

Product Data Sheet

Product Name: Doxorubicin

Cat. No.: GC16994

Chemical Properties

Cas. No. 23214-92-8

Chemical Name (7S,9S)-7-[(2R,4S,5S,6S)-4-amino-5-hydroxy-6-methyloxan-2-yl]oxy-6,9,11-trihydroxy-9-(2-hydroxyacetyl)-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)CO)O)N)O

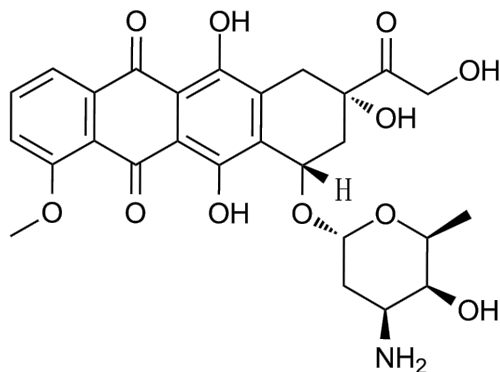
Formula $C_{27}H_{29}NO_{11}$ M.Wt 543.52

Solubility 20mg/mL in Water(Need ultrasonic); 20mg/mL in DMSO(Need ultrasonic) Storage Store at -20°C, protect from light

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C 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



Protocol

Cell experiment [1]:

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Cell lines	FU-SY-1 cells (human synovial sarcoma cell line), SW982 cells (human pleomorphic sarcoma cell line), NHDF cells (human primary dermal fibroblast cell line)
Preparation Method	FU-SY-1, SW982 and NHDF cells were maintained in appropriate culture medium at 37°C, 5% CO ₂ . Log-phase cells were washed with Hanks Balanced Salt Solution and treated with 500nM Doxorubicin in serum-free medium for 24h, then washed and recovered in 10% FBS medium for an additional 24h before RNA extraction, cDNA microarray analysis, real-time RT-PCR and senescence-associated β -galactosidase staining.
Reaction Conditions	500nM; 24h
Applications	Doxorubicin induced cell senescence preferentially over apoptosis in FU-SY-1 cells, with significantly increased senescence-associated β -galactosidase positive cells (35% vs 13% control) and overexpression of CDKN1A, CDKN2A and BTG2. Doxorubicin caused negligible senescence in SW982 and NHDF cells. Doxorubicin induced modest apoptosis only in SW982 cells via BAX overexpression relative to Bcl-2, with minimal apoptotic response in FU-SY-1 and NHDF cells.
Animal experiment [2]:	
Animal models	Female C57BL/6j mice

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Preparation Method	Mice were intraperitoneally administered Doxorubicin (2mg/kg or 10mg/kg) twice, or three times. Cardiac ultrasound was performed before first administration and before sacrifice. Mice were sacrificed 24h after single injection or 7 days after last injection for heart excision and histological, ELISA, Western blot, immunofluorescence and serum nitrite analyses.
Dosage form	2mg/kg or 10mg/kg; i.p.; every other day for 2-3 times
Applications	Doxorubicin administration reduced ejection fraction and fractional shortening, increased left ventricular end-systolic diameter, caused vacuolar and granular degeneration in myocardium, increased heart TNF- α , IL-6, IKK α , iNOS and nitrotyrosine expression, reduced heart IL-10 expression, and increased serum nitrite levels, induced heart toxicity.
References: [1] Joyner DE, Bastar JD, Randall RL. Doxorubicin induces cell senescence preferentially over apoptosis in the FU-SY-1 synovial sarcoma cell line. J Orthop Res. 2006 Jun;24(6):1163-1169. [2] Pecoraro M, Del Pizzo M, Marzocco S, et al. Inflammatory mediators in a short-time mouse model of doxorubicin-induced cardiotoxicity. Toxicol Appl Pharmacol. 2016 Mar 1;293:44-52.	

Background

Doxorubicin is an anthracycline antitumor antibiotic with broad-spectrum antitumor activity. Doxorubicin inhibits DNA topoisomerase II with an IC₅₀ of 2.67 μ M. Doxorubicin intercalates into DNA base pairs to inhibit DNA replication, inhibits topoisomerase II, and generates reactive oxygen species to induce tumor cell apoptosis. Doxorubicin can be used for cancer-related research and for establishing animal models of cardiac-related diseases^[1-4].

In vitro, treatment of A549 multicellular spheroids with 2.5 μ g/mL Doxorubicin for 4 hours increased

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Doxorubicin accumulation and inhibited Doxorubicin efflux in A549 cells^[5]. Treatment of FU-SY-1 cells with 500nM Doxorubicin for 24 hours induced FU-SY-1 cell senescence and upregulated CDKN1A, CDKN2A and BTG2 expression^[6]. Treatment of neonatal rat cardiomyocytes with 10μM Doxorubicin for 16 hours increased cellular mitochondrial iron levels and cellular reactive oxygen species levels, decreased oxygen consumption and mitochondrial membrane potential, reduced cell viability and increased cell death^[7].

In vivo, intravenous injection of 2mg/kg Doxorubicin once weekly for 6 weeks in MDA-MB-436 tumor-bearing MF1 mice inhibited tumor growth, increased tumor cell Caspase-3-positive expression, decreased Ki-67-positive tumor cell number, and increased femoral trabecular bone volume^[8]. To establish a cardiotoxicity model, intraperitoneal injection of 2-10mg/kg Doxorubicin every other day for 2-3 doses in C57BL/6j mice decreased left ventricular ejection fraction and fractional shortening, increased left ventricular end-systolic diameter, caused myocardial vacuolar and granular degeneration, increased cardiac TNF-α, IL-6, IKKα, iNOS and nitrotyrosine expression, decreased cardiac IL-10 expression, and elevated serum nitrite levels^[9]. To establish a heart failure model, intraperitoneal injection of 2.5mg/kg Doxorubicin once weekly for 6 weeks in Wistar rats increased left ventricular internal diameters at diastole and systole, elevated left ventricular end-diastolic pressure, decreased ejection fraction and fractional shortening, decreased left ventricular systolic pressure and $\pm dp/dt_{max}$, destroyed sarcomeres and caused mitochondrial swelling and damage^[10].

References:

- [1] Bisht A, Avinash D, Sahu KK, et al. A comprehensive review on doxorubicin: mechanisms, toxicity, clinical trials, combination therapies and nanoformulations in breast cancer. Drug Deliv Transl Res. 2025 Jan;15(1):102-133.
- [2] Rivankar S. An overview of doxorubicin formulations in cancer therapy. J Cancer Res Ther. 2014 Oct-Dec;10(4):853-8.
- [3] Sun Y, Xiao L, Chen L, et al. Doxorubicin-Induced Cardiac Remodeling: Mechanisms and Mitigation Strategies. Cardiovasc Drugs Ther. 2026 Feb;40(1):317-345.
- [4] Armstrong J, Dass CR. Doxorubicin Action on Mitochondria: Relevance to Osteosarcoma Therapy? Curr Drug Targets. 2018;19(5):432-438.

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[5] Xu WH, Han M, Dong Q, et al. Doxorubicin-mediated radiosensitivity in multicellular spheroids from a lung cancer cell line is enhanced by composite micelle encapsulation. *Int J Nanomedicine*. 2012;7:2661-2671.

[6] Joyner DE, Bastar JD, Randall RL, et al. Doxorubicin induces cell senescence preferentially over apoptosis in the FU-SY-1 synovial sarcoma cell line. *J Orthop Res*. 2006 Jun;24(6):1163-1169.

[7] Ichikawa Y, Ghanefar M, Bayeva M, et al. Cardiotoxicity of doxorubicin is mediated through mitochondrial iron accumulation. *J Clin Invest*. 2014 Feb;124(2):617-630.

[8] Ottewell PD, Monkkonen H, Jones M, et al. Antitumor effects of doxorubicin followed by zoledronic acid in a mouse model of breast cancer. *J Natl Cancer Inst*. 2008 Aug 20;100(16):1167-1178.

[9] Pecoraro M, Del Pizzo M, Marzocco S, et al. Inflammatory mediators in a short-time mouse model of doxorubicin-induced cardiotoxicity. *Toxicol Appl Pharmacol*. 2016 Mar 1;293:44-52.

[10] Sun X, Chen G, Xie Y, et al. Qiliqiangxin improves cardiac function and attenuates cardiac remodelling in doxorubicin-induced heart failure rats. *Pharm Biol*. 2020;58(1):417-426.

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