

# *Col17a1* Cas9-KO Strategy

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**Reviewer:**

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**Design Date:**

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# Project Overview

**Project Name**

*Col17a1*

**Project type**

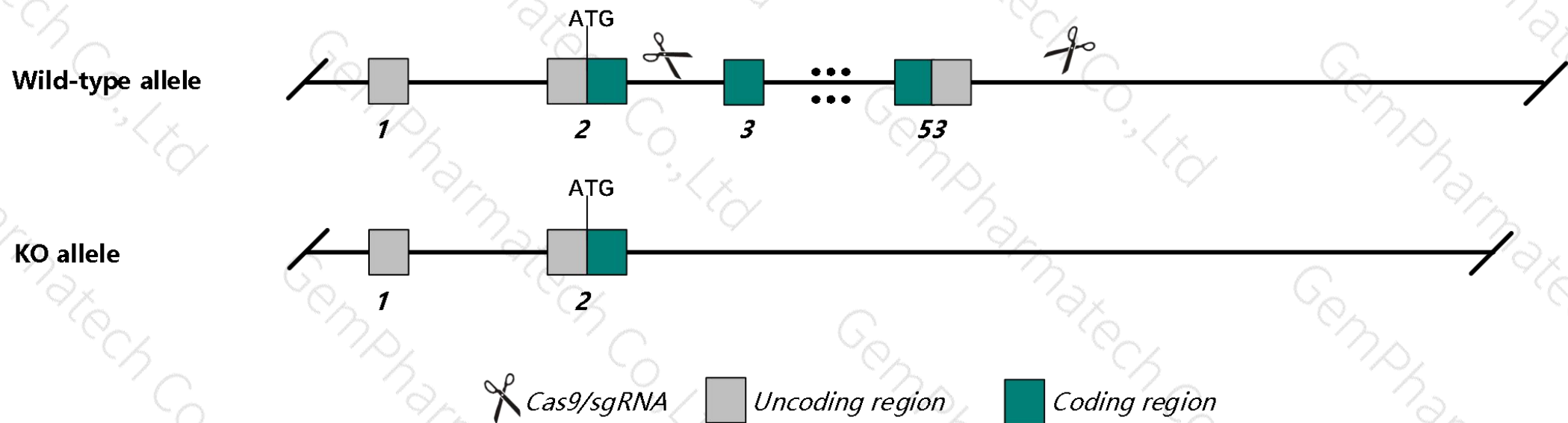
**Cas9-KO**

**Strain background**

**C57BL/6JGpt**

# Knockout strategy

This model will use CRISPR/Cas9 technology to edit the *Coll7a1* gene. The schematic diagram is as follows:



# Technical routes

- The *Coll17a1* gene has 5 transcripts. According to the structure of *Coll17a1* gene, exon3-exon53 of *Coll17a1*-202 (ENSMUST00000086923.5) 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 *Coll17a1* gene. The brief process is as follows: CRISPR/Cas9 system transfects cells 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.

- According to the existing MGI data, Mice homozygous for a knock-out allele are unable to reproduce and display postnatal growth retardation, blisters and erosion at sites of trauma, nonpigmented hair growth associated with hair loss, subepidermal blistering associated with poorly formed hemidesmosomes, and high postnatal lethality.
- The *Coll7a1* gene is located on the Chr19. 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 gene knockout on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.



# Gene information (NCBI)

## Col17a1 collagen, type XVII, alpha 1 [ *Mus musculus* (house mouse) ]

Gene ID: 12821, updated on 15-Mar-2020

### Summary

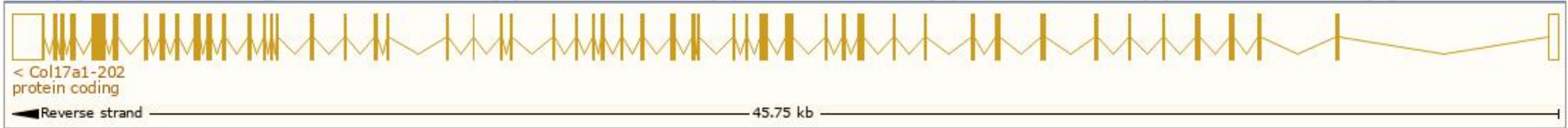
Official Symbol	Col17a1 provided by <a href="#">MGI</a>
Official Full Name	collagen, type XVII, alpha 1 provided by <a href="#">MGI</a>
Primary source	<a href="#">MGI:MGI:88450</a>
See related	<a href="#">Ensembl:ENSMUSG000000025064</a>
Gene type	protein coding
RefSeq status	VALIDATED
Organism	<a href="#">Mus musculus</a>
Lineage	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus
Also known as	Bpag; BP180; Bpag2
Expression	Biased expression in limb E14.5 (RPKM 10.4), mammary gland adult (RPKM 7.6) and 7 other tissues <a href="#">See more</a>
Orthologs	<a href="#">human</a> <a href="#">all</a>

# Transcript information (Ensembl)

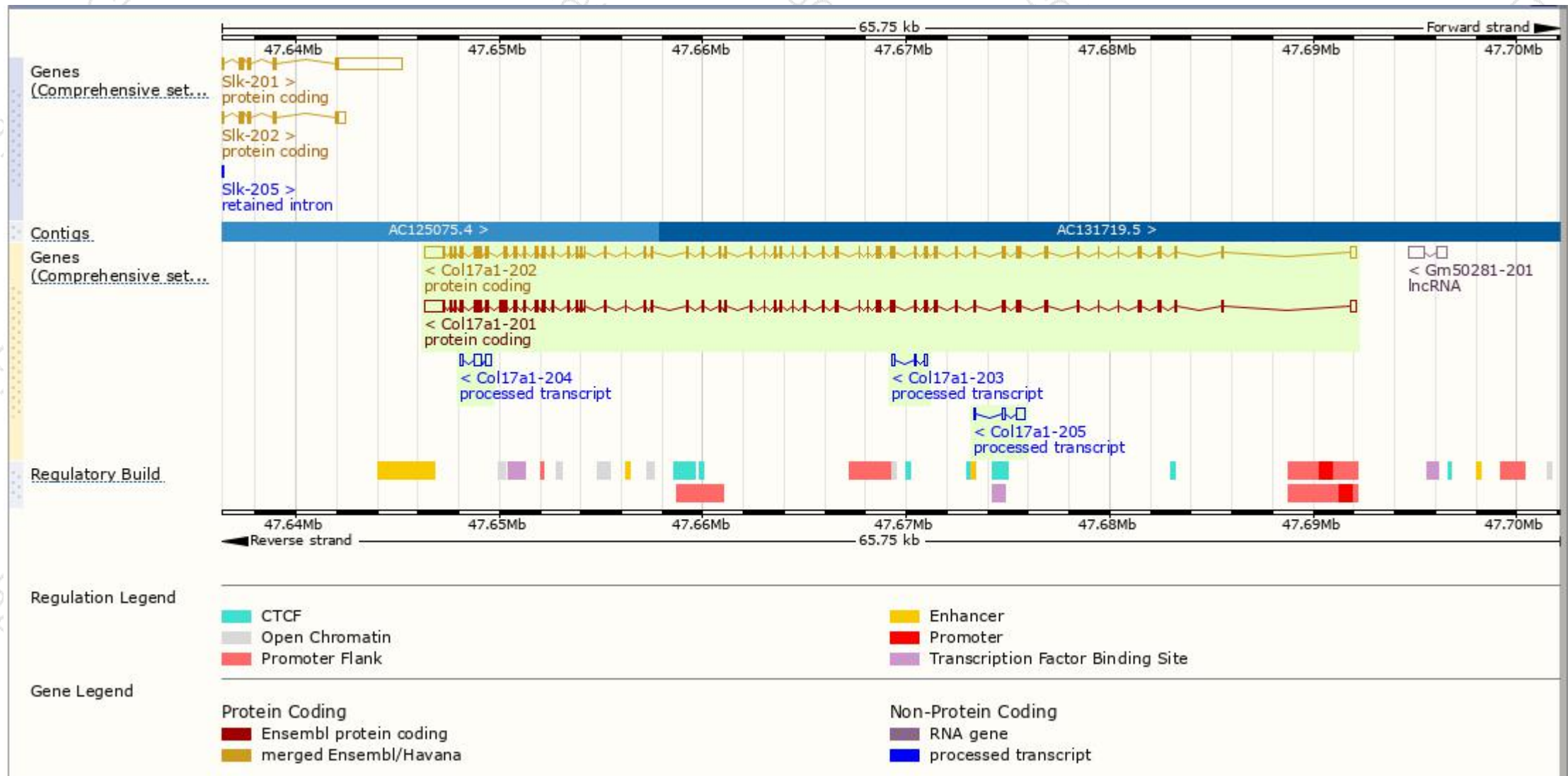
The gene has 5 transcripts,all transcripts are shown below:

Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Col17a1-201	<a href="#">ENSMUST00000026045.13</a>	5588	<a href="#">1470aa</a>	Protein coding	<a href="#">CCDS70959</a>	<a href="#">Q07563</a>	TSL:1 Gencode basic APPRIS ALT2
Col17a1-202	<a href="#">ENSMUST00000086923.5</a>	5477	<a href="#">1433aa</a>	Protein coding	<a href="#">CCDS38018</a>	<a href="#">Q07563</a>	TSL:1 Gencode basic APPRIS P3
Col17a1-204	<a href="#">ENSMUST00000151102.1</a>	758	No protein	Processed transcript	-	-	TSL:5
Col17a1-205	<a href="#">ENSMUST00000235883.1</a>	549	No protein	Processed transcript	-	-	-
Col17a1-203	<a href="#">ENSMUST00000145254.1</a>	357	No protein	Processed transcript	-	-	TSL:2

The strategy is based on the design of *Col17a1-202* transcript,The transcription is shown below

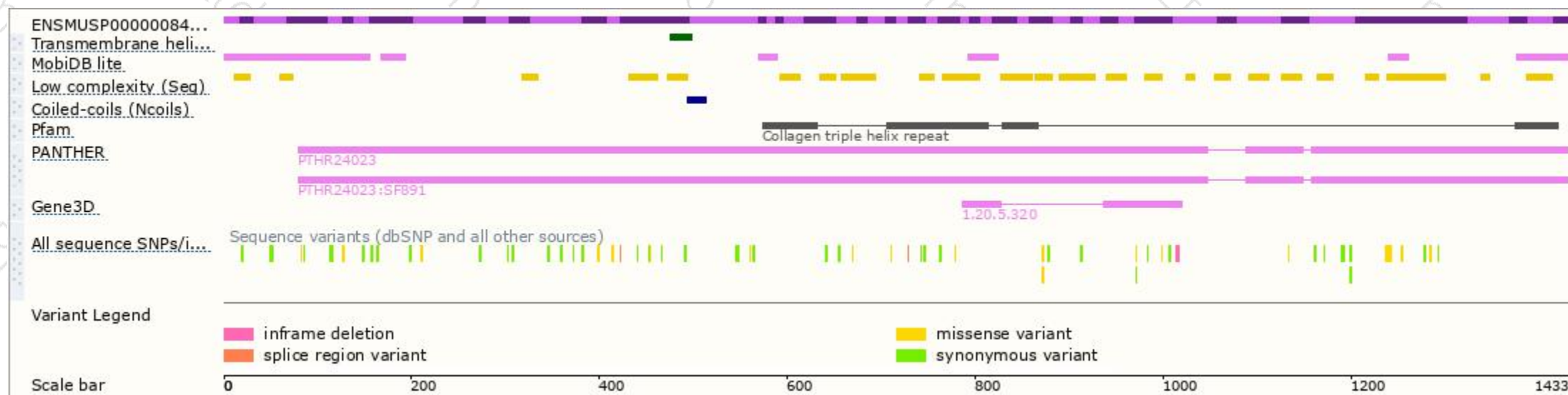


# Genomic location distribution

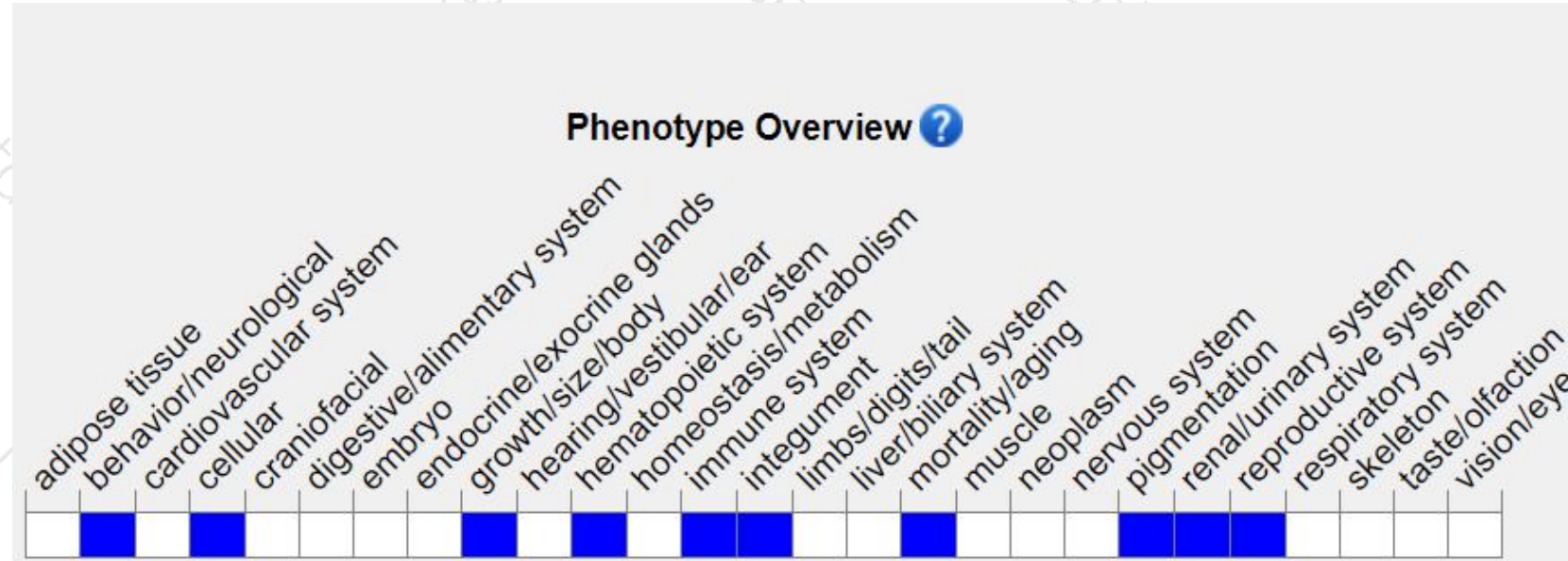




# Protein domain



# 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 are unable to reproduce and display postnatal growth retardation, blisters and erosion at sites of trauma, nonpigmented hair growth associated with hair loss, subepidermal blistering associated with poorly formed hemidesmosomes, and high postnatal lethality.

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

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