

# Cdk4-KO

<b>Nomenclature</b>	C57BL/6Smoc- <i>Cdk4</i> <sup>em1Smoc</sup>
<b>Cat. NO.</b>	NM-KO-190614
<b>Strain State</b>	Developing

## Gene Summary

<b>Gene Symbol</b> Cdk4	<b>Synonyms</b>	Crk3
	<b>NCBI ID</b>	<a href="#">12567</a>
	<b>MGI ID</b>	<a href="#">88357</a>
	<b>Ensembl ID</b>	<a href="#">ENSMUSG000000006728</a>
	<b>Human Ortholog</b>	CDK4

## Model Description

Exon 3-7 of Cdk4 gene was deleted to generate Cdk4 knockout mice.

**Research Application:** The protein encoded by this gene is a member of the Ser/Thr protein kinase family. It is a catalytic subunit of the protein kinase complex that is important for cell cycle G1 phase progression. The activity of this kinase is restricted to the G1-S phase, which is controlled by the regulatory subunits D-type cyclins and CDK inhibitor p16(INK4a). This kinase was shown to be responsible for the phosphorylation of retinoblastoma gene product (Rb).

\*Literature published using this strain should indicate: Cdk4-KO mice (Cat. NO. NM-KO-190614) were purchased from Shanghai Model Organisms Center, Inc..

## Disease Connection

<b>Type 1 Diabetes Mellitus</b>	<b>Phenotype(s)</b>	<a href="#">MGI:2386959</a>
	<b>Reference(s)</b>	Rane SG, Dubus P, Mettus RV, Galbreath EJ, Boden G, Reddy EP, Barbacid M, Loss of Cdk4 expression causes insulin-deficient diabetes and Cdk4 activation results in beta-islet cell hyperplasia. Nat Genet. 1999 May;22(1):44-52

## Validation Data

No data

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