# Prinomastat

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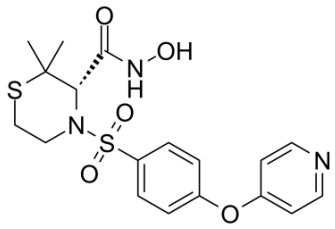
| **Cat. No.:** | HY-12170 |
| **CAS No.:** | 192329-42-3 |
| **Molecular Formula:** | $C_{18}H_{21}N_3O_5S_2$ |
| **Molecular Weight:** | 423.51 |
| **Target:** | MMP |
| **Pathway:** | Metabolic Enzyme/Protease |
| **Storage:** | Powder -20°C 3 years<br>4°C 2 years<br>In solvent -80°C 6 months<br>-20°C 1 month |



## 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 **$K_i$**s of 0.05 nM, 0.3 nM and 0.26 nM, respectively. Antitumor avtivity[1][2][3][4].

**$IC_{50}$ & Target**

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| MMP-9<br>5 nM ($IC_{50}$) | MMP-9<br>0.26 nM (Ki) | MMP-2<br>0.05 nM (Ki) | MMP-1<br>79 nM ($IC_{50}$) |
| MMP-3<br>6..3 nM ($IC_{50}$) | MMP-3<br>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].

**Western Blot Analysis[1]**

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| 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. |

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| **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]. |

## 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.





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