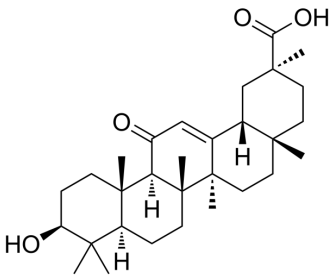
18β-Glycyrrhetinic acid

Cat. No.:	HY-N0180
CAS No.:	471-53-4
Molecular Formula:	$C_{30}H_{46}O_4$
Molecular Weight:	470.68
Target:	Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 1 year -20°C 6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (265.57 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1246 mL	10.6229 mL	21.2459 mL
	5 mM	0.4249 mL	2.1246 mL	4.2492 mL
	10 mM	0.2125 mL	1.0623 mL	2.1246 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 25 mg/mL (53.11 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.17 mg/mL (4.61 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.17 mg/mL (4.61 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

18β-Glycyrrhetinic acid is the major bioactive component of Glycyrrhiza uralensis and possesses anti-ulcerative, anti-inflammatory and antiproliferative properties.

In Vitro

18β-Glycyrrhetinic acid is the major bioactive component of Glycyrrhizae Radix and possesses anti-ulcerative, anti-inflammatory and antiproliferative properties. MTS assay demonstrates that 24 h treatment of 18β-Glycyrrhetinic acid suppresses cell proliferation in both cell lines in a dose-dependent manner. 18β-Glycyrrhetinic acid at 160 μM significantly decreases the percentage of viable cells to around 40.5±10.5% in A549 and 38.3±4.6% in NCI-H460 ($p<0.01$ respectively).

	When the cells are treated with 320 μM 18β-Glycyrrhetinic acid, a greater inhibitory effects on cell proliferation is shown, as the percentage of viable cells is below 30% compare with untreated controls ($p<0.001$). Treatment with 18β-Glycyrrhetinic acid at 160 μM and 320 μM decreases the levels of full-length PARP and increases the levels of cleaved-PARP[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Rats in 18β-Glycyrrhetinic acid+Triptolide (TP) group which receive low-dose 18β-Glycyrrhetinic acid (50 mg/kg) have significant reductions in the three serum parameters when compare with TP rats. Rats in 18β-Glycyrrhetinic acid+TP group which receive the high-dose 18β-Glycyrrhetinic acid (100 mg/kg) have slightly lowered the levels of three liver enzymes, the reductions do not reach statistical significance compare with TP group. Contrastingly, preadministration of low-dose 18β-Glycyrrhetinic acid protects animals from TP-induced hepatic lesions. On the contrary, low-dose 18β-Glycyrrhetinic acid (50 mg/kg) markedly suppresses the release of the four cytokines above[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Primary microglia cultures are used in this study. For treatment assay, microglia are incubated with complete DMEM and stimulated with or without 100 ng/mL IFN-γ in the presence or absence of 18β-Glycyrrhetinic acid (25 μM and 50 μM) at 37°C in a humidified incubator with 5% CO_2. For cell migration assay, the isolated primary microglia that seeded in complete DMEM medium are stimulated with or without IFN-γ (100 ng/mL), and treated with different doses of 18β-Glycyrrhetinic acid , 24 h later, the microglia culture supernatants are collected and added to the lower chambers of Transwell inserts[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Healthy Wistar rats (male, 200±20 g) are used and divided into five groups with 10 individuals for each group randomly. Animals in normal control (NC) group receive distilled water for 6 days and 0.5% CMC-Na for the last 3 days. Rats in Triptolide model group (TP), 18β-Glycyrrhetinic acid low-dose group (GAL+TP), and 18β-Glycyrrhetinic acid high-dose group (GAH+TP) receive distilled water, 18β-Glycyrrhetinic acid (50 mg/kg, p.o., dissolved in distilled water), or 18β-Glycyrrhetinic acid (100 mg/kg, p.o., dissolved in distilled water) for consecutive 6 days, respectively, and liver injury is induced by TP (2.4 mg/kg, p.o., suspended in 0.5% CMC-Na) for the last 3 days. Animals in the above three groups receive TP 6 hours after distilled water or 18β-Glycyrrhetinic acid treatment on the last 3 days[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2024 Jan 6:e2305260.
- Chemosphere. 2023 Feb 24;138249.
- Cell Prolif. 2023 May 4;e13494.
- Environ Toxicol. 2022 Aug 3.
- World J Gastroenterol. 2023 Jun 21; 29(23): 3622-3644.

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REFERENCES

[1]. Huang RY, et al. 18β-Glycyrrhetinic acid suppresses cell proliferation through inhibiting thromboxane synthase in non-small cell lung cancer. PLoS One. 2014 Apr 2;9(4):e93690.

[2]. Zhou J, et al. 18β-glycyrrhetinic acid suppresses experimental autoimmune encephalomyelitis through inhibition of microglia activation and promotion of remyelination. Sci Rep. 2015 Sep 2;5:13713.

[3]. Yang G, et al. Protective Effect of 18β-Glycyrrhetinic Acid against Triptolide-Induced Hepatotoxicity in Rats. Evid Based Complement Alternat Med. 2017;2017:3470320.

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