

# *Sema3c* Cas9-KO Strategy

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# Project Overview

**Project Name**

*Sema3c*

**Project type**

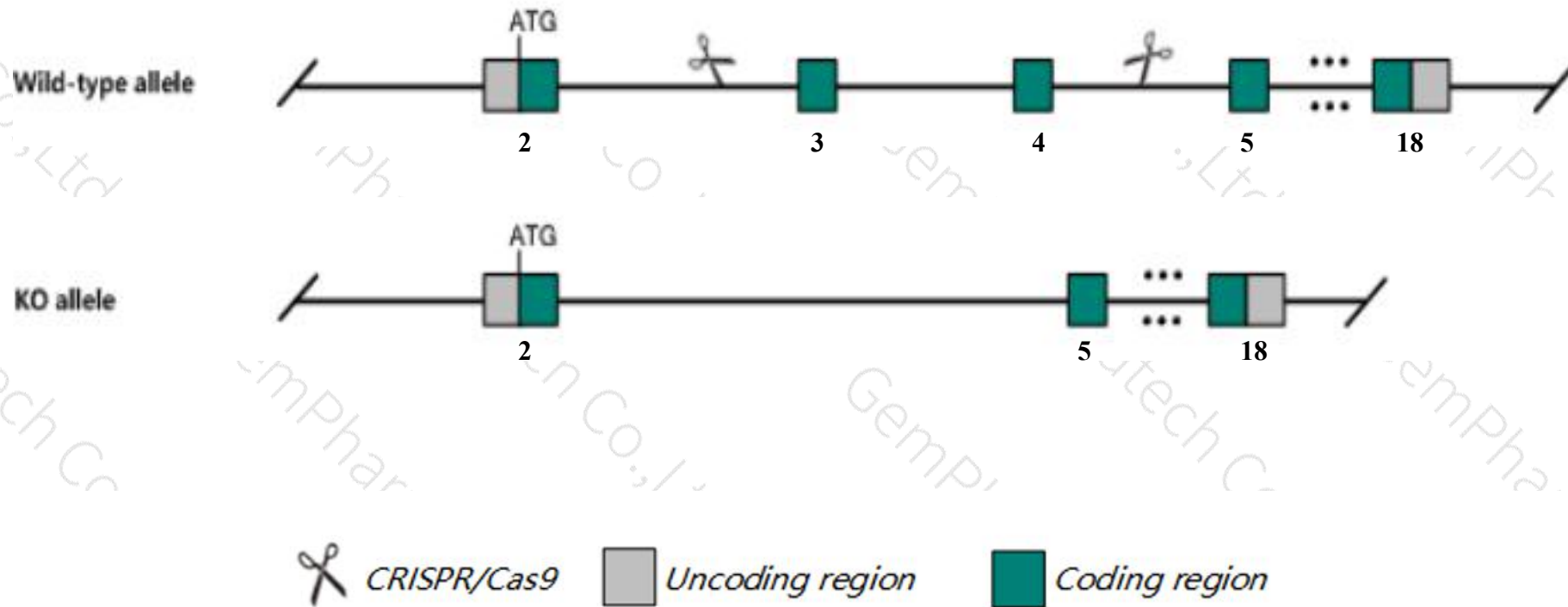
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Sema3c* gene. The schematic diagram is as follows:



# Technical routes

- The *Sema3c* gene has 5 transcripts. According to the structure of *Sema3c* gene, exon3-exon4 of *Sema3c*-201(ENSMUST00000030568.13) transcript is recommended as the knockout region. The region contains 224bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Sema3c* gene. The brief process is as follows: CRISPR/Cas9 system 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.

- According to the existing MGI data, mice homozygous for an ENU mutation exhibit perinatal lethality, hypopigmentation and abnormal heart development. Mice homozygous for a knock-out allele exhibit prenatal lethality associated with heart defects.
- The *Sema3c* gene is located on the Chr5. 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)

## Sema3c sema domain, immunoglobulin domain (Ig), short basic domain, secreted, (semaphorin) 3C [Mus musculus (house mouse)]

Gene ID: 20348, updated on 13-Mar-2020

### Summary



<b>Official Symbol</b>	Sema3c provided by <a href="#">MGI</a>
<b>Official Full Name</b>	sema domain, immunoglobulin domain (Ig), short basic domain, secreted, (semaphorin) 3C provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:107557</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000028780</a>
<b>Gene type</b>	protein coding
<b>RefSeq status</b>	PROVISIONAL
<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>	1110036B02Rik, SemE, Semae
<b>Expression</b>	Broad expression in bladder adult (RPKM 26.3), lung adult (RPKM 25.9) and 17 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

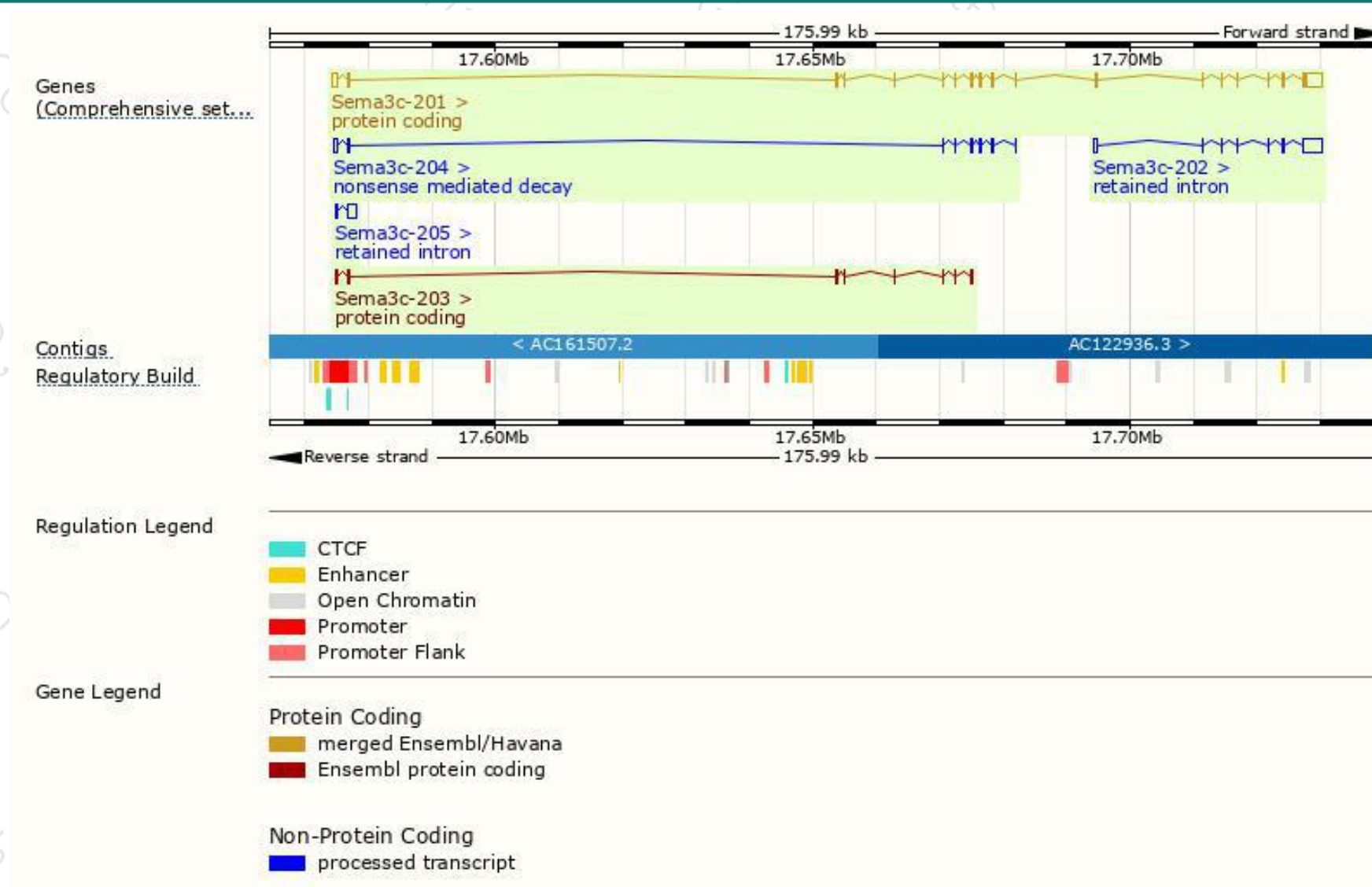
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Sema3c-201	<a href="#">ENSMUST00000030568.13</a>	5491	<a href="#">751aa</a>	Protein coding	<a href="#">CCDS19099</a>	<a href="#">Q62181</a>	TSL:1 GENCODE basic APPRIS P1
Sema3c-203	<a href="#">ENSMUST00000169603.1</a>	785	<a href="#">226aa</a>	Protein coding	-	<a href="#">E9Q0Z0</a>	CDS 3' incomplete TSL:3
Sema3c-204	<a href="#">ENSMUST00000170181.7</a>	1142	<a href="#">42aa</a>	Nonsense mediated decay	-	<a href="#">E9PXL2</a>	TSL:5
Sema3c-202	<a href="#">ENSMUST00000115271.2</a>	4075	No protein	Retained intron	-	-	TSL:1
Sema3c-205	<a href="#">ENSMUST00000170348.1</a>	1549	No protein	Retained intron	-	-	TSL:1

The strategy is based on the design of *Sema3c-201* transcript,the transcription is shown below:

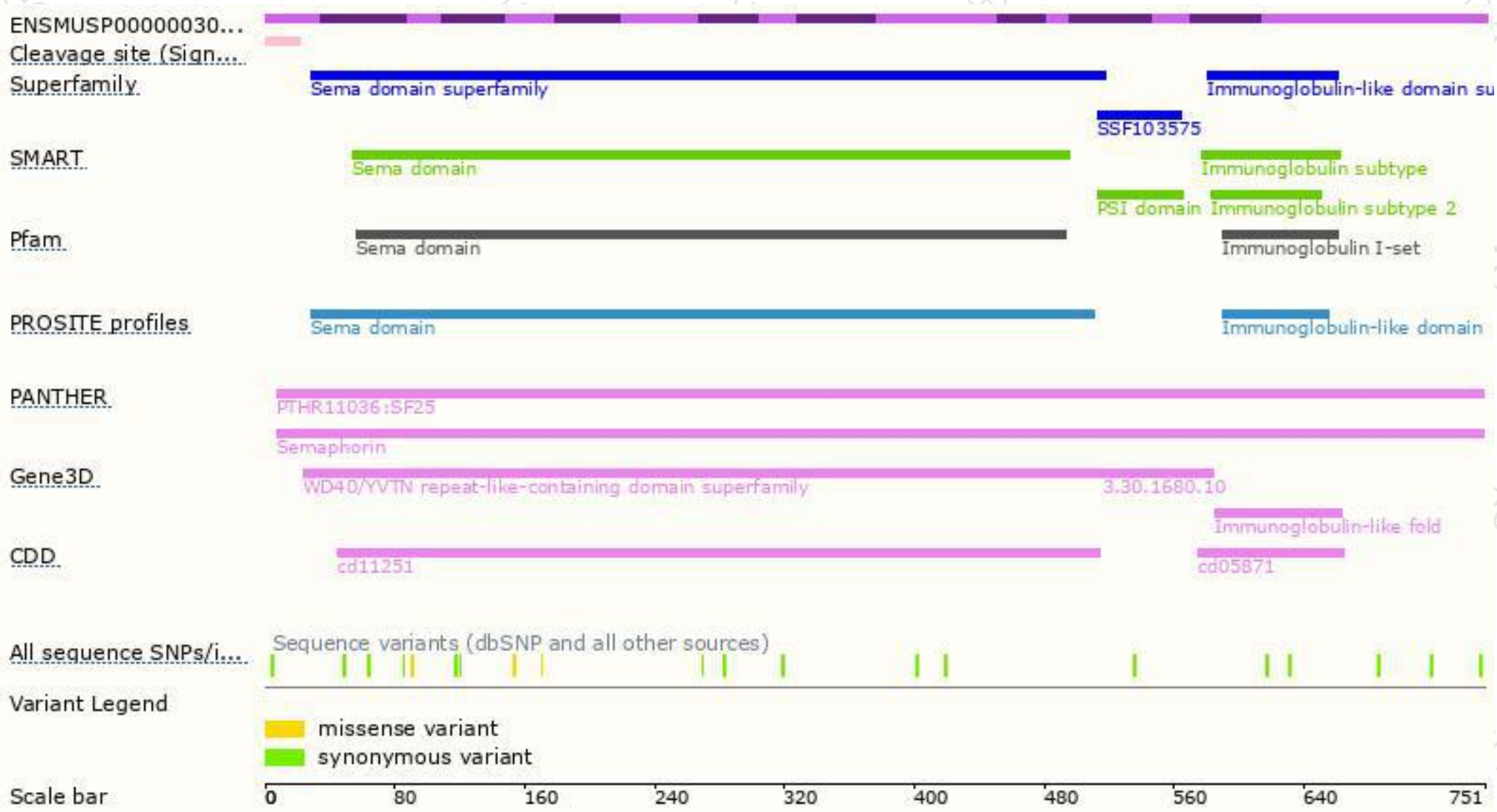


# Genomic location distribution

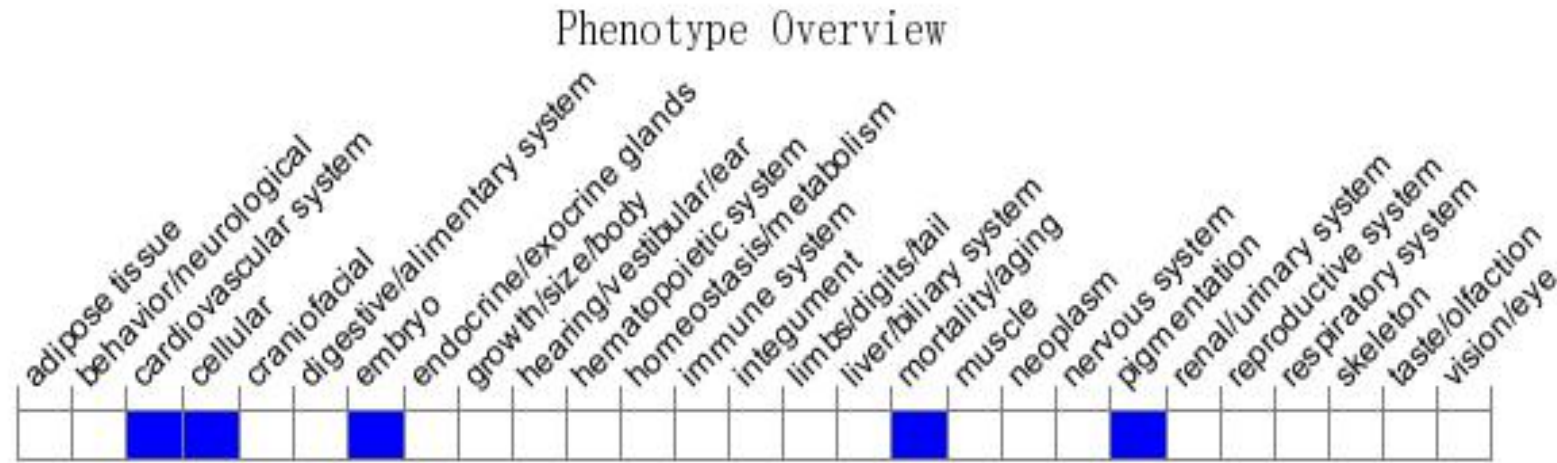




# Protein domain



# 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, mice homozygous for an ENU mutation exhibit perinatal lethality, hypopigmentation and abnormal heart development. Mice homozygous for a knock-out allele exhibit prenatal lethality associated with heart defects.

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

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