

Doxorubicin (hydrochloride)

Catalog No: tcsc1239

Available Sizes

Size:	50mg
Size:	100mg
Size:	200mg
Size:	500mg
Size:	1g
	Specifications
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CAS No:

25316-40-9

Formula:

 $\mathsf{C_{27}H_{30}CINO}_{11}$

Pathway:

Antibody-drug Conjugate/ADC Related; Anti-infection; Apoptosis; Autophagy; Cell Cycle/DNA Damage; Epigenetics; PI3K/Akt/mTOR

Target:

ADC Cytotoxin; AMPK; Antibiotic; Apoptosis; Autophagy; Bacterial; HBV; HIV; Mitophagy; Topoisomerase

Form:

Red to orange (Solid)

Purity / Grade:

99.60%

Solubility:

DMSO : 35.71 mg/mL (61.57 mM; ultrasonic and warming and heat to 60°C); H2O : 20 mg/mL (34.48 mM; Need ultrasonic)

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Storage Instruction:

2-8°C, sealed storage, away from moisture and light

Alternative Names:

Adriamycin; Hydroxydaunorubicin HCl

Observed Molecular Weight:

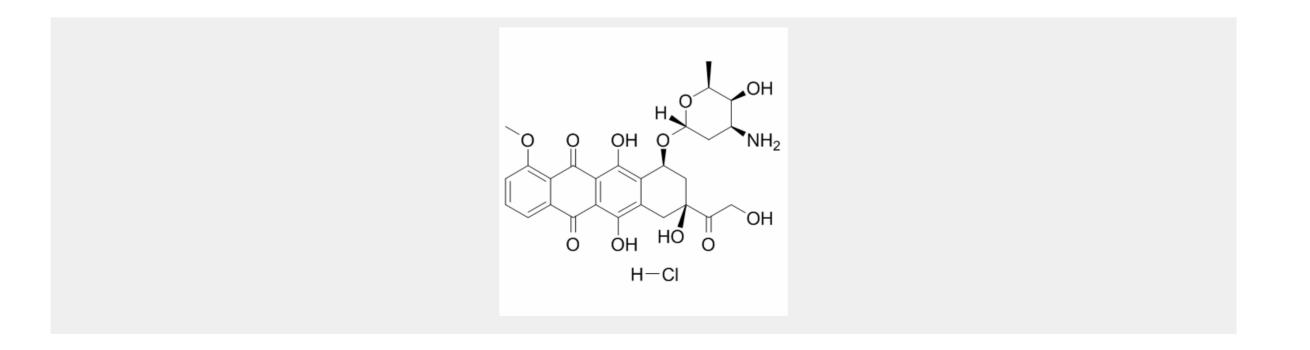
579.98

References

[1]. John L. Nitiss, et al. Targeting DNA topoisomerase II in cancer chemotherapy.Nat Rev Cancer. 2009 May;9(5):338-50. [2]. Hee-KyungRhee,et al. Synthesis, cytotoxicity, and DNA topoisomerase II inhibitory activity of benzofuroquinolinediones. Bioorg Med Chem. 2007 Feb 15;15(4):1651-8. [3]. P D Foglesong, et al. Doxorubicin inhibits human DNA topoisomerase I. Cancer Chemother Pharmacol. 1992;30(2):123-5. [4]. Nesstor Pilco-Ferreto, et al. Influence of doxorubicin on apoptosis and oxidative stress in breast cancer cell lines. Int J Oncol. 2016 Aug;49(2):753-62. [5]. Regine Lüpertz, et al. Dose- and time-dependent effects of doxorubicin on cytotoxicity, cell cycle and apoptotic cell death in human colon cancer cells. Toxicology. 2010 May 27;271(3):115-21. [6]. Penelope D Ottewell, 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):1167-78.

Product Description

Doxorubicin is a widely-used cancer chemotherapy active against a range of cancers including solid tumors, leukemias, and lymphomas. Doxorubicin can bind to topoisomerase II, inhibit DNA and RNA synthesis, as well as produce single stranded DNA breakage and membrane damage. Treatment with 345nM doxorubicin can activate the ATM signaling pathway, shown as the increased pATM-s1981 (at 2-24h post treatment), pNBS1-s343 (at 8-24h), pCHK2-T68 (at 4-24h), pCHK1-s296 (8-24h) in MCF-7 cells, and subsequently up-regulated endogenous Cyclin G2 and its subcellular localization in both MCF10a and MCF7 cells can be observed after treatment with 345nM doxorubicin for 16h or 24h, and potent DSB-induced G1- and G2-phase checkpoint arrest at 24h. Several signaling pathway, including AKT, PI3K, calcineurin, p38 and JNK has been proved to be responsible for doxorubicin toxicity, and p53, p21 and G2/M cell cycle arrest have been shown involved in doxorubicin-induced ROS generation and apoptosis. The autophagy induced by doxorubicin was shown to contributed to doxorubicin cardiotoxicity via decreasing GATA-4 protein expression.



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