

Apc2 Cas9-KO Strategy

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Project Overview

Project Name

Apc2

Project type

Cas9-KO

Strain background

C57BL/6JGpt

Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Apc2* gene. The schematic diagram is as follows:



- The *Apc2* gene has 6 transcripts. According to the structure of *Apc2* gene, exon4-exon12 of *Apc2-201* (ENSMUST00000020349.6) transcript is recommended as the knockout region. The region contains 1204bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Apc2* gene. The brief process is as follows: CRISPR/Cas9 system v

- According to the existing MGI data, Mice homozygous for a null allele display gradual postnatal growth retardation, abnormal lamination of the cerebral cortex, hippocampus, olfactory bulb and cerebellum, impaired neuronal migration and impaired coordination.
- Transcript *Apc2*-204 may not be affected.
- The effect on transcript *Apc2*-203&205&206 is unknown.
- The N-terminal of *Apc2* gene will remain several amino acids, it may remain the partial function of *Apc2* gene.
- The *Apc2* 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)

Apc2 APC regulator of WNT signaling pathway 2 [*Mus musculus* (house mouse)]

Gene ID: 23805, updated on 21-Dec-2019

Summary

- Official Symbol** Apc2 provided by [MGI](#)
- Official Full Name** APC regulator of WNT signaling pathway 2 provided by [MGI](#)
- Primary source** [MGI:MGI:1346052](#)
- See related** [Ensembl:ENSMUSG00000020135](#)
- 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** APCL; R75424; AI852447
- Expression** Biased expression in whole brain E14.5 (RPKM 41.1), CNS E14 (RPKM 27.7) and 7 other tissues [See more](#)
- Orthologs** [human](#) [all](#)

Genomic context

Location: 10; 10 C1 See Apc2 in [Genome Data Viewer](#)

Exon count: 16

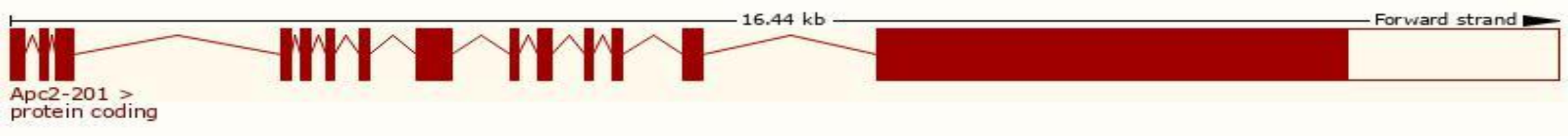
Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	10	NC_000076.6 (80295949..80318257)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	10	NC_000076.5 (79764565..79781001)

Transcript information (Ensembl)

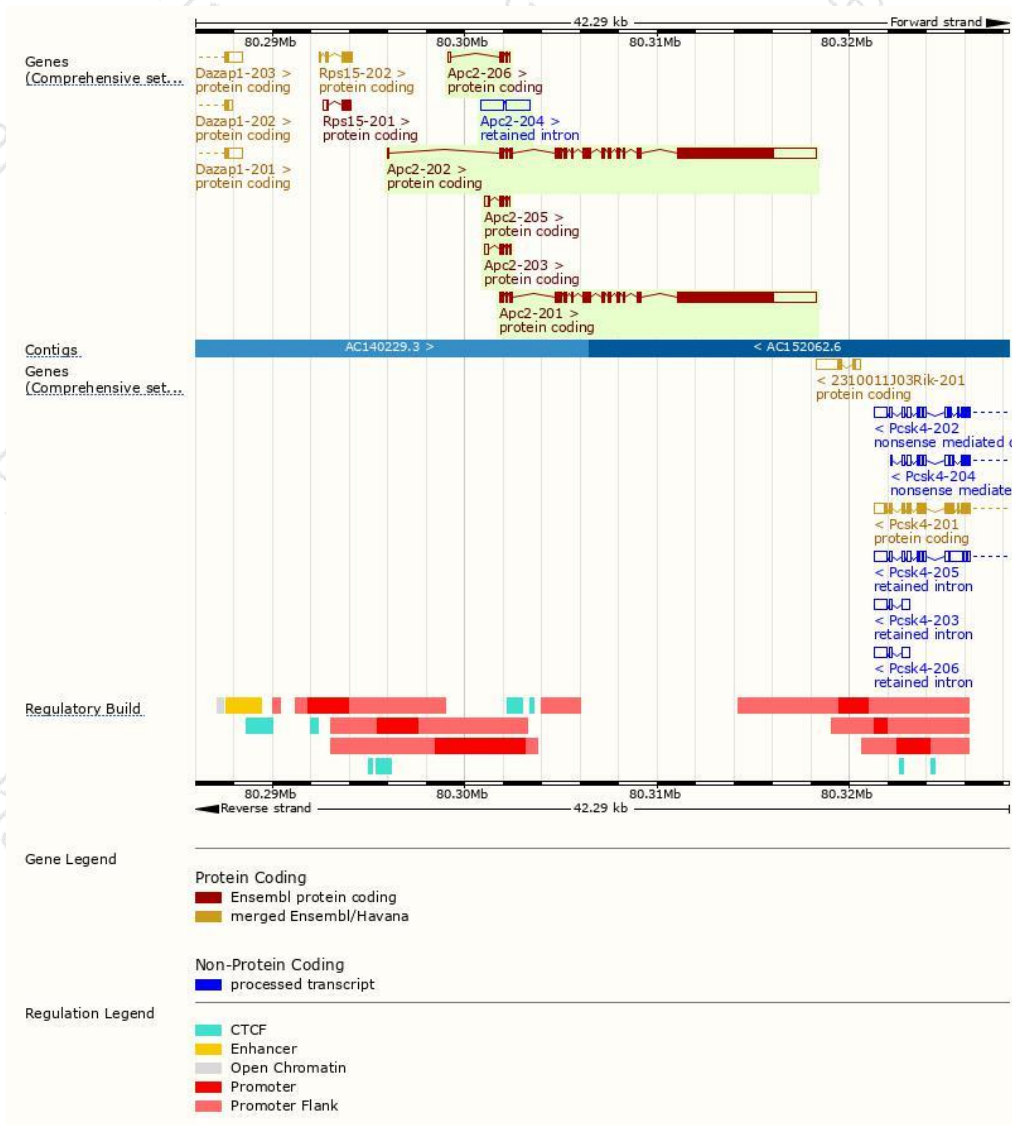
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Apc2-201	ENSMUST00000020349.6	9074	2274aa	Protein coding	CCDS24016	G5E832	TSL:5 GENCODE basic APPRIS P2
Apc2-202	ENSMUST00000105359.7	9173	2303aa	Protein coding	-	D3YTR0	TSL:5 GENCODE basic APPRIS ALT2
Apc2-205	ENSMUST00000140828.7	566	125aa	Protein coding	-	D3Z344	CDS 3' incomplete TSL:1
Apc2-203	ENSMUST00000138909.7	558	123aa	Protein coding	-	D3Z3K9	CDS 3' incomplete TSL:3
Apc2-206	ENSMUST00000154212.7	486	93aa	Protein coding	-	D3YYQ9	CDS 3' incomplete TSL:2
Apc2-204	ENSMUST00000140658.1	2351	No protein	Retained intron	-	-	TSL:1

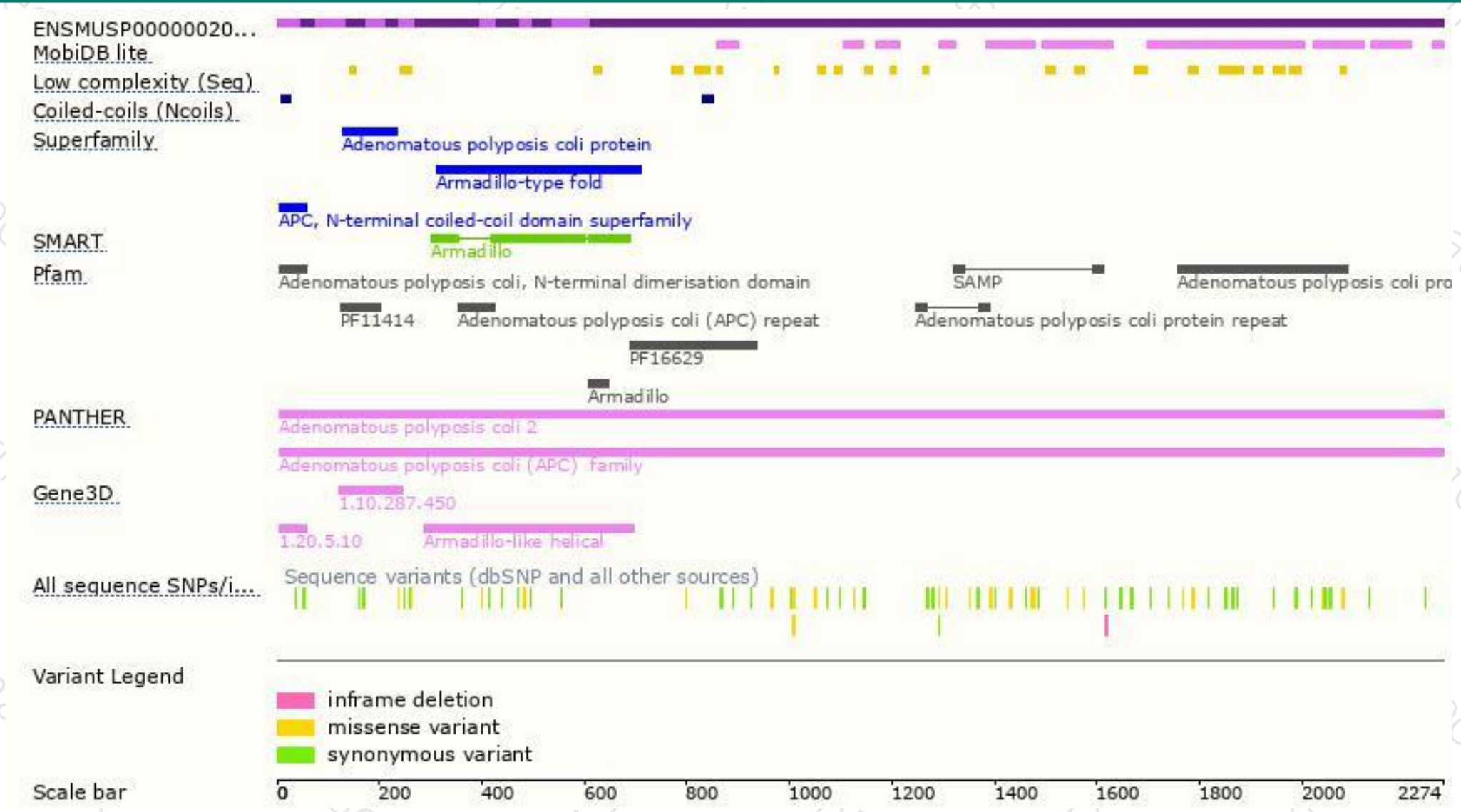
The strategy is based on the design of *Apc2-201* transcript,The transcription is shown below



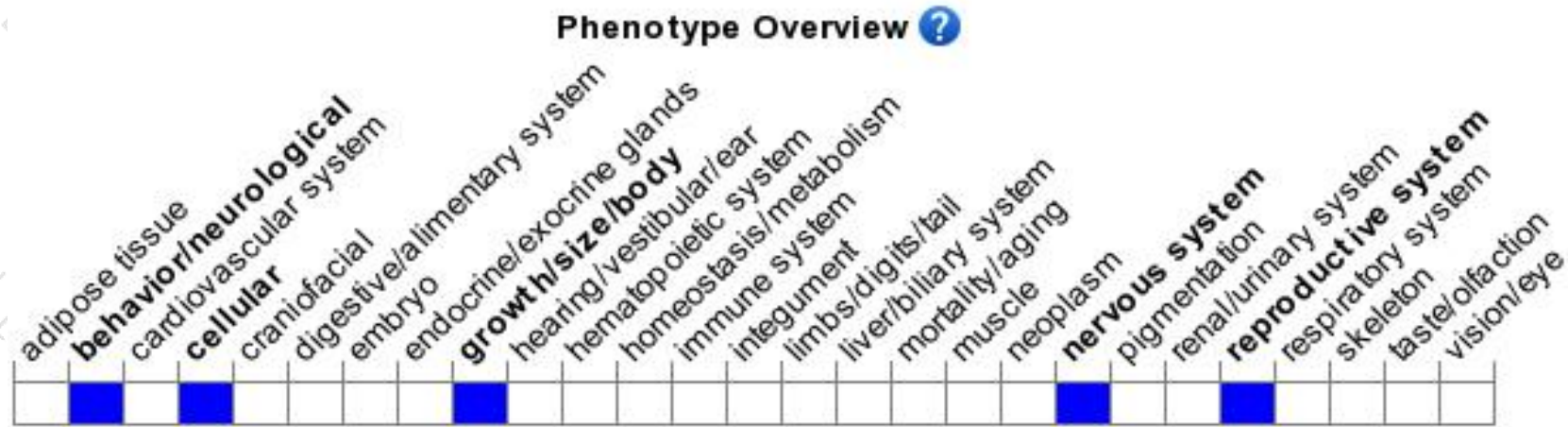
Genomic location distribution



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, Mice homozygous for a null allele display gradual postnatal growth retardation, abnormal lamination of the cerebral cortex, hippocampus, olfactory bulb and cerebellum, impaired neuronal migration and impaired coordination.

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

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