Ginsenoside Rd

Cat. No.: HY-N0043

CAS No.: 52705-93-8

Molecular Formula: $C_{48}H_{82}O_{18}$

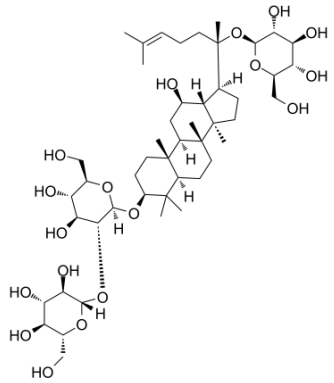
Molecular Weight: 947.15

Target: NF-κB; COX; Calcium Channel; Cytochrome P450; Endogenous Metabolite

Pathway: NF-κB; Immunology/Inflammation; Membrane Transporter/Ion Channel; Neuronal Signaling; Metabolic Enzyme/Protease

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 (105.58 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	1.0558 mL	5.2790 mL	10.5580 mL
5 mM	0.2112 mL	1.0558 mL	2.1116 mL
10 mM	0.1056 mL	0.5279 mL	1.0558 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 (2.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.64 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ginsenoside Rd inhibits TNFα-induced NF-κB transcriptional activity with an IC_{50} of 12.05±0.82 μM in HepG2 cells. Ginsenoside Rd inhibits expression of COX-2 and iNOS mRNA. Ginsenoside Rd also inhibits Ca^{2+} influx. Ginsenoside Rd inhibits CYP2D6, CYP1A2, CYP3A4, and CYP2C9, with IC_{50}s of 58.0±4.5 μM, 78.4±5.3 μM, 81.7±2.6 μM, and 85.1±9.1 μM, respectively.

IC_{50} & Target	NF-κB 12.05 μM (IC_{50}, in HepG2 cells)	COX-2	L-type calcium channel	CYP2D6 58 μM (IC_{50})
	CYP1A2 78.4 μM (IC_{50})	CYP3A4 81.7 μM (IC_{50})	CYP2C9 85.1 μM (IC_{50})	Human Endogenous Metabolite

In Vitro

Ginsenoside Rd is one of the most abundant ingredients of Panax ginseng. Ginsenoside Rd significantly inhibits TNF-α-induced NF-κB transcriptional activity with an IC_{50} of 12.05±0.82 in HepG2 cells. Ginsenoside Rd also inhibits expression of COX-2 and iNOS mRNA and iNOS promoter activity in a dose-dependent manner. To determine nontoxic concentrations, HepG2 cells are treated with various concentrations (0.1, 1, and 10 μM) of compounds (e.g., Ginsenoside Rd) and cell viability is measured using an MTS assay. No compounds are significantly cytotoxic at up to 10 μM, indicating that NF-κB inhibition is not due to cell toxicity[1]. Ginsenoside Rd is one of the most abundant ingredients of Panax ginseng, protects the heart via multiple mechanisms including the inhibition of Ca^{2+} influx. Ginsenoside Rd reduces $I_{Ca,L}$ peak amplitude in a concentration-dependent manner (IC_{50}=32.4±7.1 μM)[2]. Ginsenoside Rd exhibits an inhibition against the activity of CYP2D6 in human liver microsomes with an IC_{50} of 58.0±4.5 μM, a weak inhibition against the activity of CYP1A2, CYP3A4, and CYP2C9 in human liver microsomes with IC_{50}s of 78.4±5.3, 81.7±2.6, and 85.1±9.1, respectively, and an even weaker inhibition against the activity of CYP2A6 in human liver microsomes with an IC_{50} value of more than 100 μM[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Ginsenosides Rd 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 Ginsenoside Rd 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/ Ginsenoside 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 Ginsenoside Rd treatments[3].

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PROTOCOL

Cell Assay [1]

An MTS assay is used to analyze the effects of the compounds on cell viability. HepG2 cells are cultured overnight in a 96-well plate (1×10^4 cells/well). Cell viability is assessed after adding the compounds (e.g., Ginsenoside Rd; 0.1, 1, and 10 μM) for 24 h. The number of viable cells is determined by the A490nm of the dissolved formazan product, after addition of MTS for 30 min[1].

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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 Ginsenoside 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]. Song SB, et al. Inhibition of TNF-α-mediated NF-κB Transcriptional Activity in HepG2 Cells by Dammarane-type Saponins from Panax ginseng Leaves. J Ginseng Res. 2012 Apr;36(2):146-52.

[2]. Lu C, et al. Inhibition of L-type Ca^{2+} current by ginsenoside Rd in rat ventricular myocytes. J Ginseng Res. 2015 Apr;39(2):169-77.

[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.

[4]. Liu Y, et al. Ginsenoside metabolites, rather than naturally occurring ginsenosides, lead to inhibition of human cytochrome P450 enzymes. Toxicol Sci. 2006 Jun;91(2):356-64.

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