Ginsenoside Rh2

Cat. No.: HY-N0605

CAS No.: 78214-33-2

Molecular Formula: $C_{36}H_{62}O_8$

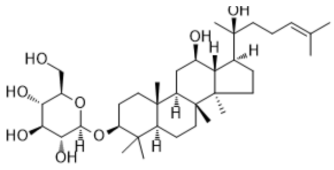
Molecular Weight: 622.87

Target: Caspase; Apoptosis; Endogenous Metabolite

Pathway: Apoptosis; 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 (80.27 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6055 mL	8.0274 mL	16.0547 mL
	5 mM	0.3211 mL	1.6055 mL	3.2109 mL
	10 mM	0.1605 mL	0.8027 mL	1.6055 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.01 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.25 mg/mL (2.01 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (2.01 mM); Suspended solution

BIOLOGICAL ACTIVITY

Description

Ginsenoside Rh2 induces the activation of caspase-8 and caspase-9. Ginsenoside Rh2 induces cancer cell apoptosis in a multi-path manner.

IC_{50} & Target			
	Caspase-8	Caspase-9	Apoptosis

In Vitro

Ginsenoside Rh2 induces the activation of two initiator caspases, caspase-8 and caspase-9 in human cancer cells. Ginsenoside Rh2 induces cancer cell apoptosis in a multi-path manner and is therefore a promising candidate for anti-tumor

	drug development. Ginsenoside Rh2 triggers p53-dependent Fas expression and consequent activation of caspase-8 and p53-independent caspase-9-mediated intrinsic pathway to cause cancer cell death.The cytotoxic activity of Ginsenoside Rh2 in the human tumor cell lines HeLa, SK-HEP-1, SW480, and PC-3 is assessed by MTT. The cell viability of HeLa cells is remarkably inhibited by Ginsenoside Rh2, with an IC_{50} value of 2.52 μg/mL, whereas SK-HEP-1 and SW480 cells are less sensitive to Ginsenoside Rh2, with IC_{50} values of 3.15 μg/mL and 4.06 μg/mL, respectively. PC-3 cells are the least vulnerable to Ginsenoside Rh2, with an IC_{50} value of 7.85 μg/mL, 3-fold higher than HeLa cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	A total of 15 days following B16-F10 cell injection, tumor sizes from the 3 tumor bearing groups are measured. The tumor sizes in the G-L group and G-H group (G-L and G-H refer to a low or high dose of ginsenoside Rh2 injection) are reduced compared with the tumor group ($P<0.05$). The survival analysis reveals that the Ginsenoside Rh2 treated groups survive longer than the untreated tumor group and the effect is dose-dependent ($P<0.05$)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	HeLa, SK-HEP-1, SW480, and PC-3 cells are treated with Ginsenoside Rh2 (7.5 μg/mL) in serum free media for indicated time periods and then are harvested. Fifty micrograms of cell lysates are incubated with 200 nM Ac-DEVD-AFC (for caspase-3), Ac-IETD-AFC (for caspase-8), and Ac-LEHD-AFC (for caspase-9) in a reaction buffer containing 20 mM HEPES, pH 7.4, 100 mM NaCl, 10 mM DTT, 0.1% CHAPS, and 10% sucrose at 37°C for 1 h. The reaction is monitored by fluorescence emission at 535 nm and excitation at 405 nm[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	Determination of cell viability is performed by using MTT assay, which is used to calculate the growth inhibition induced by increasing concentrations of drug. Briefly, exponentially growing HeLa, SK-HEP-1, SW480, and PC-3 cells are seeded into a 96-well plate at 1×10^4 cells/well in triplicate. After incubation for 24 h, cells are treated with increasing concentration of Ginsenoside Rh2 (1, 2.5, 5, 7.5 and 10 μg/mL) in serum free media for 48 h. At the end of treatment, 20 μL of MTT (5 mg/mL) is added to each well and incubated for an additional 4 h. The formazan grains formed by viable cells are solubilized with DMSO, and the color intensity is measured at 550 nm with an ELISA reader[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Male C57BL6 mice (3-4 weeks old) are randomly arranged into 4 groups of 80 mice: Tumor group, G-L group, G-H group and Control group. G-L and G-H refer to a low or high dose of ginsenoside Rh2 injection. For the tumor group, G-L group and G-H group, the B16-F10 cell line is injected into the mice. These 3 groups become tumor bearing groups. For the control group, the same volume of PBS is injected instead. Ginsenoside Rh2 is injected into the left back of mice in the G-L and G-H groups. The dose for the G-H group is 0.5 mg/kg or 0.2 mg/kg for G-L group, every 2 days after day 5. PBS is injected in the tumor and control groups at the same time points. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Pharmacol Res. 2020 May;155:104751.
- J Ginseng Res. 2021 May 25.
- Viruses. 2022, 14(12), 2724.

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REFERENCES

[1]. Guo XX, et al. p53-dependent Fas expression is critical for Ginsenoside Rh2 triggered caspase-8 activation in HeLa cells. Protein Cell. 2014 Mar;5(3):224-34.

[2]. Wang M, et al. Ginsenoside Rh2 enhances the antitumor immunological response of a melanoma mice model. Oncol Lett. 2017 Feb;13(2):681-685.

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