

Dock2 Cas9-CKO Strategy

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Project Overview

Project Name

Dock2

Project type

Cas9-CKO

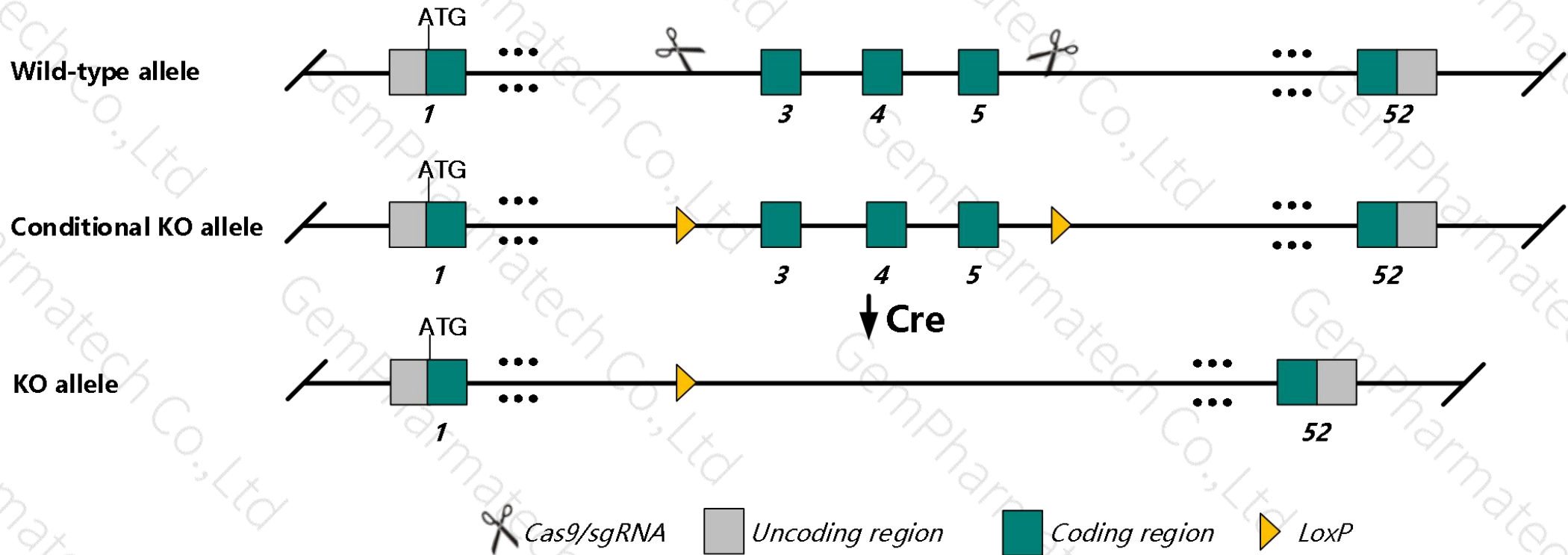
Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Dock2* gene. The schematic diagram is as follows:

Donor and CRISPR/Cas9 System



- The *Dock2* gene has 7 transcripts. According to the structure of *Dock2* gene, exon3-5 of *Dock2-201* (ENSMUST00000093193.11) transcript is recommended as the knockout region. The region contains 194bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Dock2* gene. The brief process is as follows: CRISPR/Cas9 system and Donor were microinjected into the fertilized eggs of C57BL/6JGpt 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/6JGpt mice.
- The flox mice will be knocked out after mating with mice expressing Cre recombinase, resulting in the loss of function of the target gene in specific tissues and cell types.

- According to the existing MGI data, Homozygous mutants are defective in the migration of T and B lymphocytes in response to chemokines, and thus display immune defects such as lymphocytopenia, atrophy of lymphoid follicles and loss of marginal-zone B cells.
- The *Dock2* gene is located on the Chr11. 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.

Gene information (NCBI)

Dock2 dedicator of cyto-kinesis 2 [*Mus musculus* (house mouse)]

Gene ID: 94176, updated on 21-Oct-2019

Summary

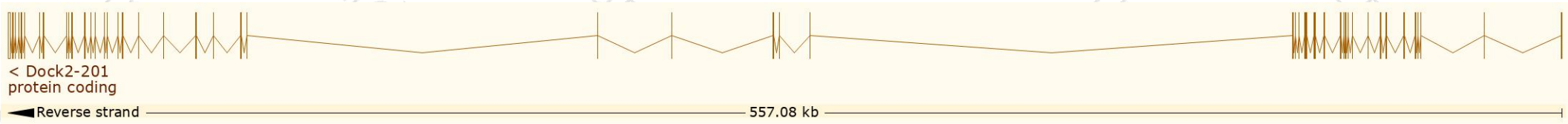
Official Symbol	Dock2 provided by MGI
Official Full Name	dedicator of cyto-kinesis 2 provided by MGI
Primary source	MGI:MGI:2149010
See related	Ensembl:ENSMUSG00000020143
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Hch; MBC; CED-5; AI662014; AW122239
Annotation information	Annotation category: suggests misassembly
Expression	Biased expression in spleen adult (RPKM 15.3), thymus adult (RPKM 14.5) and 11 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

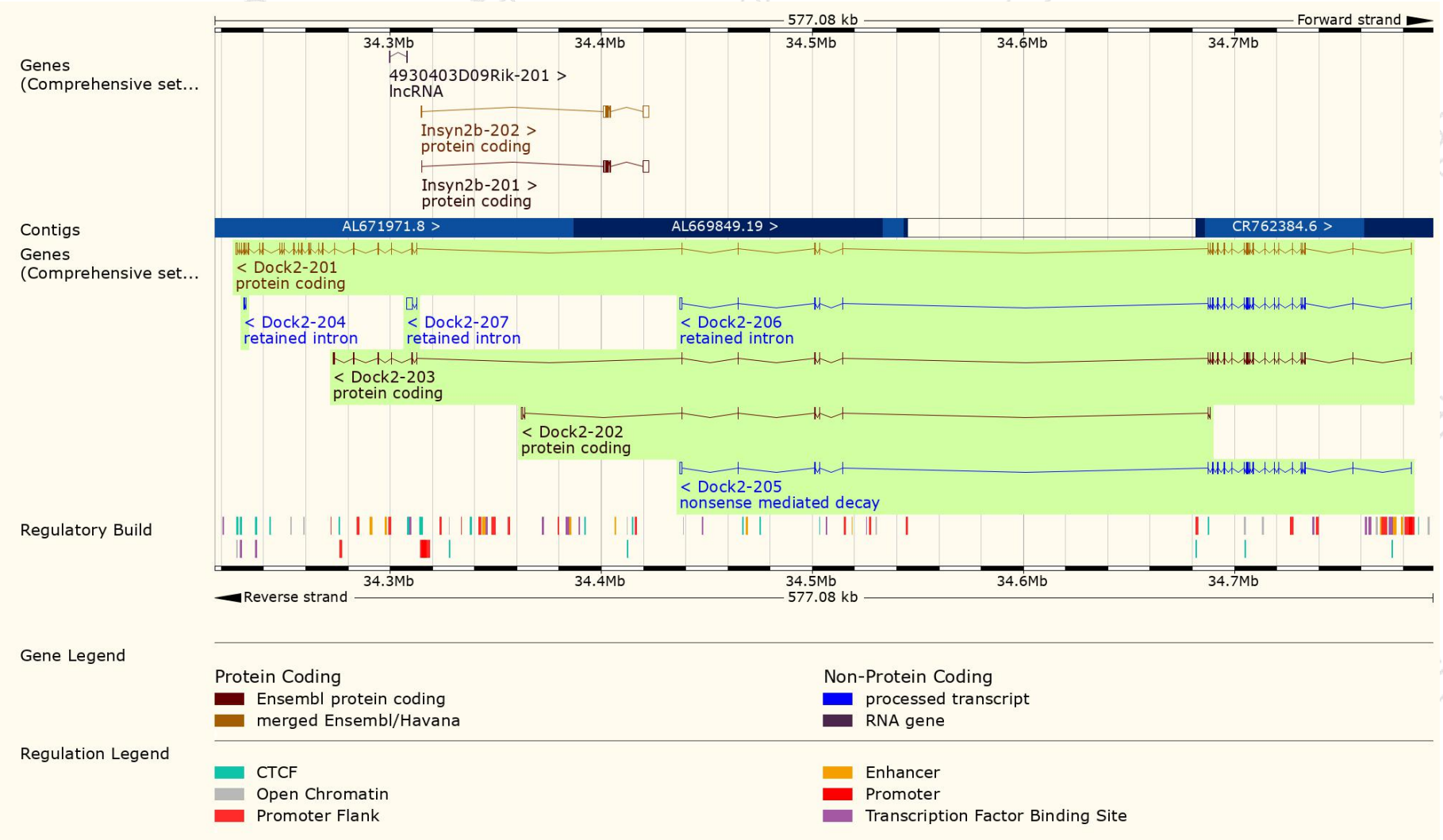
The gene has 7 transcripts,all transcripts are shown below:

Name ▲	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Dock2-201	ENSMUST00000093193.11	6409	1828aa	Protein coding	CCDS83791	Q8C3J5	TSL:1 GENCODE basic APPRIS P1
Dock2-202	ENSMUST00000101364.2	1616	295aa	Protein coding	-	Q3TMS1	TSL:1 GENCODE basic
Dock2-203	ENSMUST00000101365.8	3785	1175aa	Protein coding	-	Q5SRI3	TSL:1 GENCODE basic
Dock2-204	ENSMUST00000127846.1	366	No protein	Retained intron	-	-	TSL:3
Dock2-205	ENSMUST00000143540.7	3727	732aa	Nonsense mediated decay	-	D6RGU3	TSL:2
Dock2-206	ENSMUST00000154178.1	3819	No protein	Retained intron	-	-	TSL:2
Dock2-207	ENSMUST00000157036.1	2973	No protein	Retained intron	-	-	TSL:1

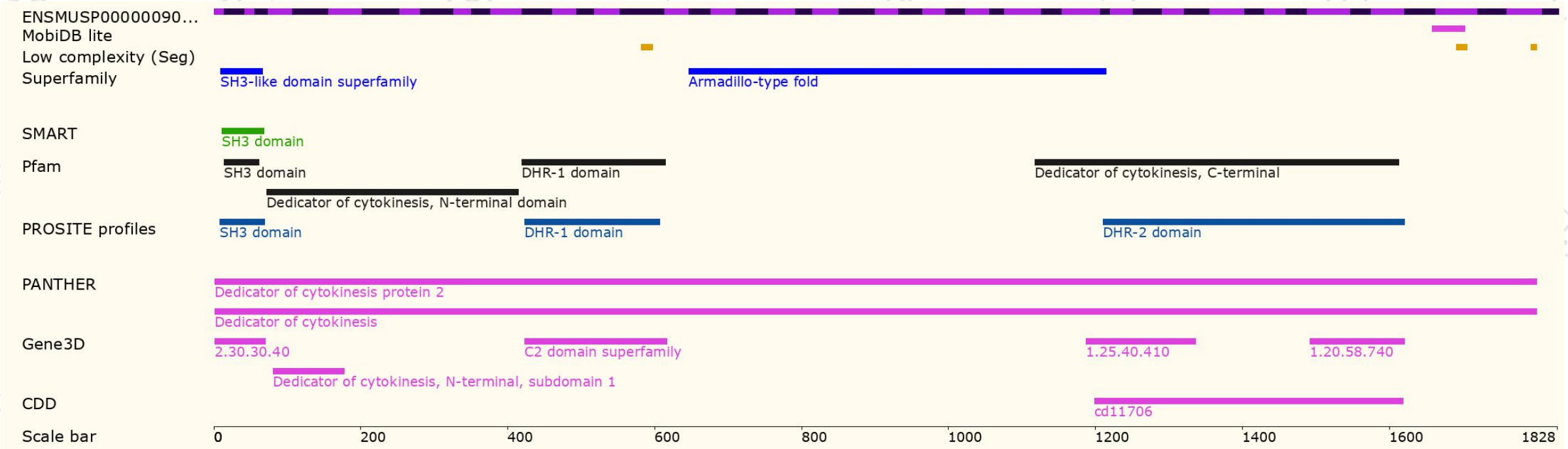
The strategy is based on the design of *Dock2-201* transcript,The transcription is shown below



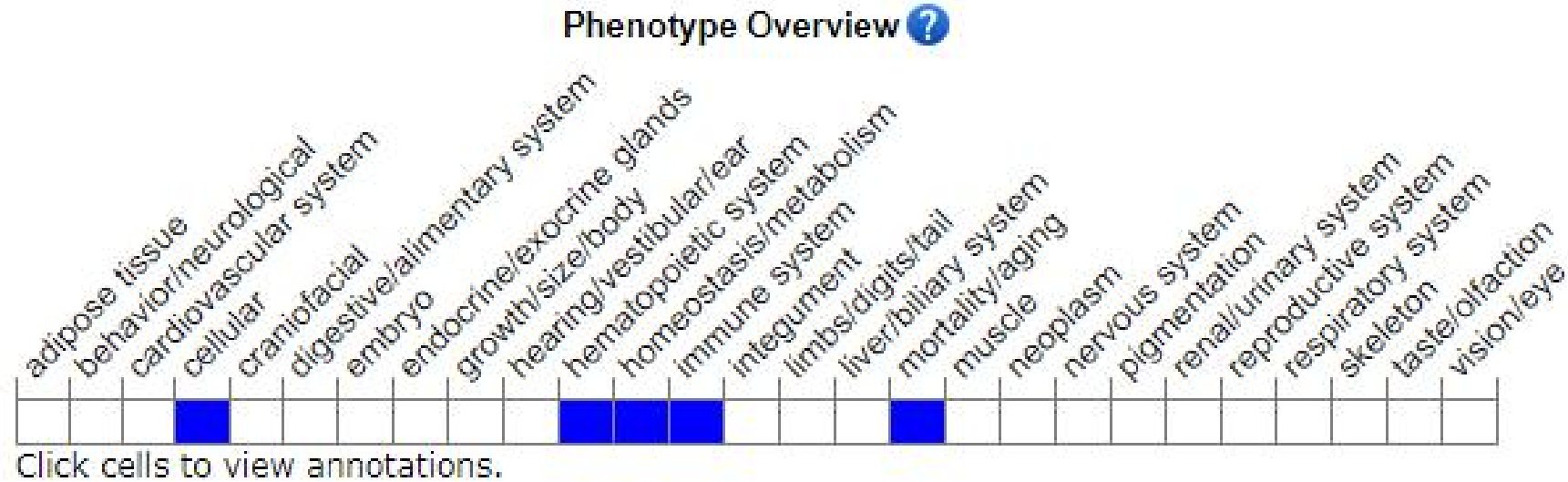
Genomic location distribution



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/>).

Homozygous mutants are defective in the migration of T and B lymphocytes in response to chemokines, and thus display immune defects such as lymphocytopenia, atrophy of lymphoid follicles and loss of marginal-zone B cells.

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

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