

Ddr2 Cas9-CKO Strategy

Designer:

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Design Date:

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Project Overview

Project Name

Ddr2

Project type

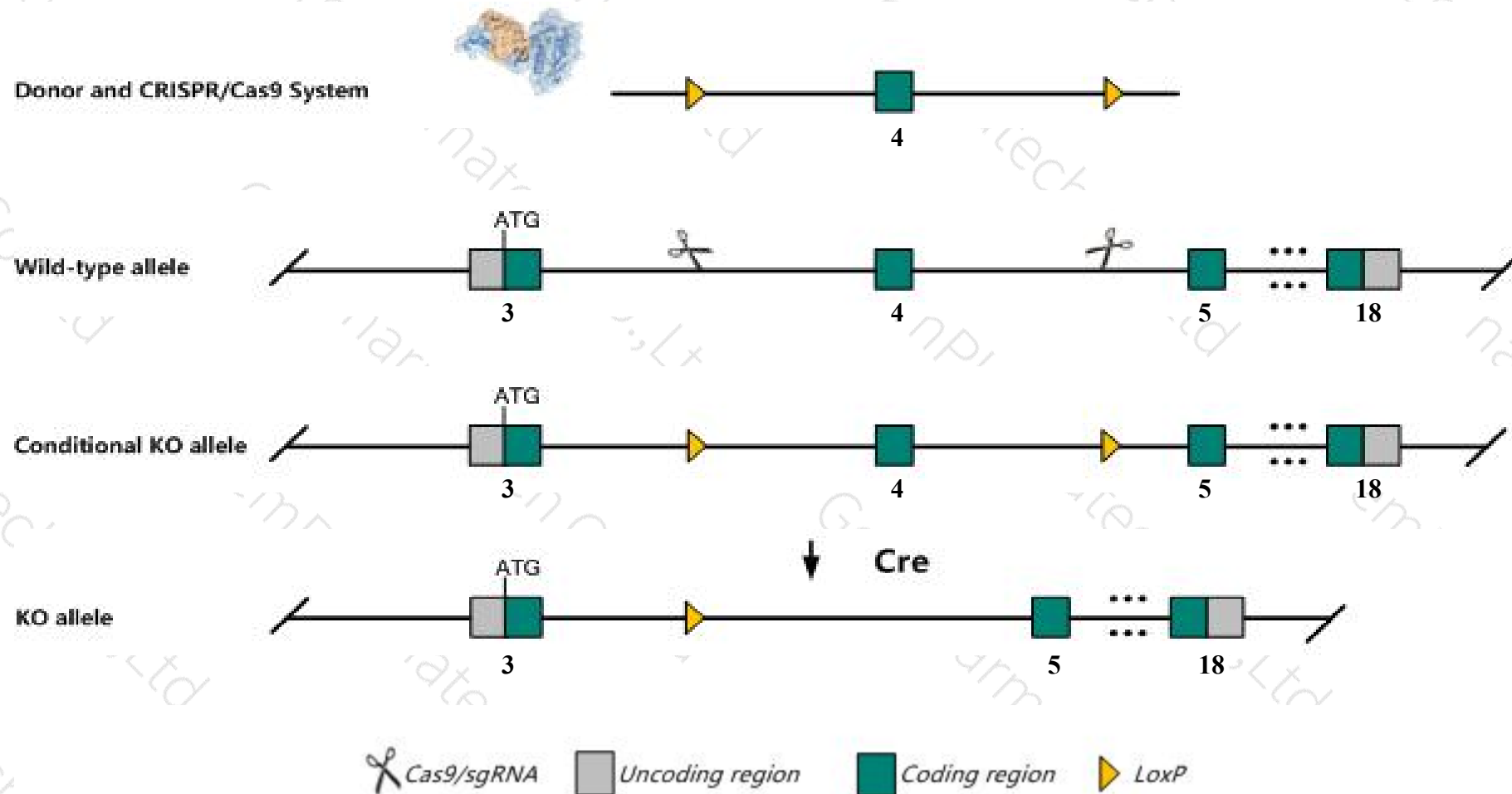
Cas9-CKO

Strain background

C57BL/6J

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Ddr2* gene. The schematic diagram is as follows:



Technical routes

- The *Ddr2* gene has 6 transcripts. According to the structure of *Ddr2* gene, exon4 of *Ddr2*-205 (ENSMUST00000194690.5) transcript is recommended as the knockout region. The region contains 103bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Ddr2* gene. The brief process is as follows: sgRNA was transcribed in vitro, donor vector was constructed. Cas9, sgRNA and Donor 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.
- The flox mice was 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, Homozygotes for a null allele show dwarfism, reduced chondrocyte proliferation, shortened long bones and snout, and skull anomalies. Homozygotes for another null allele show similar skeletal defects, small hearts, short cardiomyocytes, lower cardiac collagen density, and altered cardiac function.
- The *Ddr2* gene is located on the Chr1. 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)

Ddr2 discoidin domain receptor family, member 2 [*Mus musculus* (house mouse)]

Gene ID: 18214, updated on 10-Oct-2019

Summary

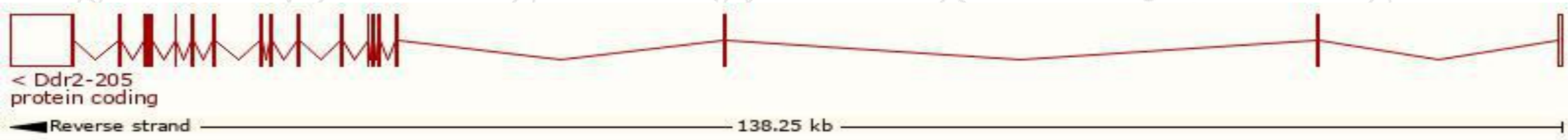
Official Symbol	Ddr2 provided by MGI
Official Full Name	discoidin domain receptor family, member 2 provided by MGI
Primary source	MGI:MGI:1345277
See related	Ensembl:ENSMUSG00000026674
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Ntrkr3; tyro10; AW495251
Expression	Biased expression in bladder adult (RPKM 28.6), limb E14.5 (RPKM 18.7) and 13 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

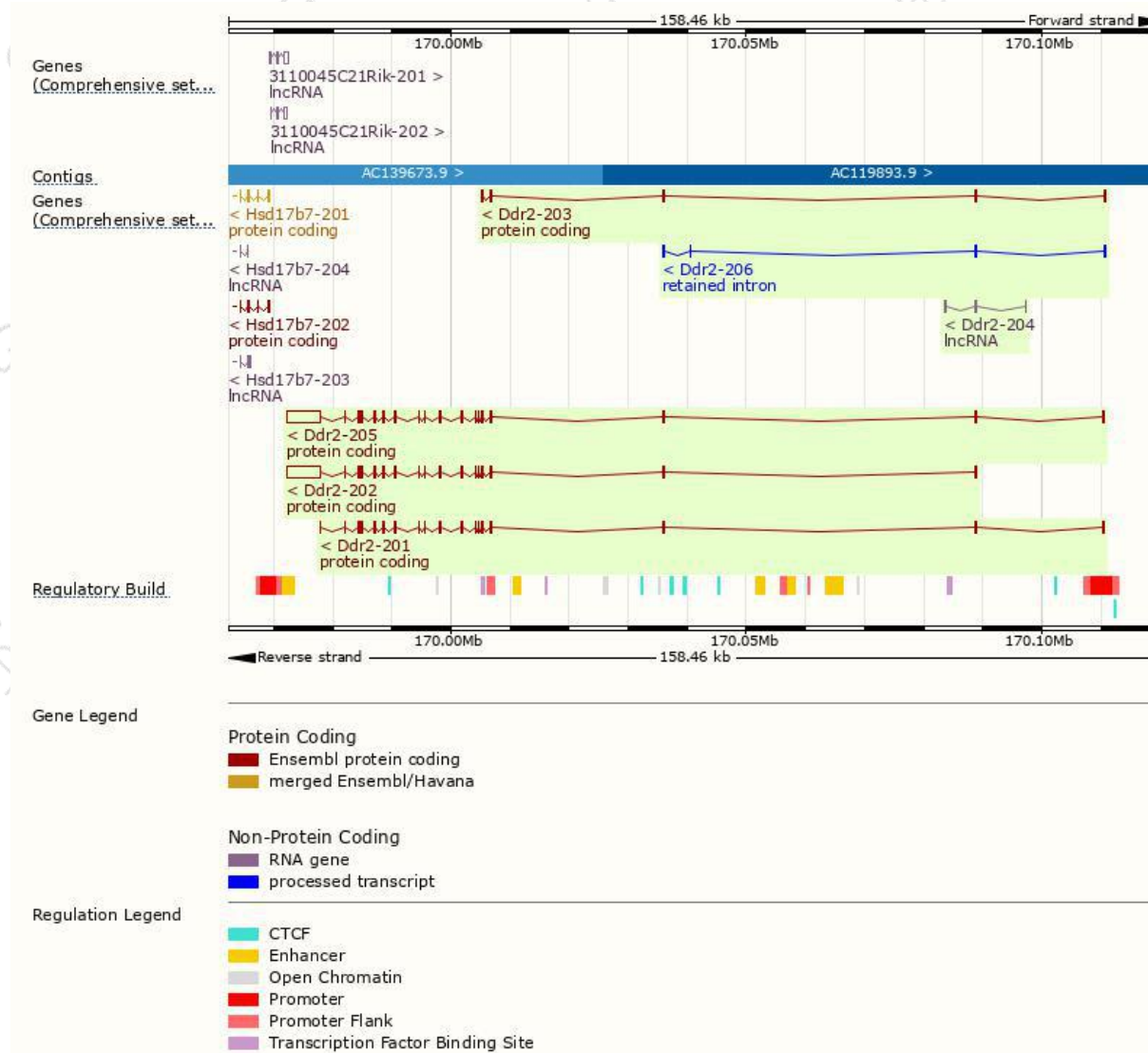
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Ddr2-205	ENSMUST00000194690.5	8590	854aa	Protein coding	CCDS48436	Q62371	TSL:1 GENCODE basic APPRIS P1
Ddr2-202	ENSMUST00000170800.7	8289	854aa	Protein coding	CCDS48436	Q62371	TSL:5 GENCODE basic APPRIS P1
Ddr2-201	ENSMUST00000027985.7	2928	854aa	Protein coding	CCDS48436	Q62371	TSL:1 GENCODE basic APPRIS P1
Ddr2-203	ENSMUST00000192312.5	672	129aa	Protein coding	-	A0A0A6YXY2	CDS 3' incomplete TSL:3
Ddr2-206	ENSMUST00000195867.5	641	No protein	Retained intron	-	-	TSL:3
Ddr2-204	ENSMUST00000194619.1	442	No protein	lncRNA	-	-	TSL:5

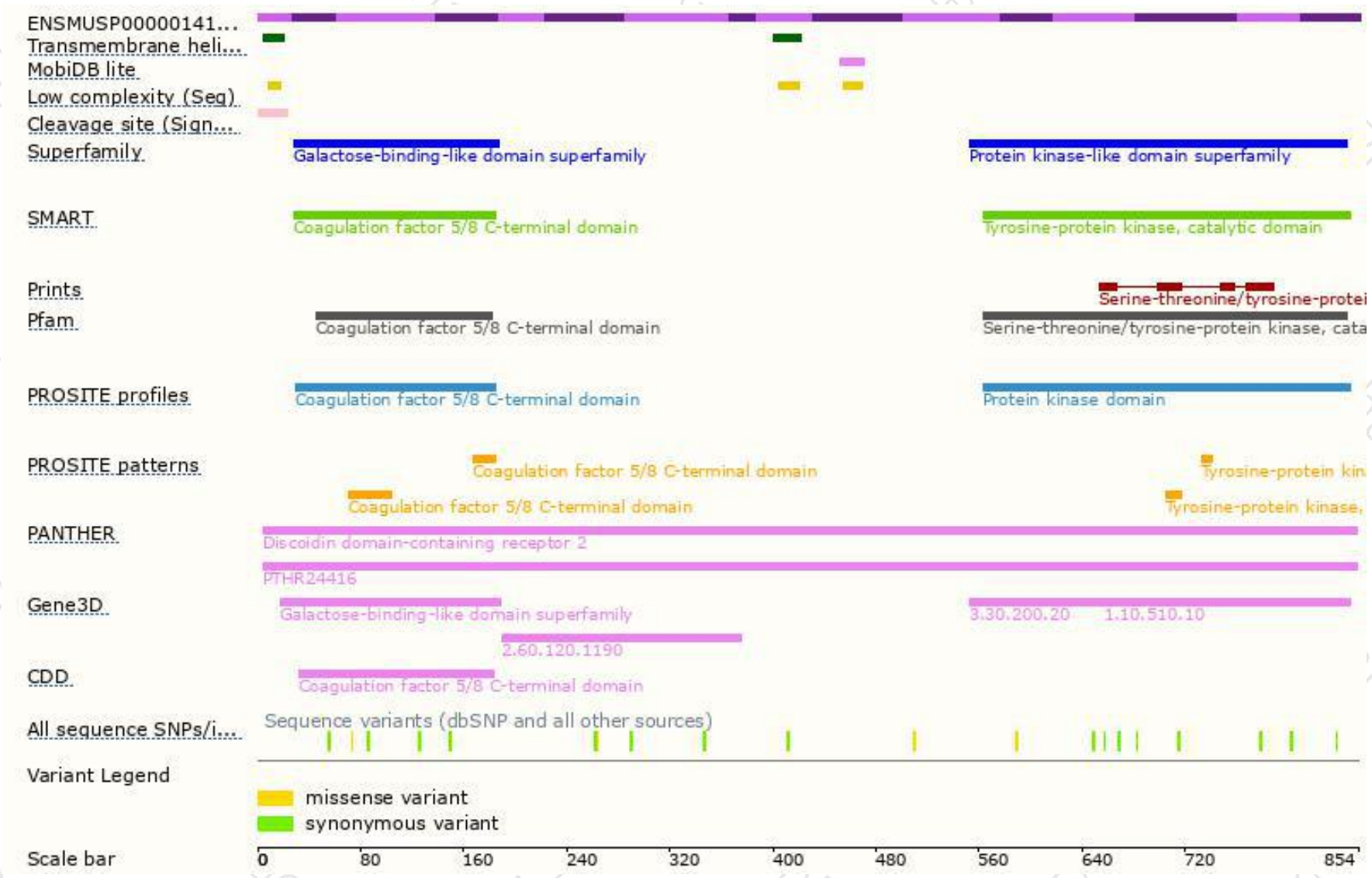
The strategy is based on the design of *Ddr2-205* transcript,The transcription is shown below



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, Homozygotes for a null allele show dwarfism, reduced chondrocyte proliferation, shortened long bones and snout, and skull anomalies. Homozygotes for another null allele show similar skeletal defects, small hearts, short cardiomyocytes, lower cardiac collagen density, and altered cardiac function.

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

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