

# ***Neurog3 Cas9-KO Strategy***

**Designer:**

**Qiong Zhou**

# Project Overview

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**Project Name**

***Neurog3***

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**Project type**

**Cas9-KO**

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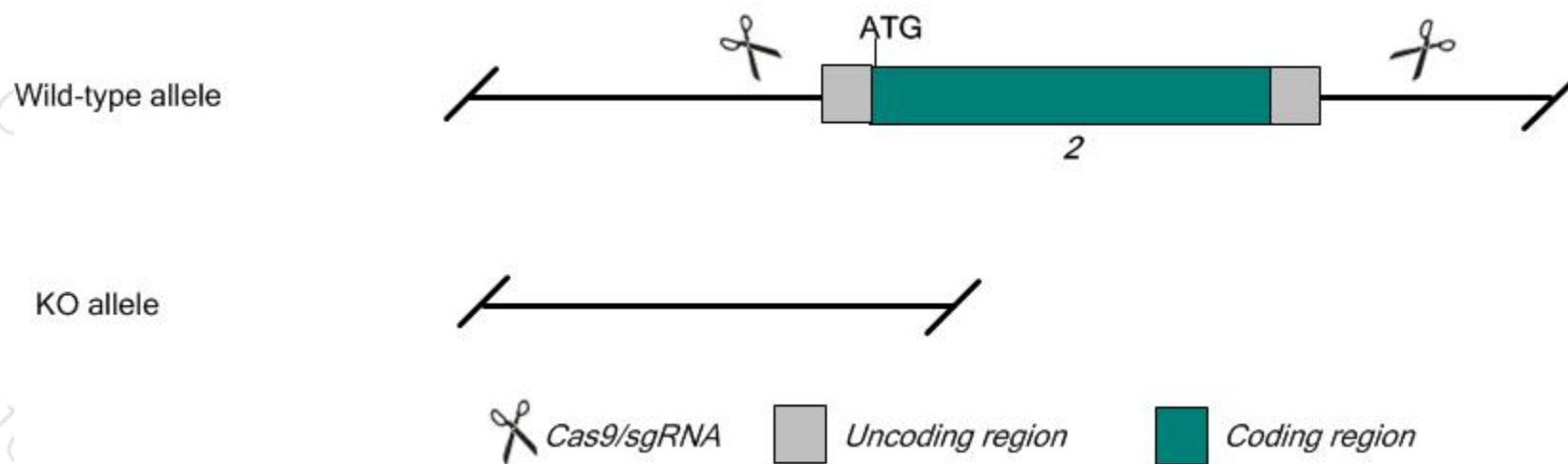
**Strain background**

**C57BL/6J**

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# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Neurog3* gene. The schematic diagram is as follows:



- The *Neurog3* gene has 3 transcripts. According to the structure of *Neurog3* gene, exon2 of *Neurog3*-201 (ENSMUST00000050103.1) transcript is recommended as the knockout region. The region contains all coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Neurog3* gene. The brief process is as follows: sgRNA was transcribed in vitro. Cas9 and sgRNA were microinjected into the fertilized eggs of C57BL/6J 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/6J mice.

- According to the existing MGI data, Homozygotes for targeted null mutations are deficient in endocrine cells of the glandular stomach and intestinal epithelium, and lack glucagon- and insulin-producing cells of the pancreas. Mutants die postnatally from diabetes.
- The KO region contains functional region of the *Fam241b* gene. Knockout the region may affect the function of *Fam241b* gene.
- The *Neurog3* 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.

## Neurog3 neurogenin 3 [ *Mus musculus* (house mouse) ]

Gene ID: 11925, updated on 23-Apr-2019

### Summary

<b>Official Symbol</b>	Neurog3 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	neurogenin 3 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:893591</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000044312</a>
<b>Gene type</b>	protein coding
<b>RefSeq status</b>	VALIDATED
<b>Organism</b>	<a href="#">Mus musculus</a>
<b>Lineage</b>	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
<b>Also known as</b>	ngn3; Atoh5; Math4B; bHLHa7
<b>Expression</b>	Biased expression in duodenum adult (RPKM 2.0), large intestine adult (RPKM 1.3) and 5 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

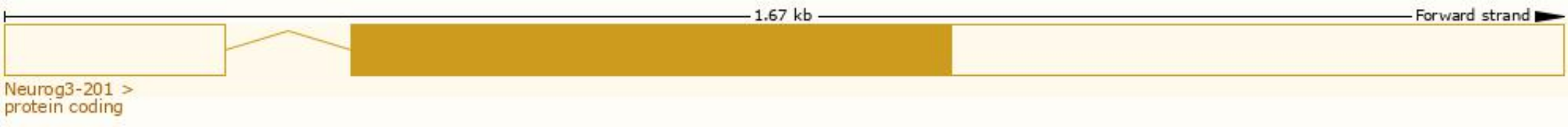


# Transcript information (Ensembl)

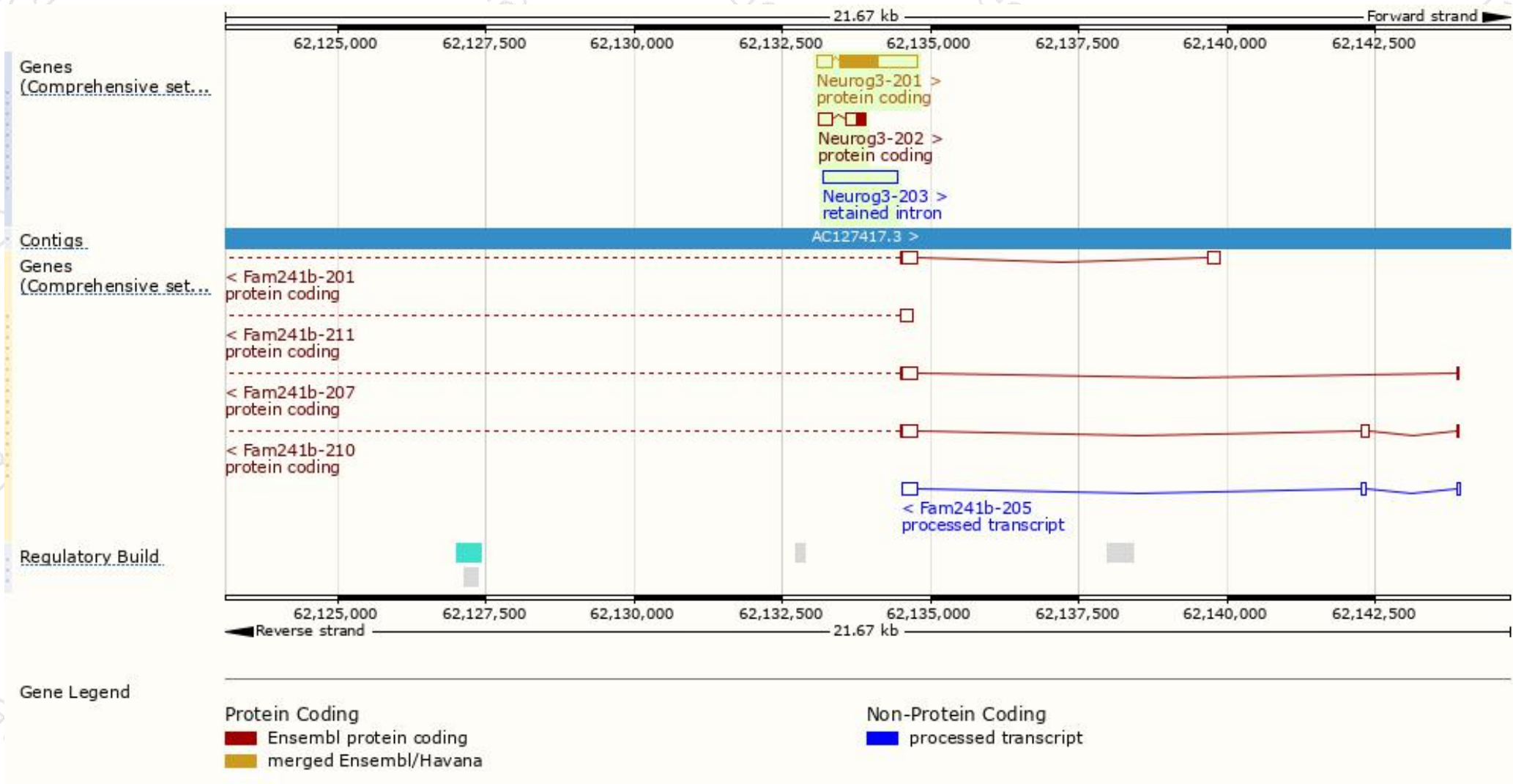
The gene has 3 transcripts, and all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Neurog3-201	<a href="#">ENSMUST00000050103.1</a>	1540	<a href="#">214aa</a>	Protein coding	<a href="#">CCDS23887</a>	<a href="#">P70661</a> <a href="#">Q548G3</a>	TSL:1 GENCODE basic APPRIS P1
Neurog3-202	<a href="#">ENSMUST00000218121.1</a>	553	<a href="#">50aa</a>	Protein coding	-	<a href="#">A0A1W2P770</a>	CDS 3' incomplete TSL:5
Neurog3-203	<a href="#">ENSMUST00000218216.1</a>	1259	No protein	Retained intron	-	-	TSL:NA

The strategy is based on the design of *Neurog3-201* transcript, The transcription is shown below

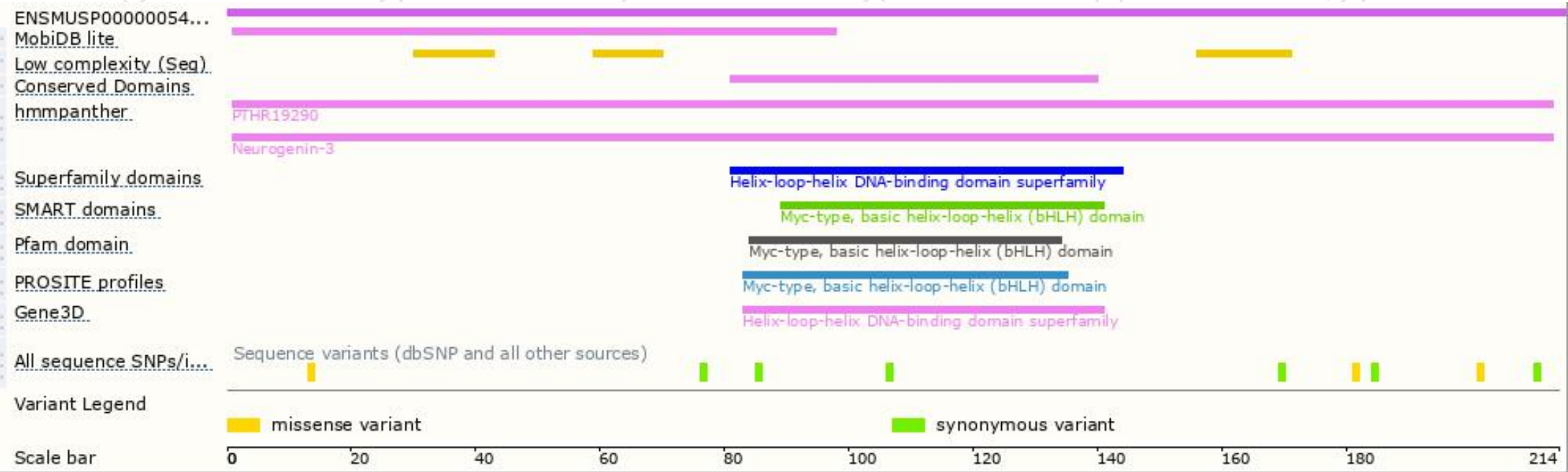


# Genomic location (Ensembl)

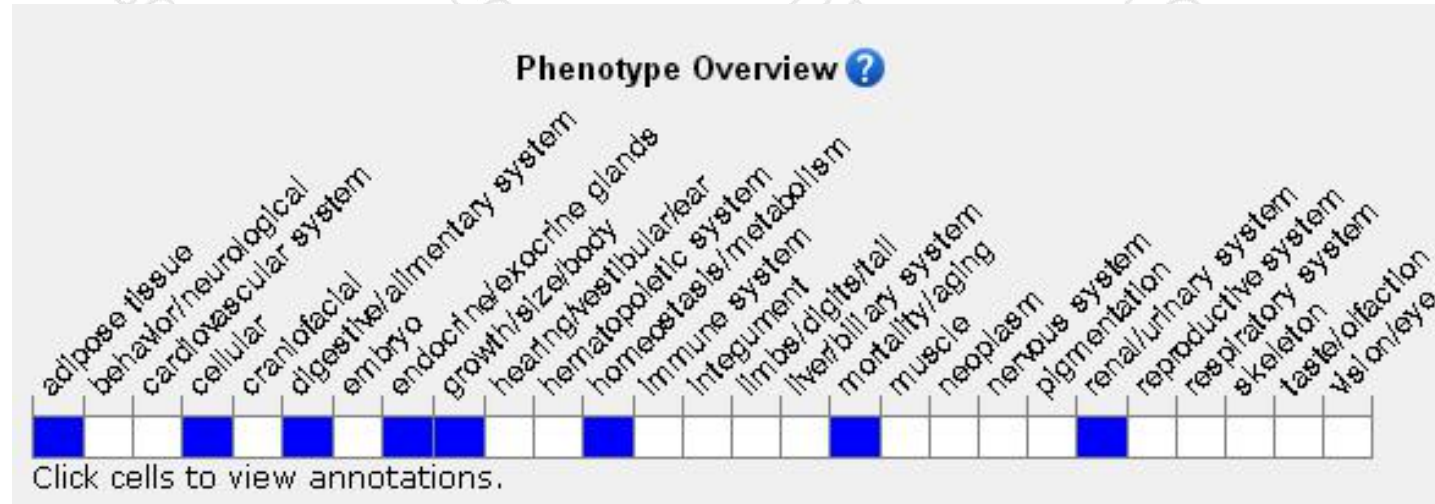




# Protein domain (Ensembl)



# 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 for targeted null mutations are deficient in endocrine cells of the glandular stomach and intestinal epithelium, and lack glucagon- and insulin-producing cells of the pancreas.

Mutants die postnatally from diabetes.

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

Tel: 025-5864 1534



集萃药康生物科技

GemPharmatech Co.,Ltd

