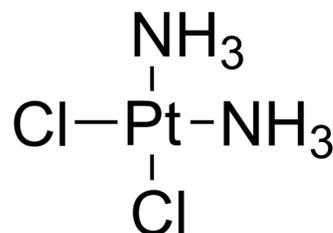


Cisplatin

Cat. No.:	HY-17394
CAS No.:	15663-27-1
Molecular Formula:	Cl ₂ H ₆ N ₂ Pt
Molecular Weight:	300.05
Target:	DNA Alkylator/Crosslinker; Ferroptosis; Autophagy
Pathway:	Cell Cycle/DNA Damage; Apoptosis; Autophagy
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro	DMF : 14.17 mg/mL (47.23 mM; Need ultrasonic and warming; DMSO can inactivate Cisplatin's activity)					
	H ₂ O : 1 mg/mL (3.33 mM; ultrasonic and warming and heat to 60°C; DMSO can inactivate Cisplatin's activity)					
	Preparing Stock Solutions	Solvent	Mass	1 mg	5 mg	10 mg
		Concentration				
		1 mM		3.3328 mL	16.6639 mL	33.3278 mL
5 mM			0.6666 mL	3.3328 mL	6.6656 mL	
10 mM		0.3333 mL	1.6664 mL	3.3328 mL		
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: 0.5% CMC-Na/saline water Solubility: 20 mg/mL (66.66 mM); Suspended solution; Need ultrasonic					
	2. Add each solvent one by one: 50% PEG300 >> 50% saline Solubility: 10 mg/mL (33.33 mM); Suspended solution; Need ultrasonic					
	3. Add each solvent one by one: PBS Solubility: 0.91 mg/mL (3.03 mM); Clear solution; Need ultrasonic and warming and heat to 60°C					

BIOLOGICAL ACTIVITY

Description	Cisplatin (CDDP) is an antineoplastic chemotherapy agent by cross-linking with DNA and causing DNA damage in cancer cells. Cisplatin activates ferroptosis and induces autophagy ^{[1][2][3]} .
IC₅₀ & Target	DNA Alkylator/Crosslinker ^[1]
In Vitro	Cisplatin (CDDP) causes apoptosis of HeLa cells in a dose-dependent manner, with a concentration of 30 μM Cisplatin resulting in death of greater than 90% of the cell population by 24 h of treatment. The kinetics of Cisplatin-induced apoptosis are examined using a 30 μM concentration. Cisplatin Activates the MEK/ERK Signaling Pathway, 20 and 30 μM

Cisplatin, both of which results in significant apoptosis, leads to strong activation of ERK^[1].

Cisplatin (50 μ M) produces time-dependent apoptosis in renal proximal tubular cell (RPTCs), causing cell shrinkage, a 50-fold increase in caspase 3 activity, a 4-fold increase in phosphatidylserine externalization, and 5- and 15-fold increases in chromatin condensation and DNA hypoploidy, respectively^[2].

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

In Vivo

In melanoma-bearing mice, Cisplatin (CDDP; 4 mg/kg B.W.) reduces the size and weight of the solid tumors, and HemoHIM supplementation with Cisplatin enhances the decrease of both the tumor size and weight^[3].

Cisplatin administration results in significant increases in the kidney weight as a percentage of the total body weight, urine volume, serum creatinine, and blood urea nitrogen by about 132, 315, 797, and 556% in comparison with the control rats, respectively^[4].

Induction of Acute Kidney Injury (AKI)^{[7][8]}

- Background

The pathogenesis of Cisplatin-induced acute kidney injury (AKI) is complex and Oxidative stress, mitochondrial dysfunction, inflammation, and apoptosis are all participating in the progression of Cisplatin-induced AKI. Oxidative stress is a predominant mechanism of injury in cisplatin-induced AKI.

- Specific Modeling Methods

Mice: C57BL/6 • male • 6-week-old (period: 2 weeks)

Administration: 5 mg/kg • ip • once daily for 2 weeks

Note

(1) Suggest using male mice as female mice are more resistant to renal injury.

(2) Mice can be deprived of food and water for 18 h prior to induction, and food and water are returned after administration.

(3) Cisplatin can be dissolved in sterile saline solution to prepare injection working solution while protecting from light.

- Modeling Indicators

Molecular changes: Increased indicators: Serum creatinine (SCr) levels, blood urea nitrogen (BUN) levels, neutrophil gelatinase-associated lipocalin (NGAL), 3-nitrotyrosine.

- Correlated Product(s): 84-B10 (HY-44307)

- Opposite Product(s):

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

PROTOCOL

Cell Assay ^[1]

HeLa and A549 cells are maintained in Dulbecco's modified Eagle's medium supplemented with 10% fetal bovine serum, 100 units of Penicillin, and 100 µg of Streptomycin/mL. They are cultured at 37°C in a humidified chamber containing 5% CO₂. For the induction of apoptosis, cells are plated in 60-mm dishes 1 day prior to Cisplatin (0-30 µM) treatment^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration ^{[3][4]}

Mice^[3]

Mice are divided randomly into three groups (Control, Cisplatin and Cisplatin+HemoHIM), and each group consists of twenty mice. B16F0 melanoma (5×10⁵ cells/mouse) is inoculated into subcutaneous femoral left region of mice at 3 days before an initial injection of Cisplatin. Cisplatin is injected intraperitoneally at 4 mg/kg body weight (B.W.) on day 0, 7 and 14 (total three injections). Experimental group is intubated with HemoHIM at a final concentration of 100 mg/kg B.W. by everyday from day -1 to day 16, while the control group received only water. On day 17 after initial injection of Cisplatin, all mice of each group are experimented, respectively, to evaluate tumor weight or tumor size. The tumor size is calculated as follows: tumor size=ab²/2, where a and b are the larger and smaller diameters, respectively.

Rats^[4]

Male Sprague-Dawley rats weighing 200 to 250 g are divided at random into 4 groups of 4 or 5 animals each. The first group (control) received a vehicle (5% carboxymethyl cellulose sodium solution (CMC-Na), 5 mL/kg body wt., p.o.) used for Capsaicin (Cap). The second group received Cap (10 mg/kg/d, p.o.) in 5% CMC-Na (5 mL/kg), and the third received 5% CMC-Na for 6 consecutive days injected with Cisplatin (5 mg/kg in physiological saline solution, i.p.). The fourth group received Cap (10 mg/kg/d, p.o.) in 5% CMC-Na for 6 consecutive days after Cisplatin injection (5 mg/kg, i.p.). For all groups, Cap or vehicle is given twice daily. The selected Cap concentration and the dose administration schedule without inducing any rat intestinal damage are chosen using data from our preliminary experiments.

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

CUSTOMER VALIDATION

- Cell Res. 2023 Jul 17.
- Signal Transduct Target Ther. 2022 Sep 12;7(1):317.
- Signal Transduct Target Ther. 2020 May 20;5(1):56.
- Mol Cancer. 2021 Apr 4;20(1):62.
- Adv Mater. 2023 May 6;e2300602.

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- [2]. Ryan M Williams, et al. Kidney-Targeted Redox Scavenger Therapy Prevents Cisplatin-Induced Acute Kidney Injury. *Front Pharmacol.* 2022 Jan 3;12:790913.
- [3]. Wang X, et al. Requirement for ERK activation in cisplatin-induced apoptosis. *J Biol Chem.* 2000 Dec 15;275(50):39435-43.
- [4]. Cummings BS, et al. Cisplatin-induced renal cell apoptosis: caspase 3-dependent and -independent pathways. *J Pharmacol Exp Ther.* 2002 Jul;302(1):8-17.
- [5]. Park HR, et al. Enhanced antitumor efficacy of cisplatin in combination with HemoHIM in tumor-bearing mice. *BMC Cancer.* 2009 Mar 17;9:85.
- [6]. Shimeda Y, et al. Protective effects of capsaicin against cisplatin-induced nephrotoxicity in rats. *Biol Pharm Bull.* 2005 Sep;28(9):1635-8.
- [7]. Hall MD, et al. Say no to DMSO: dimethylsulfoxide inactivates cisplatin, NSC 241240, and other platinum complexes. *Cancer Res.* 2014 Jul 15;74(14):3913-22.

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

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