HBX 41108

Cat. No.: HY-101666

CAS No.: 924296-39-9

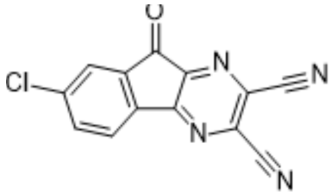
Molecular Formula: $C_{13}H_3ClN_4O$

Molecular Weight: 266.64

Target: Deubiquitinase; Apoptosis; MDM-2/p53

Pathway: Cell Cycle/DNA Damage; Apoptosis

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 : 250 mg/mL (937.59 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7504 mL	18.7519 mL	37.5037 mL
	5 mM	0.7501 mL	3.7504 mL	7.5007 mL
	10 mM	0.3750 mL	1.8752 mL	3.7504 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: ≥ 1 mg/mL (3.75 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

HBX 41108 is an inhibitor of ubiquitin-specific protease 7 (USP7) with an IC_{50} of 424 nM. HBX 41108 inhibits USP7-mediated p53 deubiquitination to stabilize p53 and inhibits cancer cell growth. BX 41108 can be used in cancer and diabetes research [1][2][3][4].

IC_{50} & Target

USP7 424 nM (IC_{50})	hTPH2

In Vitro

HBX 41108 (0-3 μM, 24 h) inhibits the proliferation of tumor cells HCT-116, induces P53 dependent apoptosis and does not affect the activity of normal hepatocytes [1].

HBX 41108 (5 μM, 24 h) can inhibit cell cycle arrest and cell senescence induced by USP7 in HUVECs[3].

HBX 41108 (5-25 μM, 48 h) can enhances the hTPH2 promoter activity in RN46A cells[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HCT-116, NIH-3T3
Concentration:	0-3 μM
Incubation Time:	24 h
Result:	HCT116 colon tumor cells were more sensitive to HBX 41108 (IC_{50} = 0.27 μmol/L) than normal diploid NIH-3T3 fibroblasts (p53 wild-type) with a 7-fold differential effect (IC_{50} = 1.77 μmol/L).

In Vivo

HBX 41108 (100 mg/kg/day for 14 days, i.p.) can promote wound healing and reduce blood sugar levels in diabetic rats [3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Commun Signal. 2023 Nov 9;21(1):319.

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REFERENCES

[1]. Li X, Wang T, et al. Inhibition of USP7 suppresses advanced glycation end-induced cell cycle arrest and senescence of human umbilical vein endothelial cells through ubiquitination of p53. Acta Biochim Biophys Sin (Shanghai). 2022 Mar 25;54(3):311-320.

[2]. Li X, Wang T, et al. Inhibition of USP7 suppresses advanced glycation end-induced cell cycle arrest and senescence of human umbilical vein endothelial cells through ubiquitination of p53. Acta Biochim Biophys Sin (Shanghai). 2022 Mar 25;54(3):311-320.

[3]. Nawa Y, et al. Functional characterization of the neuron-restrictive silencer element in the human tryptophan hydroxylase 2 gene expression. J Neurochem. 2017 Sep;142(6):827-840.

[4]. Colland F, et al. Small-molecule inhibitor of USP7/HAUSP ubiquitin protease stabilizes and activates p53 in cells. Mol Cancer Ther. 2009 Aug;8(8):2286-95.

[5]. Colombo M, et al. Synthesis and biological evaluation of 9-oxo-9H-indeno[1,2-b]pyrazine-2,3-dicarbonitrile analogues as potential inhibitors of deubiquitinating enzymes. ChemMedChem. 2010 Apr 6;5(4):552-8.

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