

Product Data Sheet

Product Name: Cisplatin

Cat. No.: GC11908

Chemical Properties

Cas. No. 15663-27-1

Chemical Name azane;dichloroplatinum(2+)

SMILES N.N.Cl[Pt+2]Cl

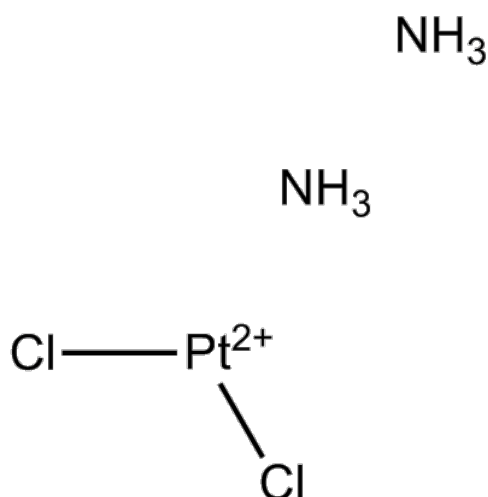
Formula Cl2H6N2Pt M.Wt 300.05

Solubility 5 mg/mL in DMF (16.66 mM; DMSO can inactivate Cisplatin's activity), 1 mg/mL in Water (3.33 mM; DMSO can inactivate Cisplatin's activity) Storage 4°C, protect from light

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure



Protocol

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

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Cell experiment [1]:	
Cell lines	Melanoma cell
Preparation Method	The melanoma cells were seeded on to 96-well plates at a density of 7×10^3 cells per well in 100 μ l of media. After incubation for 24 hours, cells were treated with various concentration of Cisplatin for 24 hours. After incubation, a CCK-8 solution was added to each well.
Reaction Conditions	0-40 μ g/ml; 24h
Applications	Cisplatin inhibited melanoma cell growth in a dose-dependent manner, with IC ₅₀ at about 15 μ g/ml.
Animal experiment [1]:	
Animal models	B16F0 melanoma-bearing mouse model
Preparation Method	B16F0 melanoma (5×10^5 cells/mouse) was inoculated into subcutaneous femoral left region of mice at 3 days before an initial injection of Cisplatin. Cisplatin was injected intraperitoneally at 4mg/kg on day 0, 7 and 14 (total three injections). On day 17 after initial injection of Cisplatin, mice were experimented, respectively, to evaluate tumor weight or tumor size.
Dosage form	4mg/kg; i.p.; 3 times
Applications	In the Cisplatin-injected group, tumor weight (2.97g (± 1.29)) and size (6.3mm ³ (± 2.32)) were reduced significantly in comparison to the control group.

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References:

[1] Park HR, Ju EJ, Jo SK, Jung U, Kim SH, Yee ST. Enhanced antitumor efficacy of cisplatin in combination with HemoHIM in tumor-bearing mice. BMC Cancer. 2009;9:85.

Background

Cisplatin, a platinum-containing anticancer drug, is one of the most commonly used cytotoxic agents for the treatment of a variety of solid malignant tumors. Cisplatin generally binds with genomic DNA (gDNA) or mitochondrial DNA (mtDNA) to create DNA lesions, block the production of DNA, mRNA and proteins, arrest DNA replication, activate several transduction pathways which finally led to necrosis or apoptosis^[1-2].

In vitro, Cisplatin (0-30 μ M; 12-24h) induced apoptosis in HeLa cells in a dose- and time-dependent manner^[1]. Cisplatin (0-40 μ g/ml; 24h) inhibited melanoma cell growth in a dose-dependent manner, with IC₅₀ at about 15 μ g/ml^[3].

In vivo, Cisplatin (4mg/kg; i.p.; 3 times) inhibited tumor growth in a B16F0 melanoma-bearing mouse model^[3]. Cisplatin (5mg/kg; i.p.; 2 weeks) can establish a mouse model of acute kidney injury, as evidenced by elevated levels of serum creatinine (SCr), blood urea nitrogen (BUN), neutrophil gelatinase-associated lipocalin (NGAL), and nitrotyrosine^[4].

References:

[1] Wang X, Martindale JL, Holbrook NJ. Requirement for ERK activation in cisplatin-induced apoptosis. J Biol Chem. 2000;275(50):39435-39443.

[2] Stordal B, et al. Understanding cisplatin resistance using cellular models. IUBMB Life. 2007 Nov;59(11):696-9.

[3] Park HR, Ju EJ, Jo SK, Jung U, Kim SH, Yee ST. Enhanced antitumor efficacy of cisplatin in combination with HemoHIM in tumor-bearing mice. BMC Cancer. 2009;9:85. Published 2009 Mar 17.

[4] Noha Alassaf, et al. Autophagy and necroptosis in cisplatin-induced acute kidney injury: Recent advances regarding their role and therapeutic potential. Front Pharmacol. 2023 Jan 30;14:1103062.

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