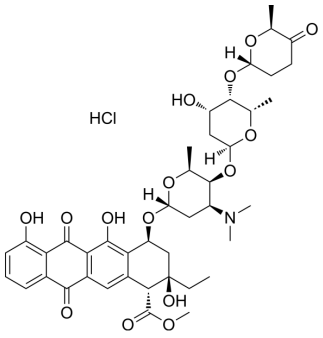
Aclacinomycin A hydrochloride

Cat. No.:	HY-N2306A
CAS No.:	75443-99-1
Molecular Formula:	$C_{42}H_{54}ClNO_{15}$
Molecular Weight:	848.33
Target:	Proteasome; Topoisomerase; Antibiotic; DNA/RNA Synthesis
Pathway:	Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; 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 : ≥ 125 mg/mL (147.35 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.1788 mL	5.8939 mL	11.7879 mL
5 mM	0.2358 mL	1.1788 mL	2.3576 mL
10 mM	0.1179 mL	0.5894 mL	1.1788 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 (2.45 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (2.45 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Aclacinomycin A (Aclarubicin) hydrochloride is an orally active and potent anthracycline antitumor antibiotic. Aclacinomycin A hydrochloride is an inhibitor of topoisomerase I and II. Aclacinomycin A hydrochloride inhibits synthesis of nucleic acid, especially RNA. Aclacinomycin A hydrochloride might inhibit the 26S protease complex as well as the ubiquitin-ATP-dependent proteolysis[1][2][3].

IC_{50} & Target	Topoisomerase I	Topoisomerase II

In Vitro

Aclacinomycin A (0-120 μM, 30 min) inhibits the ubiquitin-ATP-dependent proteolytic activity of rabbit reticulocytes in a dose-dependent manner, with an IC_{50} of 52 μM. But it does not inhibit the ubiquitination[1].
Aclacinomycin A inhibits ubiquitin-ATP-dependent proteolysis after the conjugation of ubiquitin to proteins[1].

Aclacinomycin A (0-2.4 μM, 3 h) inhibits the topo II catalytic activity[2].
Aclacinomycin A (0-1.8 μM, 3 h) has negative effect on the proliferative rate of V79 and irs-2 cells[2].
Aclacinomycin A emits fluorescence and that human-cervical cancer HeLa cells exposed to Aclacinomycin A exhibits bright fluorescence signals in the cytoplasm when fluorescence microscopy was performed using the red filter (excitation 530-550 nm/emission 575 nm)[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	V79 and irs-2 cells
Concentration:	0, 0.006, 0.12, 1.2, and 2.4 μM
Incubation Time:	3 h
Result:	Inhibited the topo II catalytic activity in a dose-dependent manner. The loss of topo II catalytic activity in ACLA-treated cells was in all cases significant compared with non-treated cells.

Cell Proliferation Assay[2]

Cell Line:	V79 and irs-2 cells
Concentration:	0, 0.12, 0.25, 0.37, 0.6, 1.2, 1.8 μM
Incubation Time:	3 h
Result:	Showed a dose-dependent negative effect on the proliferative rate of V79 and irs-2 cells, but the reduction in surviving colonies was higher in the radiosensitive irs-2 cells for most of the ACLA doses tested.

In Vivo

Aclacinomycin A (0.75-6 mg/kg, IP, daily) dose-dependently exhibits tumor growth in mice-based Leukemia P-388 model[4].
Aclacinomycin A (0.6-20 mg/kg, Orally, daily) exhibits an antitumor effect on leukemia L-1210[4].
Aclacinomycin A is very well absorbed in mice, rats, and dogs after its oral administration. The oral LD_{50} (76.5 mg/kg) is about twice the iv LD_{50} (35.6 mg/kg) in mice[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	DBA/2, CDF_1 (BALB/c×DBA/2) mice with Leukemia P-388[4].
Dosage:	0.75 mg/kg, 1.5 mg/kg, 3 mg/kg, 6 mg/kg.
Administration:	Intraperitoneal administration daily for 10 days starting 3 hr after transplantation.
Result:	Inhibited tumor growth.

Animal Model:	CDF_1 mouse with Leukemia L-1210[4]
Dosage:	0.6 mg/kg, 1.25 mg/kg, 2.5 mg/kg, 5 mg/kg, 10 mg/kg, 20 mg/kg
Administration:	Orally, daily for days 1-9
Result:	Exhibited an antitumor effect on leukemia L-1210.

CUSTOMER VALIDATION

- Bioengineered. 2022 Feb;13(2):2207-2216.
- Int J Hyperthermia. 2022;39(1):998-1009.

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REFERENCES

[1]. Isoe T, et al. Inhibition of different steps of the ubiquitin system by CDDP and aclarubicin. Biochim Biophys Acta. 1992 Sep 15;1117(2):131-5.

[2]. Hajji N, et al. Induction of genotoxic and cytotoxic damage by aclarubicin, a dual topoisomerase inhibitor. Mutat Res. 2005 May 2;583(1):26-35.

[3]. Iihoshi H, et al. Aclarubicin, an anthracycline anti-cancer drug, fluorescently contrasts mitochondria and reduces the oxygen consumption rate in living human cells. Toxicol Lett. 2017 Aug 5;277:109-114.

[4]. Hori S, et al. Antitumor activity of new anthracycline antibiotics, aclacinomycin-A and its analogs, and their toxicity. Gan. 1977 Oct;68(5):685-90.

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