Product Data Sheet

Product Name: Daunorubicin
Cat. No.: GC12828

Chemical Properties

Cas. No.	20830-81-3		
Chemical Name	(7S,9S)-9-acetyl-7-[(2R,4S,5S,6S)-4-amino-5-hydroxy-6-methyloxan-2-yl]oxy-6,9,11-trihydroxy-4-methoxy-8,10-dihydro-7H-tetracene-5,12-dione		
SMILES	CC1C(C(CC(O1)OC2CC(CC3=C(C4=C(C(=C23)O)C(=O)C5=C(C4=O)C=CC=C5OC)O)(C(=O)C)O)N)O		
Formula	$C_{27}H_{29}NO_{10}$	M.Wt	527.52
Solubility	≥ 83.3mg/mL in DMSO	Storage	Store at -20°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Daunorubicin		

Protocol

Cell experiment [1]:

Cell lines	Human acute myeloid leukemia HL-60 cells
Preparation Method	HL-60 cells were cultured in RPMI-1640 with 10% FCS at 37°C, 5% CO_2. For experiments, cells were seeded at 0.5 × 10^6cells/mL and exposed to Daunorubicin for 1h, then washed and maintained in drug-free medium for the indicated chase periods.
Reaction Conditions	0.5–1μM Daunorubicin for 1h followed by 6–24h drug-free incubation.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

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Applications	Daunorubicin rapidly induced apoptotic cell death in HL-60 cells, evidenced by pyknotic nuclei, internucleosomal DNA fragmentation, and activation of caspases-3/8/9 and PARP cleavage. Cell cycle analysis revealed an early S-phase block and accumulation of sub-G1 apoptotic cells, confirming Daunorubicin-mediated caspase-dependent apoptosis.

Animal experiment [2]:

Animal models	BALB/c nude mice bearing HCT116 colorectal-cancer xenografts.
Preparation Method	HCT116 cells (2×10^6) were subcutaneously implanted into the dorsal flank of 5-week-old female mice. Once tumors reached ≈100mm^3, animals were randomized and treated with either vehicle (DMSO) or Daunorubicin every other day for 15 days.
Dosage form	2mg/kg; intraperitoneal injection, every 48h for 15 days.
Applications	Daunorubicin profoundly inhibited tumor growth and reduced tumor weight without affecting body weight. Immunohistochemistry revealed marked suppression of GLI1 and elevation of p53 within tumor tissues, while TUNEL staining confirmed extensive apoptosis.

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References:
[1] Côme MG, Skladanowski A, Larsen AK, et al. Dual mechanism of daunorubicin-induced cell death in both sensitive and MDR-resistant HL-60 cells. Br J Cancer. 1999 Mar;79(7-8):1090-7.
[2] Kim BR, Kim DY, Tran NL, et al. Daunorubicin induces GLI1-dependent apoptosis in colorectal cancer cell lines. Int J Oncol. 2024 Jun;64(6):66.

Background

Daunorubicin is a natural anthracycline antibiotic that intercalates into DNA double strands and inhibits topoisomerase II activity, thereby blocking DNA replication and transcription and inducing tumor cell apoptosis[1]. Clinically, Daunorubicin is primarily used for the treatment of acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL)[2-3]. In addition, Daunorubicin has shown therapeutic potential against gliomas[4].

In vitro, treatment of human acute myeloid leukemia HL-60 cells with 0.1–1μM Daunorubicin for 1h, followed by incubation in drug-free medium for 6–24h, rapidly induces apoptotic cell death[5]. Exposure of human acute myeloid leukemia U937 cells to 0.5–1μM Daunorubicin for 2–24h significantly activates the PI3K/Akt signaling pathway, leading to cell cycle arrest and enhanced apoptosis[6].

In vivo, intraperitoneal administration of 2mg/kg Daunorubicin every other day for 15 days in HCT116 colorectal cancer xenograft BALB/c nude mice markedly inhibited tumor growth, reduced GLI1 expression in tumor tissues, and induced tumor cell apoptosis[7]. Intravenous tail-vein injection of 10mg/kg Daunorubicin for three consecutive days in C57BL/6 wild-type and Trp53-knockout mice elicited early, transient apoptosis in wild-type mice, with recovery to normal morphology by day 4. In contrast, Trp53-knockout mice exhibited delayed and persistent extensive necrosis and structural disruption despite the absence of early apoptosis[8].

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References:
[1] Di Marco A, Cassinelli G, Arcamone F. The discovery of daunorubicin. Cancer Treat Rep. 1981;65 Suppl 4:3-8.
[2] Cann ML, Herring LE, Haar LL, et al. Dasatinib Is Preferentially Active in the Activated B-Cell Subtype of Diffuse Large B-Cell Lymphoma. J Proteome Res. 2019 Jan 4;18(1):522-534.
[3] Lin TY, Zhu Y, Li Y, et al. Daunorubicin-containing CLL1-targeting nanomicelles have anti-leukemia stem cell activity in acute myeloid leukemia. Nanomedicine. 2019 Aug;20:102004.
[4] Casazza AM, Pratesi G, Giuliani F, et al. Antileukemic activity of 4-demethoxydaunorubicin in mice. Tumori. 1980 Oct 31;66(5):549-64.
[5] Côme MG, Skladanowski A, Larsen AK, et al. Dual mechanism of daunorubicin-induced cell death in both sensitive and MDR-resistant HL-60 cells. Br J Cancer. 1999 Mar;79(7-8):1090-7.
[6] Plo I, Bettaïeb A, Payrastre B, et al. The phosphoinositide 3-kinase/Akt pathway is activated by daunorubicin in human acute myeloid leukemia cell lines. FEBS Lett. 1999 Jun 11;452(3):150-4.
[7] Kim BR, Kim DY, Tran NL, et al. Daunorubicin induces GLI1-dependent apoptosis in colorectal cancer cell lines. Int J Oncol. 2024 Jun;64(6):66.
[8] Herfindal L, Myhren L, Gjertsen BT, et al. Functional p53 is required for rapid restoration of daunorubicin-induced lesions of the spleen. BMC Cancer. 2013 Jul 11;13:341.

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