

# **F9-KO**

**Nomenclature** C57BL/6Smoc-*F9*<sup>em1Smoc</sup>

**Cat. NO.** NM-KO-18046

**Strain State** Embryo cryopreservation

# **Gene Summary**

Gene Symbol F9	Synonyms	Cf9; Cf-9; AW111646
	NCBI ID	<u>14071</u>
	MGI ID	88384
	Ensembl ID	ENSMUSG00000031138
	Human Ortholog	F9

# **Model Description**

The F9 gene is located on the X chromosome. In this F9 knockout mouse model, gRNAs were designed targeting Exon8 of F9 gene via CRISPR gene editing technology. Loss of F9 caused coagulopathy in mice. Mice that are homozygous for F9 knockout are viable, fertile and normal in size. Normal fighting in the cage may cause bleeding or even death due to massive internal hemorrhaging. After the tail cutting, wounds must be cauterized to prevent homozygous knockout mice from blood loss and death. This strain is a powerful model for studying coagulopathy, gene therapy methods and function of factor IX mutations. F9-KO mice (Stock No.NM-KO-18046) carry a knockout allele derived from the targeted deletion of exon 8. While F9-KO(2) mice (Stock No.NM-KO-200607) carrying the exon 1-8 deletion.

**Research Application**: factor IX (F9) function and gene therapy

\*Literature published using this strain should indicate: F9-KO mice (Cat. NO. NM-KO-18046) were purchased from Shanghai Model Organisms Center, Inc..

#### **Disease Connection**

	Phenotype(s)	MGI:2175873
Hemophilia B	Reference(s)	Wang L, Zoppe M, Hackeng TM, Griffin JH, Lee KF, Verma IM, A factor IX-deficient mouse model for hemophilia B gene therapy. Proc Natl Acad Sci U S A. 1997 Oct 14;94(21):11563-6



# **Validation Data**

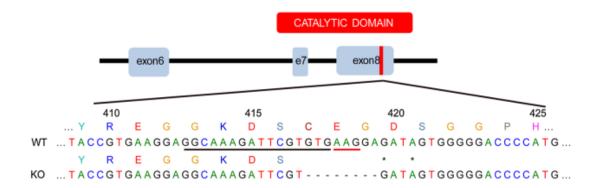


Fig7 Generation of F9 mutant mouse strain.

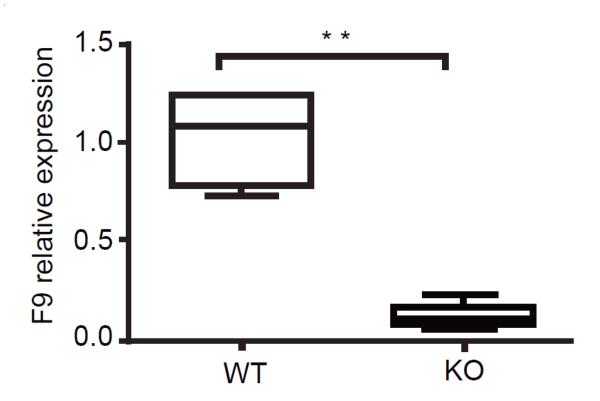


Fig8 Expression of F9 mRNA in the hepatic tissue of WT and KO mouse strains.



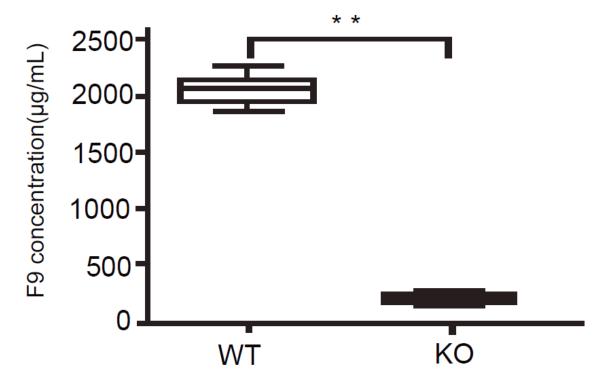


Fig9 Concentration of FIX protein in the plasma of WT and KO mouse strains.

Data in c and d were acquired from 8 mice per group, and are shown as the mean ± the s.d.

Twotailed unpaired Student's t tests were used to determine the P value.\*\*P < 0.01

# **Publications**

<u>CRISPR/Cas9-mediated somatic and germline gene correction to restore hemostasis in hemophilia B mice</u>

References: Springer Link