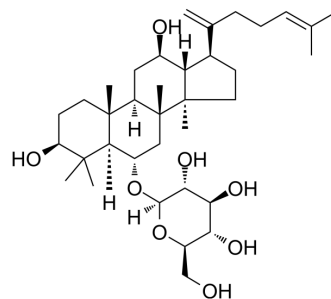


## Ginsenoside Rk3

Cat. No.:	HY-N0906
CAS No.:	364779-15-7
Molecular Formula:	C <sub>36</sub> H <sub>60</sub> O <sub>8</sub>
Molecular Weight:	620.86
Target:	NF-κB
Pathway:	NF-κB
Storage:	-20°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



### SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (161.07 mM; Need ultrasonic)						
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg	
				1 mM	1.6107 mL	8.0533 mL	16.1067 mL
				5 mM	0.3221 mL	1.6107 mL	3.2213 mL
				10 mM	0.1611 mL	0.8053 mL	1.6107 mL
Please refer to the solubility information to select the appropriate solvent.							
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (4.03 mM); Clear solution						
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (4.03 mM); Clear solution						
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (4.03 mM); Clear solution						

### BIOLOGICAL ACTIVITY

Description	Ginsenoside Rk3 is present in the roots Panax ginseng herbs. Ginsenoside Rk3 significantly inhibits TNF-α-induced NF-κB transcriptional activity, with an IC <sub>50</sub> of 14.24±1.30 μM in HepG2 cells.	
IC <sub>50</sub> & Target	NF-κB 14.24 μM (IC <sub>50</sub> , in HepG2 cells)	NF-κB 15.32 μM (IC <sub>50</sub> , in SK-Hep1 cell)
In Vitro	Ginsenoside Rk3 exerts the strong activity inhibiting NF-κB in a dose-dependent manner. HepG2 cells are pre-treated with different ginsenosides at concentrations ranging from 0.01 to 10 μM for 1 h, and induced with TNF-α for 20 h. Ginsenoside	

Rk3 significantly inhibits TNF- $\alpha$ -induced NF- $\kappa$ B transcriptional activity, with an IC<sub>50</sub> of 14.24 $\pm$ 1.30  $\mu$ M. Ginsenoside Rk3 significantly inhibits TNF- $\alpha$ -induced NF- $\kappa$ B transcriptional activity, with an IC<sub>50</sub> of 15.32 $\pm$ 0.29  $\mu$ M in SK-Hep1 cells, consistent with the data from HepG2 cells. Consistent with the inhibition of NF- $\kappa$ B, Ginsenoside Rk3 inhibits the induction of IL8, CXCL1, iNOS, and ICAM1 mRNA significantly in a dose-dependent manner<sup>[1]</sup>.  
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### In Vivo

The inhibitory effects of Ginsenoside Rk3 (Rk3) on tumor progression are studied in vivo using a H460 xenograft model in nude mice. Compared with the control group, a significant inhibition of tumor growth (volume) is observed in the Ginsenoside Rk3-treated group. Twenty-one days after treatment initiation, tumor growth is significantly inhibited by approximately 62.99% in the mice receiving 20 mg/kg Ginsenoside Rk3, similar to the inhibitory effect observed in the 20 mg/kg Gefitinib-treated group (57.21%). Compared with the control group, tumor growth is moderately inhibited in the mice receiving 10 and 5 mg/kg Ginsenoside Rk3, with inhibition rates of 32.54% and 11.84%, respectively<sup>[2]</sup>.  
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## PROTOCOL

#### Cell Assay <sup>[1]</sup>

HepG2 and SK-Hep1 cells are maintained in Dulbecco's modified Eagle's medium containing 10% heat-inactivated fetal bovine serum, 100 units/mL Penicillin, and 10  $\mu$ g/mL Streptomycin, at 37°C and 5% CO<sub>2</sub>. Cell-Counting Kit (CCK)-8 is used to analyze the effect of compounds (e.g., Ginsenoside Rk3; 0.01, 0.1, 1 and 10  $\mu$ M) on cell toxicity. Cells are cultured overnight in 96-well plate ( $\sim$ 1 $\times$ 10<sup>4</sup> cells/well). Cell toxicity is assessed after the addition of compounds on dose-dependent manner. After 24 h of treatment, 10  $\mu$ L of the CCK-8 solution is added to triplicate wells, and incubated for 1 h. Absorbance is measured at 450 nm to determine viable cell numbers in wells<sup>[1]</sup>.  
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### Animal Administration <sup>[2]</sup>

Mice<sup>[2]</sup>  
Four- to 5-week-old male nude mice are selected and housed under aseptic conditions. The animals are exposed to a phase shift of the light/dark cycle for one week and allowed free access to a normal diet. All animal handling is performed under a laminar flow hood. The H460 xenograft model is established. Cell suspensions at a density of 4-5 $\times$ 10<sup>6</sup> cells are subcutaneously implanted into the left axilla of each mouse. Tumor engraftment is considered successful when the tumors are clearly visible, which occurs after one to two weeks. The tumor-bearing mice are randomly divided into the following 5 groups (5 mice per group) according to tumor size and body weight: control group (0.9% saline solution), Ginsenoside Rk3-treated group (5/10/20 mg/kg), and Gefitinib-treated group (20 mg/kg). The indicated doses (5-20 mg/kg) of Ginsenoside Rk3 are safe for mice as determined by preliminary acute oral toxicity tests. The dose of Gefitinib is based on the results from another study. Mice are intragastrically treated daily for 21 days. The tumor volumes are estimated. The body weights and tumor volumes of the mice in each group are measured twice per week. After 21 days, all animals are euthanized, and all tumor tissues are removed, weighed, and collected.  
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## REFERENCES

- [1]. Cho K, et al. Inhibition of TNF- $\alpha$ -Mediated NF- $\kappa$ B Transcriptional Activity by Dammarane-Type Ginsenosides from Steamed Flower Buds of Panax ginseng in HepG2 and SK-Hep1 Cells. *Biomol Ther (Seoul)*. 2014 Jan;22(1):55-61.
- [2]. Duan Z, et al. Anticancer effects of ginsenoside Rk3 on non-small cell lung cancer cells: in vitro and in vivo. *Food Funct*. 2017 Oct 18;8(10):3723-3736.

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**Caution: Product has not been fully validated for medical applications. For research use only.**

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