Ginsenoside Re

Cat. No.: HY-N0044

CAS No.: 52286-59-6

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

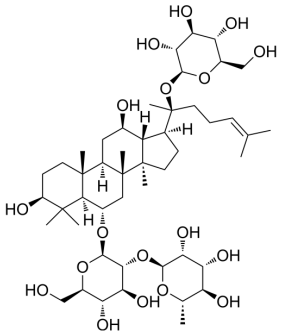
Molecular Weight: 947.15

Target: Amyloid-β; NF-κB; JNK; Endogenous Metabolite

Pathway: Neuronal Signaling; NF-κB; MAPK/ERK Pathway; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (52.79 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / 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 Re (Ginsenoside B2) is an extract from Panax notoginseng. Ginsenoside Re decreases the β-amyloid protein (Aβ). Ginsenoside Re plays a role in antiinflammation through inhibition of JNK and NF-κB.

IC_{50} & Target	$Aβ_{1-40}$	$Aβ_{1-42}$	NF-κB	JNK

In Vitro

Ginsenoside Re is a well-known traditional Chinese medicine, which decreases the β-site amyloid precursor protein cleaving

enzyme 1 (BACE1) mRNA and protein levels and inhibits BACE1 activity in the N2a/APP695 cells. Ginsenoside Re also significantly increases the PPARγ protein and mRNA levels.To prevent Ginsenoside Re from having a cytotoxic effect on the N2a/APP695 cells, the cell viability is first determined by the MTT assay. The N2a/WT and N2a/APP695 cells are treated with increasing concentrations of Ginsenoside Re (0-200 μM) for 24 h. Ginsenoside Re concentrations under 100 μM do not affect the viability of the N2a/WT and N2a/APP695 cells, whereas the 150 μM Ginsenoside Re concentration markedly decreases the survival rate of the N2a/WT and N2a/APP695 cells. Incubation with Ginsenoside Re at a 200 μM concentration for 24 h reduces the viability of the N2a/WT and N2a/APP695 cells by 15.58% and 26.82%, respectively. These data indicate that Ginsenoside Re treatment within the range of 0-100 μM for 24 h is safe for the N2a/WT and N2a/APP695 cells ($P>0.05$)[1].

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

In Vivo

Ginsenoside Re reduces insulin resistance in 3T3-L1 adipocytes and high-fat diet (HFD) rats through inhibition of JNK and NF-κB activation[2]. Intraperitoneal injection of lipopolysaccharide (LPS) at a dose of 20 mg/kg is lethal to mice, and 70% to 80% of the mice die within 60 h. However, pretreatment of the mice with Rg1 or Ginsenoside Re increases their survival rates in a dose-dependent manner. With the doses of Rg1 or Ginsenoside Re increase from 2.5 to 5 mg/kg, the survival rate is elevated from 60% to 90% (Rg1) or from 30% to 40% (Ginsenoside Re). All the mice administered Rg1 at a minimal dose of 10 mg/kg are protected from death compared to 80% survival of mice treated with an equal dose of Ginsenoside Re. To protect all the mice, 20 mg/kg Ginsenoside Re is needed. To investigate the anti-inflammatory potential of Rg1 and Ginsenoside Re, 1 mg/kg Rg1 or Ginsenoside Re is injected into rats and then challenged the animals with LPS. The injection procedure itself causes a transient stress-induced increase in body temperature of ~1.2°C in each group. Thereafter, LPS-challenged rats without pretreatment develope a robust biphasic fever, with the first peak reaching ~1.5°C at 2 h and the second peak reaching 1.8°C at 4 h. In contrast, the temperature changes for the Rg1-, Ginsenoside Re-, and TAK-242-treated groups are only 0.9, 1.2, and 0.8°C at 2 h and 1.3, 1.4, and 1.0°C at 4 h, respectively. Pretreatment with Rg1, Ginsenoside Re, or TAK-242 significantly attenuates LPS-induced alterations in body temperature[3].

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PROTOCOL

Cell Assay [1]

To explore the cytotoxic effect of Ginsenoside Re on N2a/APP695 cells, cell proliferation is assessed using the MTT assay. The cells are treated with increasing doses of Ginsenoside Re (0, 25, 50, 100, 150, and 200 μM) for 24 h. The MTT assay is performed after the treatments. The cells are incubated for 4 h at 37 °C with 0.5 mg/mL of MTT dissolved in fresh complete medium. The dark blue formazan crystals are dissolved in DMSO, and the absorbance is measured on a microplate reader using a reference wavelength of 630 nm and a test wavelength of 490 nm. The data are expressed as the mean percentages of viable cells versus the control[1].

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Animal Administration [3]

Mice[3]

To examine the prophylactic effect of Rg1 and Ginsenoside Re on LPS-induced lethality, 6- to 8-week-old BALB/c mice are randomly assigned to 7 groups with 10 mice in each group. The mice are either left untreated or subcutaneously injected with 2.5, 5, 10, or 20 mg/kg Rg1, 20 mg/kg Ginsenoside Re, or 5 mg/kg TAK-242 3 times at 30-min intervals and then challenged with LPS (20 mg/kg) 15 min later. To test the therapeutic effect of Rg1 and Ginsenoside Re on LPS-induced lethality, BALB/c mice are assigned to 5 groups with 10 mice in each group. The mice are injected intraperitoneally (i.p.) with 20 mg/kg LPS. Fifteen minutes later, mice are subcutaneously injected with 10 mg/kg Rg1, 20 mg/kg Ginsenoside Re, or 5 mg/kg TAK-242 3 times at 30-min intervals or left untreated. Survival rates are recorded for 60 h.

Rats[3]

Sprague Dawley rats are intravenously administered saline solution, 1 mg of Rg1/kg body weight (1 mg/kg Rg1), 1 mg/kg Ginsenoside Re, or 1 mg/kg TAK-242. Fifteen minutes later, rats are challenged with 2.5 μg/kg LPS. Body temperature is measured before and after drug administration. Anticoagulated blood samples with EDTA are collected for white blood cell (WBC) counts at indicated time points using a ProCyte Dx automatic blood cell analyzer. Additional blood samples are collected at 4 h post-drug injection and used for preparation of serum to analyze proinflammatory mediators. The proinflammatory cytokine responses are detected by Western blot assay.

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CUSTOMER VALIDATION

- J Neurosci Res. 2019 Dec;97(12):1689-1705.

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REFERENCES

[1]. Cao G, et al. Ginsenoside Re reduces Aβ production by activating PPARγ to inhibit BACE1 in N2a/APP695 cells. Eur J Pharmacol. 2016 Dec 15;793:101-108.

[2]. Zhang Z, et al. Ginsenoside Re reduces insulin resistance through inhibition of c-Jun NH2-terminal kinase and nuclear factor-kappaB. Mol Endocrinol. 2008 Jan;22(1):186-95.

[3]. Su F, et al. Protective effect of ginsenosides Rg1 and Re on lipopolysaccharide-induced sepsis by competitive binding to Toll-like receptor 4. Antimicrob Agents Chemother. 2015 Sep;59(9):5654-63.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA