Dehydrocorydaline chloride

Cat. No.: HY-N0674A

CAS No.: 10605-03-5

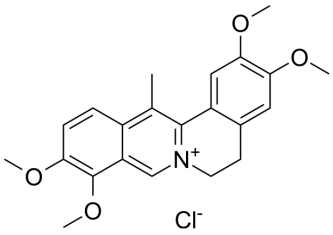
Molecular Formula: $C_{22}H_{24}ClNO_4$

Molecular Weight: 401.88

Target: p38 MAPK; Autophagy; Bcl-2 Family; Caspase; PARP; Parasite

Pathway: MAPK/ERK Pathway; Autophagy; Apoptosis; Cell Cycle/DNA Damage; Epigenetics; Anti-infection

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (62.21 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4883 mL	12.4415 mL	24.8830 mL
	5 mM	0.4977 mL	2.4883 mL	4.9766 mL
	10 mM	0.2488 mL	1.2442 mL	2.4883 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 (6.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.22 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dehydrocorydaline chloride (13-Methylpalmatine chloride) is an alkaloid that regulates protein expression of Bax, Bcl-2; activates caspase-7, caspase-8, and inactivates PARP[1]. Dehydrocorydaline chloride elevates p38 MAPK activation. Anti-inflammatory and anti-cancer activities[2]. Dehydrocorydaline chloride shows strong anti-malarial effects (IC_{50} =38 nM), and low cytotoxicity (cell viability > 90%) using P. falciparum 3D7 strain[3].

IC_{50} & Target

Plasmodium

In Vitro

Treatment of C2C12 myoblasts with 500 nM Dehydrocorydaline increases the expression levels of muscle-specific proteins,

	including MyoD, myogenin and myosin heavy chain. Treatment with Dehydrocorydaline elevates p38 MAPK activation and the interaction of MyoD with an E protein. Furthermore, defects in differentiation-induced p38 MAPK activation and myoblast differentiation induced by depletion of the promyogenic receptor protein Cdo in C2C12 myoblasts are restored by Dehydrocorydaline treatment[2]. Dehydrocorydaline significantly inhibits MCF-7 cell proliferation in a dose- dependent manner, which can be reversed by a caspase-8 inhibitor, Z-IETD-FMK. Dehydrocorydaline increases DNA fragments without affecting ΔΨm. Western blotting assay shows that dehydrocorydaline dose-dependently increases Bax protein expression and decreases Bcl-2 protein expression. Furthermore, dehydrocorydaline induces activation of caspase-7,-8 and the cleavage of PARP without affecting caspase-9. These results show that dehydrocorydaline inhibits MCF-7 cell proliferation by inducing apoptosis mediated by regulating Bax/Bcl-2, activating caspases as well as cleaving PARP[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Dehydrocorydaline (3.6, 6 or 10 mg/kg, i.p.) shows a dose-dependent antinociceptive effect in the acetic acid-induced writhing test and significantly attenuates the formalin-induced pain responses in mice. In the formalin test, dehydrocorydaline decreases the expression of caspase 6 (CASP6), TNF-α, IL-1β and IL-6 proteins in the spinal cord. These findings confirm that Dehydrocorydaline has antinociceptive effects in mice[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Briefly, MCF-7 cells (1×10^4 cells/well) are seeded in 96-well plates and treated with different concentrations of dehydrocorydaline (0-200 μM) for 24 h. The cell viability is determined. To explore the role of caspase-8 in dehydrocorydaline induced cytotoxicity, a caspase-8 inhibitor Z-IETD-FMK (10 μM) is co-incubated with 200 μM dehydrocorydaline. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Briefly, the mice are placed individually in glass beakers and are allowed to acclimate for 30 min before the test. The vehicle or Dehydrocorydaline (3.6, 6 or 10 mg/kg) are injected (10 ml/kg, i.p.) 15 min prior to the formalin injection. Morphine (10 mg/kg) or diclofenac sodium (20 mg/kg) are injected 15 and 30 min, respectively, prior to the formalin injection as positive controls. Then, 25 μL of a 5% formalin solution is injected into the plantar surface of the right hind paw of each mouse. Immediately after the formalin injection, the mice are placed individually in the beakers, and a mirror is placed under the beaker to allow clear observation of the paws of the animals. The time that the animals spent on biting/licking the injected paw is measured with a stopwatch every 5 min and considered as indication of nociception. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Phytomedicine. 8 September 2021, 153740.
- J Agric Food Chem. 2023 Oct 12.
- Front Pharmacol. 31 May 2021.
- J Cell Physiol. 2019 Dec;234(12):22463-22476.
- Aging (Albany NY). 2021 Oct 7;13(19):23133-23148.

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REFERENCES

[1]. Yoo M, et al. Dehydrocorydaline promotes myogenic differentiation via p38 MAPK activation. Mol Med Rep. 2016 Oct;14(4):3029-36.

[2]. Yin ZY, et al. Antinociceptive effects of dehydrocorydaline in mouse models of inflammatory pain involve the opioid receptor and inflammatory cytokines. Sci Rep. 2016 Jun 7;6:27129

[3]. Xu Z, et al. Dehydrocorydaline inhibits breast cancer cells proliferation by inducing apoptosis in MCF-7 cells. Am J Chin Med. 2012;40(1):177-85.

[4]. Nonaka M, et al. Screening of a library of traditional Chinese medicines to identify anti-malarial compounds and extracts. Malar J. 2018 Jun 25;17(1):244.

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