

# *Atg5* Cas9-KO Strategy

**Designer:**

**Huan Wang**

**Design Date:**

**2019-7-22**

# Project Overview

**Project Name**

*Atg5*

**Project type**

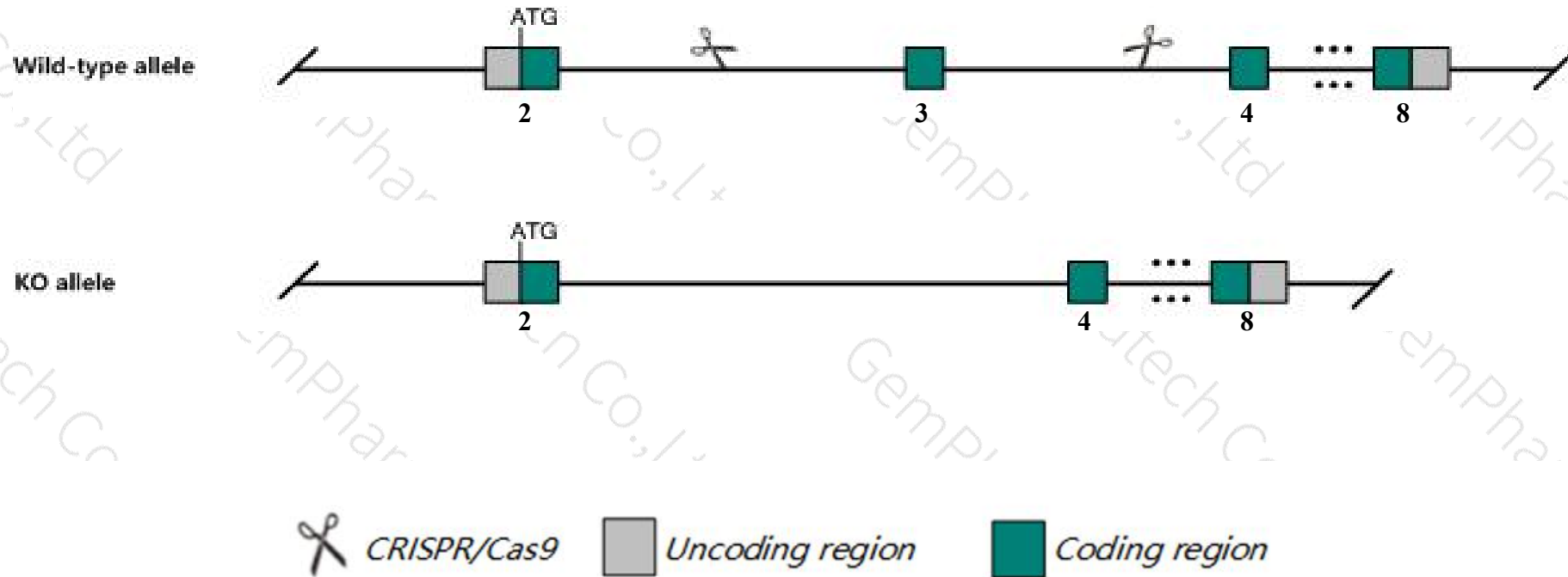
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Atg5* gene. The schematic diagram is as follows:



- The *Atg5* gene has 2 transcripts. According to the structure of *Atg5* gene, exon3 of *Atg5-201* (ENSMUST00000039286.4) transcript is recommended as the knockout region. The region contains 128bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Atg5* gene. The brief process is as follows: CRISPR/Cas9 system v

- According to the existing MGI data, Mutation of this gene results in impaired autophagy due to absence of autolysosomes. Homozygotes die within 1 day of birth, have shorter survival times and reduced amino acid levels under fasting conditions. Homozygotes for a gene trap insertion mutation in this gene show no abnormal phenotype.
- The *Atg5* gene is located on the Chr10. 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)

## Atg5 autophagy related 5 [Mus musculus (house mouse)]

Gene ID: 11793, updated on 2-Apr-2019

### Summary



**Official Symbol** Atg5 provided by [MGI](#)

**Official Full Name** autophagy related 5 provided by [MGI](#)

**Primary source** [MGI:MGI:1277186](#)

**See related** [Ensembl:ENSMUSG00000038160](#)

**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** 2010107M05Rik, 3110067M24Rik, AW319544, Apg5l, Atg5l, C88337, Paddy

**Summary** The protein encoded by this gene, in combination with autophagy protein 12, functions as an E1-like activating enzyme in a ubiquitin-like conjugating system. The encoded protein is involved in several cellular processes, including autophagic vesicle formation, mitochondrial quality control after oxidative damage, negative regulation of the innate antiviral immune response, lymphocyte development and proliferation, MHC II antigen presentation, adipocyte differentiation, and apoptosis. Two transcript variants encoding different protein isoforms have been found for this gene. [provided by RefSeq, Sep 2015]

**Expression** Ubiquitous expression in placenta adult (RPKM 9.7), bladder adult (RPKM 9.2) and 28 other tissues [See more](#)

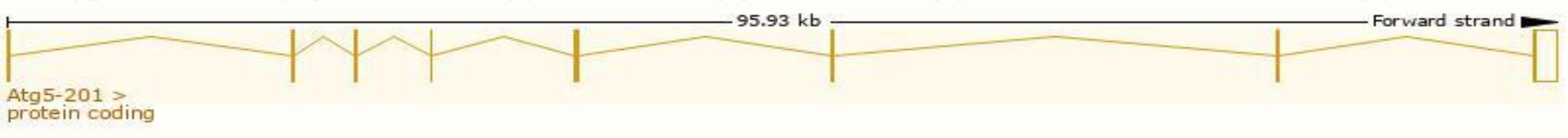
**Orthologs** [human](#) [all](#)

# Transcript information (Ensembl)

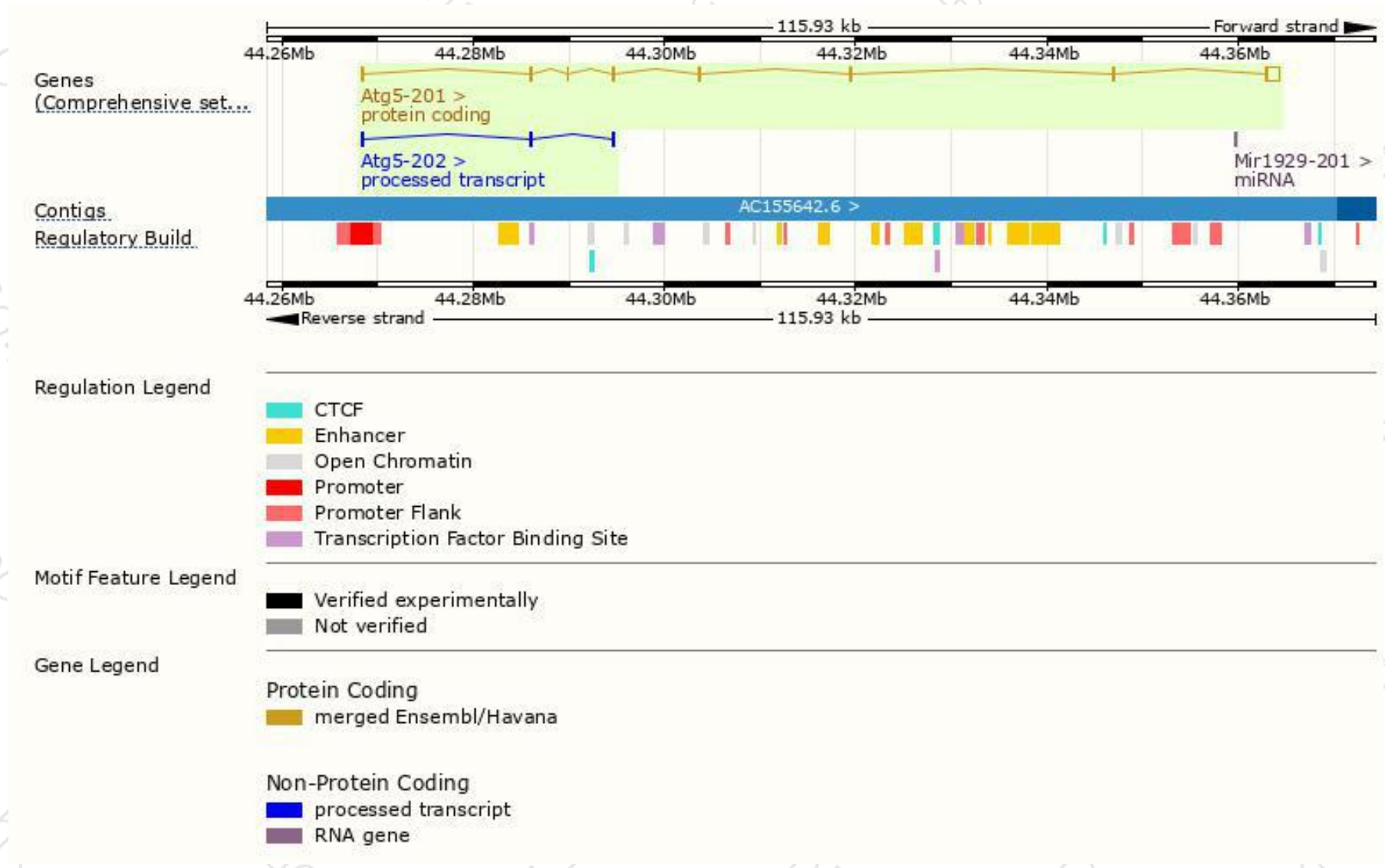
The gene has 2 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Atg5-201	<a href="#">ENSMUST00000039286.4</a>	2352	<a href="#">275aa</a>	Protein coding	<a href="#">CCDS23824</a>	<a href="#">Q99J83</a>	TSL:1 GENCODE basic APPRIS P1
Atg5-202	<a href="#">ENSMUST00000217412.1</a>	415	No protein	Processed transcript	-	-	TSL:3

The strategy is based on the design of *Atg5-201* transcript,The transcription is shown below

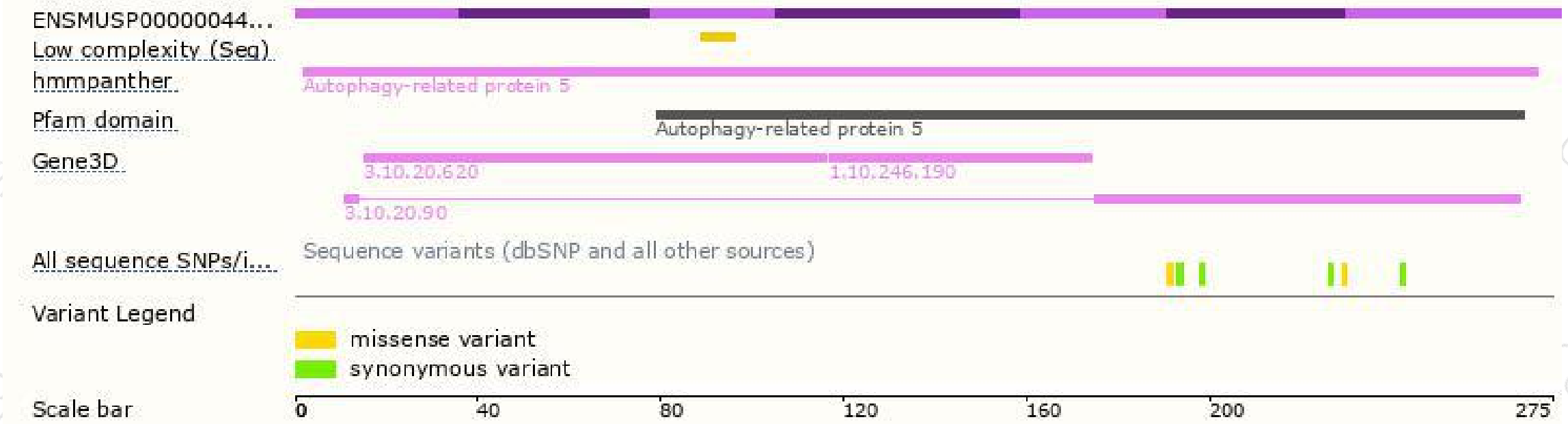


# Genomic location distribution

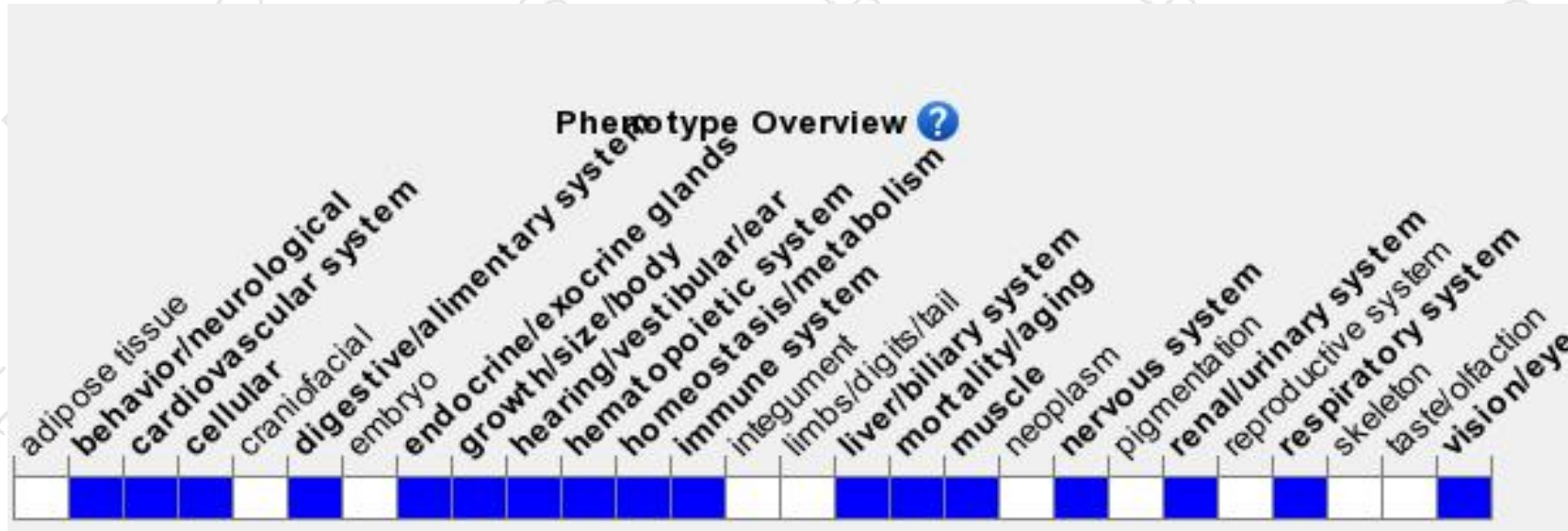




# Protein domain



# 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, Mutation of this gene results in impaired autophagy due to absence of autolysosomes.

Homozygotes die within 1 day of birth, have shorter survival times and reduced amino acid levels under fasting conditions. Homozygotes for a gene trap insertion mutation in this gene show no abnormal phenotype.

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

Tel: 400-9660890

