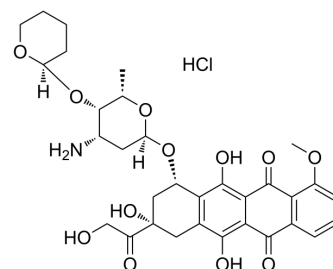


Pirarubicin Hydrochloride

Cat. No.:	HY-13725A
CAS No.:	95343-20-7
Molecular Formula:	C ₃₂ H ₃₈ ClNO ₁₂
Molecular Weight:	664
Target:	Topoisomerase; Autophagy; Bacterial; Antibiotic
Pathway:	Cell Cycle/DNA Damage; Autophagy; Anti-infection
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 20.83 mg/mL (31.37 mM; Need ultrasonic)
H₂O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.5060 mL	7.5301 mL	15.0602 mL
	5 mM	0.3012 mL	1.5060 mL	3.0120 mL
	10 mM	0.1506 mL	0.7530 mL	1.5060 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.08 mg/mL (3.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pirarubicin Hydrochloride is an anthracycline antibiotics, acts as a topoisomerase II inhibitor, and is a widely used for treatment of various cancers, in particular, solid tumors.

IC₅₀ & Target

Topoisomerase II

In Vitro

Pirarubicin is a topoisomerase II inhibitor^[1].

Pirarubicin shows inhibitory activities against M5076 and Ehrlich cells, with IC₅₀s of 0.366 and 0.078 μM, respectively. The cytotoxicity of Pirarubicin toward M5076 cells is lower than toward Ehrlich cells, and this is due to the much lower expression of topoisomerase II in M5076 cells than in Ehrlich cells^[2].

Pirarubicin (2.5, 5, 10 μg/mL) significantly induces autophagy in a dose dependent manner in bladder cancer (T24, EJ, 5637, J82 and UM-UC-3) cells. Furthermore, Pirarubicin (5 μg/mL) induces apoptosis through inhibition of mTOR/p70S6K/4E-BP1 in bladder cancer cells, and this effect is enhanced by inhibition of autophagy^[3].

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

In Vivo

Pirarubicin (18 mg/kg, i.v.) significantly elevates serum level of BNP, CK-MB, CTnT, LDH, and MDA compared with those in the control group in acute cardiac toxicity rats. Pirarubicin also lowers heart rate, and depresses R-wave voltage, and prolongation of QT intervals in the acute cardiac toxicity model^[4].

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

PROTOCOL

Cell Assay ^[3]

MTS is used to analyze cell survival. Briefly, cells are plated in 96-well plates in triplicate at 2×10^3 cells per well and cultured in growth medium. Then cells are treated with pirarubicin at different concentrations (2.5 $\mu\text{g}/\text{mL}$, 5 $\mu\text{g}/\text{mL}$, 10 $\mu\text{g}/\text{mL}$) for 24 h. MTS reagent (5 mg/mL) is added and incubated at 37°C for 4 h. The absorbance is monitored at 490 nm using a microplate reader^[3].

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

Animal Administration ^[4]

An acute cardiac toxicity model is established by a single dose of 18 mg/kg pirarubicin through the caudal vein injection. Thirty-six rats are randomized equally to six groups: normal control, cardiac injury (THP) model, dexrazoxane (180 mg/kg), low-dose rutin (25 mg/kg), middle-dose rutin (50 mg/kg), and high-dose rutin (100 mg/kg). Rats in the rutin-treated group are administered different doses of rutin and CMC-Na for 7 days by gavage and a single dose of 18 mg/kg pirarubicin through caudal vein injection. Rats in the dexrazoxane-treated group receive sodium carboxymethylcellulose (CMC-Na) by gavage for six days. 40 mg/kg dexrazoxane is then administered to rats by intraperitoneal injection and 18 mg/kg pirarubicin is administered by caudal vein injection on day 7. Rats in the THP model group receive CMC-Na by gavage for seven days, followed by pirarubicin 18 mg/kg through the caudal vein injection on day 7. Rats in the normal control group receive CMC-Na by gavage for seven days, followed by saline through caudal vein injection on day 7^[4].

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

CUSTOMER VALIDATION

- J Mol Liq. 2023 Jun 12, 122324.
- J Mol Liq. 2020, 114633.
- Int J Pharm. 2022 May 25;620:121761.
- Cancers (Basel). 2023 Apr 26, 15(9), 2487.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

[1]. Takigawa N, et al. Cytotoxic effect of topoisomerase II inhibitors against adriamycin- and etoposide-resistant small cell lung cancer sublines. Gan To Kagaku Ryoho. 1993 May;20(7):929-35.

[2]. Nagai K, et al. Relationships between the in vitro cytotoxicity and transport characteristics of pirarubicin and doxorubicin in M5076 ovarian sarcoma cells, and comparison with those in Ehrlich ascites carcinoma cells. Cancer Chemother Pharmacol. 2002 Mar;49(3):244-50. Epub 2002 Jan 8.

[3]. Li K, et al. Pirarubicin induces an autophagic cytoprotective response through suppression of the mammalian target of rapamycin signaling pathway in human bladder cancer cells. Biochem Biophys Res Commun. 2015 May 1;460(2):380-5.

[4]. Wang YD, et al. Cardioprotective effects of rutin in rats exposed to pirarubicin toxicity. J Asian Nat Prod Res. 2017 Oct 27:1-13.

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

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