

# *Sat1* Cas9-KO Strategy

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**Reviewer:**

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# Project Overview

**Project Name**

*Sat1*

**Project type**

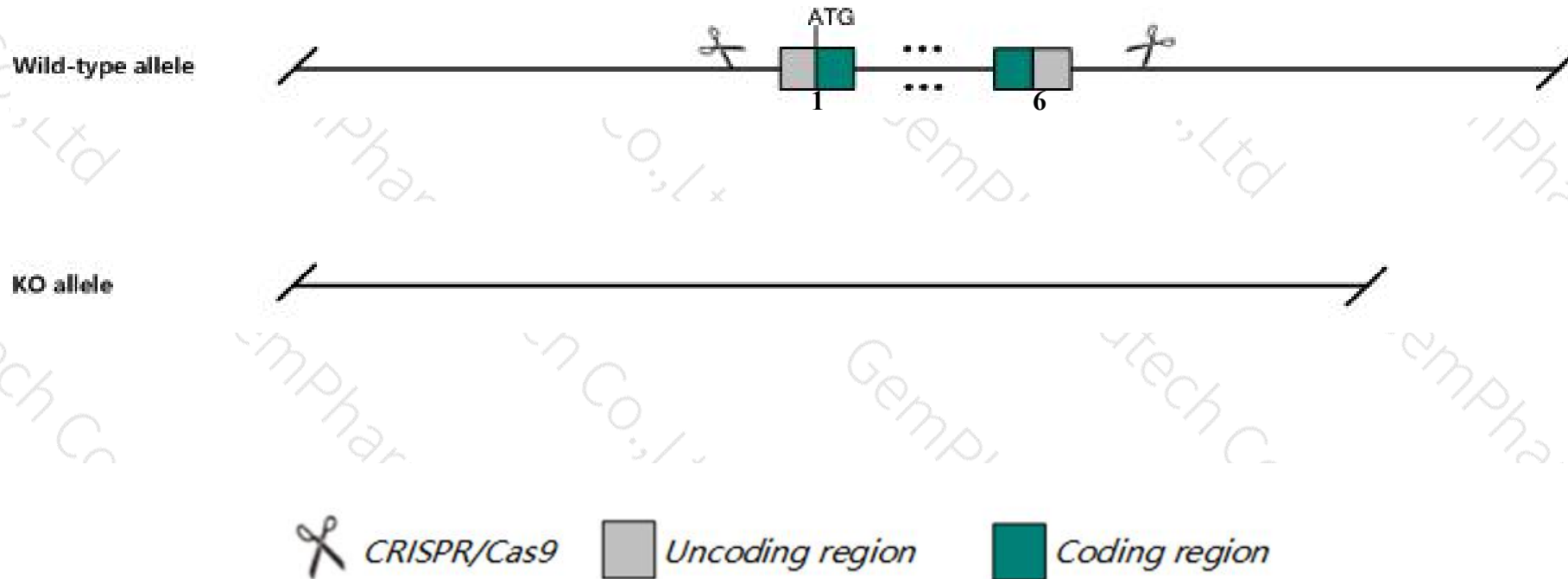
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Sat1* gene. The schematic diagram is as follows:



- The *Sat1* gene has 7 transcripts. According to the structure of *Sat1* gene, exon1-exon6 of *Sat1-201* (ENSMUST00000026318.14) transcript is recommended as the knockout region. The region contains all of the coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Sat1* gene. The brief process is as follows: CRISPR/Cas9 system w

- According to the existing MGI data, Mice homozygous for a knock-out allele exhibit reduced female fertility, increased percent body fat and total fat pad weight, abnormal liver and white adipose tissue physiology, abnormal aerobic energy metabolism, increased serum leptin levels, and increased weight gain on a high-fat diet.
- The *Sat1* gene is located on the ChrX. 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)

## Sat1 spermidine/spermine N1-acetyl transferase 1 [ *Mus musculus* (house mouse) ]

Gene ID: 20229, updated on 12-Aug-2019

### Summary

<b>Official Symbol</b>	Sat1 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	spermidine/spermine N1-acetyl transferase 1 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:98233</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000025283</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>	Sat; SSAT; AA617398
<b>Expression</b>	Broad expression in bladder adult (RPKM 89.1), placenta adult (RPKM 43.0) and 22 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

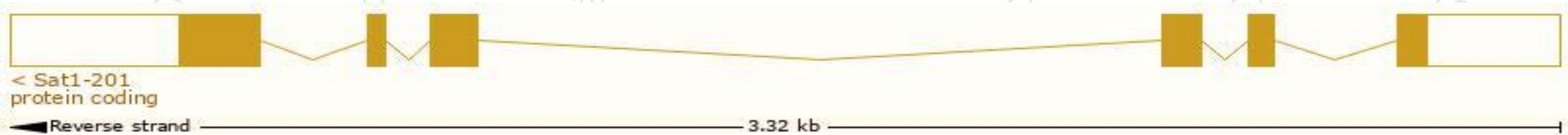


# Transcript information (Ensembl)

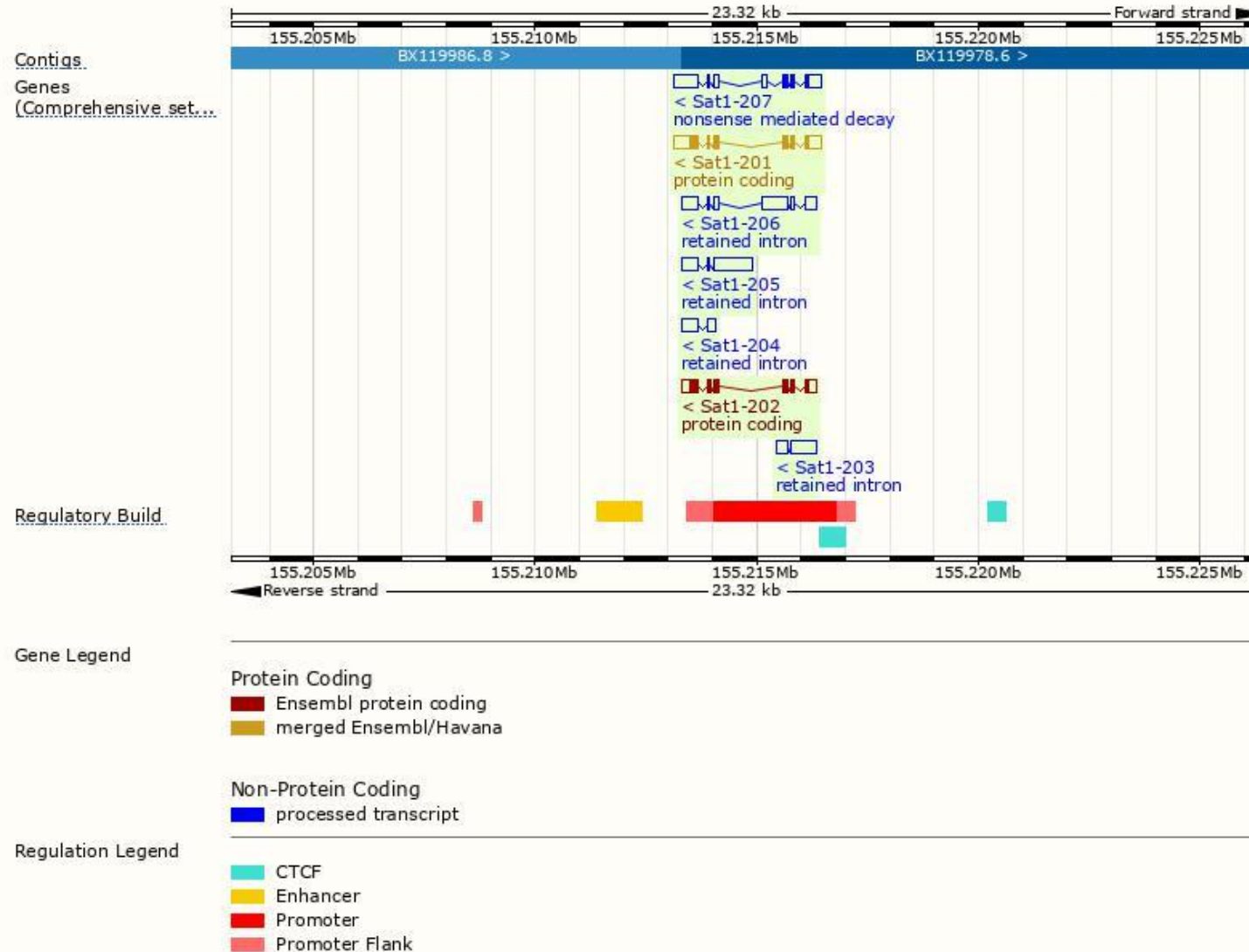
The gene has 7 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Translation ID	Biotype	CCDS	UniProt	Flags
Sat1-201	<a href="#">ENSMUST00000026318.14</a>	1162	<a href="#">171aa</a>	<a href="#">ENSMUSP00000026318.8</a>	Protein coding	<a href="#">CCDS30495</a>	<a href="#">P48026</a>	TSL:1 GENCODE basic APPRIS P1
Sat1-202	<a href="#">ENSMUST00000112551.3</a>	896	<a href="#">178aa</a>	<a href="#">ENSMUSP00000108170.3</a>	Protein coding	-	<a href="#">A2BES2</a>	TSL:2 GENCODE basic
Sat1-207	<a href="#">ENSMUST00000152463.7</a>	1272	<a href="#">71aa</a>	<a href="#">ENSMUSP00000138776.1</a>	Nonsense mediated decay	-	<a href="#">S4R2T2</a>	TSL:5
Sat1-206	<a href="#">ENSMUST00000150046.1</a>	1352	No protein	-	Retained intron	-	-	TSL:5
Sat1-205	<a href="#">ENSMUST00000138944.7</a>	1275	No protein	-	Retained intron	-	-	TSL:1
Sat1-203	<a href="#">ENSMUST00000123337.1</a>	773	No protein	-	Retained intron	-	-	TSL:3
Sat1-204	<a href="#">ENSMUST00000134855.7</a>	525	No protein	-	Retained intron	-	-	TSL:2

The strategy is based on the design of *Sat1-201* transcript,The transcription is shown below

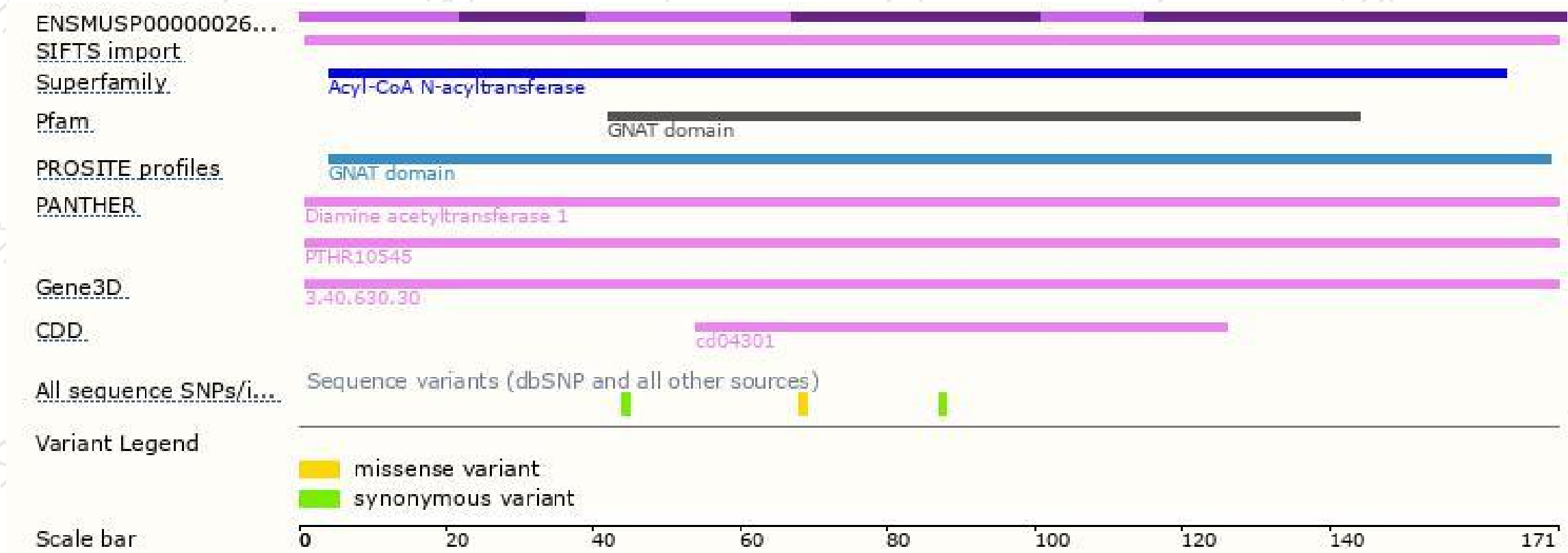


# Genomic location distribution



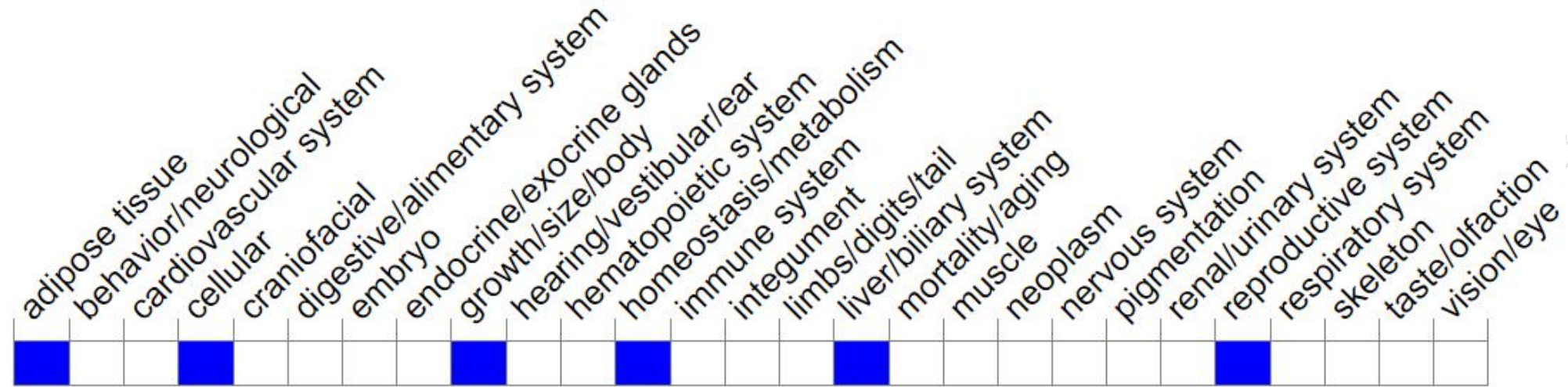


# Protein domain



# Mouse phenotype description(MGI)

## Phenotype Overview ?



*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 reduced female fertility, increased percent body fat and total fat pad weight, abnormal liver and white adipose tissue physiology, abnormal aerobic energy metabolism, increased serum leptin levels, and increased weight gain on a high-fat diet.

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

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