Ginsenoside Rg5

Cat. No.: HY-N0908

CAS No.: 186763-78-0

Molecular Formula: $C_{42}H_{70}O_{12}$

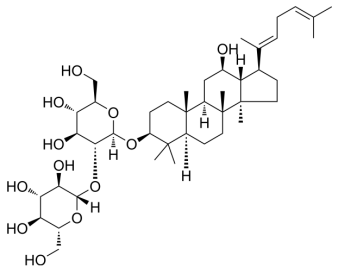
Molecular Weight: 767

Target: IGF-1R; NF-κB; COX

Pathway: Protein Tyrosine Kinase/RTK; NF-κB; Immunology/Inflammation

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (65.19 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.3038 mL	6.5189 mL	13.0378 mL
5 mM	0.2608 mL	1.3038 mL	2.6076 mL
10 mM	0.1304 mL	0.6519 mL	1.3038 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 (3.26 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.26 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ginsenoside Rg5 is the main component of Red ginseng and IGF-1R agonist. Ginsenoside Rg5 competes for the binding site of IGF-1R and blocks the binding of IGF-1 to IGF-1R (IC_{50} about 90 nM). Ginsenoside Rg5 also inhibits the mRNA expression of COX-2 via suppression of the DNA binding activities of NF-κB p65.

IC_{50} & Target

IGF-1R 90 nM (IC_{50})	p65	COX-2

In Vitro

Ginsenoside Rg5 plays a novel role as an IGF-1R agonist. Ginsenoside Rg5 has angiogenic activity, which is inhibited by IGF-

	1R knockdown. To investigate the possible interaction of Ginsenoside Rg5 with IGF-1R, a docking analysis is performed. Docking results show that Ginsenoside Rg5 binds strongly at two sites, A and B, with K_d values of 20 and 27 nM, respectively, to the cysteine-rich domain of IGF-1R. Pretreatment with Rg5 blocks the binding of radiolabeled IGF-1 to HUVECs with an IC_{50} value of ~90 μM, which is greater than an IC_{50} value of ~1.4 nM for unlabeled IGF-1[1]. The results from MTT assay show that MCF-7 cell proliferation is inhibited by Ginsenoside Rg5 treatment for 24, 48 and 72 h in a dose-dependent manner. Ginsenoside Rg5 at different concentrations (0, 25, 50 and 100 μM), induce cell cycle arrest in G0/G1 phase through regulation of cell cycle-related proteins in MCF-7 cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Ginsenoside Rg5 inhibits the mRNA expression of COX-2 via suppression of the DNA binding activities of NF-κB p65 in lipopolysaccharides (LPS)-stimulated BV2 microglial cells. Rg5 pretreated group mice show declined expression of NF-κB p65 and COX-2. In the group treated with low dose of Ginsenoside Rg5 (10 mg/kg), there is remarkable tubular damage and infiltration of inflammatory cells. However, at the higher dose of Ginsenoside Rg5 (20 mg/kg), tubules markedly appeare histologically normal and no inflammation and cast formation is observed in kidney tissues[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	HUVECs are cultured in 24-well plates overnight. The cells are changed to serum-free M199 and incubated for 1 h. The medium is removed, and cells are incubated with fresh serum-free medium containing 0.1 μM-50 mM Ginsenoside Rg5 at 37°C for 20 min followed by the addition of 50 μL (1 μCi) of [^{125}I]IGF-1 and then further incubated for 10 min. The medium is decanted, and cell plates are washed twice with serum-free medium. Cells are lysed in 300 μL of 0.1 N NaOH solution containing 0.1% SDS, transferred to scintillation vials, and mixed with 1 mL of Ultima Gold mixture solution. Cell-associated [^{125}I]IGF-1 is analyzed in a scintillation counter. The nonspecific binding is determined by coincubation with unlabeled IGF-1 (50 nM)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [3]	MCF-7 (HER2$^-$/ER$^+$) and MDA-MB-453 (HER2$^+$/ER$^-$) human breast cancer cell lines are maintained using RPMI 1640 medium supplemented with 10% (vol/vol) FBS plus 100 units/mL Penicillin and Streptomycin in a 5% carbon dioxide air incubator at 37°C. Cell cytotoxicity is measured by MTT assay. Cells are seeded in 96-well tissue culture plates at the density of 0.2×10^4 cells per well with 100 μL medium, and are allowed to become attached for 24 h. One hundred microliters of the medium with different concentrations of Ginsenoside Rg5 (e.g., 0 μM, 25 μM, 50 μM, and 100 μM) are added to each well. At indicated times, 30 μL MTT stock solution (3 mg/mL) are added to each well. After culturing the cells at 37°C for 2 h, DMSO is added to dissolve the formazan crystals. The absorbance is read at the wavelength of 540 nm with a microplate reader[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Male ICR mice (6 to 8 weeks old), weighing 25-27 g, are used. After acclimation for one week, mice are randomly assigned into 4 experimental groups with 8 mice in each group: normal control, Cisplatin control, and Cisplatin+Ginsenoside Rg5 groups (10 and 20 mg/kg, respectively). Ginsenoside Rg5 is administered intragastrically at the dose of 10 and 20 mg/kg for 10 days. On the 7th day, animals in Cisplatin control and Ginsenoside Rg5-treated groups receive a single intraperitoneal injection of Cisplatin (25 mg/kg) to induce nephrotoxicity in mice. Mice are anaesthetized with pentobarbital, subsequently sacrificed at 72 h after Cisplatin injection (Day 10). Blood samples are collected and then centrifuged at 3000 rpm to separate the serum and stored at -20 °C for determining blood urea nitrogen (BUN) and creatinine (CRE) levels. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Ginseng Res. 2023 Jun 30.

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REFERENCES

[1]. Cho YL, et al. Specific activation of insulin-like growth factor-1 receptor by ginsenoside Rg5 promotes angiogenesis and vasorelaxation. J Biol Chem. 2015 Jan 2;290(1):467-77.

[2]. Li W, et al. Ginsenoside Rg5 Ameliorates Cisplatin-Induced Nephrotoxicity in Mice through Inhibition of Inflammation, Oxidative Stress, and Apoptosis. Nutrients. 2016 Sep 13;8(9). pii: E566.

[3]. Kim SJ, et al. Anti-breast cancer activity of Fine Black ginseng (Panax ginseng Meyer) and ginsenoside Rg5. J Ginseng Res. 2015 Apr;39(2):125-34.

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