

***Trip10* Cas9-CKO Strategy**

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Reviewer:

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Design Date:

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Project Overview

Project Name

Trip10

Project type

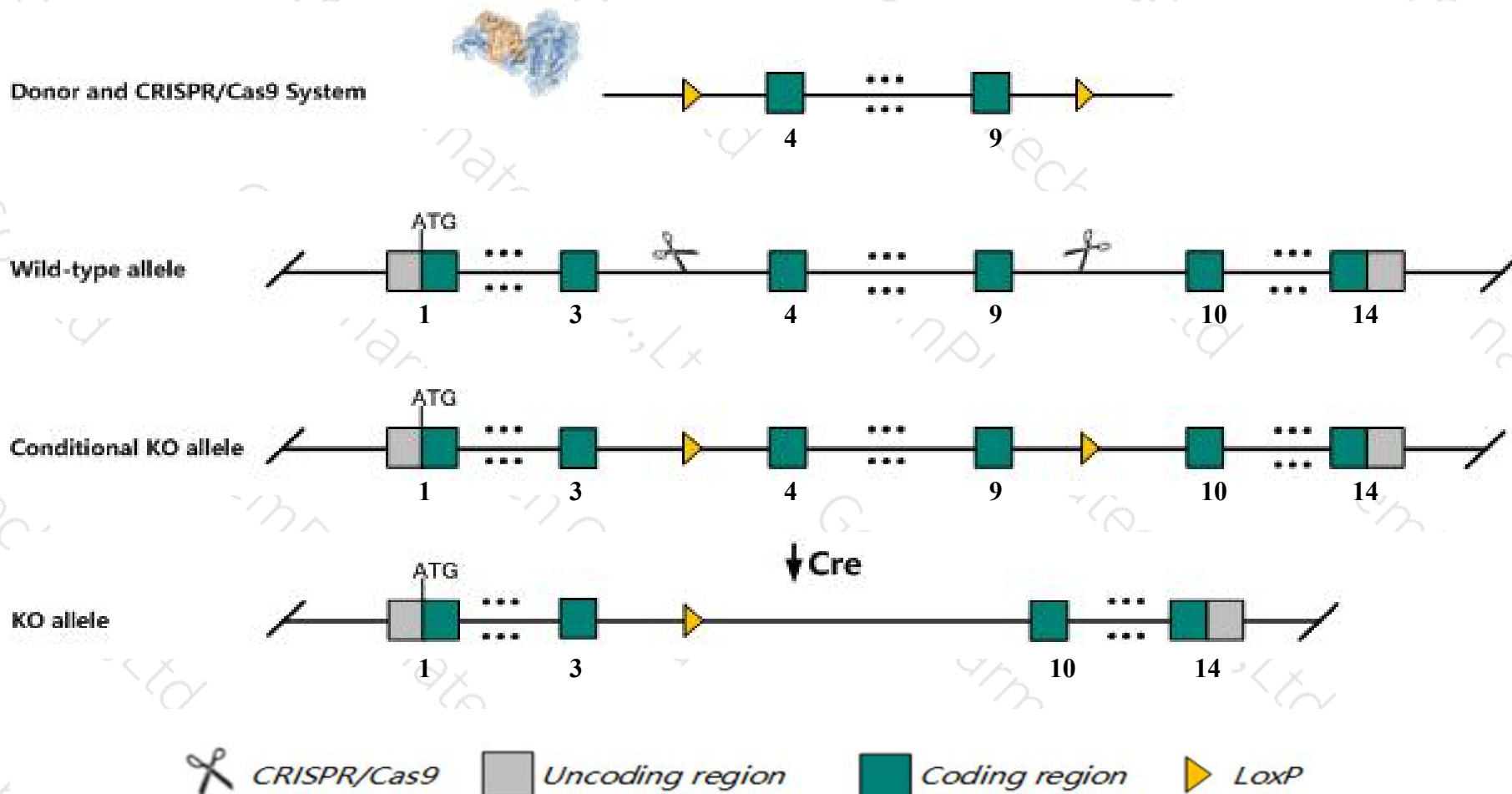
Cas9-CKO

Strain background

C57BL/6JGpt

Conditional Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Trip10* gene. The schematic diagram is as follows:



- The *Trip10* gene has 9 transcripts. According to the structure of *Trip10* gene, exon4-exon9 of *Trip10*-207 (ENSMUST00000224947.1) transcript is recommended as the knockout region. The region contains 787bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Trip10* gene. The brief process is as follows: CRISPR/Cas9 system and Donor were microinjected into the fertilized eggs of C57BL/6JGpt mice. Fertilized eggs were transplanted to obtain positive F0 mice which were confirmed by PCR and sequencing. A stable F1 generation mouse model was obtained by mating positive F0 generation mice with C57BL/6JGpt mice.
- The flox mice will be knocked out after mating with mice expressing Cre recombinase, resulting in the loss of function of the target gene in specific tissues and cell types.

- According to the existing MGI data, Mice homozygous for a knock-out allele exhibit increased insulin-stimulated glucose uptake in adipocytes and decreased circulating glucose levels. Mice homozygous for another knock-out allele exhibit impaired integrin-dependent T-cell trafficking.
- Some amino acids will remain at the N-terminus and some functions may be retained.
- The *Trip10* gene is located on the Chr17. 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 loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.

Gene information (NCBI)

Trip10 thyroid hormone receptor interactor 10 [Mus musculus (house mouse)]

Gene ID: 106628, updated on 31-Jan-2019

Summary



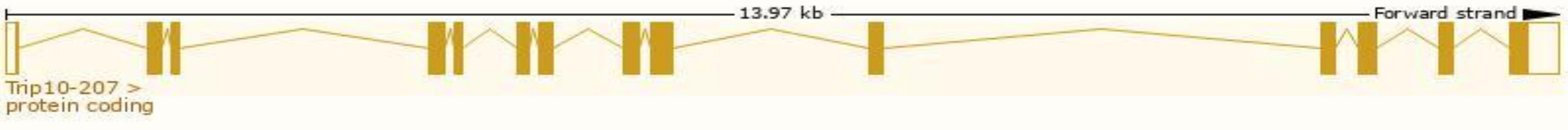
Official Symbol	Trip10 provided by MGI
Official Full Name	thyroid hormone receptor interactor 10 provided by MGI
Primary source	MGI:MGI:2146901
See related	Ensembl:ENSMUSG00000019487
Gene type	protein coding
RefSeq status	VALIDATED
Organism	Mus musculus
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	AI646975, Cip4
Expression	Ubiquitous expression in bladder adult (RPKM 19.3), heart adult (RPKM 17.6) and 28 other tissues See more
Orthologs	human all

Transcript information (Ensembl)

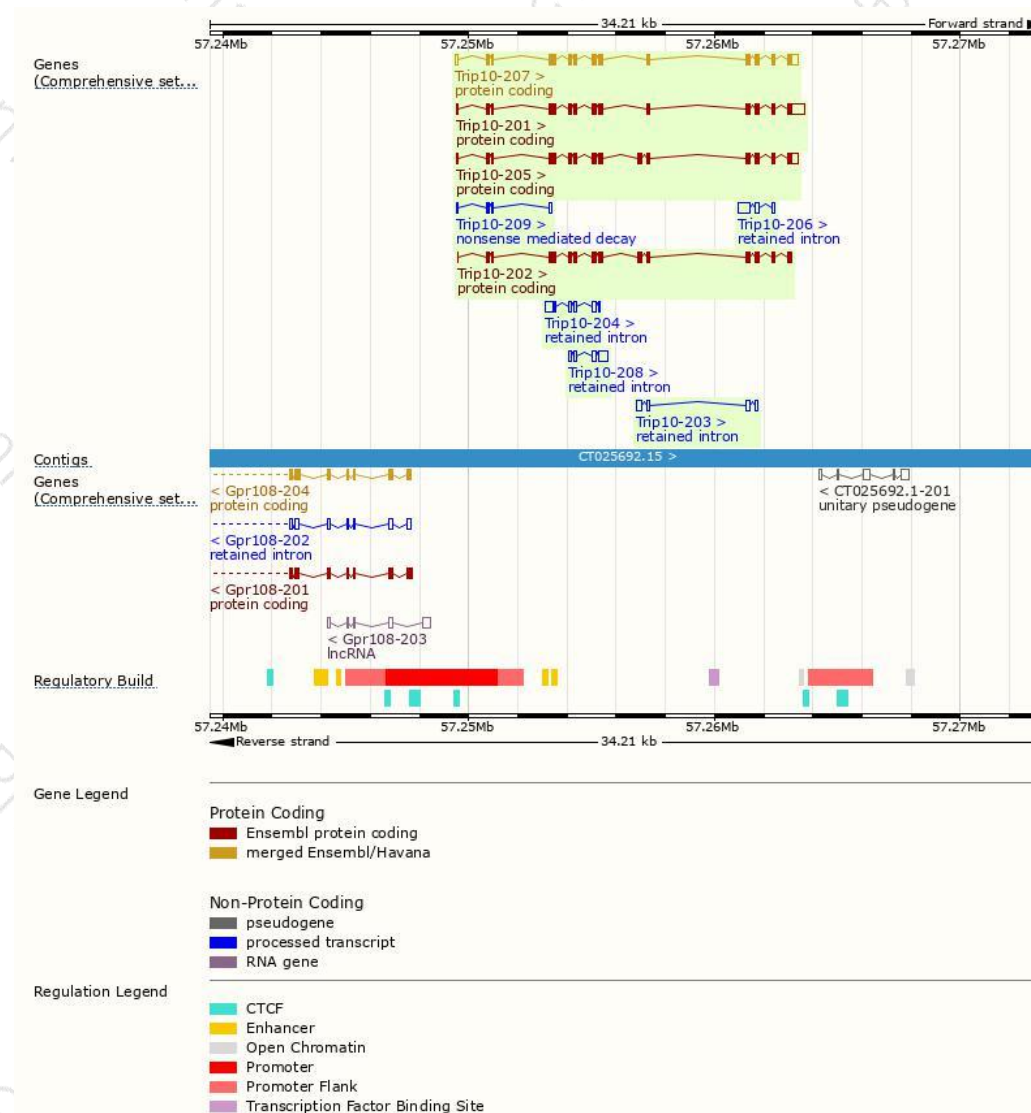
The gene has 9 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Trip10-207	ENSMUST00000224947.1	2016	547aa	Protein coding	CCDS28930	Q8CJ53	GENCODE basic APPRIS P2
Trip10-201	ENSMUST00000019631.10	2224	546aa	Protein coding	-	Q8CJ53	TSL:1 GENCODE basic APPRIS ALT2
Trip10-205	ENSMUST00000224885.1	2136	602aa	Protein coding	-	Q8CJ53	GENCODE basic APPRIS ALT2
Trip10-202	ENSMUST00000224152.1	1812	603aa	Protein coding	-	Q8CJ53	GENCODE basic APPRIS ALT2
Trip10-209	ENSMUST00000225664.1	422	41aa	Nonsense mediated decay	-	A0A286YEA1	
Trip10-204	ENSMUST00000224586.1	799	No protein	Retained intron	-	-	
Trip10-208	ENSMUST00000225523.1	768	No protein	Retained intron	-	-	
Trip10-206	ENSMUST00000224904.1	683	No protein	Retained intron	-	-	
Trip10-203	ENSMUST00000224261.1	572	No protein	Retained intron	-	-	

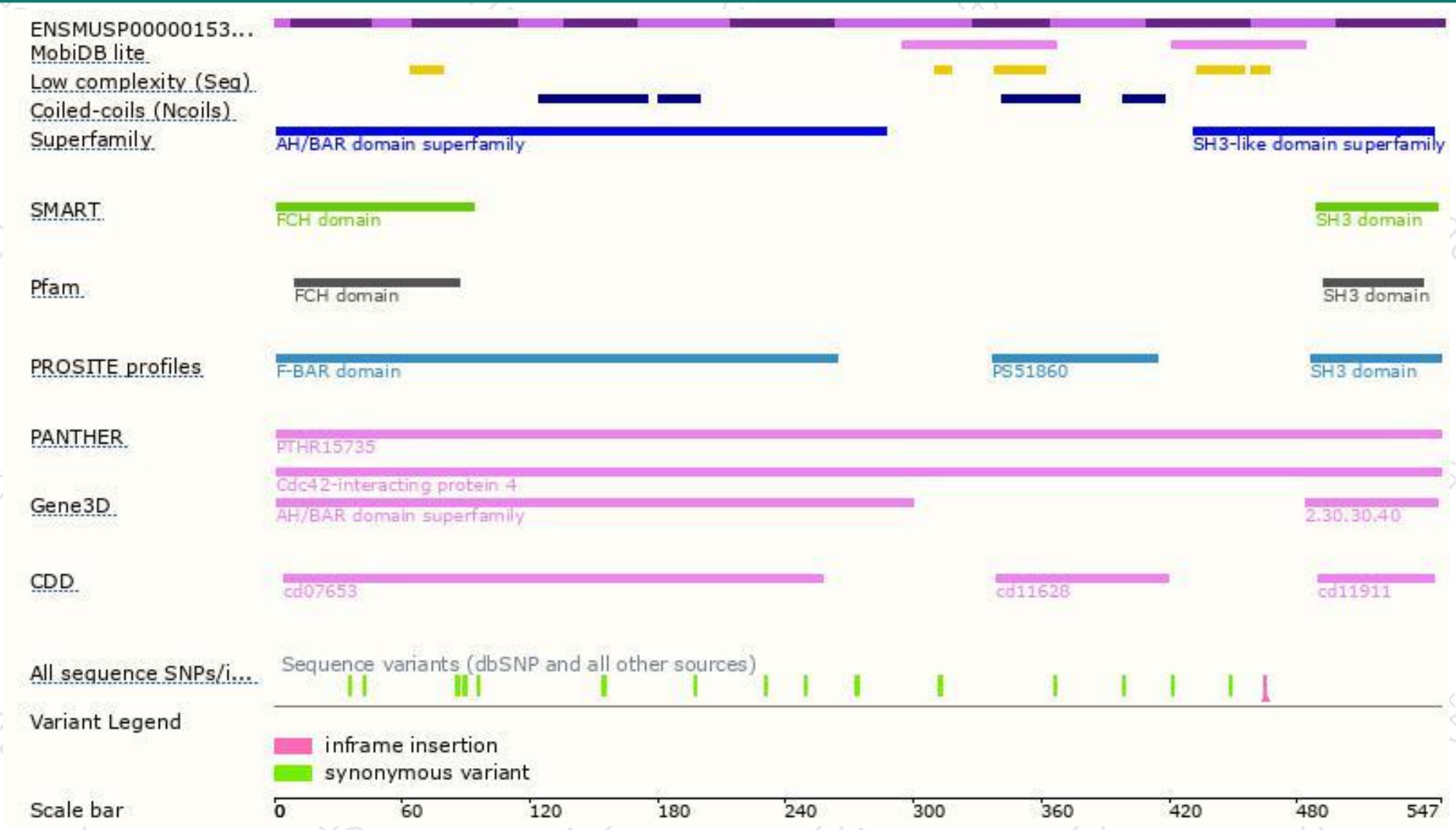
The strategy is based on the design of *Trip10-207* transcript,The transcription is shown below



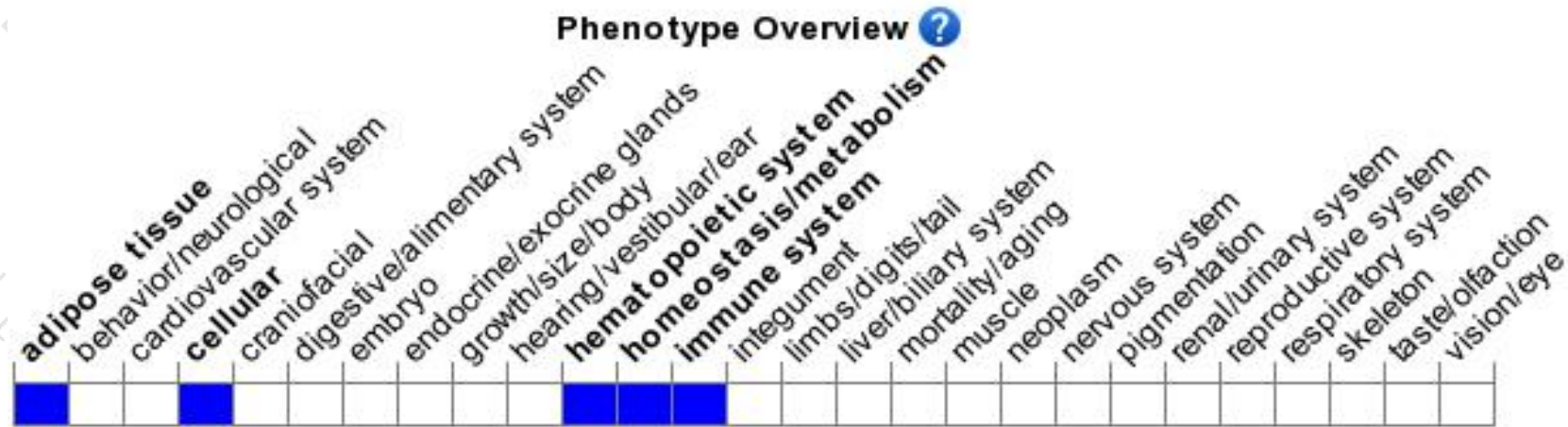
Genomic location distribution



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 knock-out allele exhibit increased insulin-stimulated glucose uptake in adipocytes and decreased circulating glucose levels. Mice homozygous for another knock-out allele exhibit impaired integrin-dependent T-cell trafficking.

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

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