

Gdf11 Cas9-CKO Strategy

Designer:

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Design Date:

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Project Overview

Project Name

Gdf11

Project type

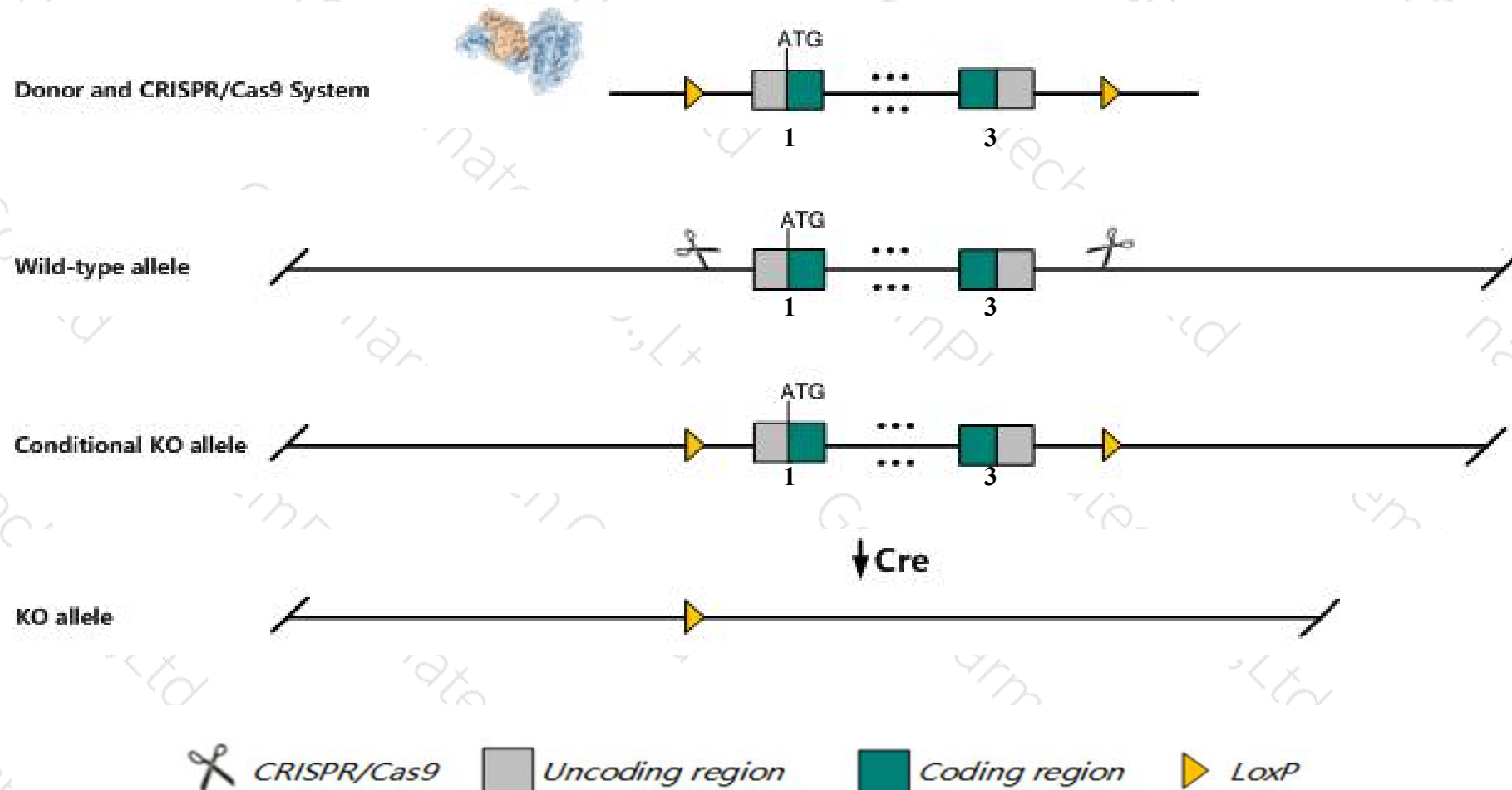
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Gdf11* gene. The schematic diagram is as follows:



Technical routes

- The *Gdf11* gene has 1 transcript. According to the structure of *Gdf11* gene, exon1-exon3 of *Gdf11*-201 (ENSMUST00000026408.6) transcript is recommended as the knockout region. The region contains all of the coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Gdf11* 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, Homozygotes for a null allele die neonatally showing altered patterning of the axial skeleton and impaired renal, palate, stomach, spleen and pancreatic development. A second null allele also alters retinal and olfactory epithelium neurogenesis. A third null allele causes extra thoracic vertebrae.
- The *Gdf11* 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.

Gene information (NCBI)

Gdf11 growth differentiation factor 11 [Mus musculus (house mouse)]

Gene ID: 14561, updated on 19-Mar-2019

Summary



Official Symbol Gdf11 provided by [MGI](#)

Official Full Name growth differentiation factor 11 provided by [MGI](#)

Primary source [MGI:MGI:1338027](#)

See related [Ensembl:ENSMUSG000000025352](#)

Gene type protein coding

RefSeq status REVIEWED

Organism [Mus musculus](#)

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus

Also known as BMP-11, Bmp11

Summary This gene encodes a secreted ligand of the TGF-beta (transforming growth factor-beta) superfamily of proteins. Ligands of this family bind various TGF-beta receptors leading to recruitment and activation of SMAD family transcription factors that regulate gene expression. The encoded preproprotein is proteolytically processed to generate each subunit of the disulfide-linked homodimer. This protein plays a role in the development of the nervous and other organ systems, and may regulate aging. Mice lacking a functional copy of this gene exhibit impaired anteroposterior patterning and other developmental defects. [provided by RefSeq, Aug 2016]

Expression Broad expression in CNS E11.5 (RPKM 8.5), whole brain E14.5 (RPKM 5.3) and 23 other tissues [See more](#)

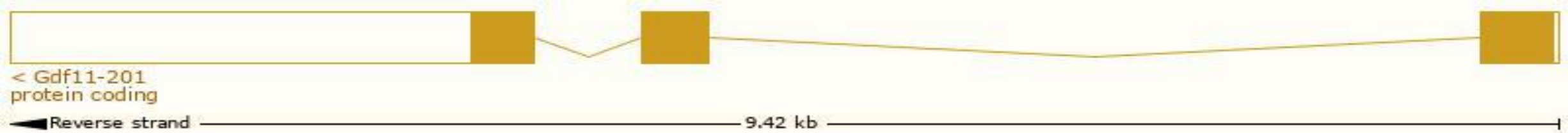
Orthologs [human](#) [all](#)

Transcript information (Ensembl)

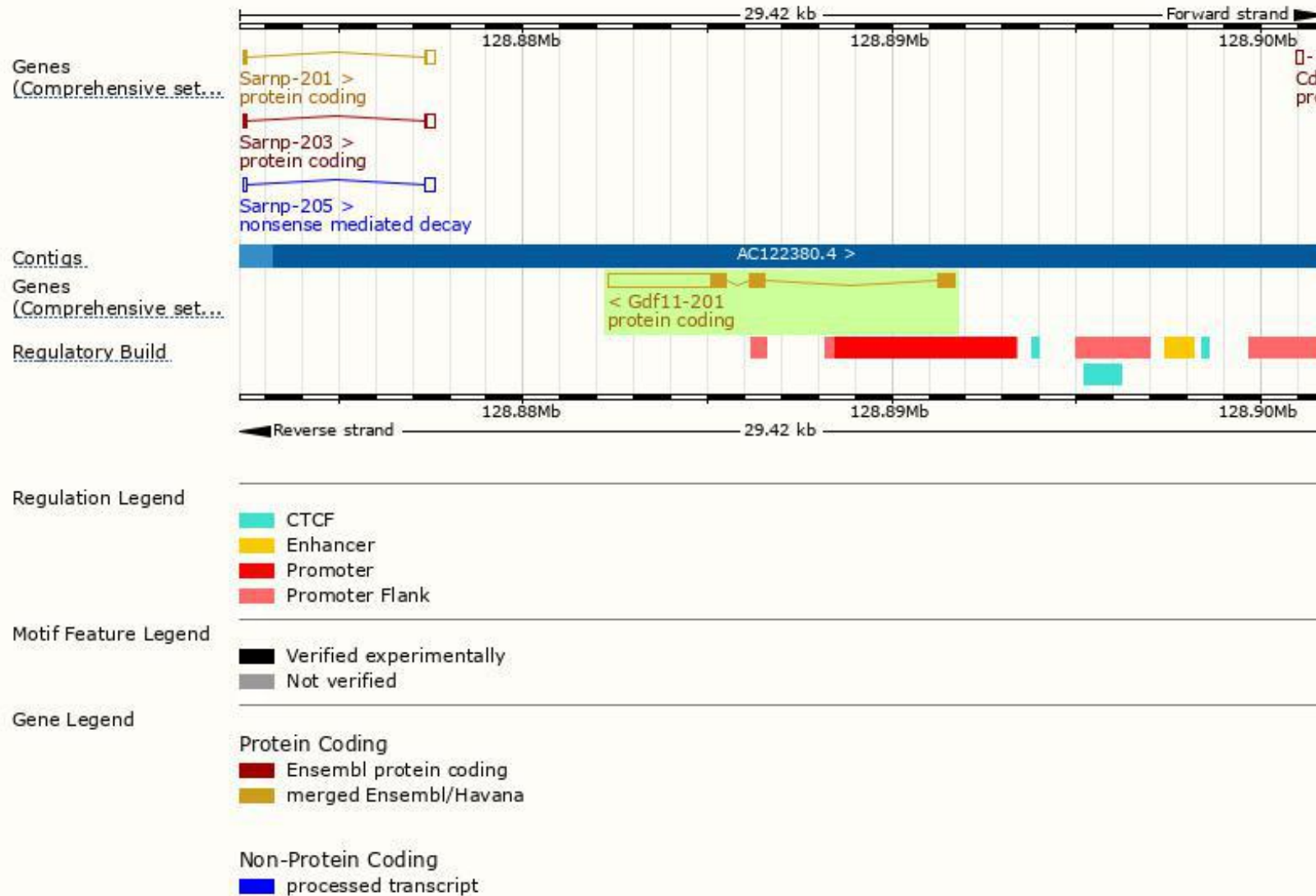
The gene has 1 transcript, and the transcript is shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Gdf11-201	ENSMUST00000026408.6	4062	405aa	Protein coding	CCDS24296	Q9Z1W4	TSL:1 GENCODE basic APPRIS P1

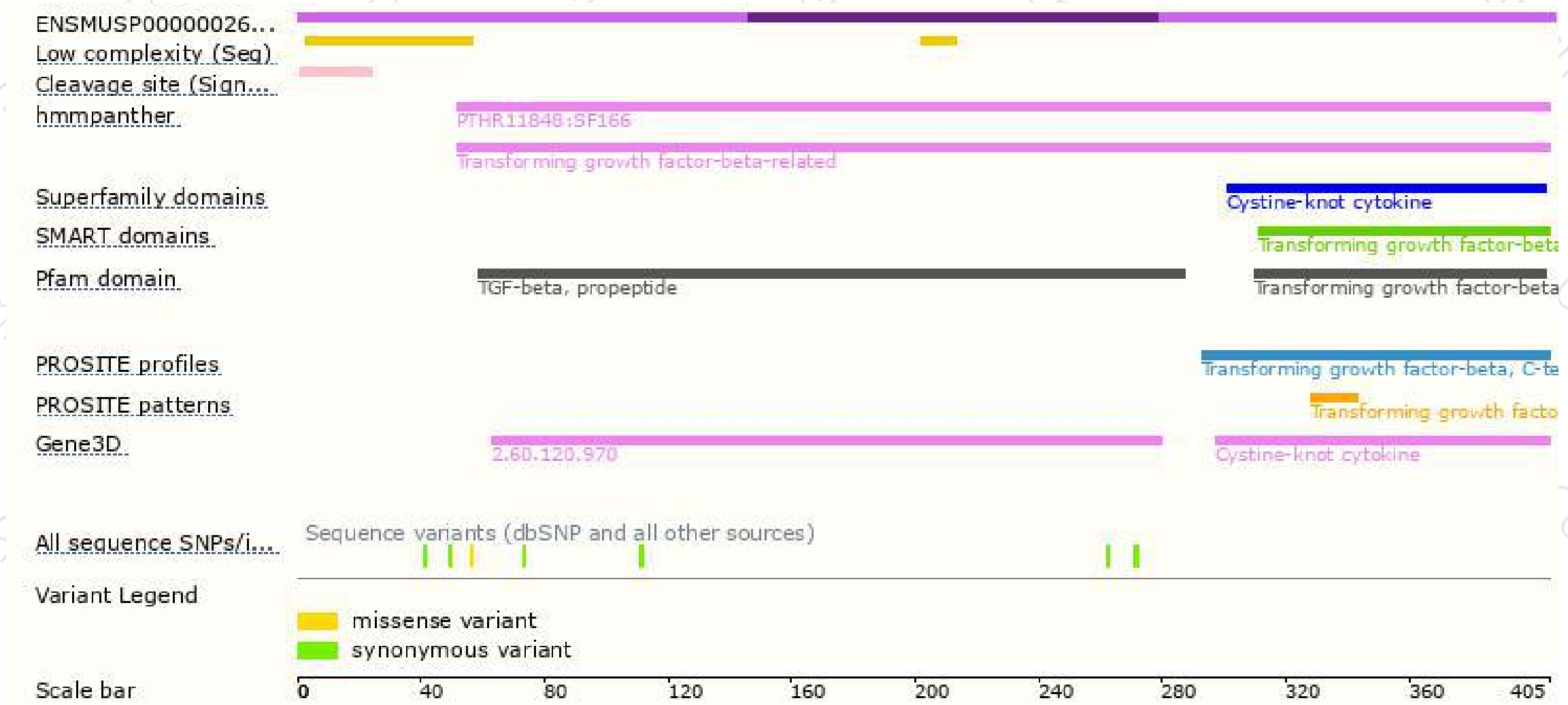
The strategy is based on the design of *Gdf11-201* transcript, The transcription is shown below



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, Homozygotes for a null allele die neonatally showing altered patterning of the axial skeleton and impaired renal, palate, stomach, spleen and pancreatic development. A second null allele also alters retinal and olfactory epithelium neurogenesis. A third null allele causes extra thoracic vertebrae.

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

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