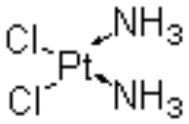
Product Introduction

Cisplatin

Cisplatin is an inorganic platinum complex, which is able to inhibit **DNA synthesis** by conforming DNA adducts.

Technical Data:

Molecular Weight (MW):	300.05	
Formula:	$Cl_2H_6N_2Pt$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 60 mg/mL	
	Water <1 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	15663-27-1	

Biological Activity

Cisplatin induces cytotoxic by interaction with DNA to form DNA adducts which activate several signal transduction pathways, including Erk, p53, p73, and MAPK, which culminates in the activation of apoptosis. [1] Cisplatin (30 mM) treated for 6 h induces an apparent activation of Erk in HeLa cells, which is sustained over the following 14 h period. Cisplatin also shows an effective antineoplastic activity by inducing tumor cells death. Cisplatin displays ability to cause renal proximal tubular cell (RPTC) apoptosis, 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. [4] Cisplatin (800 μM) causes typical features of necrosis of RPTC after treatment for 4 hr. [5]

Cisplatin has been demonstrated to be efficient in regression tumor growth in a wide variety of animal tumors models, including head and neck cancer xenografts, cervical squamous carcinoma xenografts, testicular carcinoma xenografts, ovarian cancer xenografts, breast carcinoma xenografts, colonic carcinoma, heterotransplanted hepatoblastoma, and so on. Cisplatin (5 mg/kg) given weekly i.v. at the day 1 and 7 induces a tumor growth inhibition (GI) of 77.5% and 85.1% of the serous xenografts Ov.Ri(C) and OVCAR-3, respectively. [6]

One of the most widely used and most potent chemotherapeutic agents.

References

[1] Siddik ZH. Oncogene, 2003, 22(47), 7265-7279.

[2] Wang X, et al. J Biol Chem, 2000, 275(50), 39435-39443.

[3] Sorenson CM, et al. Cancer Res, 1988, 48(16), 4484-4488.

[4] Cummings BS, et al. J Pharmacol Exp Ther, 2002, 302(1):8-17.

[5] Lieberthal W, et al. Am J Physiol, 1996, 270(4 Pt 2), F700-708.

[6] Molthoff CF, et al. Int J Cancer, 1992, 51(1), 108-115.

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