Prinomastat

Cat. No.: HY-12170

CAS No.: 192329-42-3

Molecular Formula: $C_{18}H_{21}N_3O_5S_2$

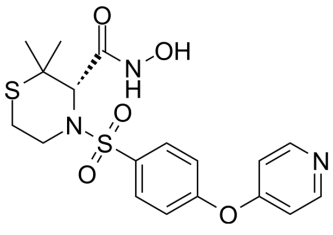
Molecular Weight: 423.51

Target: MMP; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 2.5 mg/mL (5.90 mM; ultrasonic and adjust pH to 4 with 1M HCl)

H_2O : 2.5 mg/mL (5.90 mM; ultrasonic and adjust pH to 3 with HCl)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3612 mL	11.8061 mL	23.6122 mL
5 mM	0.4722 mL	2.3612 mL	4.7224 mL
10 mM	---	---	---

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

BIOLOGICAL ACTIVITY

Description

Prinomastat (AG3340) 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 inhibits MMP-2, MMP-3 and MMP-9 with K_is of 0.05 nM, 0.3 nM and 0.26 nM, respectively. Prinomastat crosses 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-3 6..3 nM (IC_{50})	MMP-3 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.

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