H11-CAG-LSL-Myc

Nomenclature	C57BL/6Smoc- <i>Igs2</i> ^{em1(CAG-LSL-Myc)Smoc}	
Cat. NO.	NM-KI-00039	
Strain State	Repository Live	

Gene Summary

Gene Symbol	Synonyms	H11, Hipp11, lis2
	NCBI ID	Null
	MGI ID	<u>5461148</u>
	Ensembl ID	Null

Model Description

The c-Myc gene (also known as Myc) is abnormally expressed in many tumors and plays a critical role in the regulation of cell proliferation, growth and metabolism, gene instability, stimulation of angiogenesis, malignant transformation, cell differentiation and apoptosis. These mice harbor a loxP-flanked STOP cassette preventing transcription of a CAG promoter-driven Myc oncogene. The targeted mutation was inserted into the Hipp11(H11) locus by homologous recombination. Myc is expressed when bred to mice that express Cre recombinase.

Research Application: tumor-bearing model, cancer research

*Literature published using this strain should indicate: H11-CAG-LSL-Myc mice (Cat. NO. NM-KI-00039) were purchased from Shanghai Model Organisms Center, Inc..

Validation Data

Using the Crispr technology, the conditional overexpression structure of CAG promoter-loxp-STOP-loxp-Myc-polyA was inserted into the H11 locus to generate an H11-LSL-Myc mouse model. Located on mouse chromosome 11, the H11 locus is similar to Rosa26 and can be used to express a wide range of exogenous genes. The Myc gene can be highly expressed in Cre-expressing tissues after mating the H11-LSL-Myc mice with Cre mice.

For example: After mating H11-LSL-Myc mice with Alb-cre mice, a liver cancer can be spontaneously developed at 2 months old. This mouse model can be used in the establishment of tumor models and tumor research.



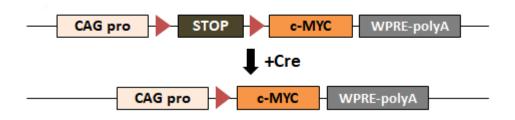


Fig1. Schematic diagram of H11-LSL-Myc knockin cassette.

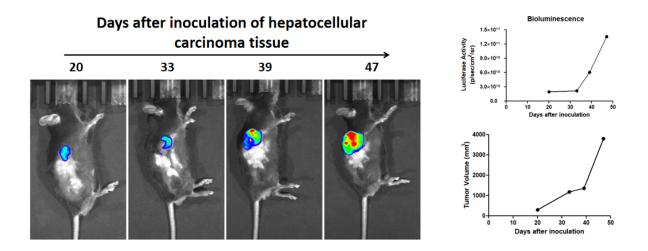


Fig2 Representative in vivo bioluminescence imaging showing the growth of tumors in C57BL/6 mice inoculated with hepatocellular carcinoma tissue from H11-LSL-Myc; Rosa26-LSL-Luc-EGFP; Alb-Cre mice.

Publications

<u>A method to establish a c-Myc transgenic mouse model of hepatocellular carcinoma</u> References: MethodsX

Loss of Mettl3 enhances liver tumorigenesis by inducing hepatocyte dedifferentiation and hyperproliferation References: Cell Reports