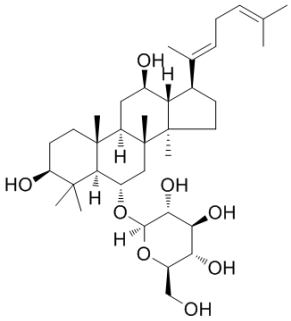
Ginsenoside Rh4

Cat. No.:	HY-N0905
CAS No.:	174721-08-5
Molecular Formula:	$C_{36}H_{60}O_8$
Molecular Weight:	620.86
Target:	Bcl-2 Family; Caspase; Apoptosis; Autophagy
Pathway:	Apoptosis; Autophagy
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



BIOLOGICAL ACTIVITY

Description	Ginsenoside Rh4 is a rare saponin obtained from Panax notoginseng. Ginsenoside Rh4 activates **Bax**, **caspase 3**, **caspase 8**, and **caspase 9**. Ginsenoside Rh4 also induces **autophagy**.
IC_{50} & Target	Bax; Caspase 3; Caspase 8; Caspase 9 Apoptosis; Autophagy
In Vitro	Ginsenoside Rh4 causes cytochrome C release and activates the death receptor Fas, the pro-apoptotic protein Bax, and caspase 3, caspase 8, and caspase 9. Ginsenoside Rh4 induces caspase-dependent apoptosis via both the intrinsic and extrinsic pathways in Caco-2 and HCT-116 cells. The CCK-8 assay reveals that Ginsenoside Rh4 can significantly inhibit the growth of colorectal carcinoma cells, such as Caco-2 and HCT-116 cells, to varying degrees. Ginsenoside Rh4 dramatically reduces cell viability in a concentration- and time-dependent manner. In particular, treatment with 120 and 180 μM Ginsenoside Rh4 results in marked Caco-2 cell death of 44.51±1.23% and 75.74±2.91%, respectively, after incubation for 48 h. Similar results are observed in HCT-116 cells treated with concentrations of 120 μM (33.62±1.98%) and 180 μM Ginsenoside Rh4 (59.88±2.31%). In contrast, concentrations of Rh4 from 120 to 300 μM cause no obvious toxic effects on the normal human colon epithelial cell line CCD-18Co, and a slight difference in the effect is observed between 24 and 48 h of treatment. In the colony formation assay, the number of colonies is found to be significantly decreased in Caco-2 and HCT-116 cells, whereas the number of colonies is almost unchanged in CCD-18Co cells[1].
In Vivo	To assess whether Ginsenoside Rh4 can inhibit the growth of colorectal cancer, a colorectal cancer xenograft model is established by inoculating nude mice with Caco-2 cells. Mice treated with 20 and 40 mg/kg Ginsenoside Rh4 and 40 mg/kg CAMPTO exhibit smaller tumors than the control after 30 days of treatment, showing inhibition rates of 29.91%, 66.30% and 77.82%, respectively. However, there is a significant difference in body weight between the Ginsenoside Rh4-treated group and the CAMPTO-treated group. The body weights of the 20 and 40 mg/kg Ginsenoside Rh4-treated groups (15.95±0.35 g and 18.35±0.44 g) are markedly higher, whereas the body weight of the CAMPTO-treated group is lower (10.85±0.28 g) than that of the solvent control group (14.19±0.25 g)[1].

PROTOCOL

Cell Assay [1]	Cell viability is measured via the CCK-8 assay. For these assays, **Caco-2, HCT116 and CCD-18Co cells** are seeded onto 96-well plates at a density of 1×10^4 cells per well and incubated overnight, followed by treatment with 0.1% (vol/vol) DMSO as vehicle or **Ginsenoside Rh4 (60, 120, 180, 240 and 300 μM)** for 24 or 48 h. Subsequently, the cells are incubated with 10% (vol/vol) the CCK-8 solution for 2 h. The absorbance at 450 nm is then read with a microplate reader. Cell viability is calculated[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] **Four-week-old female nude mice (12±2 g)** are used. After acclimation for one week, the mice are inoculated with Caco-2 cells (1×10^7 cells/each) in the left forelimb pit. When the tumors reach 100 mm^3, the mice are randomized into four groups (n=5): a control group (solvent), two **Ginsenoside Rh4-treated groups (20 and 40 mg/kg/d)** , and a positive control CAMPTO-treated group (40 mg/kg/3 d). The body weights and tumor sizes of the mice are measured every three days, and the tumor volume is calculated. After 30 days of treatment, the mice are sacrificed, and the xenograft tumors are removed and partially lysed to analyze the expression of cleaved-caspase 3, LC3, p-JNK and p-p53. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Wu Q, et al. Ginsenoside Rh4 induces apoptosis and autophagic cell death through activation of the ROS/JNK/p53 pathway in colorectal cancer cells. Biochem Pharmacol. 2018 Feb;148:64-74.

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