Anxa7 Cas9-CKO Strategy

Designer: Huimin Su

Reviewer: Ruiuri Zhang

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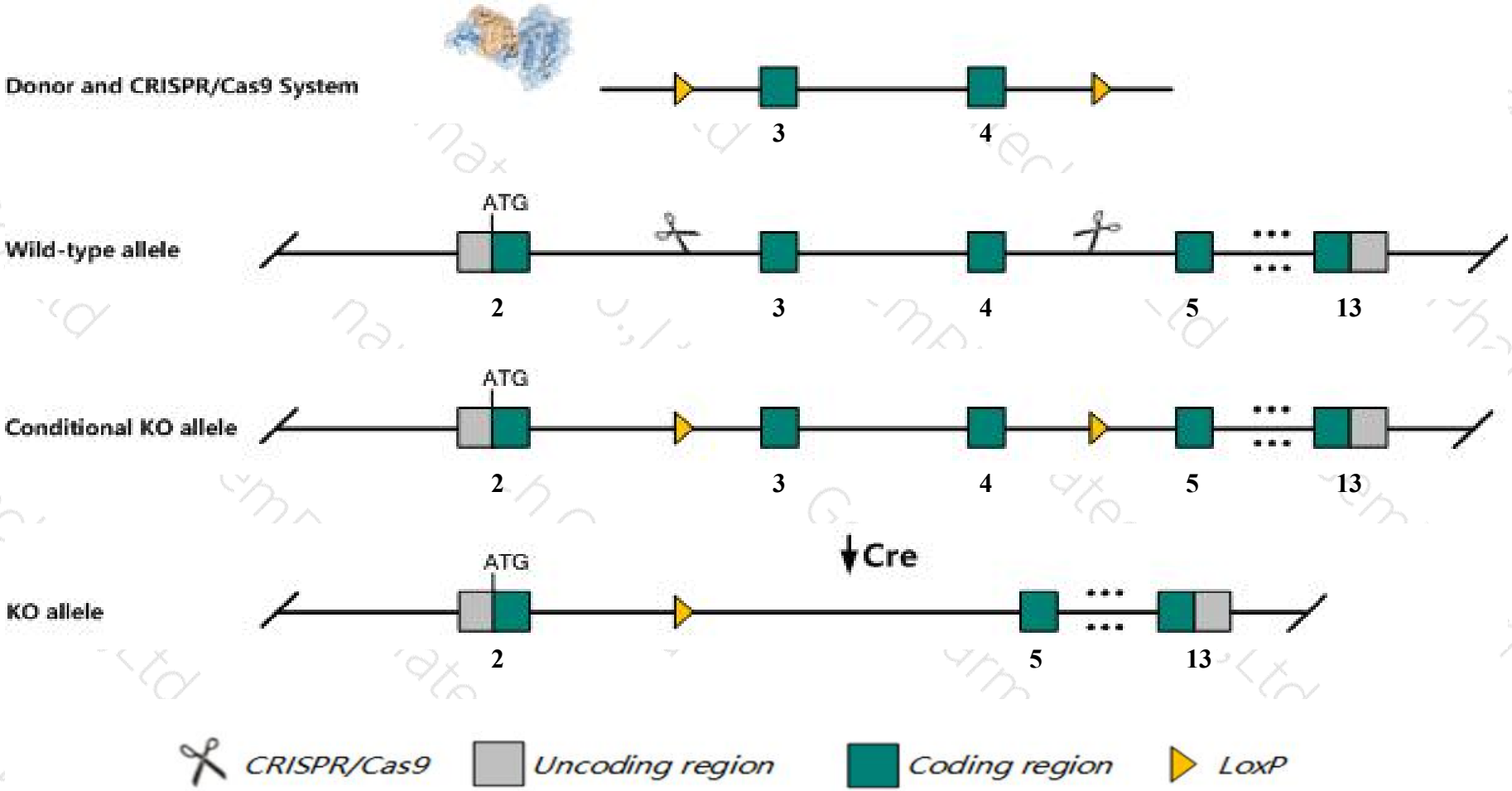
Project Overview

Project Name	***Anxa7***
Project type	**Cas9-CKO**
Strain background	**C57BL/6JGpt**

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Anxa7* gene. The schematic diagram is as follows:



Technical routes

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- The *Anxa7* gene has 11 transcripts. According to the structure of *Anxa7* gene, exon3-exon4 of *Anxa7-201* (ENSMUST00000065504.16) transcript is recommended as the knockout region.The region contains 307bp coding sequence. Knock out the region will result in disruption of protein function.

- In this project we use CRISPR/Cas9 technology to modify *Anxa7* 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.

Notice

- According to the existing MGI data,homozygotes for a null allele are viable but exhibit altered ca2+ signaling and/or homeostasis in cardiomyocytes and glia cells, and changes in erythrocyte shape, osmotic resistance, platelet number and aggregation velocity. homozygotes for another null allele die at ~e10 with cerebral hemorrhage.
- *Anxa7-210* transcript is incomplete, so the effect on it is unknown.
- The *Anxa7* gene is located on the Chr14. 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）

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Anxa7 annexin A7 [*Mus musculus* (house mouse)]

Gene ID: 11750, updated on 3-May-2020

Summary

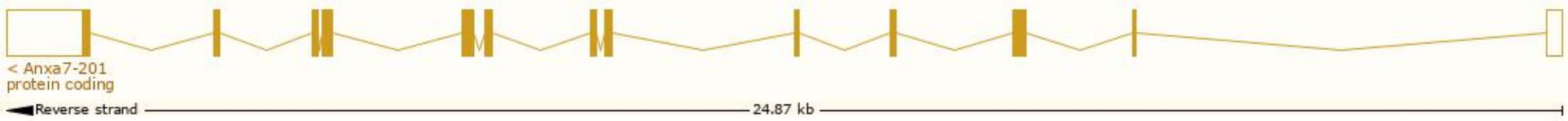
Official Symbol	Anxa7 provided by MGI
Official Full Name	annexin A7 provided by MGI
Primary source	MGI:MGI:88031
See related	Ensembl:ENSMUSG00000021814
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Anx7; synexin; AI265384; AI316497
Expression	Ubiquitous expression in placenta adult (RPKM 48.4), bladder adult (RPKM 40.7) and 28 other tissues See more
Orthologs	human all

Transcript information（Ensembl）

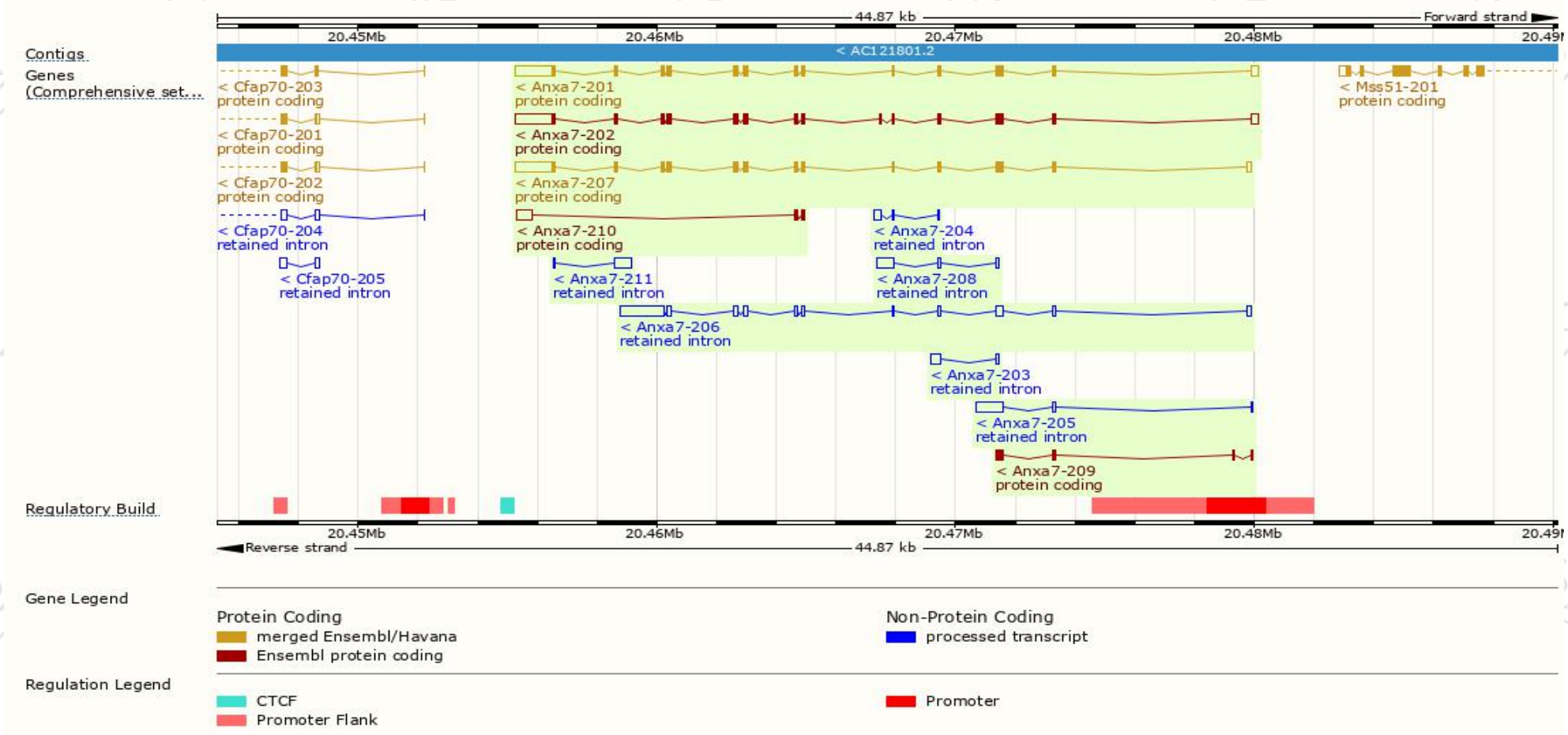
The gene has 11 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Anxa7-201	ENSMUST00000065504.16	2851	463aa	Protein coding	CCDS26845	Q07076	TSL:1 GENCODE basic APPRIS P2
Anxa7-207	ENSMUST00000224975.1	2727	463aa	Protein coding	CCDS26845	Q07076	GENCODE basic APPRIS P2
Anxa7-202	ENSMUST00000100844.5	2904	485aa	Protein coding	-	A0A2C9F2D2	TSL:1 GENCODE basic APPRIS ALT2
Anxa7-210	ENSMUST00000225941.1	739	72aa	Protein coding	-	A0A286YCW4	CDS 5' incomplete
Anxa7-209	ENSMUST00000225132.1	364	86aa	Protein coding	-	A0A286YCS5	CDS 3' incomplete
Anxa7-206	ENSMUST00000224410.1	2653	No protein	Retained intron	-	-	-
Anxa7-205	ENSMUST00000224344.1	988	No protein	Retained intron	-	-	-
Anxa7-208	ENSMUST00000225118.1	761	No protein	Retained intron	-	-	-
Anxa7-211	ENSMUST00000226001.1	673	No protein	Retained intron	-	-	-
Anxa7-203	ENSMUST00000223681.1	420	No protein	Retained intron	-	-	-
Anxa7-204	ENSMUST00000223960.1	387	No protein	Retained intron	-	-	-

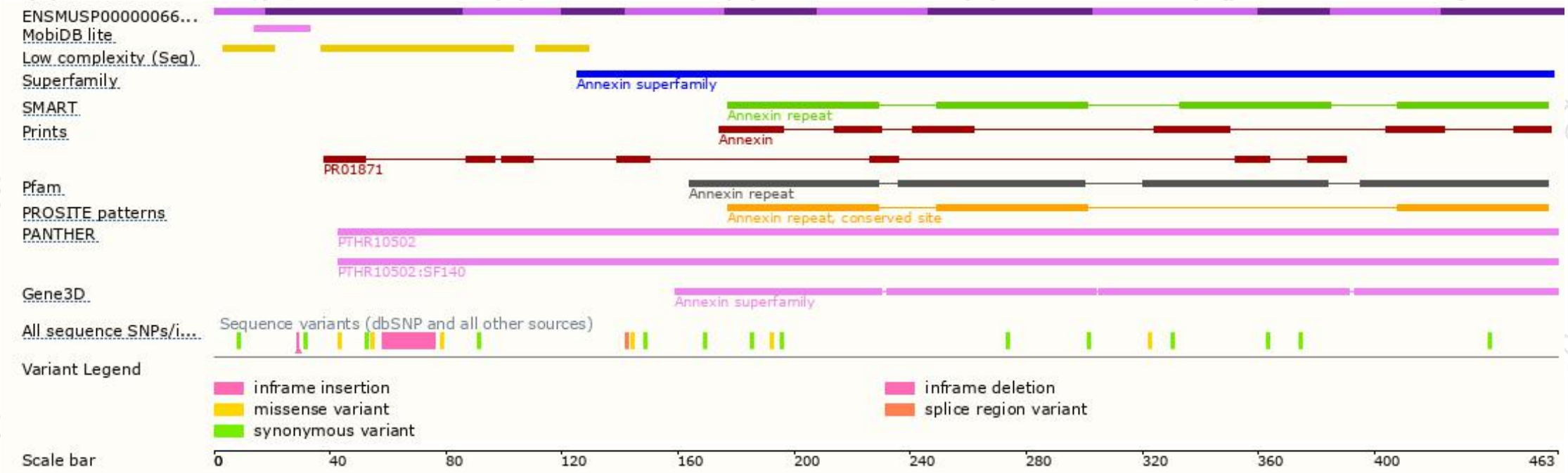
The strategy is based on the design of *Anxa7-201* transcript, the transcription is shown below



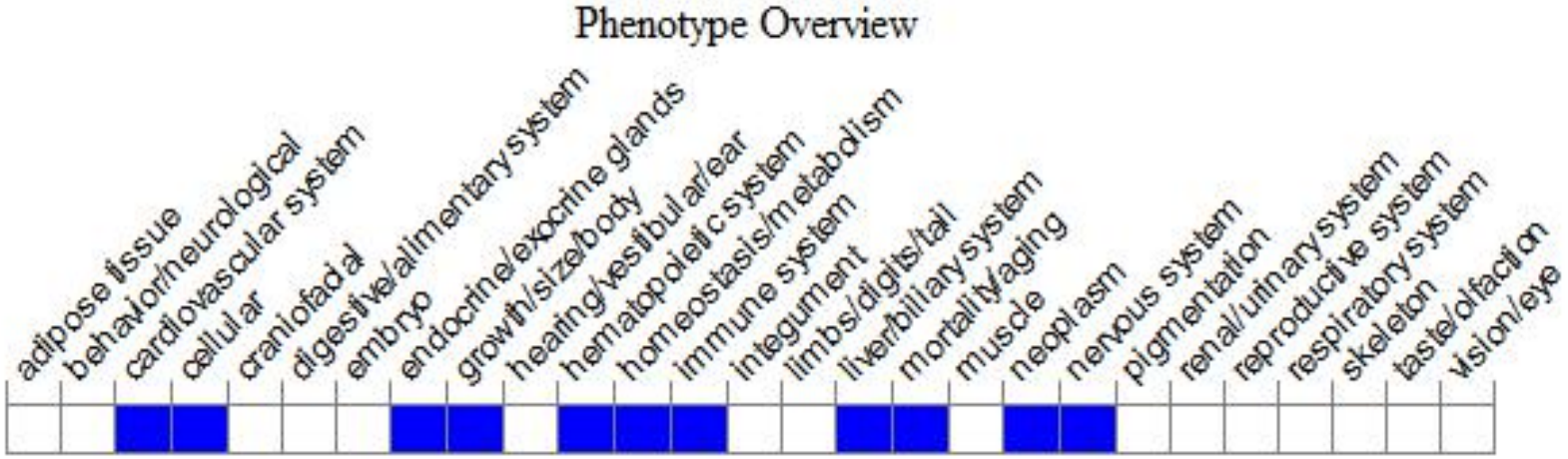
Genomic location distribution



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 are viable but exhibit altered Ca2+ signaling and/or homeostasis in cardiomyocytes and glia cells, and changes in erythrocyte shape, osmotic resistance, platelet number and aggregation velocity. Homozygotes for another null allele die at ~E10 with cerebral hemorrhage.

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

Tel: 400-9660890

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