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Laborgeräte & Service

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### SZABO-SCANDIC HandelsgmbH

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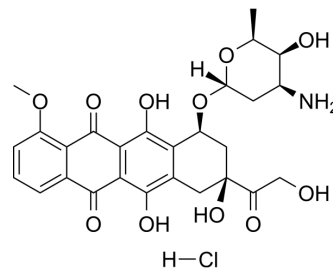
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## Doxorubicin hydrochloride

<b>Cat. No.:</b>	HY-15142
<b>CAS No.:</b>	25316-40-9
<b>Molecular Formula:</b>	C <sub>27</sub> H <sub>30</sub> ClNO <sub>11</sub>
<b>Molecular Weight:</b>	579.98
<b>Target:</b>	ADC Cytotoxin; Autophagy; AMPK; Apoptosis; HBV; HIV; Mitophagy; Bacterial; Antibiotic; Topoisomerase
<b>Pathway:</b>	Antibody-drug Conjugate/ADC Related; Autophagy; Epigenetics; PI3K/Akt/mTOR; Apoptosis; Anti-infection; Cell Cycle/DNA Damage
<b>Storage:</b>	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : 83.33 mg/mL (143.68 mM; Need ultrasonic)  
H<sub>2</sub>O : 50 mg/mL (86.21 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.7242 mL	8.6210 mL	17.2420 mL
	5 mM	0.3448 mL	1.7242 mL	3.4484 mL
	10 mM	0.1724 mL	0.8621 mL	1.7242 mL

Please refer to the solubility information to select the appropriate solvent.

#### In Vivo

- Add each solvent one by one: Saline  
Solubility: 8.33 mg/mL (14.36 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
- Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline  
Solubility: ≥ 2.75 mg/mL (4.74 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
Solubility: ≥ 2.08 mg/mL (3.59 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
Solubility: ≥ 2.08 mg/mL (3.59 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

Doxorubicin (Hydroxydaunorubicin) hydrochloride, a cytotoxic anthracycline antibiotic, is an anti-cancer chemotherapy agent. Doxorubicin hydrochloride is a potent human DNA topoisomerase I and topoisomerase II inhibitor with IC<sub>50</sub>s of 0.8 μM and 2.67 μM, respectively. Doxorubicin hydrochloride reduces basal phosphorylation of AMPK and its downstream target

	acetyl-CoA carboxylase. Doxorubicin hydrochloride induces apoptosis and autophagy <sup>[1][2][3]</sup> .																											
<b>IC<sub>50</sub> &amp; Target</b>	Topoisomerase I 0.8 μM (IC <sub>50</sub> )	Topoisomerase II 2.67 μM (IC <sub>50</sub> )	Daunorubicins/Doxorubicins	HIV-1																								
<b>In Vitro</b>	<p>Doxorubicin hydrochloride (1-8 μM; 24 and 48 hours) decreases the viability of MCF-10F, MCF-7 and MDA-MB-231 cells in a time- and dose-dependent manner<sup>[4]</sup>.</p> <p>Doxorubicin hydrochloride (1 μM; 3 and 24 hours) results in Hct-116 human colon carcinoma cells reduction in G0/G1 phase and accumulation in G2 phase<sup>[5]</sup>.</p> <p>Doxorubicin hydrochloride (1 μM for MCF-10F and MDA-MB-231 cells, 4 μM for MCF-7 cells; 48 hours) induces apoptosis by upregulating Bax, caspase-8 and caspase-3 and downregulation of Bcl-2 protein expression<sup>[4]</sup>.</p> <p>Doxorubicin can label neuron cells, and it is bright red under Rhodamine filter bag, and light red-orange under catecholamine filter bag<sup>[7]</sup>.</p> <p>Doxorubicin (5 μM; 10-30 min) can be accumulated in B16-F10 melanoma cell line CRL-6475 in a time-dependent manner, and can be detected by green or red fluorescence (green fluorescence has higher detection sensitivity) with a maximum excitation wavelength (λ<sub>ex</sub>) and a maximum emission wavelength (λ<sub>em</sub>) of 470 nm and 560 nm, respectively<sup>[8]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Viability Assay<sup>[4]</sup></p> <table border="1"> <tr> <td>Cell Line:</td> <td>Breast cancer cell lines MCF-10F, MCF-7 and MDA-MB-231</td> </tr> <tr> <td>Concentration:</td> <td>0, 1, 2, 4 and 8 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>24 and 48 hours</td> </tr> <tr> <td>Result:</td> <td>IC<sub>50</sub> was 1 μM for both MCF-10F and MDA-MB-231 cell lines. IC<sub>50</sub> was 4 μM for MCF-7 cell line.</td> </tr> </table> <p>Cell Cycle Analysis<sup>[5]</sup></p> <table border="1"> <tr> <td>Cell Line:</td> <td>Hct-116 human colon carcinoma cells</td> </tr> <tr> <td>Concentration:</td> <td>1 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>3 hours and 24 hours</td> </tr> <tr> <td>Result:</td> <td>Both, bolus (3 h) and continuous (24 h) incubation led to a significant reduction of cells in G0/G1 and accumulation in G2 phase.</td> </tr> </table> <p>Western Blot Analysis<sup>[4]</sup></p> <table border="1"> <tr> <td>Cell Line:</td> <td>Breast cancer cell lines MCF-10F, MCF-7 and MDA-MB-231</td> </tr> <tr> <td>Concentration:</td> <td>1 μM for MCF-10F and MDA-MB-231 cells, 4 μM for MCF-7 cells</td> </tr> <tr> <td>Incubation Time:</td> <td>48 hours</td> </tr> <tr> <td>Result:</td> <td>Bax protein expression was upregulated in MCF-10F and MDA-MB-231 cell lines but MCF-7 cells did not show any significant increase. Caspase-8 gene expression was upregulated in MCF-10F, but it was downregulated in MCF-7 and MDA-MB-231 cells.</td> </tr> </table>				Cell Line:	Breast cancer cell lines MCF-10F, MCF-7 and MDA-MB-231	Concentration:	0, 1, 2, 4 and 8 μM	Incubation Time:	24 and 48 hours	Result:	IC <sub>50</sub> was 1 μM for both MCF-10F and MDA-MB-231 cell lines. IC <sub>50</sub> was 4 μM for MCF-7 cell line.	Cell Line:	Hct-116 human colon carcinoma cells	Concentration:	1 μM	Incubation Time:	3 hours and 24 hours	Result:	Both, bolus (3 h) and continuous (24 h) incubation led to a significant reduction of cells in G0/G1 and accumulation in G2 phase.	Cell Line:	Breast cancer cell lines MCF-10F, MCF-7 and MDA-MB-231	Concentration:	1 μM for MCF-10F and MDA-MB-231 cells, 4 μM for MCF-7 cells	Incubation Time:	48 hours	Result:	Bax protein expression was upregulated in MCF-10F and MDA-MB-231 cell lines but MCF-7 cells did not show any significant increase. Caspase-8 gene expression was upregulated in MCF-10F, but it was downregulated in MCF-7 and MDA-MB-231 cells.
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<b>In Vivo</b>	<p>Doxorubicin hydrochloride can be used in animal modeling to construct animal heart failure models.</p> <p>Treatment with Doxorubicin (2 mg/kg) or Zoledronic acid (100 μg/kg) alone does not statistically significantly decrease final tumor volume compared with saline. Mice treated with Doxorubicin plus Zoledronic acid have statistically significantly</p>																											

smaller final tumor volumes than those treated with Doxorubicin alone<sup>[6]</sup>. Doxorubicin (4%-20%; Intrastriatal injection; Single dose) is neurotoxic in Sprague-Dawley mice<sup>[7]</sup>. Doxorubicin can be coupled to gold nanoparticles (Au NPs) by PH-sensitive bonding under acidic conditions, allowing it to pass through the blood-brain barrier with a maximum absorption wavelength of 528 nm<sup>[9]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female MF1 nu/nu mice bearing MDA-G8 breast tumor xenograft (6-week-old) <sup>[6]</sup>
Dosage:	Doxorubicin (2 mg/kg); Zoledronic acid (100 µg/kg)
Administration:	Intravenous injection; once a week; 6 weeks
Result:	Moderate inhibition of subcutaneous tumor growth in mice that were treated with 2 mg/kg Doxorubicin alone or with 100 µg/kg Zoledronic acid alone compared to the saline control. Mice treated with Zoledronic acid and Doxorubicin together had statistically significant smaller mean tumor volumes on day 42 than those treated with Doxorubicin alone.

Animal Model:	Male Sprague-Dawley rats <sup>[7]</sup>
Dosage:	1%, 2%, 4%, 5%, 6%, 10%, 20%
Administration:	Intrastriatal injection; Single dose
Result:	In doses of 4, 5, 6, 10 or 20% caused obvious loss of ipsilateral SNc and VTA neurons and doses of 1 or 2% failed to produce obvious neuron loss.

## CUSTOMER VALIDATION

- Nat Med. 2016 May;22(5):547-56.
- Nature. 2023 Jun;618(7964):374-382.
- Cell Res. 2018 Dec;28(12):1171-1185.
- Signal Transduct Target Ther. 2023 Feb 3;8(1):51.
- Cell Metab. 2022 Feb 7;34(3):424-440.e7.

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## REFERENCES

- [1]. Koda LY, Van der Kooy D. Doxorubicin: a fluorescent neurotoxin retrogradely transported in the central nervous system. *Neurosci Lett*. 1983 Mar 28;36(1):1-8. doi: 10.1016/0304-3940(83)90476-7. PMID: 6190113. 9
- [2]. Kauffman MK, Kauffman ME, Zhu H, Jia Z, Li YR. Fluorescence-Based Assays for Measuring Doxorubicin in Biological Systems. *React Oxyg Species (Apex)*. 2016;2(6):432-439. doi: 10.20455/ros.2016.873. PMID: 29707647; PMCID: PMC5921830.
- [3]. Mirza A Z, Shamshad H. Preparation and characterization of doxorubicin functionalized gold nanoparticles[J]. *European journal of medicinal chemistry*, 2011, 46(5): 1857-1860.
- [4]. John L. Nitiss, et al. Targeting DNA topoisomerase II in cancer chemotherapy. *Nat Rev Cancer*. 2009 May;9(5):338-50.
- [5]. Hee-Kyung Rhee, et al. Synthesis, cytotoxicity, and DNA topoisomerase II inhibitory activity of benzofuroquinolinediones. *Bioorg Med Chem*. 2007 Feb 15;15(4):1651-8.
- [6]. P D Foglesong, et al. Doxorubicin inhibits human DNA topoisomerase I. *Cancer Chemother Pharmacol*. 1992;30(2):123-5.

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[7]. 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.

[8]. 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.

[9]. 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.

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

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