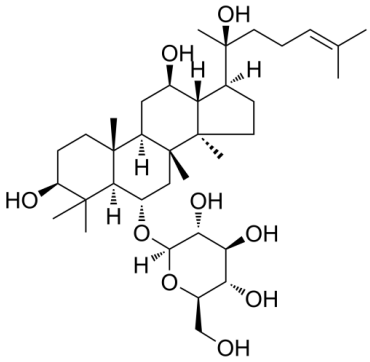
Data Sheet

Product Name:	Ginsenoside Rh1
Cat. No.:	CS-3834
CAS No.:	63223-86-9
Molecular Formula:	$C_{36}H_{62}O_9$
Molecular Weight:	638.87
Target:	Endogenous Metabolite; Interleukin Related; PPAR; TNF Receptor
Pathway:	Apoptosis; Cell Cycle/DNA Damage; Immunology/Inflammation; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor
Solubility:	DMSO : 100 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

Ginsenoside Rh1 (Prosapogenin A2) inhibits the expression of **PPAR-γ**, **TNF-α**, **IL-6**, and **IL-1β**. IC50 & Target: PPAR-γ, TNF-α, IL-6, and IL-1β[1] ***In Vitro:*** The effect of Ginsenoside Rh1 is examined on adipogenesis in 3T3-L1 cells. Ginsenoside Rh1 potently inhibits the adipogenesis, as assessed by Oil-red O staining and lipid contents in 3T3-L1 adipocytes. Ginsenoside Rh1, at concentrations of 50 μM and 100 μM, inhibit the adipogenesis by 50% and 63%, respectively.The expression levels of adipocytespecific genes such as PPAR-γ, C/EBP-α, FAS, aFABP and some genes are examined during early phase of differentiation such as Pref-1, C/EBP-δ and Glucocorticoid receptor (GR). After the treatment with Ginsenoside Rh1 in 3T3-L1 cells, mRNA is extracted on 18 h and 24 h for Pref-1, C/EBP-δ and GR and day 8 for PPAR-γ, C/EBP-α, FAS, aFABP. Then, the expression profiles of adipocyte-specific genes are investigated by RT-PCR. PPAR-γ, C/EBP-α, FAS, and aFABP expressions are significantly increased in DMI-stimulated differentiated adipocyte compared to those of non-stimulated adipocyte cells. However, treatment with DMI in the presence of Ginsenoside Rh1 significantly suppresses the expression levels of PPAR-γ, C/EBP-α, FAS, and aFABP in a dose- dependent manner, whereas the expression levels of Pref-1, C/EBP-δ and GR are not affected[1]. ***In Vivo:*** When high-fat diet (HFD) fed mice for 8 weeks, body and epididymal fat weight gains are significantly increased compared to those of low-fat diet (LFD)-fed mice. However, when Ginsenoside Rh1 is treated in HFD-fed mice, body and epididymal fat weight gains are significantly decrease compared with those of the HFD-fed mice. TG, glucose, insulin, total cholesterol, and HDL levels in the blood are significantly increased in HFD-fed mice group compared to LFD-fed mice group. Treatment with Ginsenoside Rh1 in HFD-fed mice significantly lowers TG level alone[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]**Mouse embryo fibroblasts 3T3-L1 cells** are incubated in DMEM, containing 10% FBS and 1% AB, at 37°C and 5.6% CO2 atmosphere. To induce differentiation, two days after confluence, preadipocytes (designated day 0) are cultured in the differentiation medium (DM), which is consisted of DMEM, 10% FBS, 1% AB, and DMI (0.28 unit/mL insulin, 0.5 mM Isobutylmethylxanthine and 1 μM Dexamethasone) for 2 d in the presence or absence of **50 μM and 100 μM of Ginsenoside Rh1**, and switched to DM containing 10% FBS and 10 μg/mL insulin and then changed to DMEM medium with 10% FBS for every 2 d[1].

Animal Administration: [1]Mice[1]

Male C57BL/6J mice are separated into 3 groups, LFD, HFD and HFDRh1. Each group is consisted of ten mice. LFD group fed LFD for 8 weeks. HFD group fed HFD for 8 weeks. HFDRh1 group fed HFD diet for 4 weeks and then simultaneously treated with HFD and **20 mg/kg/d Ginsenoside Rh1, which is orally administrated**. Weight and food intake of mice are measured daily. After finishing treatment for 4 weeks, blood and epididymal fats are collected for further analysis.

References:

[1]. Gu W, et al. Ginsenoside Rh1 ameliorates high fat diet-induced obesity in mice by inhibiting adipocyte differentiation. Biol Pharm Bull. 2013;36(1):102-7.

CAIndexNames:

β-D-Glucopyranoside, (3β,6α,12β)-3,12,20-trihydroxydammar-24-en-6-yl

SMILES:

C[C@@]([C@@]12C)(C[C@H](O[C@]([C@@H]([C@@H](O)[C@@H]3O)O)([H])O[C@@H]3CO)[C@@]4([H])C5(C)C)[C@@](C[C@@H](O)[C@]1([H])[C@]([C@@](C)(O)CC/C=C(C)/C)([H])CC2)([H])[C@]4(CC[C@@H]5O)C

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

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