Prinomastat hydrochloride

Cat. No.: HY-12170A

CAS No.: 1435779-45-5

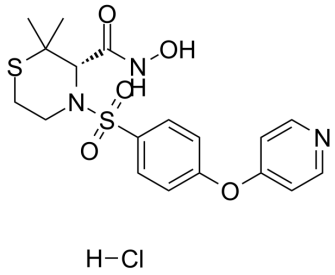
Molecular Formula: $C_{18}H_{22}ClN_3O_5S_2$

Molecular Weight: 459.97

Target: MMP; Apoptosis

Pathway: Metabolic Enzyme/Protease; 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 : 100 mg/mL (217.41 mM; Need ultrasonic)
H_2O : 50 mg/mL (108.70 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.1741 mL	10.8703 mL	21.7405 mL
5 mM	0.4348 mL	2.1741 mL	4.3481 mL
10 mM	0.2174 mL	1.0870 mL	2.1741 mL

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

BIOLOGICAL ACTIVITY

Description

Prinomastat hydrochloride (AG3340 hydrochloride) is a broad spectrum, potent, orally active metalloproteinase (MMP) inhibitor with IC_{50}s of 79, 6.3 and 5.0 nM for MMP-1, MMP-3 and MMP-9, respectively. Prinomastat hydrochloride inhibits MMP-2, MMP-3 and MMP-9 with K_is of 0.05 nM, 0.3 nM and 0.26 nM, respectively. Prinomastat hydrochloride can cross blood-brain barrier. Antitumor avtivity[1][2][3][4].

IC_{50} & Target

MMP-9 5 nM (IC_{50})	MMP-9 0.26 nM (Ki)	MMP-2 0.05 nM (Ki)	MMP-1 79 nM (IC_{50})
MMP-13 6.3 nM (IC_{50})	MMP-13 0.3 nM (Ki)		

In Vitro

Prinomastat (AG3340; 0.1-1 μg/mL; 4 days; C57MG/Wnt1 cells) inhibits Wnt1-induced MMP-3 production. Reversal of Wnt1-induced EMT and β-catenin transcriptional activity by Prinomastat[1].
Co-culture of L/Wnt3a cells and CT7 cells increases the Topflash activity in CT7 cells, and co-culturing both L/Wnt3a cells and MMP-3 overexpressing C57MG cells with CT7 cells increases the Topflash luciferase activity in CT7 cells beyond the level observed with L/Wnt3a cells, and these effects are all suppressed by Prinomastat (AG3340)[1].

Inhibition of entry of C57MG/Wnt1 cells into S phase by Prinomastat corresponds to a decrease in expression of cyclin D1 and Erk1/2 phosphorylation. The effect of Prinomastat on Wnt1-induced migration is then examined using an in vitro wound assay. As anticipated, the migration of C57MG/Wnt1 cells is increased by 1.8-fold when compared with C57MG cells.The effect of Wnt1 on the cellular distribution of vimentin is reversed by Prinomastat in C57MG/Wnt1 cells[1].

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

Western Blot Analysis[1]

Cell Line:	C57MG/Wnt1 cells
Concentration:	0.1 μg/mL, 1 μg/mL
Incubation Time:	4 days
Result:	A significant decrease in MMP-3 promoter activity in C57MG/Wnt1 cells.

In Vivo

In a human fibrosarcoma mouse model (HT1080), the mice are treated therapeutically for 14-16 days with 50 mg/kg/day ip daily starting day 3 to 6 after tumour inoculation. Prinomastat is well tolerated by the animals, and there are no signs of weight loss or other adverse effects. Prinomastat has good tumour growth inhibition, with a short $T_{1/2}$ of 1.6 hours[1].

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

CUSTOMER VALIDATION

- Sci Adv. 2023 Jan 20;9(3):eadd3867.
- J Neuropathol Exp Neurol. 2022 Jun 3;nlac041.

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REFERENCES

[1]. Sørensen MD, et al. Cyclic phosphinamides and phosphonamides, novel series of potent matrix metalloproteinase inhibitors with antitumour activity. Bioorg Med Chem. 2003 Dec 1;11(24):5461-84.

[2]. Blavier L, et al. Stromelysin-1 (MMP-3) is a target and a regulator of Wnt1-induced epithelial-mesenchymal transition (EMT). Cancer Biol Ther. 2010 Jul 15;10(2):198-208.

[3]. Shalinsky DR, et al. Broad antitumor and antiangiogenic activities of AG3340, a potent and selective MMP inhibitor undergoing advanced oncology clinical trials. Ann N Y Acad Sci. 1999 Jun 30;878:236-70.

[4]. Ozerdem U, et al. The effect of prinomastat (AG3340), a potent inhibitor of matrix metalloproteinases, on a subacute model of proliferative vitreoretinopathy. Curr Eye Res. 2000 Jun;20(6):447-53.

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