Hydrocotarnine

Cat. No.: HY-W176629

CAS No.: 550-10-7

Molecular Formula: $C_{12}H_{15}NO_3$

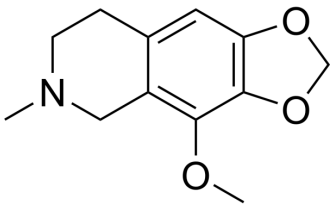
Molecular Weight: 221.25

Target: E1/E2/E3 Enzyme; Interleukin Related

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation

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 : ≥ 100 mg/mL (451.98 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent, Mass	1 mg	5 mg	10 mg
1 mM	4.5198 mL	22.5989 mL	45.1977 mL
5 mM	0.9040 mL	4.5198 mL	9.0395 mL
10 mM	0.4520 mL	2.2599 mL	4.5198 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: ≥ 2.5 mg/mL (11.30 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.30 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hydrocotarnine is a Cbl inhibitor, and results in inflammasome-mediated IL-18 secretion in colitis. Hydrocotarnine increases expression of GLUT1 and cellular glucose uptake in glycolytic metabolism. Hydrocotarnine acts as an agent that provides analgesic effect in cancer research[1][2][3].

IC_{50} & Target

Cbl[1][2]

In Vitro

Hydrocotarnine is an analgesic agent (CRIN-2), with the patent ID of WO2011160016A2[1].
Hydrocotarnine (10 μM; 1 h) elevates the secretion of IL-1β and IL-18, and (0.1-10 μM; 1 h) increases the global level of tyrosine-phosphorylated proteins in THP-1 cells[1].
Hydrocotarnine (50 μM; 0-100 min) increases the glycolytic capacity and glycolytic reserve capacity in THP-1-derived macrophages[2].
Hydrocotarnine (50 μM; 16 h) inhibits Cbl and increases the total GLUT1 protein in THP-1-derived macrophages[2].
Hydrocotarnine is known to enhance the analgesic effect of opioids, and alleviates cancer pain[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	THP-1 cells
Concentration:	0.1, 1, 10 μM
Incubation Time:	1 hour
Result:	Induced p-Pyk2 loss and increased the level of tyrosine-phosphorylated proteins in a dose-dependent manner.

In Vivo

Hydrocotarnine (10 mg/kg/d; i.p.; 9 d) shows inhibitory effect on Cbl and results in increasing IL-18 levels, indicating that NLRP3 inflammasome activation is enhanced in mice[1].
Hydrocotarnine (10 mg/kg/d; i.p.; 9 d) protects mice from DSS-induced colitis, with low scores of pathological evaluation of inflammation, epithelial defects, and crypt atrophy[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	DSS-induced colitis model in C57BL/6 mice (6-9 weeks old)[1]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; once daily; 9 days while 2.5% DSS treatment began on day 1 and ended on day 7
Result:	Significantly attenuated the weight loss of DSS-induced colitis mice compared to PBS-treated control mice, indicating that decreasing negative regulation of the NLRP3 inflammasome activation could attenuate colitis in an animal model.

REFERENCES

[1]. Chung IC, et al. Src-family kinase-Cbl axis negatively regulates NLRP3 inflammasome activation. Cell Death Dis. 2018 Oct 31;9(11):1109.

[2]. Lin HC, et al. Cbl Negatively Regulates NLRP3 Inflammasome Activation through GLUT1-Dependent Glycolysis Inhibition. Int J Mol Sci. 2020 Jul 19;21(14):5104.

[3]. Kim KU, et al. DITMD-induced mitotic defects and apoptosis in tumor cells by blocking the polo-box domain-dependent functions of polo-like kinase 1. Eur J Pharmacol. 2019 Mar 15;847:113-122.

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