Ginsenoside Rb3

Cat. No.: HY-N0041

CAS No.: 68406-26-8

Molecular Formula: $C_{53}H_{90}O_{22}$

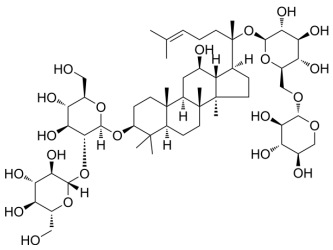
Molecular Weight: 1079.27

Target: NF-κB; COX; NO Synthase

Pathway: NF-κB; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (92.66 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.9266 mL	4.6328 mL	9.2655 mL
5 mM	0.1853 mL	0.9266 mL	1.8531 mL
10 mM	0.0927 mL	0.4633 mL	0.9266 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 (2.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ginsenoside Rb3 is extracted from steamed Panax ginseng C. A. Meyer. Ginsenoside Rb3 exhibits inhibitory effect on TNFα-induced NF-κB transcriptional activity with an IC_{50} of 8.2 μM in 293T cell lines. Ginsenoside Rb3 also inhibits the induction of COX-2 and iNOS mRNA.

IC_{50} & Target

NF-κB	iNOS	COX-2
8.2 μM (IC_{50}, in 293T cell lines)		

In Vitro

Ginsenoside Rb3 (0.1-10 μM) is tested for inhibition of tumor necrosis factor-α (TNF)-induced nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) luciferase reporter activity using a human kidney 293T cell-based assay.

	Ginsenoside Rb3 shows the significant activity with an IC_{50} of 8.2 μM. Ginsenoside Rb3 also inhibits the induction of cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) messenger Ribonucleic acid (mRNA) in a dose-dependent manner after HepG2 cells have been treated with TNF-α (10 ng/mL)[1]. Ginsenoside Rb3 (0.1-10 μM) significantly increases cell viability and inhibits lactate dehydrogenase (LDH) release in a dose-dependent manner. PC12 cell viability as determined by MTT reduction is also markedly decreased after the cell is exposed to oxygen and glucose deprivation (OGD)/OGD-Rep. But, when the cells are pretreated with Ginsenoside Rb3 (0.1, 1, and 10 μM), OGD/OGD-Rep induced cell toxicity is significantly attenuated, which is concentration-dependently attenuated by Ginsenoside Rb3 treatment. The viabilities are raised to 52.8%±5.6%, 64.6%±5.7%, and 76.4%±8.8%, respectively, compared with the control group[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Ginsenosides Rb3 is a major compound isolated from Gynostemma pentaphyllum that holistically improves gut microenvironment and induces anti-polyposis in $Apc^{Min/+}$ mice. Six-weeks-old mice are subjected to Rb3 treatment, before the appearance of the intestinal polyps. All the mice are monitored for food intake, water consumption, and weight changes. Throughout the experiment, no Rb3/Rd-associated weight loss in mice is observed. In addition, none of the treated mice show variations in food and water consumption. Whereas, the number and size of the polyps are effectively reduced by Rb3 treatments[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	HepG2 cells are seeded at a concentration of 1×10^5 cells/mL (1.5 mL) in a 12-well plate and grown for 24 h. All cells are then transfected with Pnf-κB-luc plasmid (0.5 μg/well). Transfection are performed by the lipofectamine LTX. After 23 h of transfection, medium is changed to assay medium. After 24 h of transfection, cells are treated with test compounds (e.g., Ginsenoside Rb3; 0.1, 1 and 10 uM) for another 24 hours. After 25 h of transfection, cells are treated with 10 ng/mL of TNF-α for another 23 hours. The luciferase activity of cell lysates is assayed with 100 μL of by luciferase assay kit using an LB 953 Autolumat. Transfections are performed in triplicate, and activation is normalized against α-galactosidase activity[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [2]	NGF-differentiated PC12 cells are plated at a density of 1.0×10^5 cells/mL 2 days before each experiment. To initiate OGD, the cell culture medium is removed and replaced with the glucose-free DMEM, then the cells are incubated at 37°C in an oxygen-free chamber (95% N_2 and 5% CO_2) for 4 h (OGD), and the change in oxygen levels of the culture medium are monitored during incubation in oxygen-free chamber. Following OGD, glucose is added to normal levels (final concentration: 4.5 mg/mL) and cells are incubated under normal growth conditions (95% air and 5% CO_2) for additional 24 h as OGD-reperfusion (OGD-Rep). Ginsenoside Rb3 (0.1, 1, 10 μM) is added to the culture 24 h before OGD treatment and throughout the OGD reperfusion. The control culture is always maintained in normal DMEM and put in the incubator under normal conditions[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] Heterozygous male $Apc^{Min/+}$ (C57BL/6J-$Apc^{Min/+}$) mice are used. Total 32 male $Apc^{Min/+}$ mice (aged 6 weeks) are divided into three groups; 10 mice in the control group and 22 mice equally divided for Rb3 and Rd treatments. The mice are daily gavage with a single dose of Ginsenoside Rb3 or Rd at 20 mg/kg, or solvent control. The treatments are carried out for 8 consecutive weeks. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. He F, et al. Antitumor effects of dammarane-type saponins from steamed Notoginseng. Pharmacogn Mag. 2014 Jul;10(39):314-7.

[2]. Zhu JR, et al. Protective effects of ginsenoside Rb(3) on oxygen and glucose deprivation-induced ischemic injury in PC12 cells. Acta Pharmacol Sin. 2010 Mar;31(3):273-

80.

[3]. Huang G, et al. Ginsenosides Rb3 and Rd reduce polyps formation while reinstate the dysbiotic gut microbiota and the intestinal microenvironment in $Apc^{Min/+}$ mice. Sci Rep. 2017 Oct 2;7(1):12552.

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