Plicamycin

Cat. No.: HY-A0122

CAS No.: 18378-89-7

Molecular Formula: $C_{52}H_{76}O_{24}$

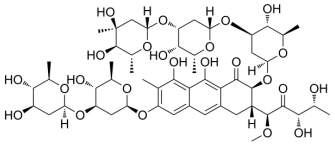
Molecular Weight: 1085.15

Target: DNA/RNA Synthesis; Bacterial; Antibiotic

Pathway: Cell Cycle/DNA Damage; Anti-infection

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (92.15 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent, Mass	1 mg	5 mg	10 mg
1 mM	0.9215 mL	4.6077 mL	9.2153 mL
5 mM	0.1843 mL	0.9215 mL	1.8431 mL
10 mM	0.0922 mL	0.4608 mL	0.9215 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: ≥ 2.5 mg/mL (2.30 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Plicamycin is a selective specificity protein 1 (Sp1) inhibitor. Plicamycin inhibits the growth of various cancers by decreasing Sp1 protein.

IC_{50} & Target

Sp1 transcription factor[1]

In Vitro

Plicamycin (Mith) decreases Sp1 protein by inducing proteasome-dependent degradation, thereby suppressing cervical cancer growth through a DR5/caspase-8/Bid signaling pathway. Plicamycin inhibits HEp-2 and KB cell growth in a concentration-dependent manner after 48 h. Apoptotic cell death is qualitatively estimated by DAPI staining for nuclear condensation and fragmentation. Plicamycin leads to significant DNA fragmentation compared to untreated controls[1].

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

In Vivo	The antitumorigenic activity of Plicamycin (0.2 mg/kg/day) is determined in a xenograft model and observed reduction in tumor volume and weight. No significant mouse body weight loss is observed in Plicamycin-treatment groups, indicating that Plicamycin-associated toxicity is minimal. Plicamycin also increases TUNEL-positive cells in tumor xenografts. No notable intergroup differences are observed among organs, indicating no marked signs of systemic toxicity at the Plicamycin dose used in this study[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	HEp-2 cells and KB cells are cultured in DMEM 100 U/mL each of Penicillin and Streptomycin and 10% FBS for HEp-2 cells and 5% FBS for KB in a humidified atmosphere containing 5% CO_2 at 37°C. Equal numbers of cells are seeded and allowed to attach. At 50-60% confluence, cells are treated with DMSO or indicated concentrations of Plicamycin (50, 100, and 200 nM for HEp-2 cells; 20, 40, and 80 nM for KB cells). Cell viability is determined using CellTiter 96 Aqueous One Solution Cell Proliferation Assay Kits. In brief, cells are seeded in 96-well plates and incubated with Plicamycin. After treatment, 30 µL MTS solution is added to each well and cells are incubated for 2 h at 37°C. MTS solution is analyzed using a microplate reader at 490 nm and 690 nm[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] Female nude mice are used. KB cells are suspended in sterile PBS and injected subcutaneously into the right flank of mice. Mice are randomized into two groups containing five mice each and treated with 0.2 mg/kg/day of Plicamycin (i.p.) three times per week for 29 days. Control mice receive an equal volume of vehicle. After 29 days, bodies, organs and tumors are weighed and tumor volumes determined. Tumors are measured[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Mol Cancer. 2022 Mar 18;21(1):77.
- Nat Commun. 2023 Feb 9;14(1):731.
- Theranostics. 2022 Jan 1;12(2):842-858.
- Cell Death Dis. 2021 Oct 21;12(11):978.
- Cell Rep. 2023 Aug 11;42(8):112975.

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REFERENCES

[1]. Choi ES, et al. Modulation of specificity protein 1 by mithramycin A as a novel therapeutic strategy for cervical cancer. Sci Rep. 2014 Nov 24;4:7162.

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