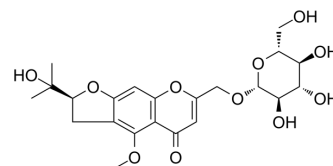


Prim-O-glucosylcimifugin

Cat. No.:	HY-N0635
CAS No.:	80681-45-4
Molecular Formula:	C ₂₂ H ₂₈ O ₁₁
Molecular Weight:	468.45
Target:	NO Synthase; COX
Pathway:	Immunology/Inflammation
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro	DMSO : ≥ 150 mg/mL (320.20 mM) * "≥" means soluble, but saturation unknown.				
	Preparing Stock Solutions	<div>Solvent Concentration</div> <div>Mass</div>	1 mg	5 mg	10 mg
		1 mM	2.1347 mL	10.6735 mL	21.3470 mL
		5 mM	0.4269 mL	2.1347 mL	4.2694 mL
		10 mM	0.2135 mL	1.0673 mL	2.1347 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: ≥ 2.08 mg/mL (4.44 mM); Clear solution				
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.08 mg/mL (4.44 mM); Suspended solution; Need ultrasonic				
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (4.44 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Prim-O-glucosylcimifugin exerts anti-inflammatory effects through the inhibition of iNOS and COX-2 expression by through regulating JAK2/STAT3 signaling.	
IC ₅₀ & Target	iNOS	COX-2
In Vitro	Prim-O-glucosylcimifugin (POG) is the highest content chromone and one of the major active constituents in Radix Saposhnikovia (RS). Prim-O-glucosylcimifugin exerts anti-inflammatory effects in RAW 264.7 macrophages through the inhibition of iNOS and COX-2 expression by inhibiting JAK2/STAT3 signaling. The cytotoxicity of Prim-O-glucosylcimifugin is	

measured to LPS-activated Raw 264.7 macrophages. Raw 264.7 macrophages are treated with LPS (1 µg/mL) and increasing concentrations of Prim-O-glucosylcimifugin (15, 50, and 100 µg/mL) for 24 h and cell viability is evaluated by CCK-8 assay. Cell viability is not significantly affected after 24 h and exposure to 15-100 µg/mL Prim-O-glucosylcimifugin as compared with DMSO-treated cells (control). To investigate the anti-inflammatory effect of Prim-O-glucosylcimifugin, whether Prim-O-glucosylcimifugin can affect NO synthesis is examined in LPS-activated RAW 264.7 cells. Macrophages are treated with LPS (1 µg/mL) and various concentrations of Prim-O-glucosylcimifugin (15, 50, and 100 µg/mL) for 24 h. NO concentrations are measured in the culture supernatants by Griess reaction. The concentrations of NO in the culture supernatants are markedly increased in response to LPS exposure, and Prim-O-glucosylcimifugin significantly inhibits LPS-induced NO production in a concentration-dependent manner^[1].

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

In Vivo

Bronchoalveolar lavage fluid (BALF) is collected at 7 h after lipopolysaccharide (LPS) administration and the cytokine levels in BALF are measured by ELISA. The levels of TNF-α, IL-1β and IL-6 in BALF are increased dramatically compared with control group. However, pretreatment with Prime-O-glucosylcimifugin (2.5, 5 or 10 mg/kg) significantly down-regulates the levels of TNF-α, IL-1β and IL-6 in a dose-dependent manner (P<0.05, P<0.01)^[1].

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

PROTOCOL

Cell Assay ^[1]

Cell Counting Kit (CCK-8) is used to determine the cytotoxic concentrations of Prim-O-glucosylcimifugin. In brief, the Raw 264.7 cells are plated at a density of 1×10^4 cells per well in a 96-well and incubated overnight. Cells are then stimulated with 1 µg/mL LPS and treated with various concentrations of Prim-O-glucosylcimifugin (15, 50, and 100 µg/mL; MedChem Express, Princeton, NJ, USA) or DMSO. After incubation at 37°C for 24 h, CCK-8 solution is added to each well and incubated for another 1 h. The absorbance is measured at 450 nm using a microplate reader^[1].

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

Animal Administration ^[1]

Mice^[1]

BALB/c male mice, 8 weeks old and weighing approximately 18 to 20 g, are used. The mice are randomly divided into five groups: Control group; LPS group; LPS+Prime-O-glucosylcimifugin (2.5, 5 or 10 mg/kg bodyweight). Prime-O-glucosylcimifugin is given intraperitoneally. One hour later, LPS group and LPS+Prime-O-glucosylcimifugin group mice are given 50 µL LPS intranasally (i.n) (200 mg/L) to induce acute lung injury. Control mice are given 50 µL PBS intranasally (i.n) without LPS^[1].

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

CUSTOMER VALIDATION

- Phytomedicine. 1 September 2022, 154427.
- Pharmacogn Mag. 2017 Jul-Sep;13(51):378-384.

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REFERENCES

- [1]. Zhou J, et al. Prim-O-glucosylcimifugin Attenuates Lipopolysaccharide-induced Inflammatory Response in RAW 264.7 Macrophages. Pharmacogn Mag. 2017 Jul-Sep;13(51):378-384.
- [2]. Chen N, et al. Prime-O-glucosylcimifugin attenuates lipopolysaccharide-induced acute lung injury in mice. Int Immunopharmacol. 2013 Jun;16(2):139-47.

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

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