Actinomycin D

Cat. No.: HY-17559

CAS No.: 50-76-0

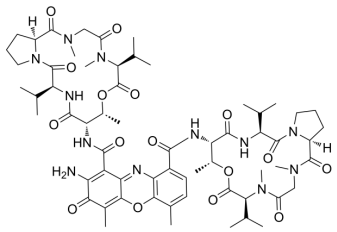
Molecular Formula: $C_{62}H_{86}N_{12}O_{16}$

Molecular Weight: 1255.42

Target: DNA/RNA Synthesis; Autophagy; Apoptosis; Bacterial; Antibiotic

Pathway: Cell Cycle/DNA Damage; Autophagy; Apoptosis; Anti-infection

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 27 mg/mL (21.51 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent, Mass	1 mg	5 mg	10 mg
1 mM	0.7965 mL	3.9827 mL	7.9655 mL
5 mM	0.1593 mL	0.7965 mL	1.5931 mL
10 mM	0.0797 mL	0.3983 mL	0.7965 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 (1.66 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (1.66 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Actinomycin D (Dactinomycin) inhibits DNA repair with an IC_{50} of 0.42 μM. Actinomycin D is an autophagy activator[1][2][3].

IC_{50} & Target

IC50: 0.42 μM (DNA repair)[1]

In Vitro

Actinomycin D (Dactinomycin) is an inhibitor of DNA transcription and replication[1]. Actinomycin D markedly reduces the vascular smooth muscle cells (SMC) proliferation via the inhibition of BrdU incorporation at 80 nM. This is further supported by the G1-phase arrest using a flowcytometric analysis. Actinomycin D is extremely potent with an inhibitory concentration IC_{50} at 0.4 nM, whereas the lethal dose LD50 is at 260 microM. The protein expression levels of proliferating cell nuclear antigen (PCNA), focal adhesion kinase (FAK), and Raf are all suppressed by Actinomycin D. Extracellular signal-regulated kinases (Erk) involved in cell-cycle arrest are found to increase by Actinomycin D[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The pluronic gel containing 80 nM and 80 μM Actinomycin D (Dactinomycin) is applied topically to surround the rat carotid adventitia, the thickness of neointima is substantially reduced (45 and 55%, respectively)[2]. Mice in the Actinomycin D and NSC 118218 group lives significantly longer than the control group with P values of <0.001 and 0.007, respectively. Interestingly, single treatment with Actinomycin D is superior to NSC 118218 regarding overall survival (P=0.026)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Actinomycin D is co-incubated for 3 h at 30°C with a reaction mixture containing: 120 mg of a whole-cell extract of HeLa cells, 70 mM KCl, 0.4 mM each of dGTP, dCTP, dATP, and digoxygenylated-dUTP in reaction buffer containing 40mM Hepes-KOH (pH 7.6), 5 mM $MgCl_2$, 0.5 mM Dithiotreitol, 2 mM EGTA, 10 mM phosphocreatine, 50 mg/mL creatine phosphate, and 360 mg/mL of bovine serum albumin. During this reaction, DNA damage is recognized and the excised patches are replaced by neosynthesized DNA fragments. Throughout this DNA synthesis, digoxygenylated-dUMPs are incorporated. The DNA repair reaction is stopped by three washes[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] The original Eμ-TCL1a transgenic mice have been backcrossed to C57BL/6 mice for >9 generations.The C57BL/6 wild-type mice are engrafted with tumor cells from Eμ-TCL-1 transgenic mice. The percentage of CD5+/CD19+ cells in the peripheral blood is routinely checked in mice by taking blood from the tail vein and analyzing it via flow cytometry. When the percentage of tumor cells in the peripheral blood reached 40-60%, treatment is started. Actinomycin D (0.06 mg/kg by 10 days) is applied daily via i.p. injections. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2019 Jan;29(1):23-41.
- Cell Res. 2018 Dec;28(12):1171-1185.
- Mol Cancer. 2023 May 9;22(1):81.
- Mol Cancer. 2022 Feb 23;21(1):60.
- Mol Cancer. 2020 Jun 17;19(1):106.

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REFERENCES

[1]. Barret JM, et al. Evaluation of DNA repair inhibition by antitumor or antibiotic drugs using a chemiluminescence microplateassay. Carcinogenesis. 1997 Dec;18(12):2441-5.

[2]. Wu CH, et al. The molecular mechanism of actinomycin D in preventing neointimal formation in rat carotid arteries after balloon injury. J Biomed Sci. 2005;12(3):503-12.

[3]. Merkel O, et al. Actinomycin D induces p53-independent cell death and prolongs survival in high-risk chronic lymphocytic leukemia. Leukemia. 2012 Dec;26(12):2508-16.

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

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