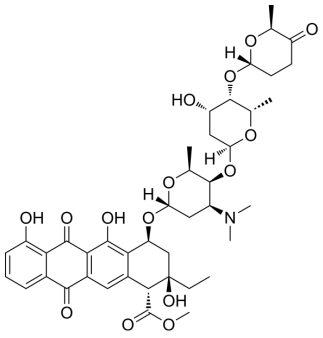
Aclacinomycin A

Cat. No.:	HY-N2306
CAS No.:	57576-44-0
Molecular Formula:	$C_{42}H_{53}NO_{15}$
Molecular Weight:	811.87
Target:	Topoisomerase; DNA/RNA Synthesis; Proteasome; Antibiotic
Pathway:	Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Anti-infection
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (61.59 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.2317 mL	6.1586 mL	12.3172 mL
	5 mM	0.2463 mL	1.2317 mL	2.4634 mL
	10 mM	0.1232 mL	0.6159 mL	1.2317 mL

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

BIOLOGICAL ACTIVITY

Description

Aclacinomycin A (Aclarubicin) is an orally active and potent anthracycline antitumor antibiotic. Aclacinomycin A is an inhibitor of topoisomerase I and II. Aclacinomycin A inhibits synthesis of nucleic acid, especially RNA. Aclacinomycin A 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 (90-110 g)[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.
- bioRxiv. 2023 Jan 13.

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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, Shirai M, Hirano S, Oki T, Inui T, Tsukagoshi S, Ishizuka M, Takeuchi T, Umezawa H. 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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