Doxycycline

Cat. No.: HY-N0565

CAS No.: 564-25-0

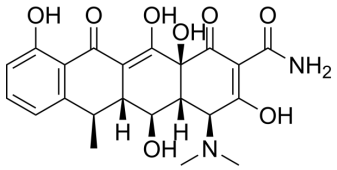
Molecular Formula: $C_{22}H_{24}N_2O_8$

Molecular Weight: 444.43

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

DMSO : 100 mg/mL (225.01 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.2501 mL	11.2504 mL	22.5007 mL
5 mM	0.4500 mL	2.2501 mL	4.5001 mL
10 mM	0.2250 mL	1.1250 mL	2.2501 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

Tetracycline

In Vitro

Doxycycline (0.01-10 μg/mL, 4 d) affects growth of glioma cells only under high concentrations[2].
Doxycycline (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 (100 ng/mL, 1 μg/mL; 24 h) reduces proliferation of human cell lines[4].
Doxycycline (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 concentration (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

Modeling ON-OFF system for gene expression[6][7]

Background

Doxycycline is often used as an inducer in molecular biology research to induce gene expression. In cells or model animals that have constructed a Tetracycline (Tet; HY-A0107) inducible expression (Tet-ON/Tet-OFF) system, the expression of the target gene can be precisely controlled by adding or removing Doxycycline. Doxycycline can act as an inhibitor of transcriptional activation in the Tetracycline (Tc)-controlled transactivation (tTA) system, and as an inducer of transcriptional activation in the "reverse tTA' system. Doxycycline and Tetracycline both act systemically after being absorbed by the upper gastrointestinal tract. In comparison, the main advantage of Doxycycline is that it has a longer activity and can be taken twice or once a day. Although the peak concentrations of the two are similar, Doxycycline takes a shorter time to reach peak

concentration and has a significantly longer half-life [6][7][8].

Specific Mmodeling Methods

Rat[8]: male • adult middle-aged (12-month-old) • Sprague-Dawley rats

Administartion (for GDNF regulation): 3g/kg (dietary with regular food) • po once daily for 6 days • monitored every day

Note

1. In this study[7], a recombinant adeno-associated virus (rAAV)-based bicistronic tetracycline (tet)-OFF construct was used for dynamic control of GDNF (target gene) expression during long-term expression.
2. 3g/kg dietary DOX produced DOX serum levels equivalent to 1mg/ml DOX in drinking water.

Modeling Record

(1) The expression level of the target gene decreases; (2) The positively correlated phenotype corresponding to the target gene is alleviated.

Correlated Product(s):

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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 active MMP-9 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 Feb;56(2):294-305.
- Nat Microbiol. 2023 Mar;8(3):410-423.

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[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]. 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.

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

[4]. 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.

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

[6]. 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.

[7]. 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.

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

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