

Dgat1 Cas9-KO Strategy

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Project Overview



Project Name

Dgat1

Project type

Cas9-KO

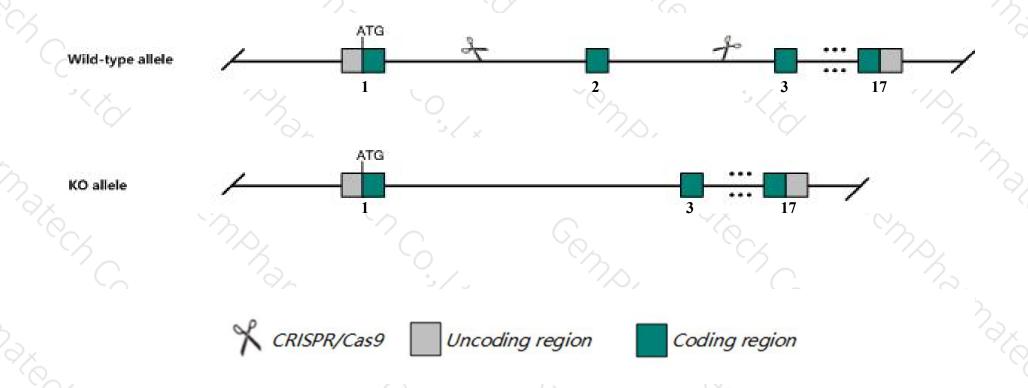
Strain background

C57BL/6JGpt

Knockout strategy



This model will use CRISPR/Cas9 technology to edit the *Dgat1* gene. The schematic diagram is as follows:



Technical routes



- ➤ The *Dgat1* gene has 12 transcripts. According to the structure of *Dgat1* gene, exon2 of *Dgat1-201*(ENSMUST00000023214.10) transcript is recommended as the knockout region. The region contains 88bp coding sequence.

 Knock out the region will result in disruption of protein function.
- > In this project we use CRISPR/Cas9 technology to modify *Dgat1* gene. The brief process is as follows: CRISPR/Cas9 system

Notice



- According to the existing MGI data, Homozygous inactivation of this gene leads to decreased percent body fat, resistance to diet-induced obesity, altered energy, glucose and triglyceride metabolism, alopecia, hair cycle and skin defects, and a lactation failure associated with impaired mammary gland growth during pregnancy.
- > The *Dgat1* gene is located on the Chr15. 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)



Dgat1 diacylglycerol O-acyltransferase 1 [Mus musculus (house mouse)]

Gene ID: 13350, updated on 31-Jan-2019

Summary

☆ ?

Official Symbol Dgat1 provided by MGI

Official Full Name diacylglycerol O-acyltransferase 1 provided by MGI

Primary source MGI:MGI:1333825

See related Ensembl:ENSMUSG00000022555

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 ARAT, C75990, D15Ertd23e, Dgat

Expression Biased expression in duodenum adult (RPKM 359.0), small intestine adult (RPKM 242.2) and 9 other tissuesSee more

Orthologs <u>human</u> all

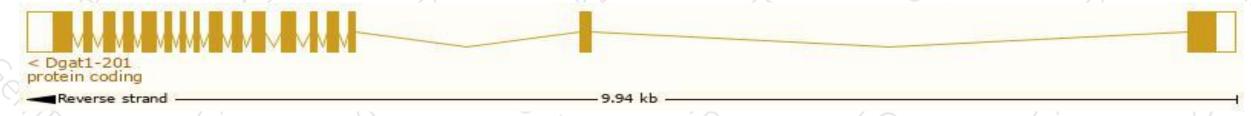
Transcript information (Ensembl)



The gene has 12 transcripts, all transcripts are shown below:

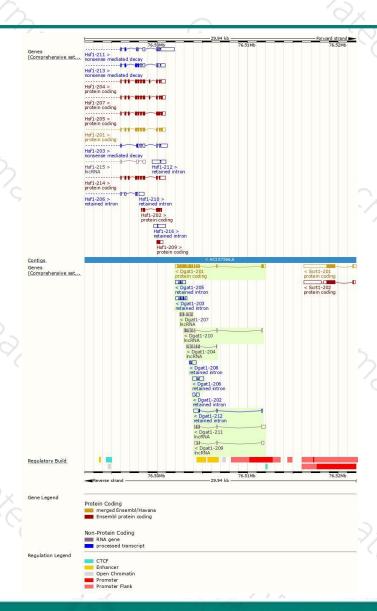
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Dgat1-201	ENSMUST00000023214.10	1888	498aa	Protein coding	CCDS27573	Q54AA6 Q9Z2A7	TSL:1 GENCODE basic APPRIS P1
Dgat1-206	ENSMUST00000161499.1	1116	No protein	Retained intron	-8	680	TSL:3
Dgat1-205	ENSMUST00000160294.7	885	No protein	Retained intron	2	(4)	TSL:1
Dgat1-212	ENSMUST00000231035.1	780	No protein	Retained intron	24	1020	
Dgat1-203	ENSMUST00000159908.1	742	No protein	Retained intron	-	150	TSL:5
Dgat1-202	ENSMUST00000159776.1	584	No protein	Retained intron		680	TSL:3
Ogat1-208	ENSMUST00000162813.1	564	No protein	Retained intron	2	(4)	TSL:2
Dgat1-210	ENSMUST00000230722.1	803	No protein	IncRNA	24	1000	
Dgat1-211	ENSMUST00000230894.1	788	No protein	IncRNA	5	150	
Dgat1-209	ENSMUST00000229569.1	659	No protein	IncRNA		680	
Dgat1-204	ENSMUST00000160293.1	602	No protein	IncRNA	2	(4)	TSL:5
Dgat1-207	ENSMUST00000162354.7	569	No protein	IncRNA	24	320	TSL:3
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The strategy is based on the design of *Dgat1-201* transcript, The transcription is shown below



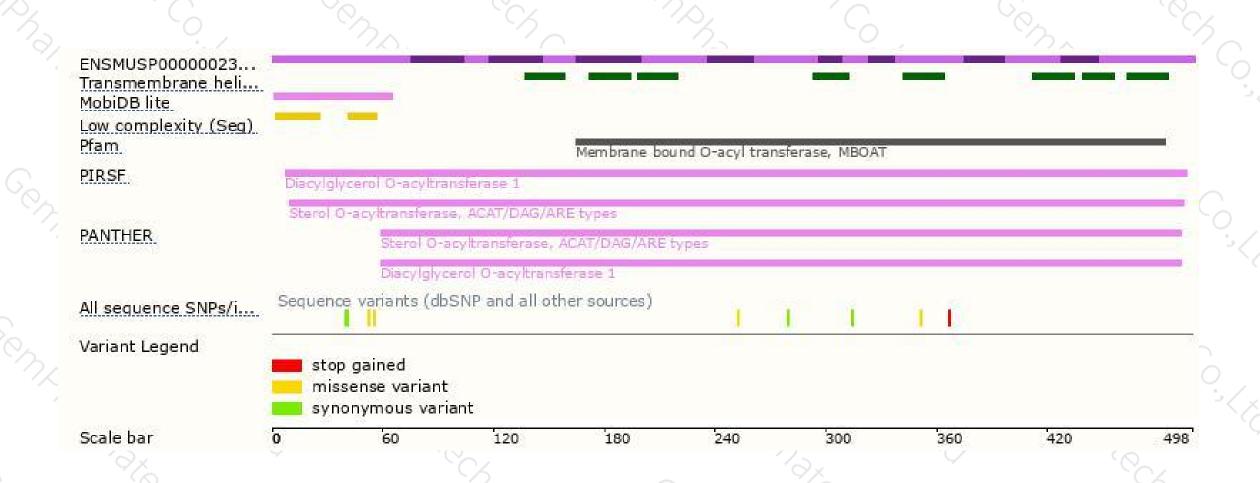
Genomic location distribution





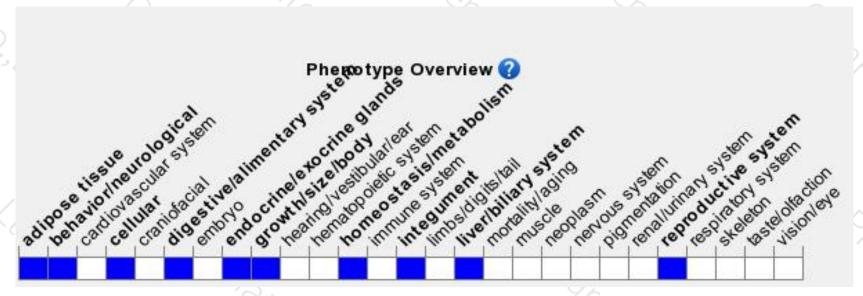
Protein domain





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, Homozygous inactivation of this gene leads to decreased percent body fat, resistance to diet-induced obesity, altered energy, glucose and triglyceride metabolism, alopecia, hair cycle and skin defects, and a lactation failure associated with impaired mammary gland growth during pregnancy.



If you have any questions, you are welcome to inquire. Tel: 400-9660890





