

# ***Fancd2* Cas9-KO Strategy**

**Designer:Fengjuan Wang**

**Reviewer:Shilei Zhu**

**Design Date:**

# Project Overview

**Project Name**

*Fancd2*

**Project type**

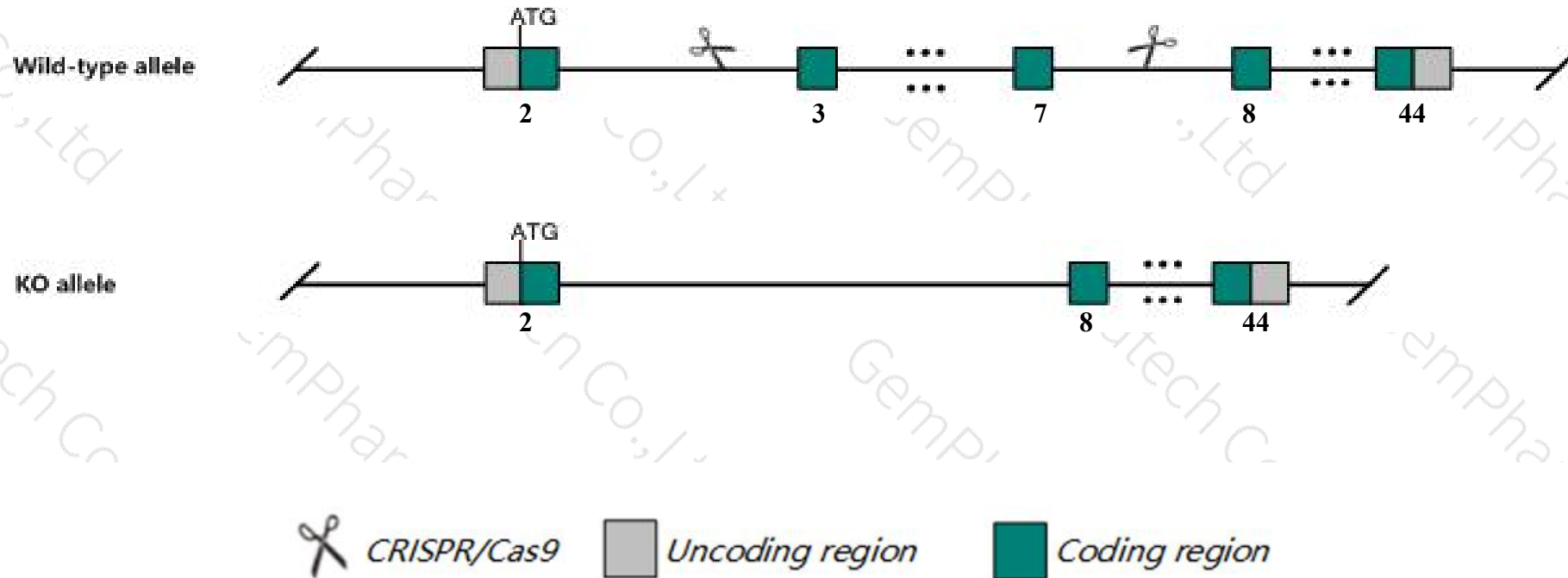
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Fancd2* gene. The schematic diagram is as follows:



- The *Fancd2* gene has 8 transcripts. According to the structure of *Fancd2* gene, exon3-exon7 of *Fancd2-201* (ENSMUST00000036340.11) transcript is recommended as the knockout region. The region contains 421bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Fancd2* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Homozygous mutant mice exhibit defects observed in human patients with Fanconi anemia (FA) meiotic defects and germ cell loss. In addition, mutant mice display perinatal lethality, susceptibility of epithelial cancer, and microphthalmia.
- The *Fancd2* 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.



# Gene information (NCBI)

## Fancd2 Fanconi anemia, complementation group D2 [Mus musculus (house mouse)]

Gene ID: 211651, updated on 31-Jan-2019

### Summary



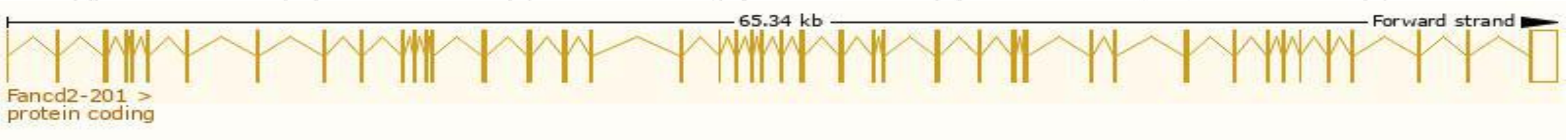
<b>Official Symbol</b>	Fancd2 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	Fanconi anemia, complementation group D2 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:2448480</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000034023</a>
<b>Gene type</b>	protein 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>	2410150O07Rik, AU015151, BB137857, FA-D2, FA4, FACD, FAD, FANCD
<b>Expression</b>	Broad expression in CNS E11.5 (RPKM 4.2), liver E14 (RPKM 4.2) and 20 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information（Ensembl）

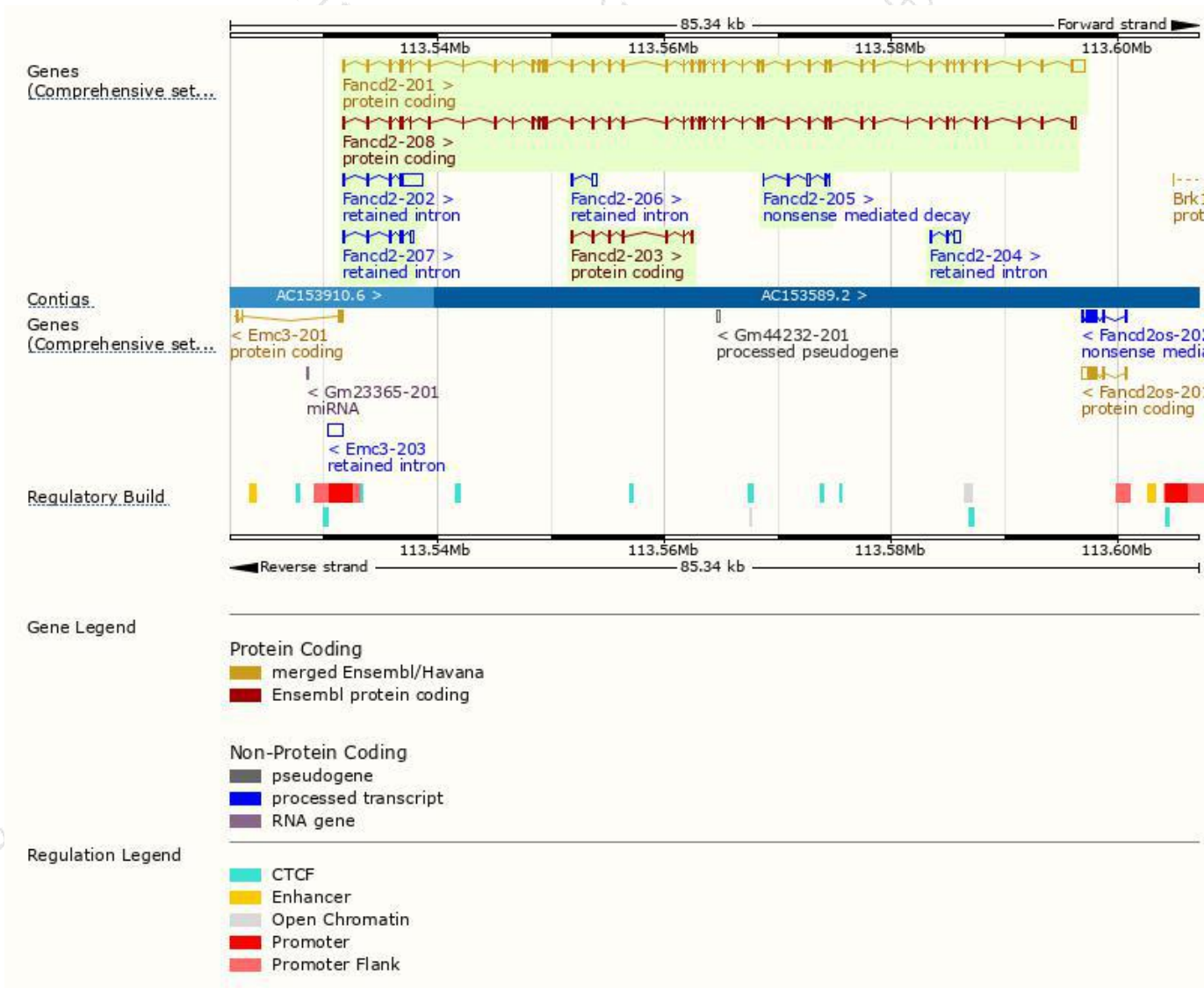
The gene has 8 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Fancd2-201	<a href="#">ENSMUST00000036340.11</a>	5512	<a href="#">1450aa</a>	Protein coding	<a href="#">CCDS20426</a>	<a href="#">B2RSU4_Q80V62</a>	TSL:1 GENCODE basic APPRIS P3
Fancd2-208	<a href="#">ENSMUST00000204827.2</a>	4707	<a href="#">1437aa</a>	Protein coding	<a href="#">CCDS85123</a>	<a href="#">A0A0N4SV29</a>	TSL:1 GENCODE basic APPRIS ALT2
Fancd2-203	<a href="#">ENSMUST00000123738.1</a>	738	<a href="#">246aa</a>	Protein coding	-	<a href="#">F7CAP1</a>	5' and 3' truncations in transcript evidence prevent annotation of the start and the end of the CDS. CDS 5' and 3' incomplete TSL:3
Fancd2-205	<a href="#">ENSMUST00000129462.2</a>	564	<a href="#">84aa</a>	Nonsense mediated decay	-	<a href="#">A0A0N4SVS0</a>	CDS 5' incomplete TSL:3
Fancd2-202	<a href="#">ENSMUST00000101051.4</a>	2142	No protein	Retained intron	-	-	TSL:1
Fancd2-204	<a href="#">ENSMUST00000124262.1</a>	764	No protein	Retained intron	-	-	TSL:2
Fancd2-207	<a href="#">ENSMUST00000143535.7</a>	680	No protein	Retained intron	-	-	TSL:2
Fancd2-206	<a href="#">ENSMUST00000142453.1</a>	391	No protein	Retained intron	-	-	TSL:2

The strategy is based on the design of *Fancd2-201* transcript,The transcription is shown below

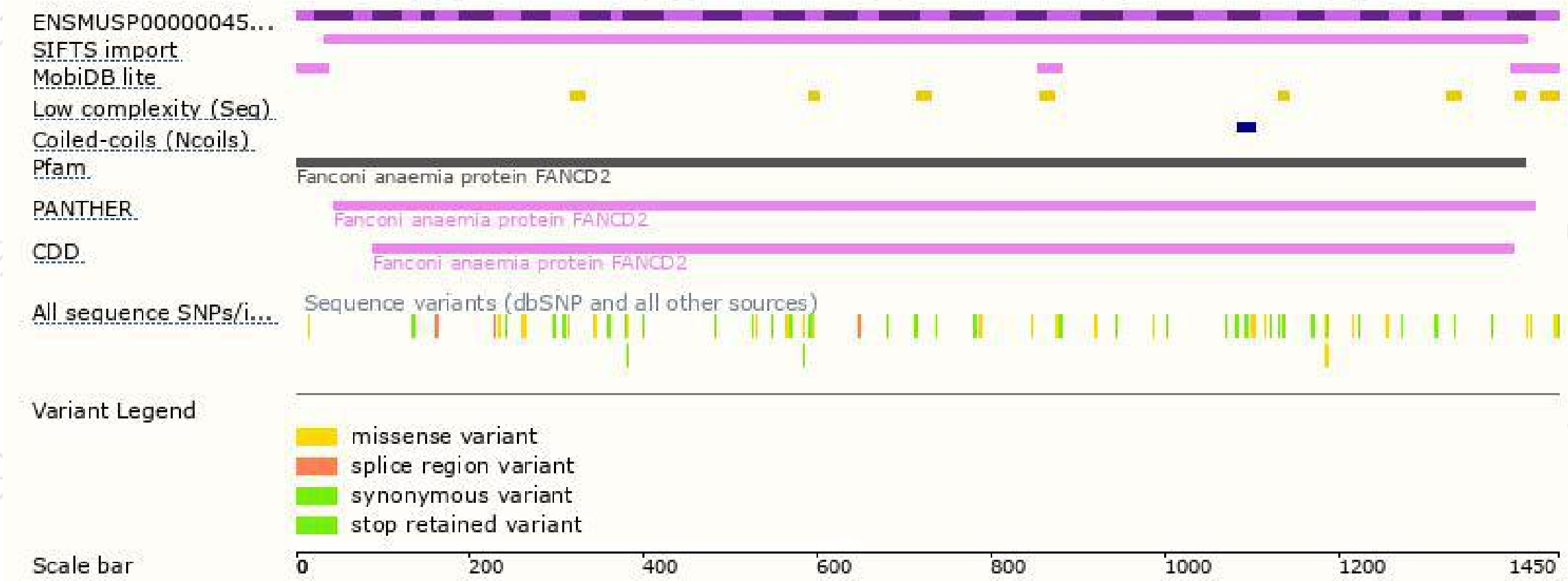


# Genomic location distribution

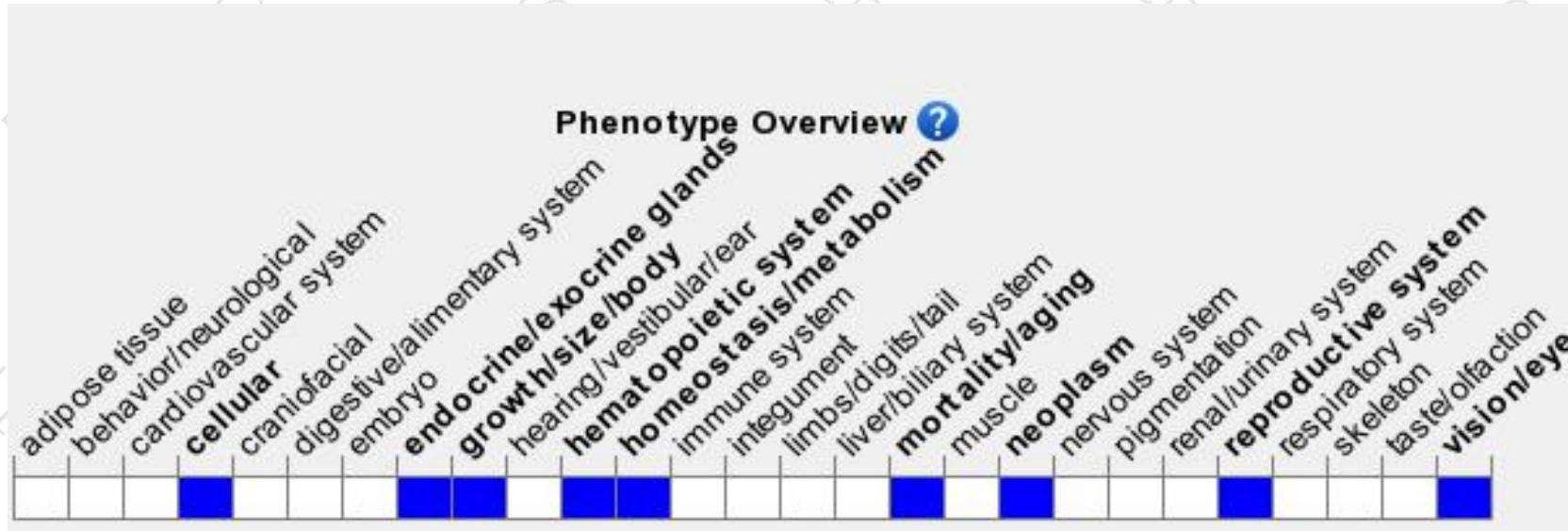




# Protein domain



# 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, Homozygous mutant mice exhibit defects observed in human patients with Fanconi anemia (FA) meiotic defects and germ cell loss. In addition, mutant mice display perinatal lethality, susceptibility to epithelial cancer, and microphthalmia.

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

Tel: 400-9660890

