



# ***Edn2 Cas9-KO Strategy***

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**Reviewer: Shilei Zhu**

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# Project Overview

<b>Project Name</b>	<i>Edn2</i>
<b>Project type</b>	Cas9-KO
<b>Strain background</b>	C57BL/6J

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Edn2* gene. The schematic diagram is as follows:



# Technical routes

- The *Edn2* gene has 1 transcript. According to the structure of *Edn2* gene, exon3-exon5 of *Edn2-201* (ENSMUST00000030384.4) transcript is recommended as the knockout region. The region contains 316bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Edn2* gene. The brief process is as follows: CRISPR/Cas9 system



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# Notice

- According to the existing MGI data, Mice homozygous for a knock-out allele exhibit growth retardation, hypothermia, hypoxic hypoxia, hypercapnia, emphysema and premature death.
- The *Edn2* gene is located on the Chr4. 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)



## Edn2 endothelin 2 [Mus musculus (house mouse)]

Gene ID: 13615, updated on 31-Jan-2019

### Summary



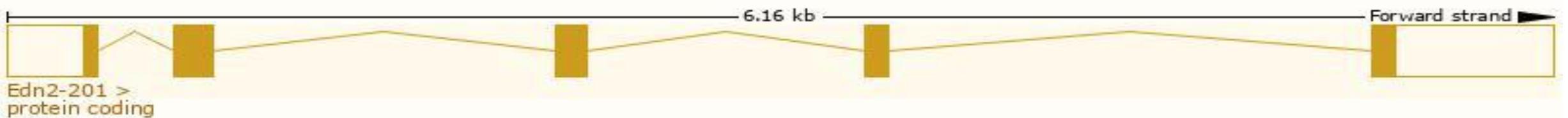
<b>Official Symbol</b>	Edn2 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	endothelin 2 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:95284</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000028635</a>
<b>Gene type</b>	protein coding
<b>RefSeq status</b>	REVIEWED
<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>	ET-2, PPET2, VIC
<b>Summary</b>	This gene encodes a member of the endothelin family of peptides. The encoded preproprotein undergoes proteolytic processing to generate a potent vasoconstrictive peptide. This gene is abundantly expressed in the gastrointestinal tract, strongly induced in photoreceptor cells in retinal diseases and injury, and produced by microglia and macrophages in the early stages of glaucoma. Mice lacking the encoded protein exhibit severe growth retardation, hypothermia and juvenile lethality. [provided by RefSeq, Feb 2016]
<b>Expression</b>	Biased expression in large intestine adult (RPKM 14.8), small intestine adult (RPKM 13.6) and 5 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

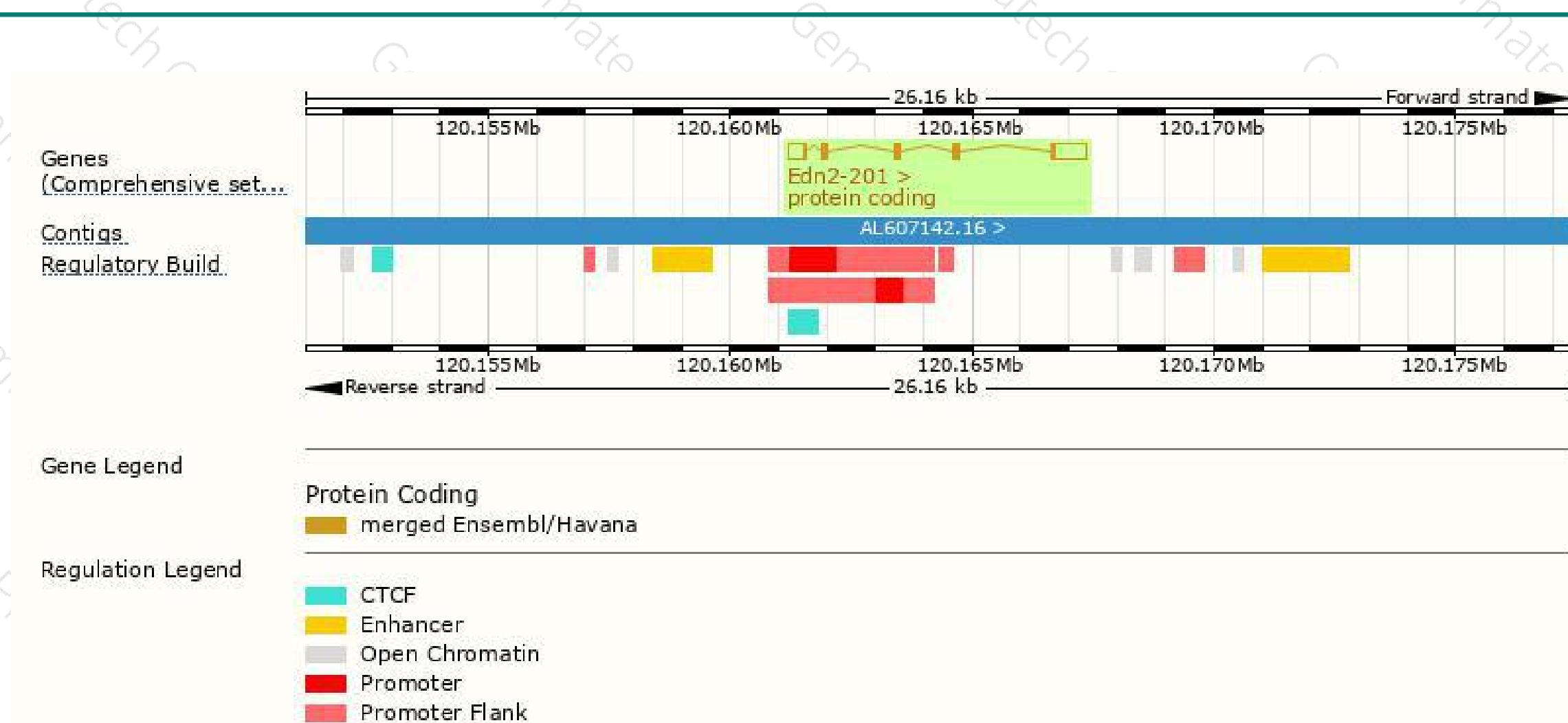
The gene has 1 transcript, and the transcript is shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Edn2-201	<a href="#">ENSMUST00000030384.4</a>	1462	<a href="#">175aa</a>	Protein coding	<a href="#">CCDS38864</a>	<a href="#">P22389</a>	TSL:1 GENCODE basic APPRIS P1

The strategy is based on the design of *Edn2-201* transcript. The transcription is shown below



# Genomic location distribution



# Protein domain

ENSMUSP000000030...

Low complexity (Seq)

Cleavage site (Sign....)

SMART

Prints

Pfam

PROSITE patterns

PANTHER

PTHR13874

Endothelin-2

All sequence SNPs/...

Sequence variants (dbSNP and all other sources)

## Variant Legend

- missense variant
- synonymous variant

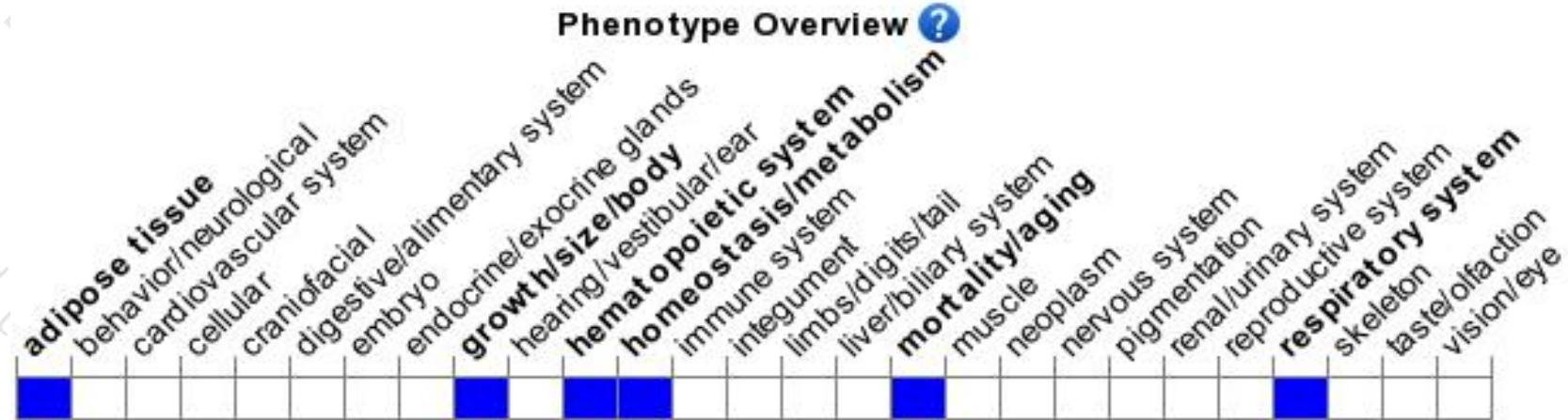
Scale bar

0 20 40 60 80 100 120 140 175



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# 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 a knock-out allele exhibit growth retardation, hypothermia, hypoxic hypoxia, hypercapnia, emphysema and premature death.



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

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