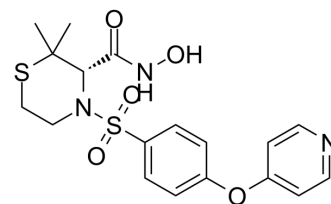


## Prinomastat hydrochloride

<b>Cat. No.:</b>	HY-12170A		
<b>CAS No.:</b>	1435779-45-5		
<b>Molecular Formula:</b>	C <sub>18</sub> H <sub>22</sub> ClN <sub>3</sub> O <sub>5</sub> S <sub>2</sub>		
<b>Molecular Weight:</b>	459.97		
<b>Target:</b>	MMP; Apoptosis		
<b>Pathway:</b>	Metabolic Enzyme/Protease; Apoptosis		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



H-Cl

### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : 100 mg/mL (217.41 mM; Need ultrasonic)  
 H<sub>2</sub>O : 50 mg/mL (108.70 mM; Need ultrasonic)

Concentration	Mass		
	1 mg	5 mg	10 mg
<b>1 mM</b>	2.1741 mL	10.8703 mL	21.7405 mL
<b>5 mM</b>	0.4348 mL	2.1741 mL	4.3481 mL
<b>10 mM</b>	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<sub>50</sub>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<sub>i</sub>s of 0.05 nM, 0.3 nM and 0.26 nM, respectively. Prinomastat hydrochloride can cross blood-brain barrier. Antitumor activity<sup>[1][2][3][4]</sup>.

#### IC<sub>50</sub> & Target

MMP-9 5 nM (IC <sub>50</sub> )	MMP-9 0.26 nM (K <sub>i</sub> )	MMP-2 0.05 nM (K <sub>i</sub> )	MMP-1 79 nM (IC <sub>50</sub> )
MMP-13 6.3 nM (IC <sub>50</sub> )	MMP-13 0.3 nM (K <sub>i</sub> )		

#### 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<sup>[1]</sup>.  
 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)<sup>[1]</sup>. 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<sup>[1]</sup>.

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

#### Western Blot Analysis<sup>[1]</sup>

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<sup>[1]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## 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.**

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