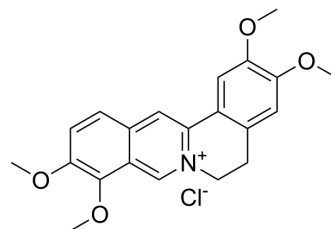


Palmatine chloride

Cat. No.:	HY-N0110
CAS No.:	10605-02-4
Molecular Formula:	C ₂₁ H ₂₂ ClNO ₄
Molecular Weight:	387.86
Target:	Indoleamine 2,3-Dioxygenase (IDO); Virus Protease; Apoptosis; Aurora Kinase; Bacterial; Parasite
Pathway:	Metabolic Enzyme/Protease; Anti-infection; Apoptosis; Cell Cycle/DNA Damage; Epigenetics
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 1 year; -20°C, 6 months (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 32 mg/mL (82.50 mM)
 H₂O : 5 mg/mL (12.89 mM; Need ultrasonic)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	2.5782 mL	12.8912 mL	25.7825 mL
	5 mM	0.5156 mL	2.5782 mL	5.1565 mL
	10 mM	0.2578 mL	1.2891 mL	2.5782 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (6.45 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (6.45 mM); Clear solution
- Add each solvent one by one: PBS
Solubility: 2.5 mg/mL (6.45 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

Palmatine chloride is an orally active and irreversible indoleamine 2,3-dioxygenase 1 (IDO-1) inhibitor with IC₅₀s of 3 μM and 157 μM against HEK 293-hIDO-1 and rhIDO-1, respectively. Palmatine chloride can also inhibit West Nile virus (WNV) NS2B-NS3 protease in an uncompetitive manner with an IC₅₀ of 96 μM. Palmatine chloride shows anti-cancer, anti-oxidation, anti-inflammatory, neuroprotection, antibacterial, anti-viral activities^{[1][2][3][4][5]}.

IC ₅₀ & Target	IDO-1 3 μM (IC ₅₀ , HEK 293-hIDO-1)	IDO-1 157 μM (IC ₅₀ , rhIDO-1)	WNV NS2B-NS3 96 μM (IC ₅₀)
In Vitro	<p>Palmatine (0-100 μM; 42 h) suppresses WNV with an EC₅₀ value of 3.6 μM, and reduce the viral titers of DENV-2 and YFV with EC₅₀ values of 26.4 μM and 7.3 μM, respectively^[3].</p> <p>?Palmatine (0-1128 μM; 24-72 h) inhibits colon cancer cell proliferation^[5].</p> <p>?Palmatine (0-704 μM; 24 h) reduces AURKA protein levels, induces G2/M phase arrest, and induces apoptosis in colon cancer cells via the mitochondrial associated pathway^[5].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Proliferation Assay^[5]</p>		
	Cell Line:	HCT-116, SW480, HT-29	
	Concentration:	0, 88, 176, 352, and 704 μM (HCT-116, SW480); 0, 141, 282, 564, and 1128 μM (HT-29)	
	Incubation Time:	24, 48 and 72 h	
	Result:	Decreased cell viability in a dose-dependent manner.	
	Western Blot Analysis ^[5]		
	Cell Line:	HCT-116, SW480, HT-29	
	Concentration:	100 nM for HCT-116, 500 nM for SW480 and HT-29	
	Incubation Time:	24 h	
	Result:	Promoted the expression of apoptosis markers such as P53 / P73, Caspase3, and Caspase9. Reduced AURKA protein levels. Increased cyt. c in the cytoplasm while reduced Bcl2 and Bcl-xl in a dose-dependent manner.	
	Cell Cycle Analysis ^[5]		
	Cell Line:	HCT-116, SW480	
Concentration:	88, 176, 352 and 704 μM		
Incubation Time:	24 h		
Result:	Induced G2/M phase arrest in a dose-dependent manner.		
Apoptosis Analysis ^[5]			
Cell Line:	HCT-116, SW480		
Concentration:	88, 176, 352 and 704 μM		
Incubation Time:	24 h		
Result:	Induced apoptosis in a dose-dependent manner.		
In Vivo	<p>Palmatine (50 or 100 mg/kg; p.o.; daily for 7 days) ameliorates DSS (dextran sulfate sodium)-induced colitis and prevents infiltration of inflammatory cells^[1].</p> <p>?Palmatine (0-200 mg/kg; i.p.; once) attenuates D-galactosamine/Lipopolysaccharides (HY-D1056)-induced fulminant hepatic failure in mice^[2].</p> <p>?Palmatine (0-1 mg/kg; i.p.; 10 days) shows memory-enhancing activity in mice^[4].</p> <p>?Palmatine (33.75-135 mg/kg; p.o.; daily for 26 days) can effectively inhibit the growth of HCT-116 xenografts in mice^[5].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		

Animal Model:	DSS- induced Colitis BALB/c mice model (8-week-old) ^[1]
Dosage:	50 or 100 mg/kg
Administration:	Orally, daily, for 7 days
Result:	Ameliorated DSS-induced colitis and prevented infiltration of inflammatory cells; remarkably extended the colon length; significantly suppressed the colonic MPO activity. Decreased the levels of colonic inflammatory cytokines (TNF- α , IFN- γ , IL-1 β , IL-6, IL-4 and IL-10); Protected mucosal integrity by modulating TJs protein and apoptosis proteins; Restored DSS-induced decreases of TJ protein ZO-1, ZO-2 and claudin-1; Reduced Bax expression and enhanced Bcl-2 expression at the dose of 100 mg/kg, prevented epithelial apoptosis and improved intestinal integrity. Prevented DSS-induced changes of gut microbiota in colitis mice.
Animal Model:	Male ICR mice (20–22 g), D-galactosamine/lipopolysaccharide (GalN/LPS)-induced fulminant hepatic failure model ^[2]
Dosage:	25, 50, 100, or 200 mg/kg
Administration:	Intraperitoneal injection, 1 h before the GalN/LPS treatment
Result:	Attenuated the mortality and serum aminotransferase activities increased by GalN/LPS. Prevented the increase of serum TNF- α and augmented that of serum IL-10. Decreased the TNF- α mRNA expression and increased the IL-10 mRNA expression. Attenuated the apoptosis of hepatocytes.
Animal Model:	Swiss young male albino mice, with Scopolamine (HY-N0296)- and diazepam-induced amnesia model ^[4]
Dosage:	0.1, 0.5, 1 mg/kg
Administration:	Intraperitoneal injection, 10 days
Result:	Significantly improved learning and memory of mice at 0.5 and 1 mg/kg and did not show any significant effect on locomotor activity of the mice. Significantly reversed scopolamine- and diazepam-induced amnesia in mice. Significantly reduced brain acetylcholinesterase activity of mice.
Animal Model:	BALB/c-nude mice, HCT-116 xenograft model ^[5]
Dosage:	33.75, 67.5 and 135 mg/kg
Administration:	Oral administration, once a day for 26 days
Result:	The tumor volume and weight of the treatment group were significantly reduced.

CUSTOMER VALIDATION

- Biol Res. 2020 Sep 14;53(1):39.

- Int Immunopharmacol. 2022 Feb 9;106:108583.
- J Ethnopharmacol. 2023 Sep 27:117238.
- Drug Dev Res. 2022 Aug 17.
- Oxid Med Cell Longev. 2021 Mar 13.

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 - [2]. Jia F, et al. Identification of palmatine as an inhibitor of West Nile virus. Arch Virol. 2010 Aug;155(8):1325-9.
 - [3]. Dhingra D, et al. Memory-enhancing activity of palmatine in mice using elevated plus maze and morris water maze. Adv Pharmacol Sci. 2012;2012:357368.
 - [4]. Liu X, et al. Palmatine induces G2/M phase arrest and mitochondrial-associated pathway apoptosis in colon cancer cells by targeting AURKA. Biochem Pharmacol. 2020 May;175:113933.
 - [5]. Long J, et al. Palmatine: A review of its pharmacology, toxicity and pharmacokinetics. Biochimie. 2019 Jul;162:176-184.
 - [6]. Zhang XJ, et al. Palmatine ameliorated murine colitis by suppressing tryptophan metabolism and regulating gut microbiota. Pharmacol Res. 2018 Nov;137:34-46.
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