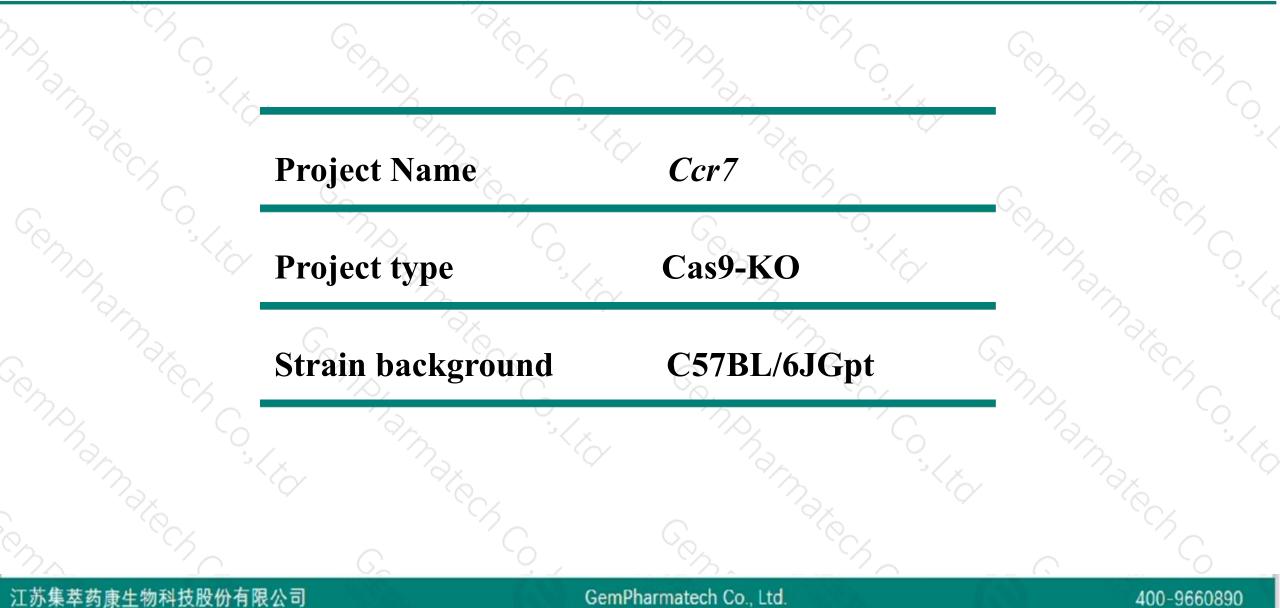


Ccr7 Cas9-KO Strategy

Designer: Reviewer: Design Date: Min Guan Yang Zeng 2018-6-28

Project Overview

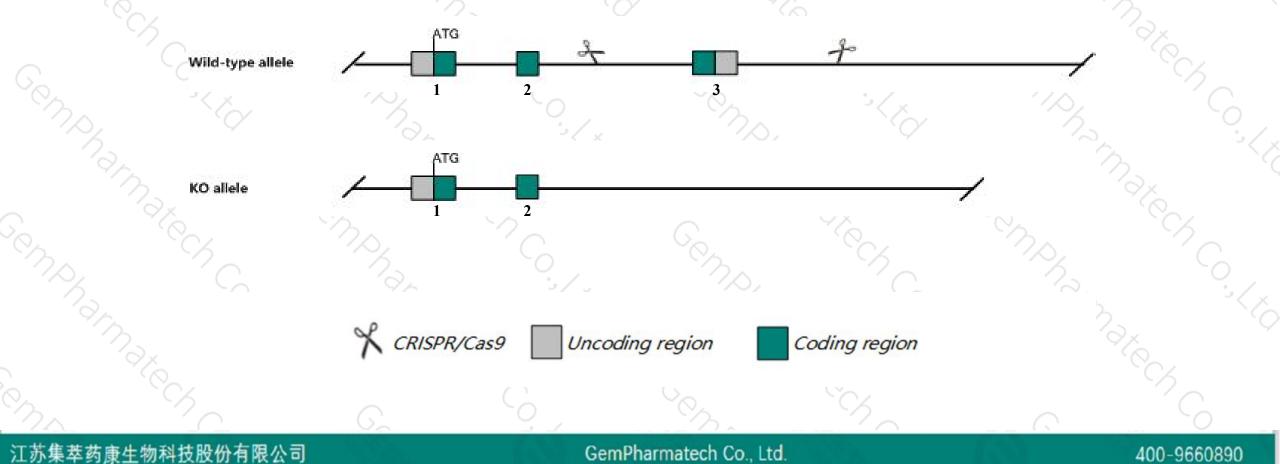




Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Ccr7 gene. The schematic diagram is as follows:





- The Ccr7 gene has 1 transcript. According to the structure of Ccr7 gene, exon3 of Ccr7-201 (ENSMUST00000103134.3) transcript is recommended as the knockout region. The region contains most 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 Ccr7 gene. The brief process is as follows: CRISPR/Cas9 system v



- According to the existing MGI data, Homozygous mice exhibit an impaired primary immune response. Dendritic cells, B, T and T regulatory cells show impaired migration to the lymph nodes and secondary lymph organs exhibit morphological abnormalities. Lymphocytic infiltrates to the pancreas, lung and stomach are observed in aged mice.
- The Ccr7 gene is located on the Chr11. 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)



六 ?

Ccr7 chemokine (C-C motif) receptor 7 [Mus musculus (house mouse)]

Gene ID: 12775, updated on 12-Aug-2019

Summary

Official Symbol	Ccr7 provided by MGI							
Official Full Name	chemokine (C-C motif) receptor 7 provided by MGI							
Primary source	<u>MGI:MGI:103011</u>							
See related	Ensembl:ENSMUSG00000037944							
Gene type	protein coding							
RefSeq status	VALIDATED							
Organism	Mus musculus							
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia;							
	Myomorpha; Muroidea; Murinae; Mus; Mus							
Also known as	EBI1; CCR-7; CD197; Ebi1h; Cdw197; Cmkbr7; CC-CKR-7							
Expression	Biased expression in spleen adult (RPKM 74.8), thymus adult (RPKM 70.1) and 2 other tissues See more							
Orthologs	human all							
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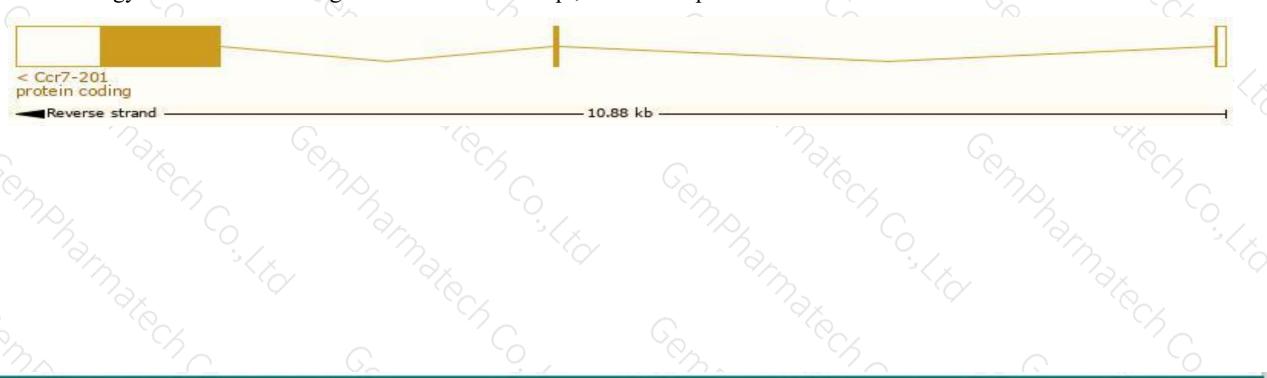
Transcript information (Ensembl)



The gene has 1 transcript, and the transcript is shown below:

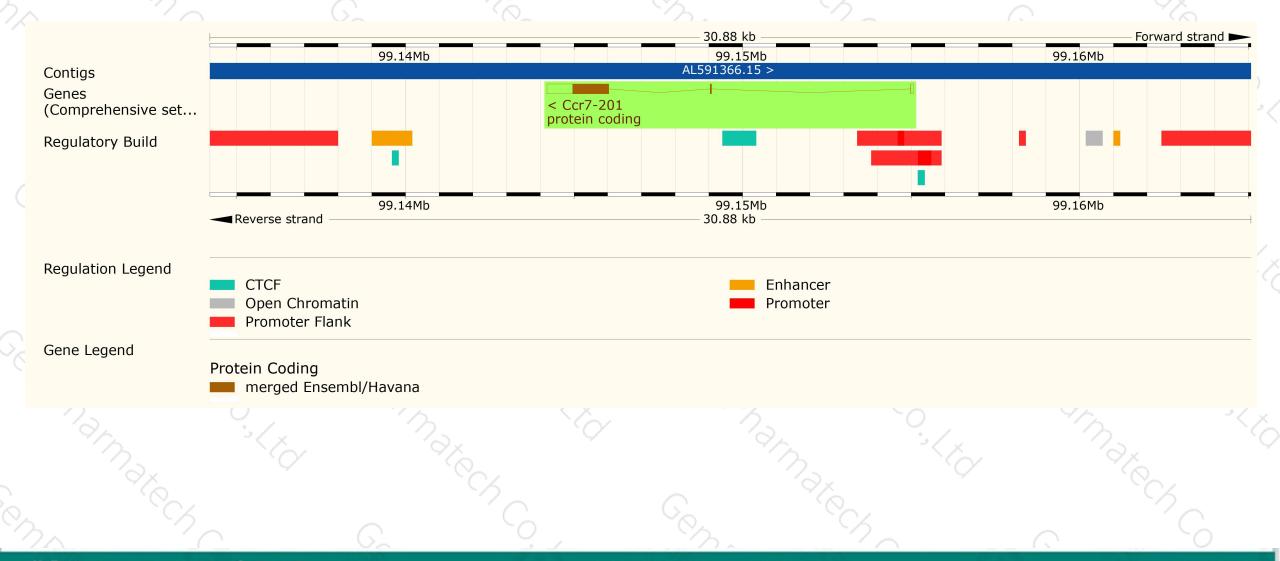
Name	Transcript ID	nscript ID bp Pr		Biotype	CCDS	UniProt	Flags		
Ccr7-201	ENSMUST00000103134.3	1978	<u>378aa</u>	Protein coding	CCDS25373	<u>P47774</u>	TSL:1 GENCODE basic APPRIS P1	Ŀ	

The strategy is based on the design of *Ccr7-201* transcript, The transcription is shown below



Genomic location distribution





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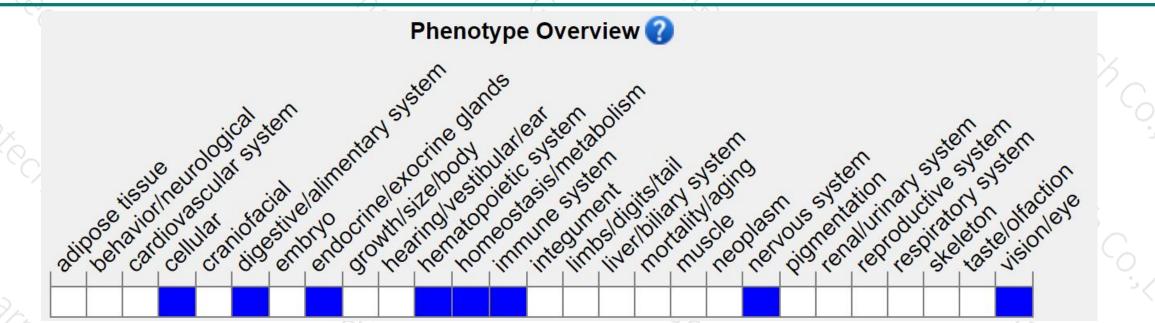
Protein domain



~°C4			(Ox	Чю.	14	\sim				12	
ENSMUSP00000099 Transmembrane heli					L						
Low complexity (Seg) Cleavage site (Sign											
Superfamily	SSF81321										
Prints		Chemokine receptor family	/							-	1
		G protein-coupled re	ceptor, rhodopsin-like		-						
	CC chemokine receptor				-				·		
Pfam			upled receptor, rhodops	in-like							
PROSITE profiles		GPCR, rhodo	psin-like, 7TM								
PROSITE patterns PANTHER			G	protein-coupled recepto	or, rhodopsin-like						
PANTIER	PTHR10489										
Gene3D	PTHR10489:SF63										
	Sequence variants (db	1.20.1070.10 SNP and all other sour									
All sequence SNPs/i					11	11	10	1.1	1	1	- E
Variant Legend	missense variant				synonymou	s variant					
Scale bar	0 40	80	120	160	200	240	280		320		378
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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 mice exhibit an impaired primary immune response. Dendritic cells, B, T and T regulatory cells show impaired migration to the lymph nodes and secondary lymph organs exhibit morphological abnormalities. Lymphocytic infiltrates to the pancreas, lung and stomach are observed in aged mice.

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



