

# *Axl* Cas9-KO Strategy

Designer: Yanhua Shen

# Project Overview

---

**Project Name**

*Axl*

---

**Project type**

**Cas9-KO**

---

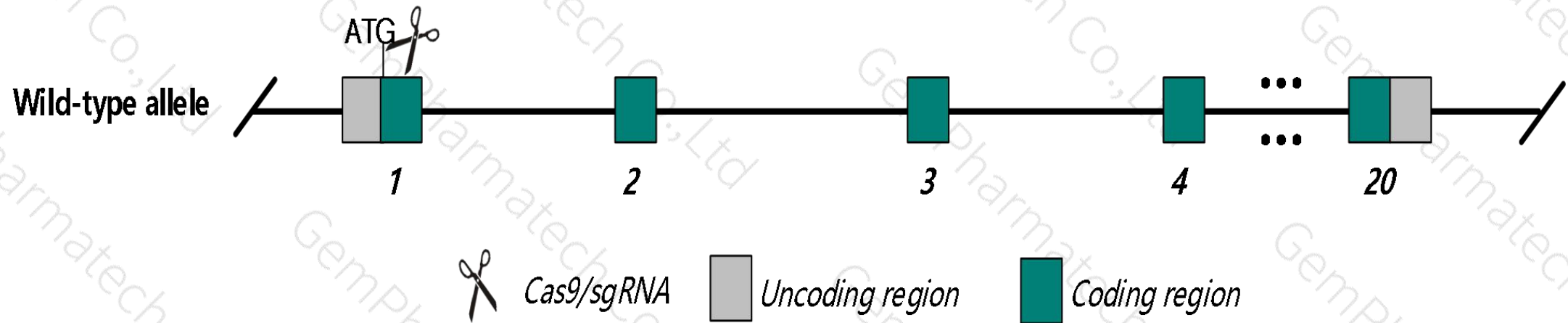
**Strain background**

**BALB/cJ**

---

# Knockout strategy

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



- The *Axl* gene has 8 transcripts. According to the structure of *Axl* gene, exon1 of MGP\_BALBcJ\_T0082410.1 transcript is recommended as the knockout region. Screening mouse model of frameshift mutation. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Axl* gene. The brief process is as follows: sgRNA was transcribed in vitro. Cas9 and sgRNA were microinjected into the fertilized eggs of BALB/cJ 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 BALB/cJ mice.

- According to the existing MGI data, Homozygous mutant mice are phenotypically normal, however in conjunction with mutations in other related receptor tyrosine kinases, mutations of this gene results in fertility defects, autoimmunity abnormalities, and aberrant apoptosis.
- The *Axl* gene is located on the Chr7. 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)

## Axl AXL receptor tyrosine kinase [ *Mus musculus* (house mouse) ]

Gene ID: 26362, updated on 21-May-2019

### Summary

Official Symbol	Axl provided by <a href="#">MGI</a>
Official Full Name	AXL receptor tyrosine kinase provided by <a href="#">MGI</a>
Primary source	<a href="#">MGI:MGI:1347244</a>
See related	<a href="#">Ensembl:ENSMUSG000000002602</a>
Gene type	protein coding
RefSeq status	VALIDATED
Organism	<a href="#">Mus musculus</a>
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Ark; Ufo; Tyro7; AI323647
Expression	Broad expression in ovary adult (RPKM 53.4), mammary gland adult (RPKM 42.5) and 28 other tissues <a href="#">See more</a>
Orthologs	<a href="#">human</a> <a href="#">all</a>

### Genomic context

Location: 7 A3; 7 14.02 cM

Exon count: 20

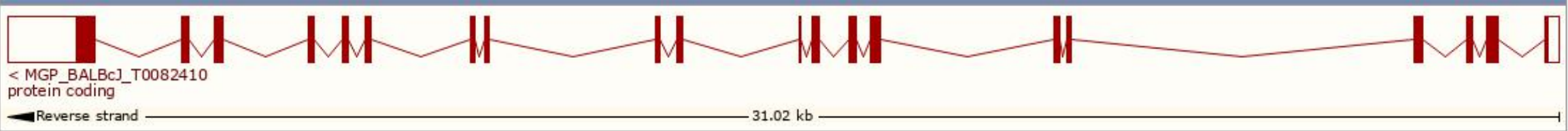
See Axl in [Genome Data Viewer](#)

# Transcript information (Ensembl)

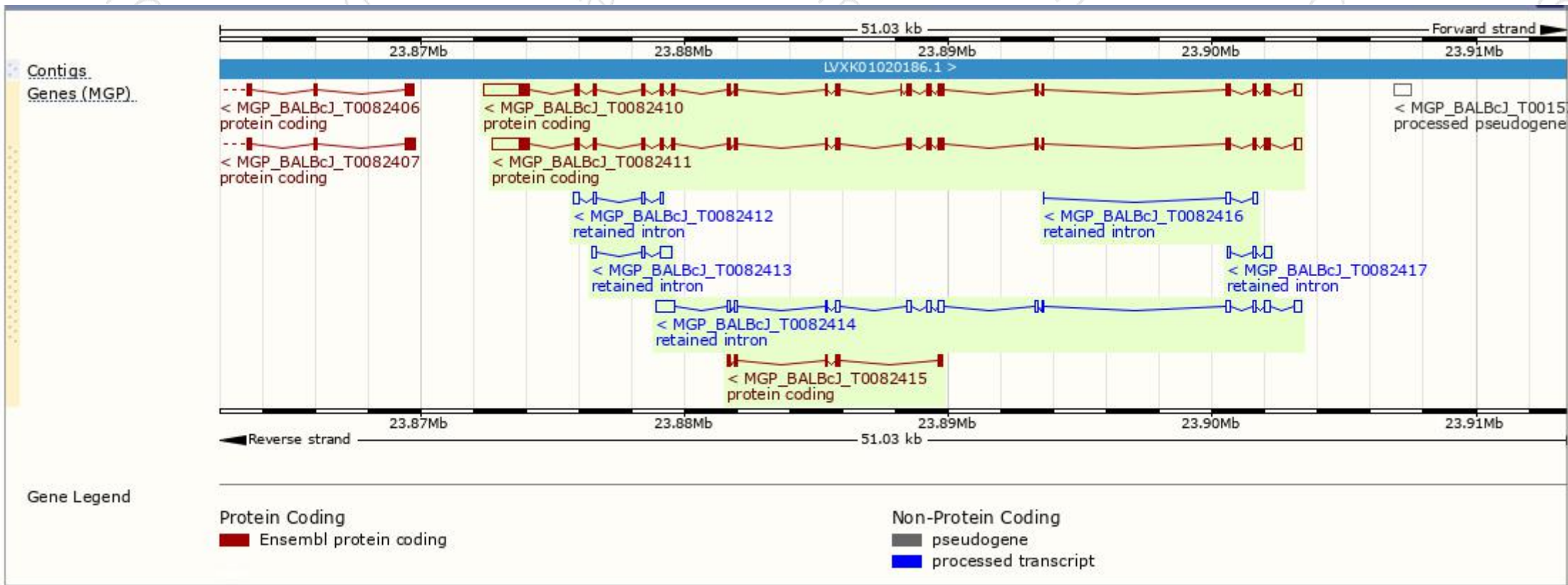
The gene has 8 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
-	<a href="#">MGP_BALBcJ_T0082410.1</a>	4265	<a href="#">888aa</a>	Protein coding	<a href="#">CCDS20996</a> , <a href="#">CCDS57528</a>	<a href="#">F6YPR4</a> <a href="#">Q00993</a> <a href="#">Q6PE80</a>	-
-	<a href="#">MGP_BALBcJ_T0082411.1</a>	3899	<a href="#">879aa</a>	Protein coding	-	-	-
-	<a href="#">MGP_BALBcJ_T0082415.1</a>	563	<a href="#">187aa</a>	Protein coding	-	-	-
-	<a href="#">MGP_BALBcJ_T0082414.1</a>	2609	No protein	Retained intron	-	-	-
-	<a href="#">MGP_BALBcJ_T0082413.1</a>	777	No protein	Retained intron	-	-	-
-	<a href="#">MGP_BALBcJ_T0082412.1</a>	602	No protein	Retained intron	-	-	-
-	<a href="#">MGP_BALBcJ_T0082417.1</a>	455	No protein	Retained intron	-	-	-
-	<a href="#">MGP_BALBcJ_T0082416.1</a>	382	No protein	Retained intron	-	-	-

The strategy is based on the design of *MGP\_BALBcJ\_T0082410.1* transcript,The transcription is shown below

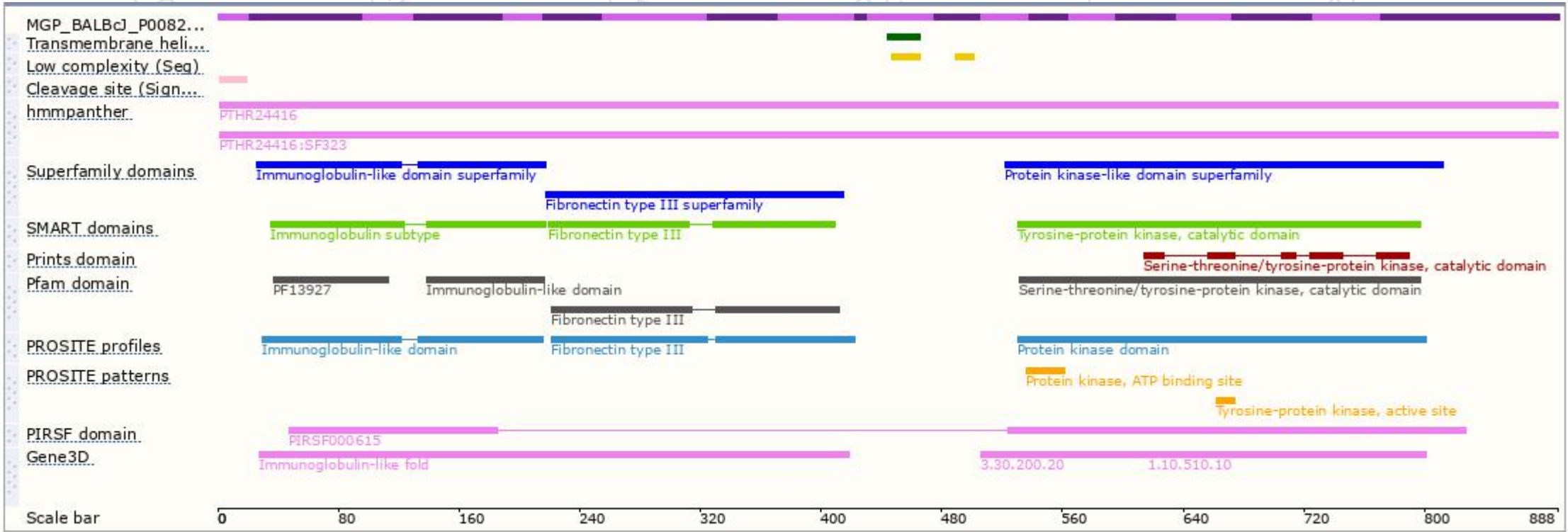


# Genomic location distribution

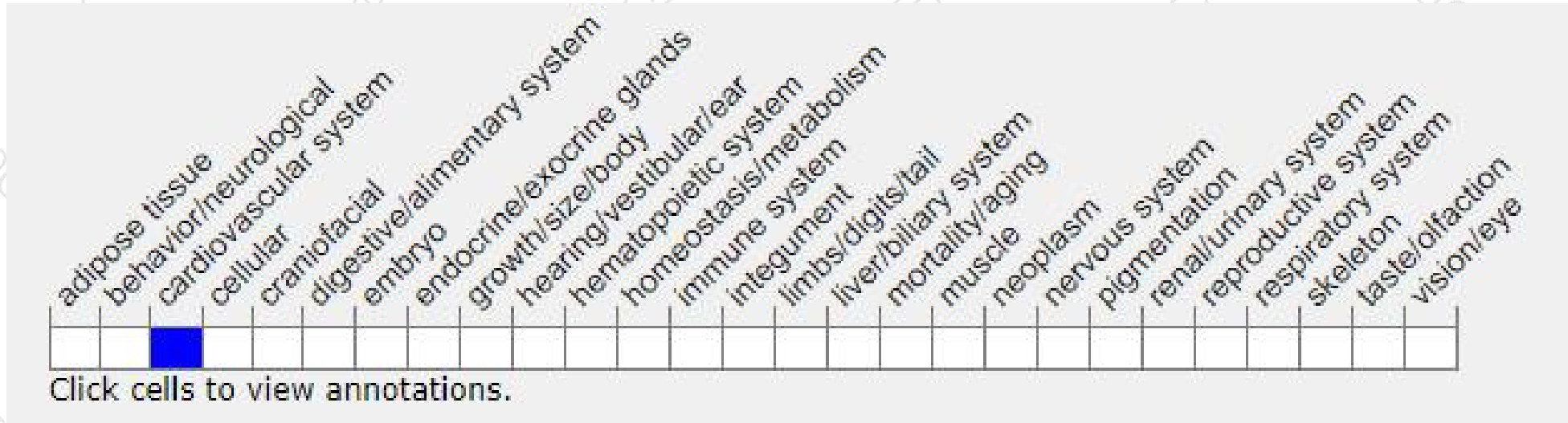




# Protein domain



# Mouse phenotype description(MGI )



*Phenotypes affected by the gene are marked in blue. Data quoted from MGI database (<http://www.informatics.jax.org/marker/MGI:1347244>).*

Homozygous mutant mice are phenotypically normal, however in conjunction with mutations in other related receptor tyrosine kinases, mutations of this gene results in fertility defects, autoimmunity abnormalities, and aberrant apoptosis.

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

Tel: 025-5864 1534

