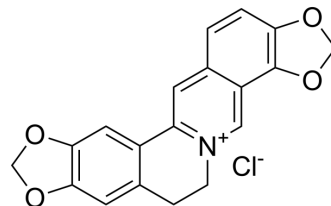


Coptisine chloride

Cat. No.:	HY-N0736
CAS No.:	6020-18-4
Molecular Formula:	C ₁₉ H ₁₄ ClNO ₄
Molecular Weight:	355.77
Target:	Indoleamine 2,3-Dioxygenase (IDO); Bacterial; Influenza Virus
Pathway:	Metabolic Enzyme/Protease; 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 : 10.42 mg/mL (29.29 mM; ultrasonic and warming and heat to 60°C)					
	Preparing Stock Solutions	<div><div>Solvent</div><div>Concentration</div></div>	Mass	1 mg	5 mg	10 mg
		1 mM	2.8108 mL	14.0540 mL	28.1080 mL	
		5 mM	0.5622 mL	2.8108 mL	5.6216 mL	
		10 mM	0.2811 mL	1.4054 mL	2.8108 mL	
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 1.04 mg/mL (2.92 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 0.5 mg/mL (1.41 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Coptisine chloride is an alkaloid from Chinese goldthread, and acts as an efficient uncompetitive IDO inhibitor with a K _i value of 5.8 μM and an IC ₅₀ value of 6.3 μM. Coptisine chloride is a potent H1N1 neuraminidase (NA-1) inhibitor with an IC ₅₀ of 104.6 μg/mL and can be used for influenza A (H1N1) infection.	
IC ₅₀ & Target	IDO 6.3 μM (IC ₅₀)	IDO 5.8 μM (K _i)
In Vitro	Coptisine chloride is an efficient uncompetitive IDO inhibitor with a K _i value of 5.8 μM and an IC ₅₀ value of 6.3 μM ^[1] . Coptisine (0.1-100 μM) inhibits the proliferation of A549, H460, H2170, MDA-MB-231 and HT-29 cells, with IC ₅₀ s of 18.09, 29.50, 21.60, 20.15 and 26.60 μM, respectively. Coptisine (12.5, 25, 50 μM) upregulates the expression of pH2AX and p21, reduces expression of cyclin B1, cdc2, and cdc25C, and induces G2/M arrest and apoptosis of A549 cells in a concentration-	

dependent manner. Coptisine (12.5, 25, 50 μ M) also induces mitochondrial dysfunction and activates caspases activities in A549 cells. Furthermore, Coptisine (50 μ M) increases ROS levels in a time-dependent manner (0.5, 1, 2, 4, 12, and 24 h)^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Coptisine shows increased toxicity in mice in a concentration dependent manner, with LD₅₀ value of 880.18 mg/kg. Coptisine (154 mg/kg/day, 90 days) shows no toxicity on SD rats. Coptisine (23.35, 46.7, 70.05 mg/kg, p.o.) dose-dependently decreases the levels of TC, TG, and LDL-c and increases HDL-c content in serum of hamsters to different degree, slows down weight gain induced by the HFHC diet, and raises the level of cholesterol and TBA in feces dose-dependently in hamsters. Coptisine (70.05 mg/kg, p.o.) suppresses HMGCR protein expression level and induces the protein expression of SREBP-2, LDLR, and CYP7A1 involved in cholesterol metabolism^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay ^[3]

A 100-mM concentration of Coptisine is dissolved in DMSO, and subsequent concentrations ranging between 100 and 0.1 μ M are prepared by diluting with cell culture medium. The final DMSO concentration used is less than 0.1% in every treatment. MTT assay is performed to assess cell proliferation effect of Coptisine. Briefly, 2500 cells/well are seeded in 96-well plate containing DMEM medium supplemented with 10% FBS and 1% penicillin-streptomycin. A series of Coptisine concentrations are added and incubated for 48 h in the presence or absence of 5-mM NAC. After 48 h of incubation, 15 μ L of MTT (5 mg/mL) is added to each well and incubated at 37°C for 4 h. Then, the supernatant is removed and 150 μ L of DMSO is added to each well to dissolve the crystals. The absorbance is measured at 595 nm by Spectramax M4 plate reader^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration ^[2]

Mice^[2]
The acute toxicity of Coptisine is tested on Kunming mice, 10 mice in each group (half each males and females). Coptisine is dissolved in distilled water and prepared for administration with eight doses (482.5, 579, 694, 833, 1,000, 1,200, 1,440, and 1,728 mg/kg). After oral administration, the reactions of each mouse including mortality are observed and recorded for 1 week to obtain the LD₅₀ value of Coptisine.

Rats^[2]
Forty SD rats are divided into the control and Coptisine groups (half each males and females) which are treated for 90 days. The animal dose is calculated by the human equivalent dose (HED) with the body surface area (BSA) normalization method. To determine the sub-chronic toxicity, the actual dosage of Coptisine on SD rats (154 mg/kg/day) is obtained using the maximum HED of Coptisine (25 mg/kg) for an adult (60 kg) as a reference. Rats in the control group are given the same volume of 0.9 % saline. In the whole experiment, all animals are fed a normal diet and water ad libitum. The general appearance and behavior of rats are recorded daily, their body weight is measured every 10 days, and clinical signs and mortality are observed twice daily. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int Immunopharmacol. 2024 Jan 4;128:111433.
- DNA Cell Biol. 2020 Oct 2.
- J Oncol. 26 Jun 2022.

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REFERENCES

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- [1]. Yu D, et al. The IDO inhibitor coptisine ameliorates cognitive impairment in a mouse model of Alzheimer's disease. J Alzheimers Dis. 2015;43(1):291-302.
- [2]. He K, et al. The safety and anti-hypercholesterolemic effect of coptisine in Syrian golden hamsters. Lipids. 2015 Feb;50(2):185-94.
- [3]. Rao PC, et al. Coptisine-induced cell cycle arrest at G2/M phase and reactive oxygen species-dependent mitochondria-mediated apoptosis in non-small-cell lung cancer A549 cells. Tumour Biol. 2017 Mar;39(3):1010428317694565.
- [4]. Zhou X, et al. Inhibition activity of a traditional Chinese herbal formula Huang-Lian-Jie-Du-Tang and its major components found in its plasma profile on neuraminidase-1. Sci Rep. 2017 Nov 14;7(1):15549.
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