

# ***Fabp5*** Cas9-KO Strategy

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**Reviewer:**

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# Project Overview

**Project Name**

***Fabp5***

**Project type**

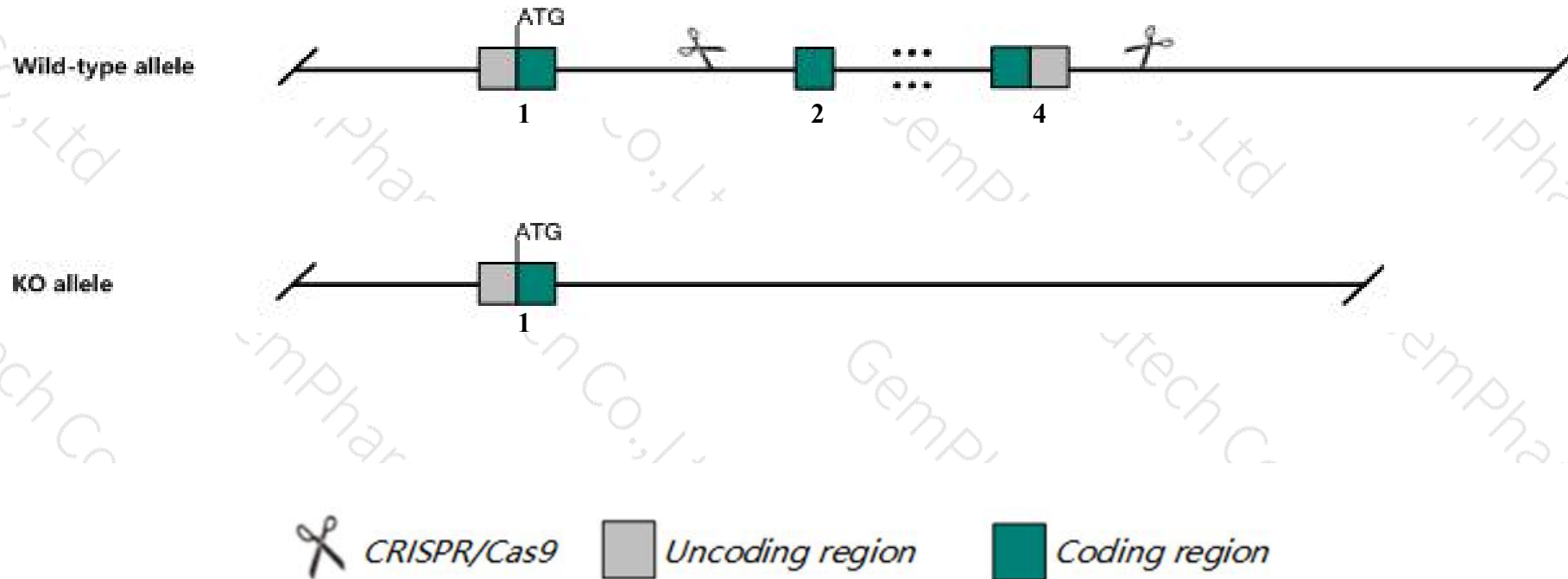
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Fabp5* gene. The schematic diagram is as follows:



- The *Fabp5* gene has 2 transcripts. According to the structure of *Fabp5* gene, exon2-exon4 of *Fabp5-201* (ENSMUST00000029046.8) transcript is recommended as the knockout region. The region contains 329bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Fabp5* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Mice homozygous for disruptions in this gene, depending on allele, display impaired skin barrier function or resistance to diet-induced obesity, showing decreased adipose tissue and improved glucose tolerance and insulin sensitivity.
- The *Fabp5* gene is located on the Chr3. 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)

## Fabp5 fatty acid binding protein 5, epidermal [Mus musculus (house mouse)]

Gene ID: 16592, updated on 31-Jan-2019

### Summary



<b>Official Symbol</b>	Fabp5 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	fatty acid binding protein 5, epidermal provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:101790</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000027533</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>	E-FABP, Fabpe, Klbp, PA-FABP, mal1
<b>Summary</b>	The protein encoded by this gene is part of the fatty acid binding protein family (FABP). FABPs are a family of small, highly conserved, cytoplasmic proteins that bind long-chain fatty acids and other hydrophobic ligands and participate in fatty acid uptake, transport, and metabolism. In humans this gene has been associated with psoriasis and type 2 diabetes. In mouse deficiency of this gene in combination with a deficiency in Fabp4 confers protection against atherosclerosis, diet-induced obesity, insulin resistance and experimental autoimmune encephalomyelitis (the mouse model for multiple sclerosis). Alternative splicing results in multiple transcript variants that encode different protein isoforms. The mouse genome contains many pseudogenes similar to this locus. [provided by RefSeq, Jan 2013]
<b>Expression</b>	Biased expression in liver E18 (RPKM 937.5), liver E14 (RPKM 371.8) and 7 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

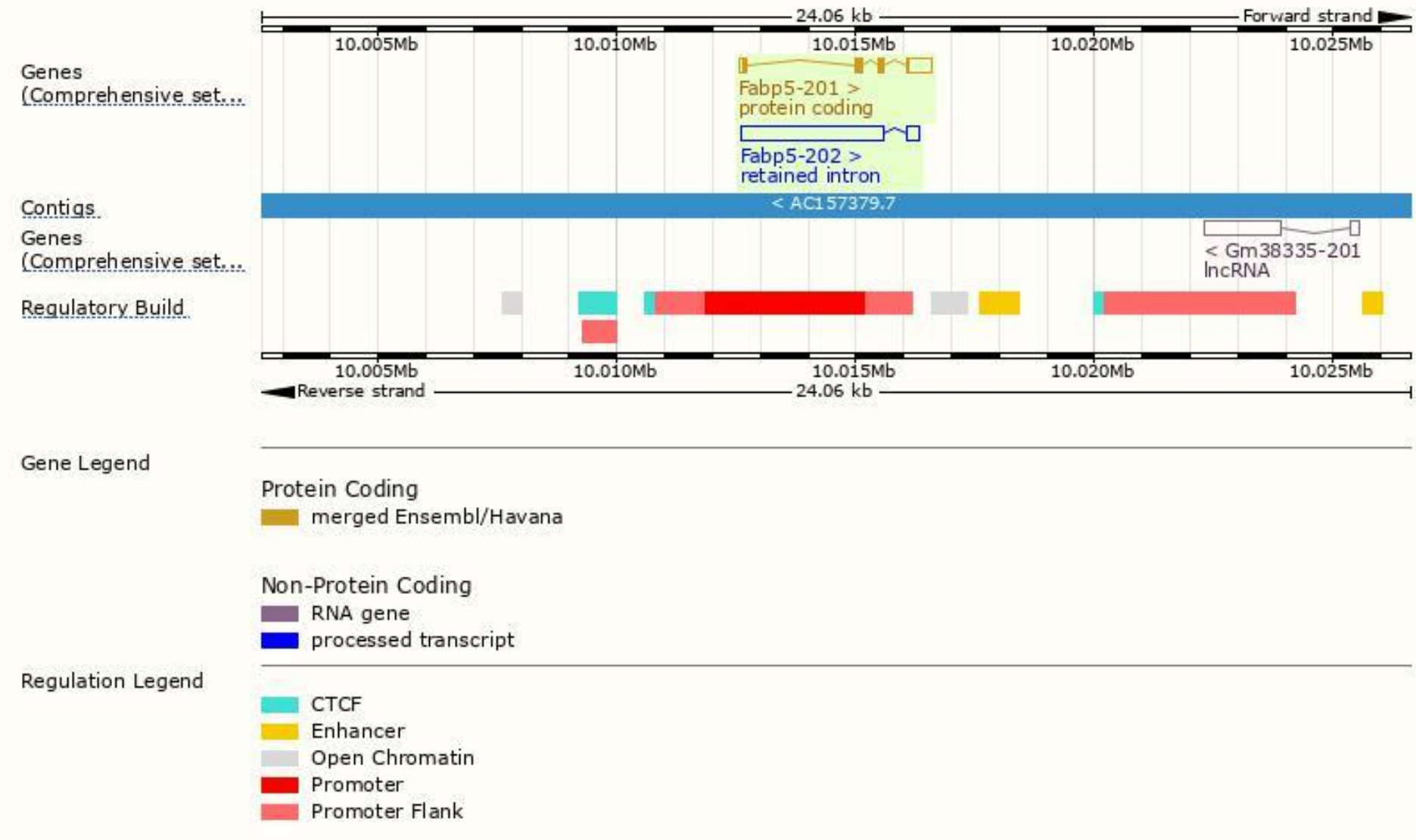
The gene has 2 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Fabp5-201	<a href="#">ENSMUST00000029046.8</a>	987	<a href="#">135aa</a>	Protein coding	<a href="#">CCDS38388</a>	<a href="#">Q05816</a> <a href="#">Q497I3</a>	TSL:1 GENCODE basic APPRIS P1
Fabp5-202	<a href="#">ENSMUST00000123744.1</a>	3238	No protein	Retained intron	-	-	TSL:2

The strategy is based on the design of *Fabp5-201* transcript,The transcription is shown below

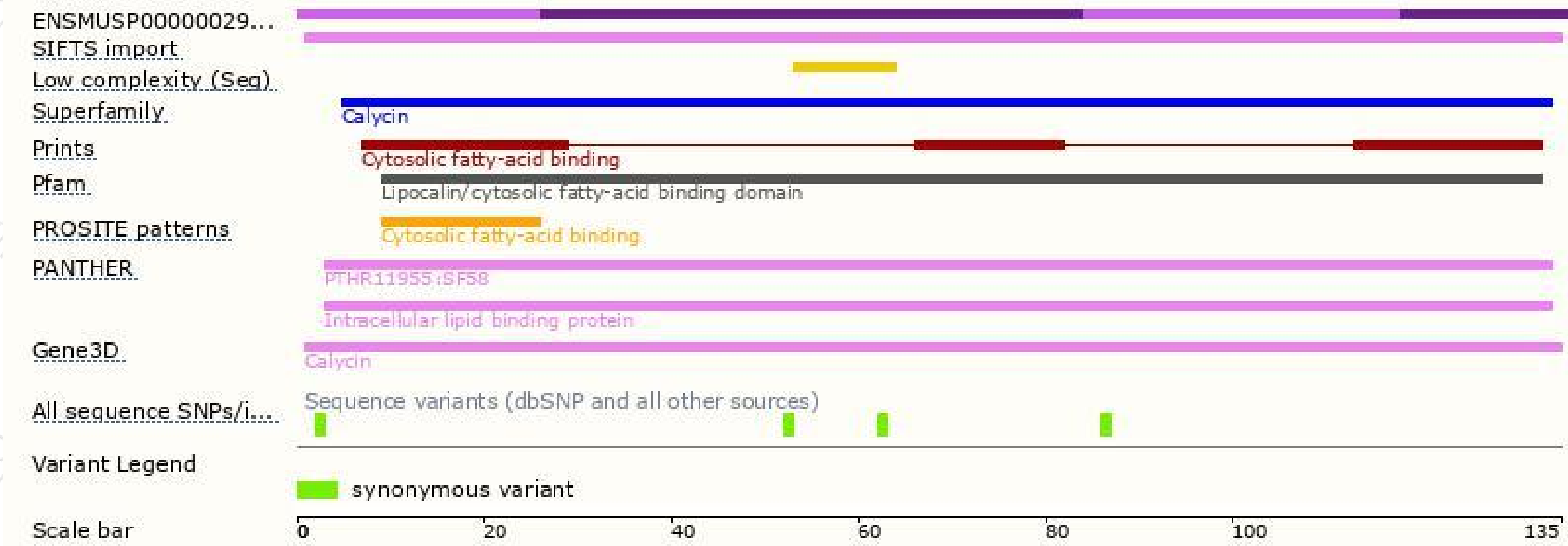


# Genomic location distribution

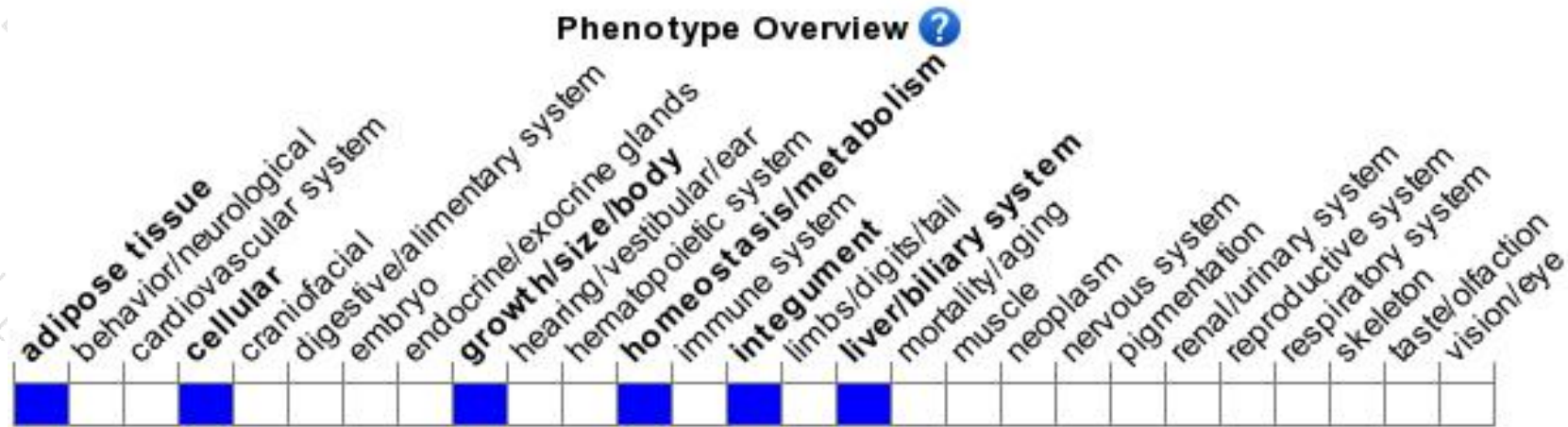




# Protein domain



# 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 disruptions in this gene, depending on allele, display impaired skin barrier function or resistance to diet-induced obesity, showing decreased adipose tissue and improved glucose tolerance and insulin sensitivity.

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

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