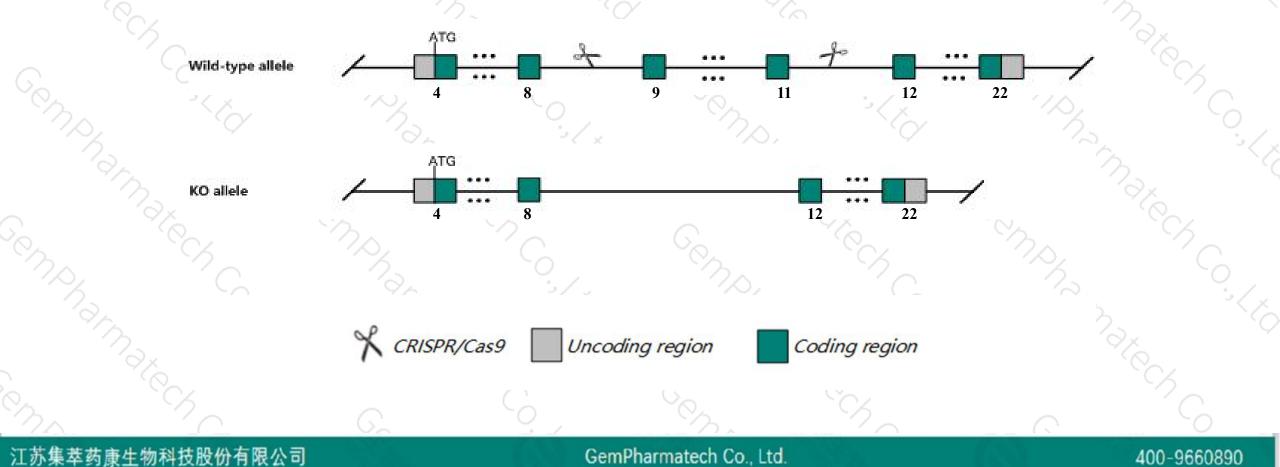




# **Knockout** strategy



This model will use CRISPR/Cas9 technology to edit the *Atp2b2* gene. The schematic diagram is as follows:





- The Atp2b2 gene has 8 transcripts. According to the structure of Atp2b2 gene, exon9-exon11 of Atp2b2-203 (ENSMUST00000101045.9) transcript is recommended as the knockout region. The region contains 617bp coding sequence. Knock out the region will result in disruption of protein function.
- > In this project we use CRISPR/Cas9 technology to modify Atp2b2 gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Homozygous mutants exhibit slower growth, balance problems, and deafness, associated with cerebellar abnormalities, an absence of otoconia, and abnormalities of the organ of Corti. Heterozygotes exhibit appreciable age-dependent hearing loss.
  The non-coding transcripts 205 and 207 are unaffected.
- > Gm44167-201 will be deleted together.
- The Atp2b2 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.

Notice

# **Gene information (NCBI)**



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# Atp2b2 ATPase, Ca++ transporting, plasma membrane 2 [Mus musculus (house mouse)]

Gene ID: 11941, updated on 7-Apr-2019

# Summary

Official Combal	Alt-alt-a
Official Symbol	Atp2b2 provided by MGI
Official Full Name	ATPase, Ca++ transporting, plasma membrane 2 provided by MGI
Primary source	MGI:MGI:105368
See related	Ensembl:ENSMUSG0000030302
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	D6Abb2e, Gena300, PMCA2, Tmy, dfw, jog, wms, wri
Expression	Biased expression in cortex adult (RPKM 49.9), cerebellum adult (RPKM 49.7) and 4 other tissues See more
Orthologs	human all

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# **Transcript information (Ensembl)**



# The gene has 8 transcripts, all transcripts are shown below:

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Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Atp2b2-203	ENSMUST00000101045.9	8877	<u>1198aa</u>	Protein coding	CCDS20433	Q3UHJ3 Q9R0K7	TSL:1 GENCODE basic APPRIS P3
Atp2b2-201	ENSMUST0000089003.11	7067	<u>1198aa</u>	Protein coding	CCDS20433	Q3UHJ3 Q9R0K7	TSL:1 GENCODE basic APPRIS P3
Atp2b2-202	ENSMUST00000101044.8	4586	<u>1243aa</u>	Protein coding	CCDS85130	F8WHB1	TSL:1 GENCODE basic APPRIS ALT1
Atp2b2-208	ENSMUST00000205052.2	5658	<u>1194aa</u>	Protein coding	N <u>17</u> 3	<u>Q3UHH0</u>	TSL:1 GENCODE basic APPRIS ALT1
Atp2b2-206	ENSMUST00000152831.7	7326	<u>1154aa</u>	Nonsense mediated decay	1731	<u>S4R1C4</u>	TSL:1
Atp2b2-207	ENSMUST00000154738.1	659	No protein	Processed transcript			TSL:5
Atp2b2-205	ENSMUST00000144507.1	617	No protein	Processed transcript	144	3 <b>4</b>	TSL:1
Atp2b2-204	ENSMUST00000135199.1	3463	No protein	Retained intron	820	62	TSL:1

The strategy is based on the design of Atp2b2-203 transcript, The transcription is shown below

### < Atp2b2-203 protein coding

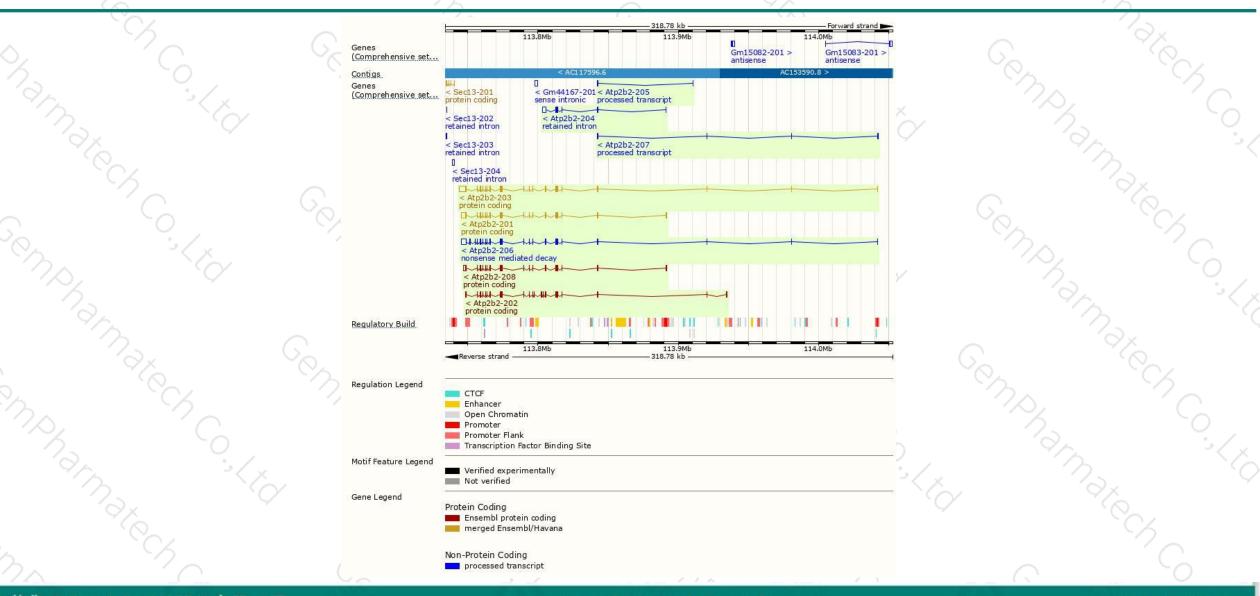
Reverse strand -

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298.20 kb

# **Genomic location distribution**



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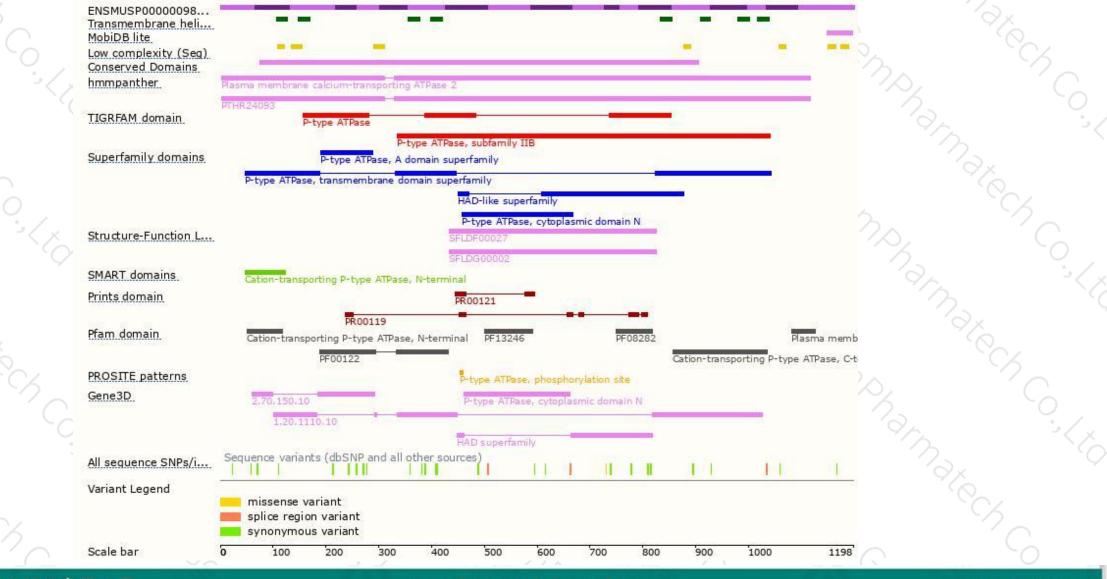
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# **Protein domain**



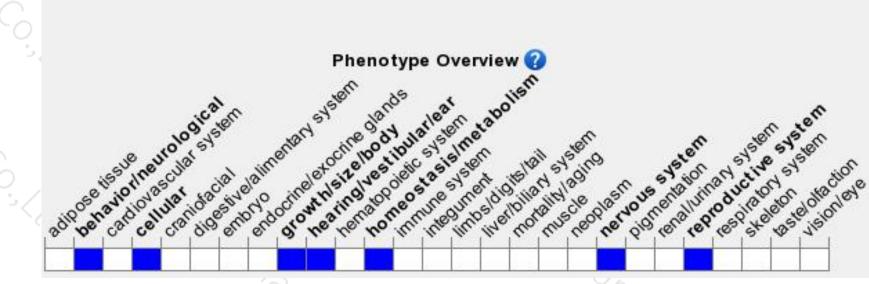


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# Mouse phenotype description(MGI)





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 mutants exhibit slower growth, balance problems, and deafness, associated with cerebellar abnormalities, an absence of otoconia, and abnormalities of the organ of Corti. Heterozygotes exhibit appreciable age-dependent hearing loss.

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



