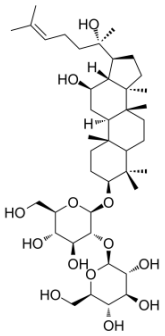
20(S)-Ginsenoside Rg3



Cat. No.: HY-N0603

CAS No.: 14197-60-5

Molecular Formula: $C_{42}H_{72}O_{13}$

Molecular Weight: 785.01

Target: Sodium Channel; Potassium Channel; NF-κB; COX; Amyloid-β; Endogenous Metabolite

Pathway: Membrane Transporter/Ion Channel; NF-κB; Immunology/Inflammation; 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 : ≥ 50 mg/mL (63.69 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.2739 mL	6.3693 mL	12.7387 mL
	5 mM	0.2548 mL	1.2739 mL	2.5477 mL
	10 mM	0.1274 mL	0.6369 mL	1.2739 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 (3.18 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

20(S)-Ginsenoside Rg3 is the main component of Red ginseng. Ginsenoside Rg3 inhibits Na^+ and hKv1.4 channel with IC_{50}s of 32.2±4.5 and 32.6±2.2 μM, respectively. 20(S)-Ginsenoside Rg3 also inhibits Aβ levels, NF-κB activity, and COX-2 expression.

IC_{50} & Target	Na^+ channel	hKv1.4 channel	p65	COX-2

	32.2 μM (IC_{50})	32.6 μM (IC_{50})		
	Aβ40	Aβ42	Human Endogenous Metabolite	

In Vitro

Ginsenoside Rg3 plays an important role in its effect on the Na^+ channel. Treatment with Ginsenoside Rg3 reversibly inhibits the inward Na+ peak current (INa) with an IC_{50} of 32.2±4.5 μM, and the inhibition is voltage-dependent[1]. Ginsenoside Rg3 at 100 μM inhibits the hKv1.4 channel currents by an average of 65%.The Ginsenoside Rg3 effect is concentration-dependent and reversible. The IC_{50} value and Hill coefficient are 32.6±2.2 μM and 1.59±0.13, respectively[2]. Ginsenoside Rg3 shows the significant inhibition of NF-κB activity thereby reduced COX-2 expression. To examine the cytotoxicity of Ginsenoside Rg3 on IL-1β-induced inflamed A549 cells, the cells are firstly treated with IL-1β (10 ng/mL) for 4 h and treated with 100 to 900 ng/mL concentration of Ginsenoside Rg3 for 12 h. Cell viability is analyzed using an MTT assay. There is no observed cytotoxicity of Ginsenoside Rg3 in IL-1β-induced inflamed A549 cells compared to only PBS-treated cells (Con).To obtain the anti-inflammatory effects of Ginsenoside Rg3 on inflammation induced human lung epithelial cells, A549 cells inflammation is induced by IL-1β (10 ng/mL) and then treated by 5 μM of Dexamethasone (Dex) or 900 nM of Rg3. The NF-κB activation is analyzed by a western blot analysis to evaluate the effect of Ginsenoside Rg3 treatment on A549 cells. Phospho-NF-κB p65/total NF-κB p65 densitometry in the cells treated with Rg3 shows the significant decrease compared to IL-1β-induced inflamed A549 cells. The meaning of reducing the ratio of p-p65/p65 by Rg3 treatment is associated with NF-κB activation. Ginsenoside Rg3 also downregulates the expression of COX-2 effectively[3].

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

In Vivo

Ginsenoside Rg3 ((20S)-Rg3) is an Aβ-lowering Natural Compound. APP/PS1 mice are treated with Ginsenoside Rg3 once a day for 4 weeks by intraperitoneal injection (10 mg/kg/day). Aβ ELISA analysis of brain tissues reveal that Ginsenoside Rg3 treatment results in a significant reduction of Aβ40 and Aβ42 in the brain[4].

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PROTOCOL

Cell Assay [3]

MTT assays are performed to evaluate the cytotoxicity of Ginsenoside Rg3 on inflamed cells. Ten thousands of A549 cells cultured each well of 96-well plate and are incubated at 37°C and 5% CO_2 overnight. After serum starvation using DMEM low glucose without FBS, the medium is changed into RPMI containing IL-1β (10 ng/mL) and the cells are incubated at 37°C and 5% CO_2 for 4 h. After 4 h incubation, the cells are treated with Ginsenoside Rg3 (100-900 nM) for 12 h. Thirty microliters of MTT solution (5 mg/mL) is added to each well and the cells are incubated for 2 h. After 2 h incubation in cell culture incubator, the medium containing MTT solution of each well is removed and 50 μL of DMSO is added. Using an automated spectrophotometric plate reader at 570 nm, the optical density of formazan is measured[3].

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Animal Administration [4]

Mice[4]

The mice used are heterozygous, double transgenic animals expressing both human APP(K670N/M671L) and PS1(M146L) proteins. These Alzheimer disease model mice are age-matched (3 months old) in all experiments with wild-type littermates. Both sets of mice are produced by crossing heterozygous APP mice with heterozygous PS1 mice and are weaned at 3 weeks and genotyped by PCR of digested tail samples. Ginsenoside Rg3 is prepared in a saline solution containing 0.01% DMSO at a concentration of 10 mg/kg of body weight. Ginsenoside Rg3 (or saline with 0.01% DMSO for controls) is administered daily via intraperitoneal injection. After sacrifice, one hemibrain from each mouse is frozen on dry ice, homogenized in sucrose buffer, and extracted via formic acid for Aβ quantification using a commercial sandwich ELISA kit.

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CUSTOMER VALIDATION

- Pharmacol Res. 2020 May;155:104751.

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REFERENCES

[1]. Kim JH, et al. A role for the carbohydrate portion of ginsenoside Rg3 in Na^+ channel inhibition. Mol Cells. 2005 Feb 28;19(1):137-42.

[2]. Lee JH, et al. Ginsenoside Rg3 inhibits human Kv1.4 channel currents by interacting with the Lys531 residue. Mol Pharmacol. 2008 Mar;73(3):619-26.

[3]. Lee IS, et al. Anti-Inflammatory Effects of Ginsenoside Rg3 via NF-κB Pathway in A549 Cells and Human Asthmatic Lung Tissue. J Immunol Res. 2016;2016:7521601.

[4]. Kang MS, et al. Modulation of lipid kinase PI4KIIα activity and lipid raft association of presenilin 1 underlies γ-secretase inhibition by ginsenoside (20S)-Rg3. J Biol Chem. 2013 Jul 19;288(29):20868-82.

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

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