

# ***Lgals3 Cas9-KO Strategy***

**Designer:**

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**Design Date:**

**2019-7-17**

# Project Overview

**Project Name**

***Lgals3***

**Project type**

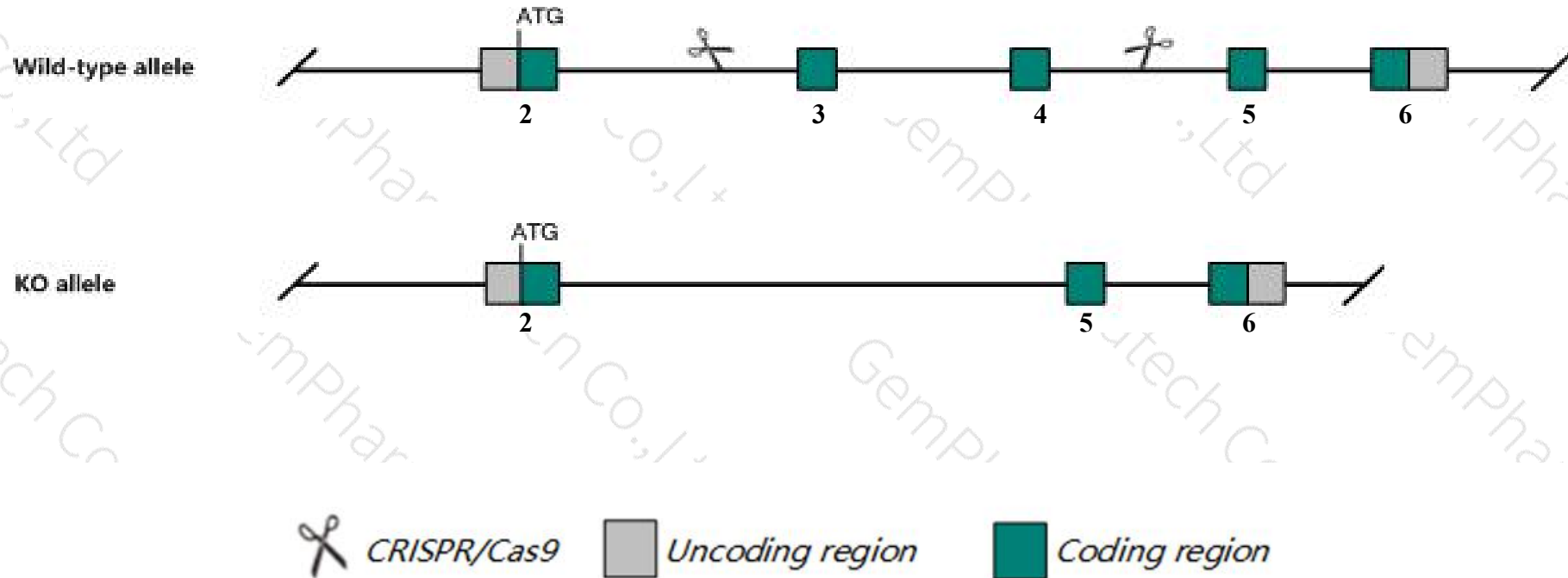
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Lgals3* gene. The schematic diagram is as follows:



- The *Lgals3* gene has 6 transcripts. According to the structure of *Lgals3* gene, exon3-exon4 of *Lgals3*-204 (ENSMUST00000150290.8) transcript is recommended as the knockout region. The region contains 455bp coding sequence. Knock out the region will result in disruption of protein function.
- In this project we use CRISPR/Cas9 technology to modify *Lgals3* gene. The brief process is as follows: CRISPR/Cas9 system

- According to the existing MGI data, Homozygotes for a null allele show susceptibility to *S. pneumoniae* infection, resistance to renal fibrosis, defects in chondrocyte differentiation, and impaired macrophage activation. Homozygotes for another null allele show altered peritoneal inflammation, and susceptibility to *T. gondii* infection.
- The *Lgals3* gene is located on the Chr14. 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)

## Lgals3 lectin, galactose binding, soluble 3 [Mus musculus (house mouse)]

Gene ID: 16854, updated on 9-Apr-2019

### Summary



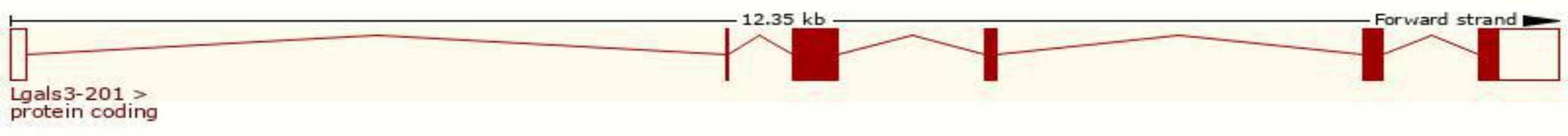
<b>Official Symbol</b>	Lgals3 provided by <a href="#">MGI</a>
<b>Official Full Name</b>	lectin, galactose binding, soluble 3 provided by <a href="#">MGI</a>
<b>Primary source</b>	<a href="#">MGI:MGI:96778</a>
<b>See related</b>	<a href="#">Ensembl:ENSMUSG00000050335</a>
<b>Gene type</b>	protein coding
<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>	GBP, L-34, Mac-2, gal3
<b>Expression</b>	Biased expression in colon adult (RPKM 931.1), duodenum adult (RPKM 311.5) and 8 other tissues <a href="#">See more</a>
<b>Orthologs</b>	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

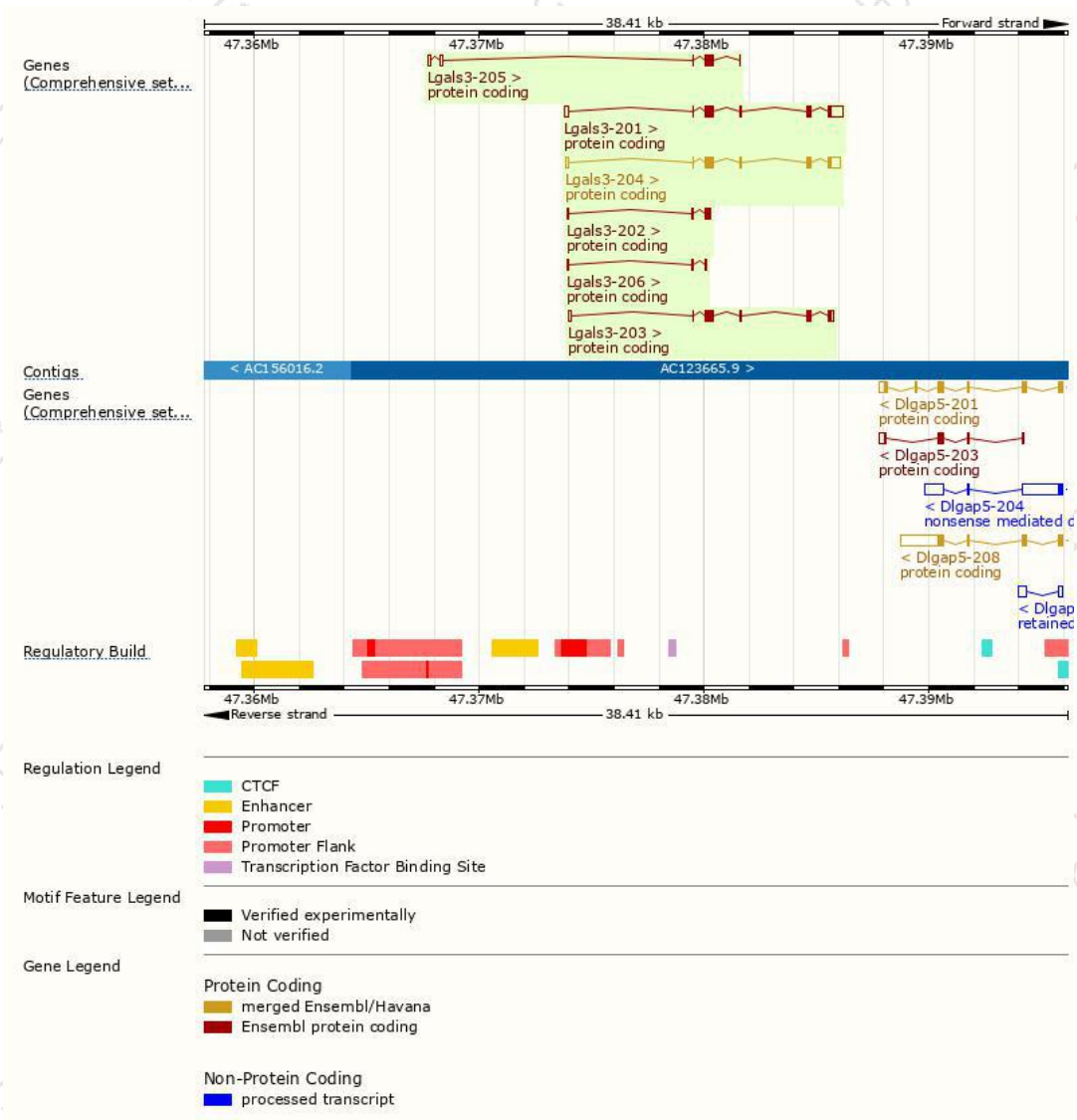
The gene has 6 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Lgals3-201	<a href="#">ENSMUST00000142734.7</a>	1419	<a href="#">264aa</a>	Protein coding	<a href="#">CCDS26986</a>	<a href="#">Q8C253</a>	TSL:1 GENCODE basic APPRIS P1
Lgals3-204	<a href="#">ENSMUST00000150290.8</a>	1298	<a href="#">264aa</a>	Protein coding	<a href="#">CCDS26986</a>	<a href="#">Q8C253</a>	TSL:1 GENCODE basic APPRIS P1
Lgals3-203	<a href="#">ENSMUST00000146468.3</a>	1072	<a href="#">264aa</a>	Protein coding	<a href="#">CCDS26986</a>	<a href="#">Q8C253</a>	TSL:1 GENCODE basic APPRIS P1
Lgals3-205	<a href="#">ENSMUST00000151405.8</a>	646	<a href="#">139aa</a>	Protein coding	-	<a href="#">D3YZJ2</a>	CDS 3' incomplete TSL:3
Lgals3-202	<a href="#">ENSMUST00000144794.1</a>	368	<a href="#">94aa</a>	Protein coding	-	<a href="#">D3YVT4</a>	CDS 3' incomplete TSL:3
Lgals3-206	<a href="#">ENSMUST00000226585.1</a>	210	<a href="#">32aa</a>	Protein coding	-	<a href="#">A0A2I3BRE5</a>	CDS 3' incomplete

The strategy is based on the design of *Lgals3-204* transcript,The transcription is shown below

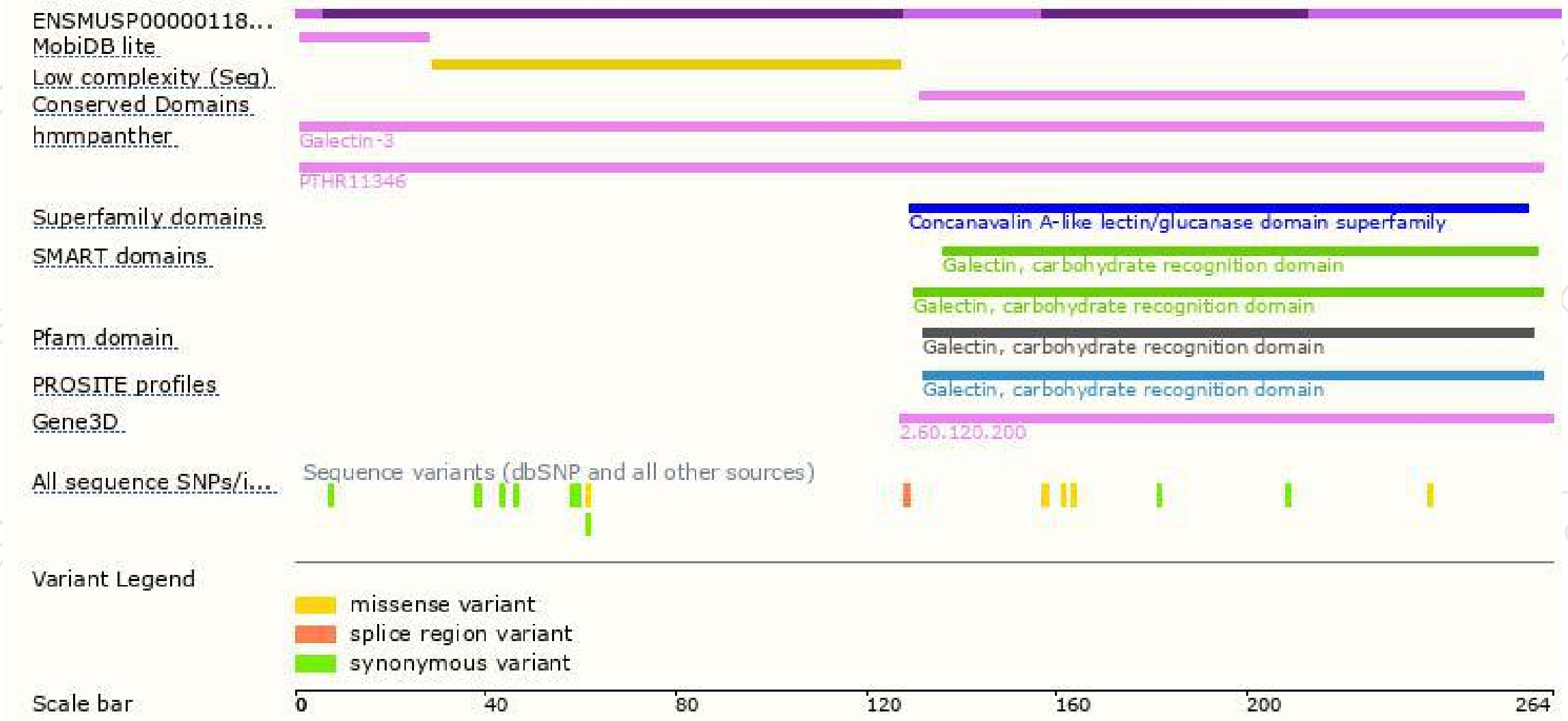


# Genomic location distribution

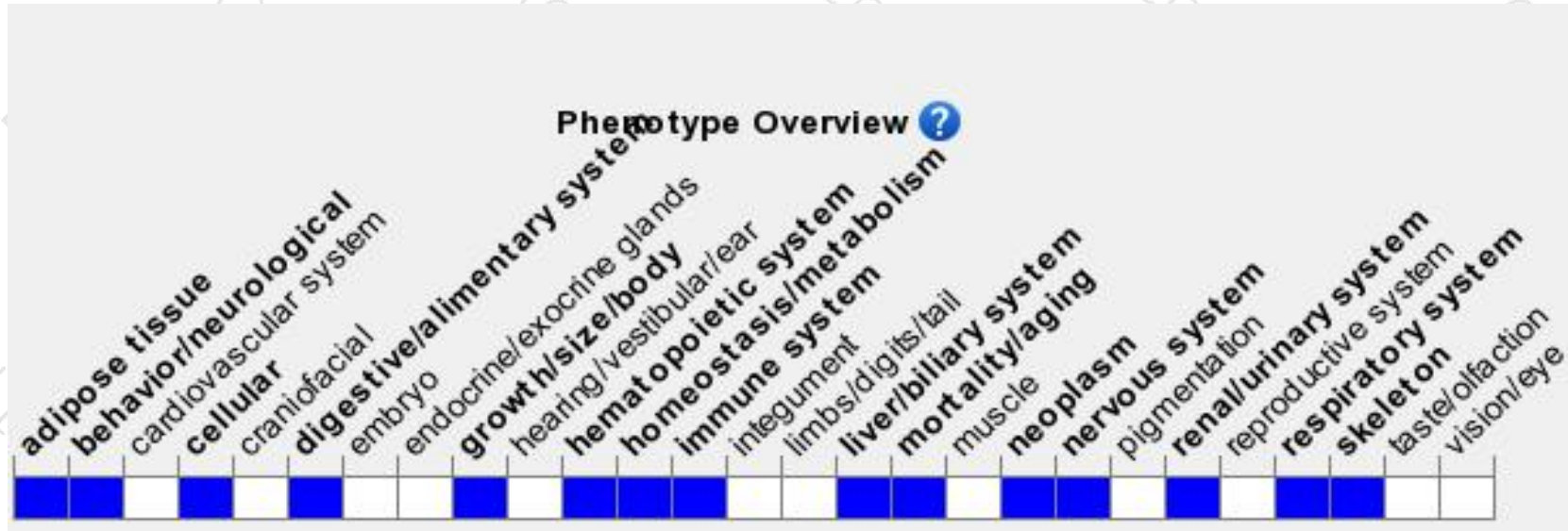




# Protein domain



# 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, Homozygotes for a null allele show susceptibility to *S. pneumoniae* infection, resistance to renal fibrosis, defects in chondrocyte differentiation, and impaired macrophage activation. Homozygotes for another null allele show altered peritoneal inflammation, and susceptibility to *T. gondii* infection.

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

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