Daunorubicin hydrochloride

Cat. No.: HY-13062

CAS No.: 23541-50-6

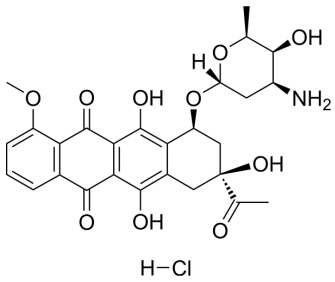
Molecular Formula: $C_{27}H_{30}ClNO_{10}$

Molecular Weight: 563.98

Target: Topoisomerase; DNA/RNA Synthesis; ADC Cytotoxin; Autophagy; Apoptosis; Bacterial; Antibiotic

Pathway: Cell Cycle/DNA Damage; Antibody-drug Conjugate/ADC Related; Autophagy; Apoptosis; 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 : 50 mg/mL (88.66 mM; Need ultrasonic)
H_2O : ≥ 34 mg/mL (60.29 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7731 mL	8.8656 mL	17.7311 mL
5 mM	0.3546 mL	1.7731 mL	3.5462 mL
10 mM	0.1773 mL	0.8866 mL	1.7731 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 33.33 mg/mL (59.10 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (3.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Daunorubicin (Daunomycin) hydrochloride is a topoisomerase II inhibitor with potent anti-tumor activity. Daunorubicin hydrochloride inhibits DNA and RNA synthesis. Daunorubicin hydrochloride is a cytotoxin that inhibits cancer cell viability and induces apoptosis and necrosis. Daunorubicin hydrochloride is also an anthracycline antibiotic. Daunorubicin hydrochloride can be used in the research of infection and variety of cancers, including leukemia, non-Hodgkin lymphomas,

	Ewing's sarcoma, Wilms' tumor[1][2][4][5].

IC_{50} & Target	Topoisomerase II	Daunorubicins/Doxorubicins

In Vitro

Daunorubicin hydrochloride (0-256 μg/mL, 30 min) inhibits DNA and RNA synthesis in sensitive and resistant Ehrlich ascites tumor cells[2].
Daunorubicin hydrochloride (7 nM-1.9 μM, 72 h) shows chemosensitivity in Molt-4 cells and L3.6 cells[3][4].
Daunorubicin hydrochloride (0.4 μM, 48 h) induces apoptotic and necrosis in L3.6 cells[4].
Daunorubicin hydrochloride (0.4 μM, 120 min) induces ROS generation in L3.6 cells[4].
Daunorubicin hydrochloride (2 μM, 24 h) induces autophagy in K562 cells (myeloid cell line)[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3][4]

Cell Line:	Molt-4 cells (a human T-lymphoblastic leukemia cell line), L3.6 cells (metastatic human pancreatic cell line)
Concentration:	7 nM-1.9 μM
Incubation Time:	72 h
Result:	Inhibited cell viability with IC_{50} values of 40 nM (Molt-4) and 400 nM (L3.6).

Apoptosis Analysis[4]

Cell Line:	L3.6 cells
Concentration:	0.4 μM
Incubation Time:	72 h
Result:	Induced necrosis without apoptosis at 24 h, induced both an apoptotic and extensive necrotic response at 48 h.

Western Blot Analysis[6]:

Cell Line:	K562 cells
Concentration:	2 μM
Incubation Time:	24 h
Result:	Enabled the switch of LC3-I into LC3-II, accompanied with a significant increased expression level of LC3.

In Vivo

Daunorubicin hydrochloride (intravenous injection, 3 mg/kg, three times at 48 h intervals.) produces cardiotoxicity and nephrotoxicity in rats[5].
Daunorubicin hydrochloride (intraperitoneal injection, 10 mg/kg) induces sister chromatid exchanges in mice[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats[5]
Dosage:	3 mg/kg
Administration:	Intravenous injection, three times at 48 h intervals.
Result:	Caused a significant increase in MDA (malondialdehyde) level in renal tissue, accompanied

	by a significant reduction in total GPx activity. Increased urinary protein excretion, serum creatinine, and BUN level.

CUSTOMER VALIDATION

- Cell Mol Immunol. 2023 Jan;20(1):51-64.
- Clin Cancer Res. 2020 Apr 15;26(8):2011-2021.
- Leukemia. 2023 Mar 28.
- Cancer Res. 2023 Dec 14.
- J Control Release. 2022 Apr 22;346:136-147.

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REFERENCES

[1]. Emeline Bollaert, et al. MiR-15a-5p Confers Chemoresistance in Acute Myeloid Leukemia by Inhibiting Autophagy Induced by Daunorubicin. Int J Mol Sci. 2021 May 13;22(10):5153.

[2]. Cheng Wu, et al. Doxorubicin suppresses chondrocyte differentiation by stimulating ROS production. Eur J Pharm Sci. 2021 Dec 1;167:106013.

[3]. Lehmann M, et al. Activity of topoisomerase inhibitors daunorubicin, idarubicin, and aclarubicin in the Drosophila Somatic Mutation and Recombination Test. Environ Mol Mutagen. 2004;43(4):250-7.

[4]. Svensson SP, et al. Melanin inhibits cytotoxic effects of Doxorubicin and Daunorubicin in MOLT 4 cells. Pigment Cell Res. 2003 Aug;16(4):351-4

[5]. Gervasoni JE Jr, et al. An effective in vitro antitumor response against human pancreatic carcinoma with paclitaxel and Daunorubicin by induction of both necrosis and apoptosis. Anticancer Res. 2004 Sep-Oct;24(5A):2617-26

[6]. Arozal W, et al. Telmisartan prevents the progression of renal injury in daunorubicin rats with the alteration of angiotensin II and endothelin-1 receptor expression associated with its PPAR-γ agonist actions. Toxicology. 2011 Jan 11;279(1-3):91-9.

[7]. Dano K, et al. Inhibition of DNA and RNA synthesis by daunorubicin in sensitive and resistant Ehrlich ascites tumor cells in vitro. Cancer Res. 1972 Jun;32(6):1307-14.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA