Doxycycline hydrochloride

Cat. No.: HY-N0565A

CAS No.: 10592-13-9

Molecular Formula: $C_{22}H_{25}ClN_2O_8$

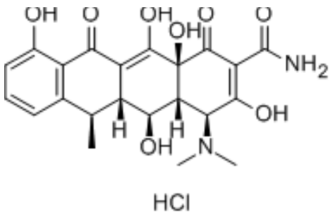
Molecular Weight: 480.9

Target: MMP; Bacterial; Antibiotic; Parasite

Pathway: Metabolic Enzyme/Protease; Anti-infection

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (207.94 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0794 mL	10.3972 mL	20.7943 mL
	5 mM	0.4159 mL	2.0794 mL	4.1589 mL
	10 mM	0.2079 mL	1.0397 mL	2.0794 mL

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

BIOLOGICAL ACTIVITY

Description

Doxycycline hydrochloride, an antibiotic, is an orally active and broad-spectrum metalloproteinase (MMP) inhibitor[1].
Doxycycline hydrochloride shows antibacterial activity and anti-cancer cell proliferation activity[1][2][3][4][5].

IC_{50} & Target

Tetracycline

In Vitro

Doxycycline hydrochloride (0.01-10 μg/mL, 4 d) affects growth of glioma cells only under high concentrations[2].
Doxycycline hydrochloride (0.01-10 μg/mL, 24 h) decreases MT-CO1 protein content with concentrations of 1 μg/mL and higher in SVG cells[2].
Doxycycline hydrochloride (100 ng/mL, 1 μg/mL; 24 h) reduces proliferation of human cell lines[4].
Doxycycline hydrochloride (0-250 μM, 72 h) inhibits cell viability of breast cancer cells [5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	LNT-229, G55, and U343 glioma cells
Concentration:	0.01, 0.1, 1 or 10 μg/mL

Incubation Time:	4 days
Result:	Affected growth of glioma cells only under high concentrations (10 μg/mL).

Cell Viability Assay[2]

Cell Line:	SVG cells
Concentration:	0.01, 0.1, 1 or 10 μg/mL
Incubation Time:	24 hours
Result:	Decreaseed MT-CO1 protein content with concentrations of 1 μg/mL and higher.

Cell Proliferation Assay[4]

Cell Line:	MCF 12A, 293T cells
Concentration:	100 ng/mL, 1 μg/mL
Incubation Time:	96 hours
Result:	Caused reduced proliferation of MCF 12A and 293T cells at 1 μg/mL.

Cell Viability Assay[5]

Cell Line:	MCF-7, MDA-MB-468 cells
Concentration:	0-250 μM
Incubation Time:	72 hours
Result:	Inhibited breast cancer cells in a dose-dependent manner with IC_{50} values for MCF-7 and MDA-MB-468 of 11.39 μM and 7.13 μM respectively.

In Vivo

Doxycycline (oral gavage; 200 or 800 mg/kg; once daily; 3 months) reduces MMP-9 activity in untreated HT mice in a dose-dependent manner[3].
Doxycycline and Tetracycline (HY-A0107), act systemically after absorption from the upper gastrointestinal tract. The main advantage of Doxycycline over Tetracycline is its longer activity, and it can be taken twice or once a day. The peak concentration of both drugs is similar, but in the case of Doxycycline the time to peak concentration is shorter, and half life is significantly longer[6].

Doxycycline (Dox) is often used as an inducer in molecular biology studies to induce gene expression. In cells or model animals that have constructed tetracycline induced expression systems (Tet-On/Tet-Off systems), the expression of target genes can be precisely controlled by adding or removing Dox[7][8][9][10].

Dose reference for Dox induction[7][8][9][10]:
(1) Model animal: male Sprague–Dawley rats
Tet regulatory system：20-3000 ppm of Dox is supplied in diet
(2) Model animal: Cags mice
Tet regulatory system：625 ppm of Dox is supplied in diet
(3) Model animal: Transgenic Wistar rats
Tet regulatory system：2 mg/mL of Dox is supplied in drinking water
(4) Model animal: Transgenic NRMI inbred mice
Tet regulatory system：2 mg/mL of Dox is supplied in drinking water

Dissolution method of Dox[9][10]:
(1) Prepare Dox working solution
Dissolve 100 mg Dox into 50 mL drinking water, add 5% sucrose or 2% saccharin to mask the bitter taste, and refresh the water every three days.
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	6-month-old female Heterozygous Col3a1-deficient (HT) mice[3]
Dosage:	200 or 800 mg/kg
Administration:	Oral gavage; 200 or 800 mg/kg; once daily; 3 months
Result:	Reduced MMP-9 activity in a dose-dependent manner.

CUSTOMER VALIDATION

- Cell. 2023 Feb 2;186(3):591-606.e23.
- Mol Cancer. 2020 Mar 30;19(1):68.
- Mol Cancer. 2020 Sep 9;19(1):139.
- Nat Genet. 2024 Jan 24.
- Nat Microbiol. 2023 Mar;8(3):410-423.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Anna-Luisa Luger, et al. Doxycycline Impairs Mitochondrial Function and Protects Human Glioma Cells from Hypoxia-Induced Cell Death: Implications of Using Tet-Inducible Systems. Int J Mol Sci. 2018 May 17;19(5):1504.

[2]. Ethan Ahler, et al. Doxycycline alters metabolism and proliferation of human cell lines. PLoS One. 2013 May 31;8(5):e64561.

[3]. Le Zhang, et al. Doxycycline inhibits the cancer stem cell phenotype and epithelial-to-mesenchymal transition in breast cancer. Cell Cycle. 2017 Apr 18;16(8):737-745.

[4]. Niv Y. Doxycycline in Eradication Therapy of Helicobacter pylori--a Systematic Review and Meta-Analysis. Digestion. 2016;93(2):167-73.

[5]. Manfredsson FP, et al. Tight Long-term dynamic doxycycline responsive nigrostriatal GDNF using a single rAAV vector. Mol Ther. 2009 Nov;17(11):1857-67.

[6]. Redelsperger IM, et al. Stability of Doxycycline in Feed and Water and Minimal Effective Doses in Tetracycline-Inducible Systems. J Am Assoc Lab Anim Sci. 2016;55(4):467-74.

[7]. Konopka W, et al. Tet system in the brain: transgenic rats and lentiviral vectors approach. Genesis. 2009 Apr;47(4):274-80.

[8]. Kistner A, et al. Doxycycline-mediated quantitative and tissue-specific control of gene expression in transgenic mice. Proc Natl Acad Sci U S A. 1996 Oct 1;93(20):10933-8.

[9]. Wilfried Briest, et al. Doxycycline ameliorates the susceptibility to aortic lesions in a mouse model for the vascular type of Ehlers-Danlos syndrome. J Pharmacol Exp Ther. 2011 Jun;337(3):621-7.

[10]. Eusebio Manchado, et al. A combinatorial strategy for treating KRAS-mutant lung cancer. Nature. 2016 Jun 30;534(7609):647-51.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA