

# *Mc3r* Cas9-KO Strategy

Designer: Yanhua Shen

Design Date: 2019-08-07

# Project Overview

**Project Name**

*Mc3r*

**Project type**

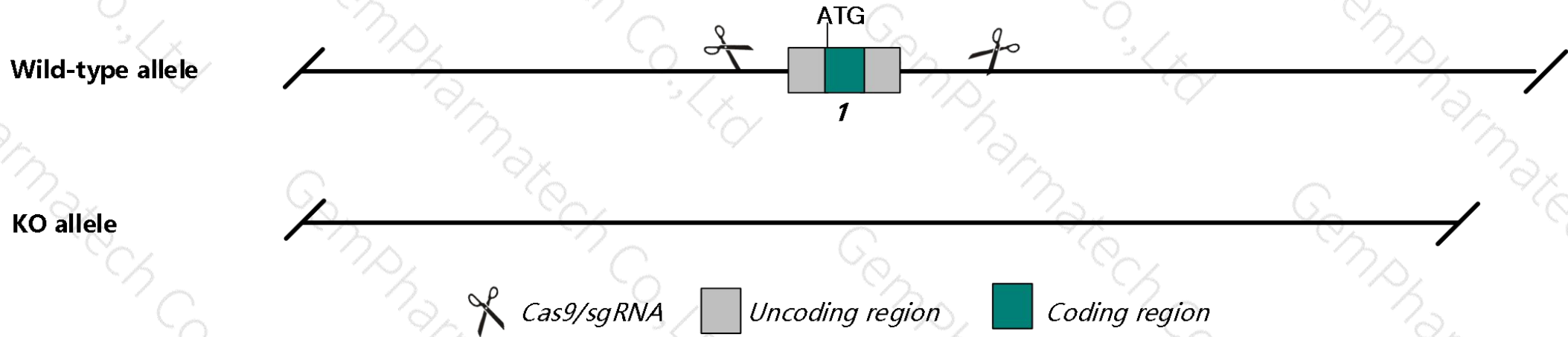
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Mc3r* gene. The schematic diagram is as follows:



- The *Mc3r* gene has 1 transcript. According to the structure of *Mc3r* gene, exon1 of *Mc3r-201* (ENSMUST00000038532.1) transcript is recommended as the knockout region. The region contains all of coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Mc3r* gene. The brief process is as follows: CRISPR/Cas9 system w

- According to the existing MGI data, Homozygotes for a null allele exhibit obesity, increased respiratory quotient on a high fat diet, and reduced energy expenditure. Homozygotes for another null allele show reduced lean mass, increased fat mass, diet-induced obesity, increased insulin and leptin levels, and reduced energy expenditure.
- The *Mc3r* gene is located on the Chr2. 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)

## Mc3r melanocortin 3 receptor [ *Mus musculus* (house mouse) ]

Gene ID: 17201, updated on 31-Jan-2019

### Summary

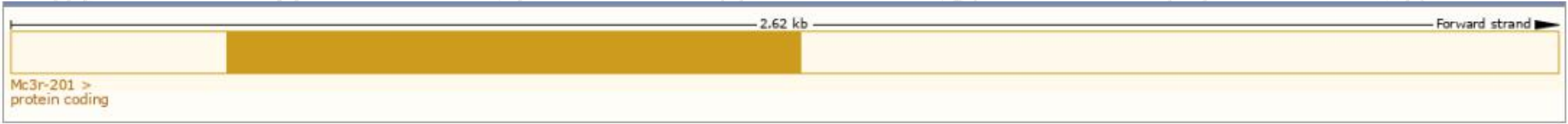
Official Symbol	Mc3r provided by <a href="#">MGI</a>
Official Full Name	melanocortin 3 receptor provided by <a href="#">MGI</a>
Primary source	<a href="#">MGI:MGI:96929</a>
See related	<a href="#">Ensembl:ENSMUSG00000038537</a>
Gene type	protein coding
RefSeq status	REVIEWED
Organism	<a href="#">Mus musculus</a>
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	MC3-R
Summary	This gene encodes a member of the melanocortin receptor family. Melanocortin receptors are transmembrane G-protein coupled receptors, which respond to small peptide hormones and exhibit diverse functions and tissue type localization. As part of the central nervous melanocortin system, the encoded protein is competitively bound by either melanocyte stimulating hormone or agouti-related protein to regulate energy homeostasis and adaptation to food restriction. Disruption of this gene results in an increased ratio of weight gain to food intake, increased fat mass, and decreased lean mass, without having a large effect on insulin sensitivity or glucose metabolism. [provided by RefSeq, Dec 2012]
Orthologs	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

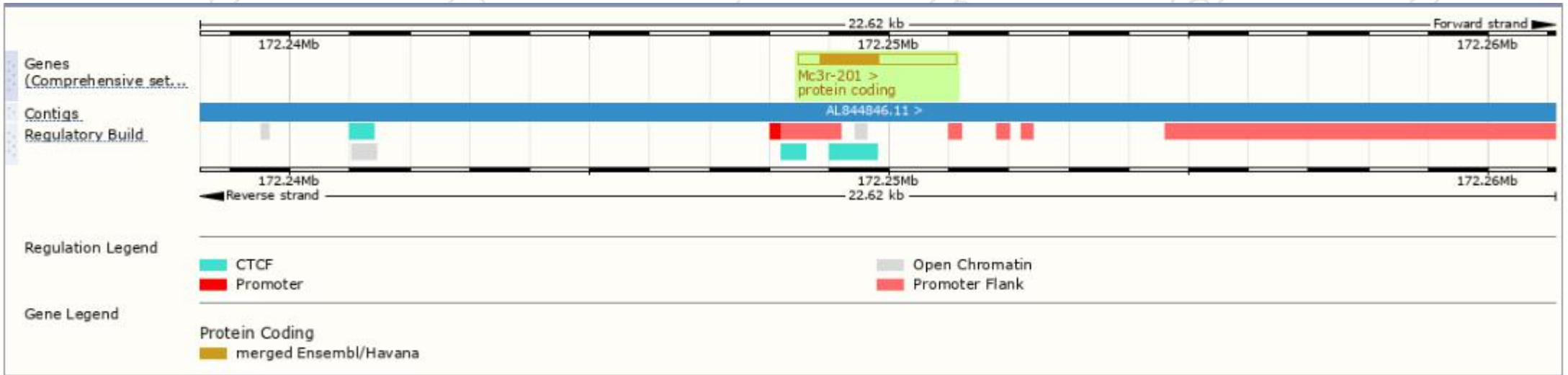
The gene has 1 transcript,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Mc3r-201	<a href="#">ENSMUST00000038532.1</a>	2623	<a href="#">323aa</a>	Protein coding	<a href="#">CCDS17127</a>	<a href="#">P33033</a> <a href="#">Q544G7</a>	TSL:NA GENCODE basic APPRIS P1

The strategy is based on the design of *Mc3r-201* transcript,The transcription is shown below

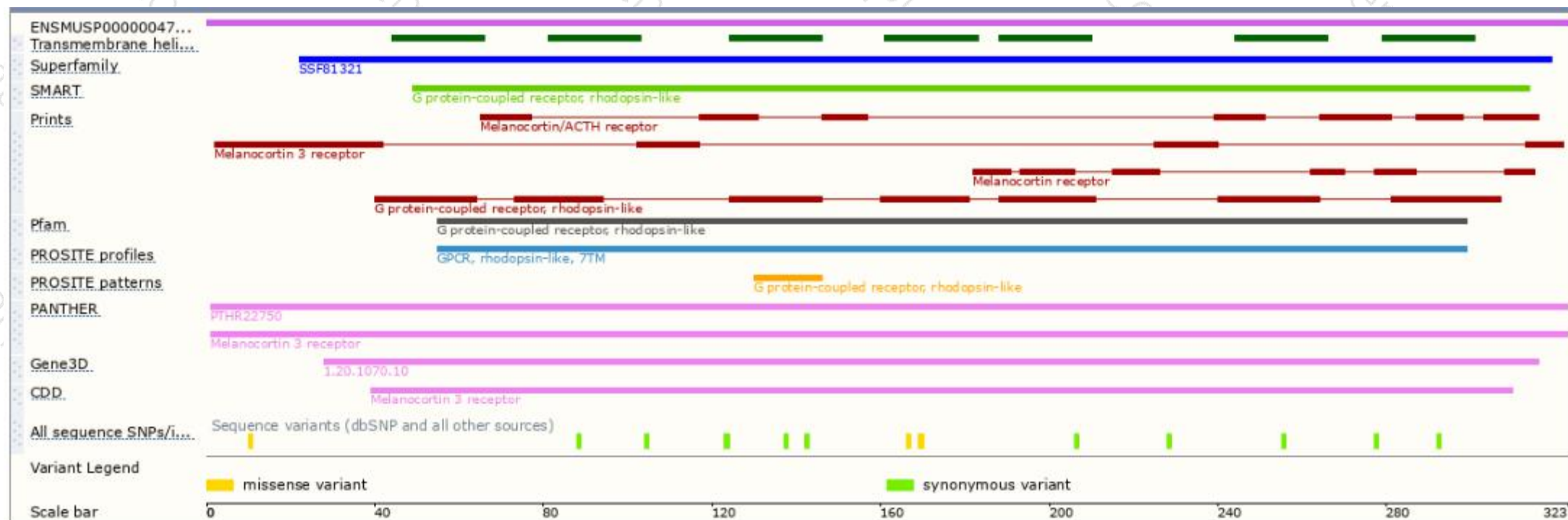


# Genomic location distribution

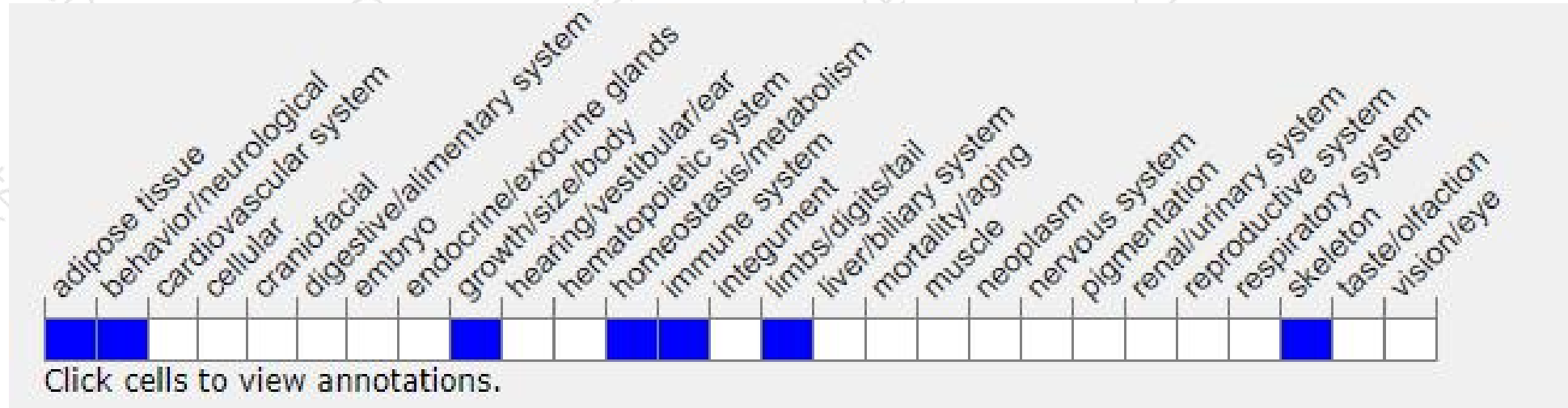




# Protein domain



# 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 exhibit obesity, increased respiratory quotient on a high fat diet, and reduced energy expenditure. Homozygotes for another null allele show reduced lean mass, increased fat mass, diet-induced obesity, increased insulin and leptin levels, and reduced energy expenditure.

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

Tel: 400-9660890

