

# *Ndufs4* Cas9-KO Strategy

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# Project Overview

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**Project Name**

*Ndufs4*

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**Project type**

**Cas9-KO**

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**Strain background**

**C57BL/6JGpt**

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# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Ndufs4* gene. The schematic diagram is as follows:



- The *Ndufs4* gene has 6 transcripts. According to the structure of *Ndufs4* gene, exon2 of *Ndufs4-201* (ENSMUST00000022286.7) transcript is recommended as the knockout region. The region contains 79bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Ndufs4* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Mice homozygous for a null allele exhibit growth retardation, lethargy, loss of motor skills, blindness and decreased mitochondrial CI complex activity beginning at 5 weeks of age followed by death at week 7.
- Transcript *Ndufs4*-205 may not be affected.
- The *Ndufs4* gene is located on the Chr13. 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)

## Ndufs4 NADH:ubiquinone oxidoreductase core subunit S4 [Mus musculus (house mouse)]

Gene ID: 17993, updated on 19-Mar-2019

### Summary



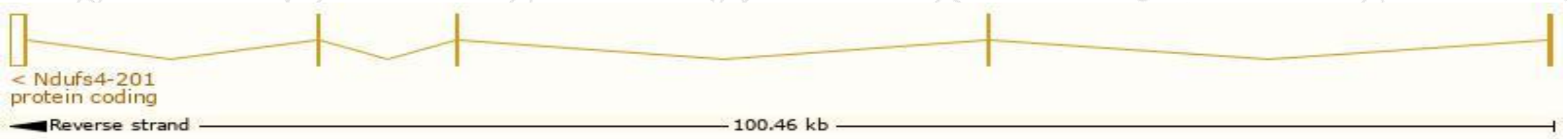
<b>Official Symbol</b>	Ndufs4 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	NADH:ubiquinone oxidoreductase core subunit S4 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:1343135</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG000000021764</a>
<b>Gene type</b>	protein coding
<b>RefSeq status</b>	VALIDATED
<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>	6720411N02Rik, C1-18k
<b>Expression</b>	Ubiquitous expression in heart adult (RPKM 45.1), CNS E18 (RPKM 31.6) and 28 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

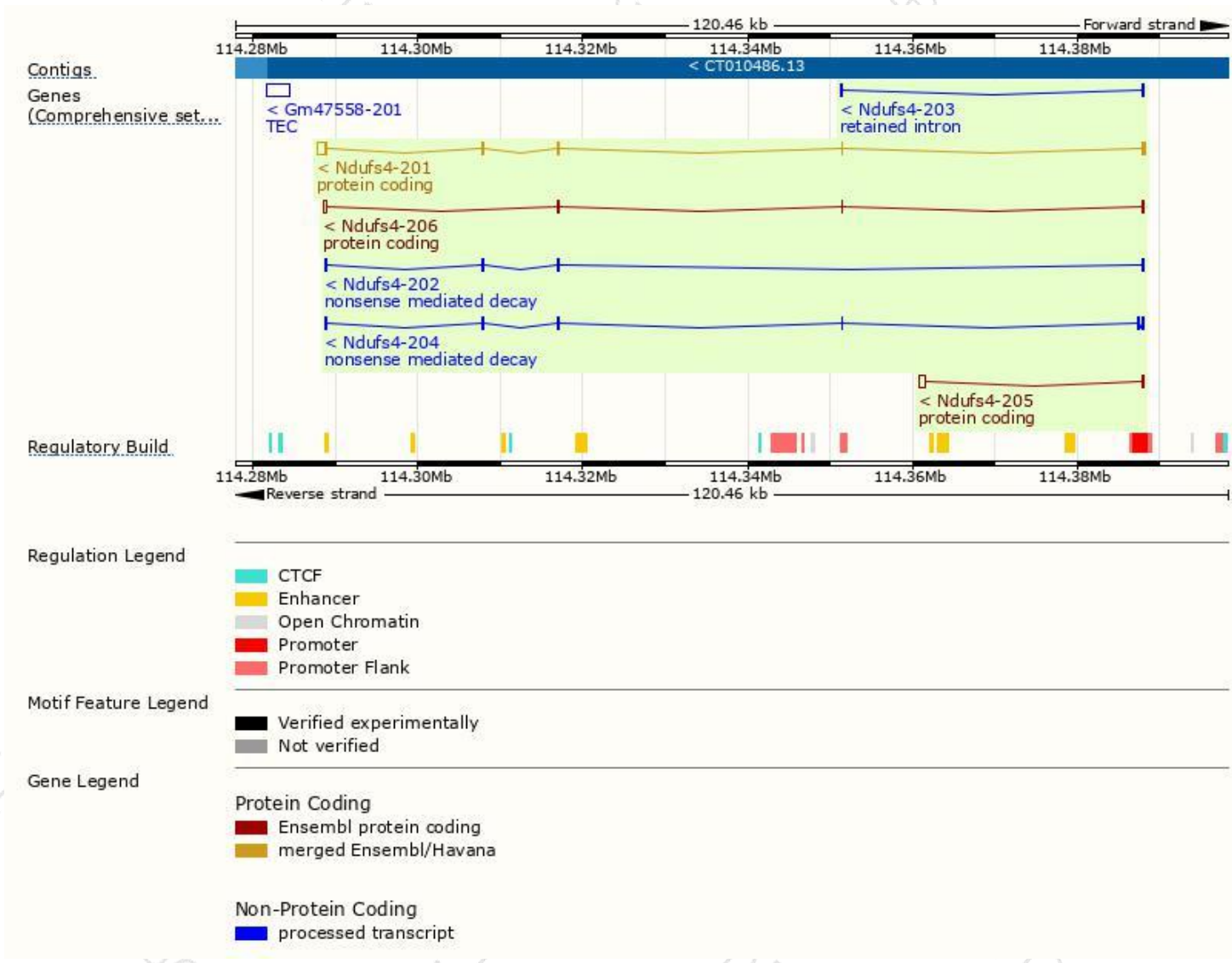
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Ndufs4-201	<a href="#">ENSMUST00000022286.7</a>	1698	<a href="#">175aa</a>	Protein coding	<a href="#">CCDS49368</a>	<a href="#">E9QPX3</a>	TSL:1 GENCODE basic APPRIS P1
Ndufs4-205	<a href="#">ENSMUST000000225978.2</a>	873	<a href="#">55aa</a>	Protein coding	-	<a href="#">A0A338P6Q8</a>	CDS 5' incomplete
Ndufs4-206	<a href="#">ENSMUST000000232101.1</a>	716	<a href="#">118aa</a>	Protein coding	-	<a href="#">A0A338P7A0</a>	CDS 5' incomplete
Ndufs4-204	<a href="#">ENSMUST000000225707.1</a>	686	<a href="#">35aa</a>	Nonsense mediated decay	-	<a href="#">A0A286YCM7</a>	CDS 5' incomplete
Ndufs4-202	<a href="#">ENSMUST000000225035.1</a>	571	<a href="#">37aa</a>	Nonsense mediated decay	-	<a href="#">A0A286YDA8</a>	
Ndufs4-203	<a href="#">ENSMUST000000225701.1</a>	383	No protein	Retained intron	-	-	

The strategy is based on the design of *Ndufs4-201* transcript,The transcription is shown below

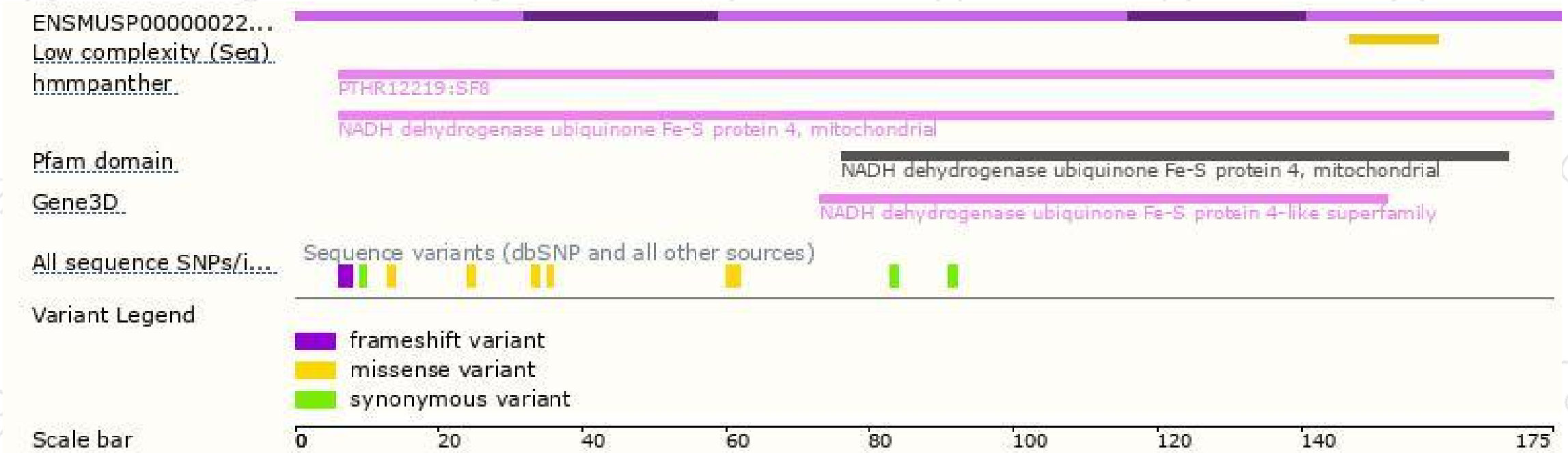


# Genomic location distribution

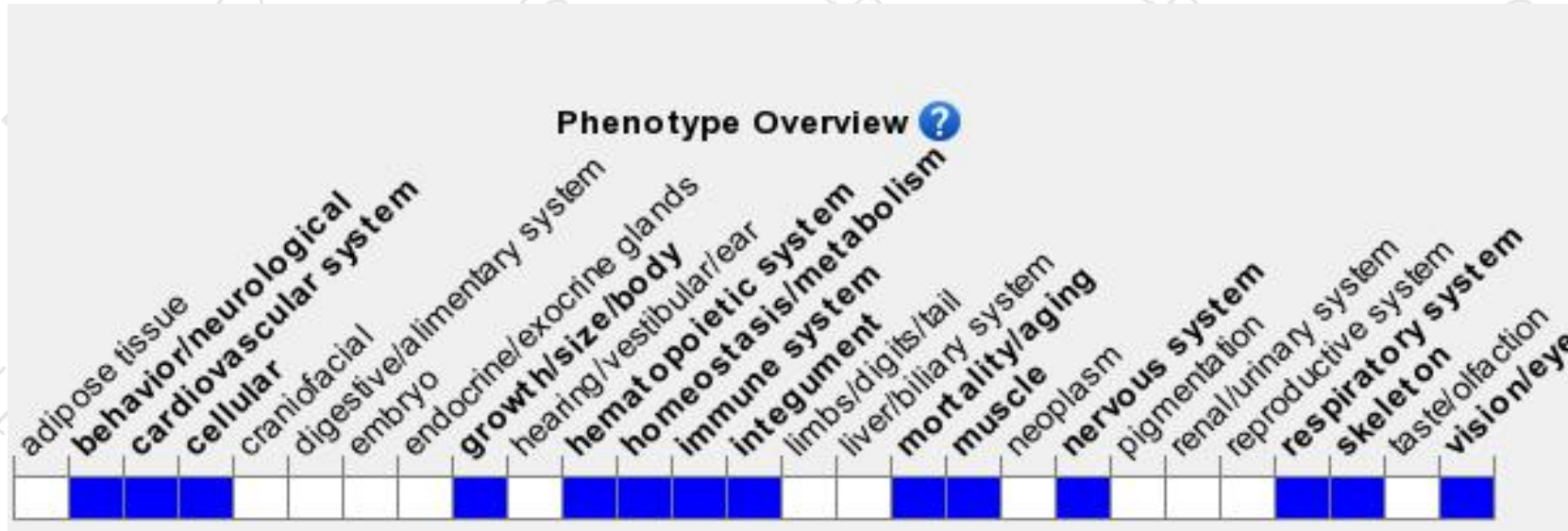




# Protein domain



# 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 a null allele exhibit growth retardation, lethargy, loss of motor skills, blindness and decreased mitochondrial CI complex activity beginning at 5 weeks of age followed by death at week 7.

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

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