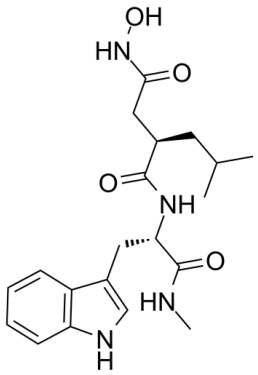
Data Sheet

Product Name:	Ilomastat
Cat. No.:	CS-1701
CAS No.:	142880-36-2
Molecular Formula:	C20H28N4O4
Molecular Weight:	388.46
Target:	MMP
Pathway:	Metabolic Enzyme/Protease
Solubility:	DMSO : ≥ 47 mg/mL (120.99 mM)



BIOLOGICAL ACTIVITY:

Ilomastat (GM6001) is a potent and broad spectrum matrix metalloprotease (**MMP**) inhibitor, inhibits MMPs (IC_{50}s, 1.5 nM for MMP-1; 1.1 nM for MMP-2; 1.9 nM for MMP-3; 0.5 nM for MMP-9), with a K_i of 0.4 nM for human skin fibroblast collagenase (MMP-1). **In Vitro:** Ilomastat (GM6001) inhibits human skin fibroblast collagenase, thermolysin and elastase with K_is of 0.4 nM, 20 nM, 20 nM, resepctively[1]. Ilomastat (0.1-10 nM) inhibits gelatinase A and gelatinase B produced by T-cells. Ilomastat inhibits T-cell homing[4]. **In Vivo:** Ilomastat (GM6001) (400 μg/mL) inhibits corneal ulceration after severe alkali injury in animals[2]. Ilomastat (GM6001) significantly suppresses intimal hyperplasia and intimalcollagen content. Ilomastat increases lumen area in stented arteries, shows no activity on proliferation rates in rabbit model after stenting[3].

PROTOCOL (Extracted from published papers and Only for reference)

Animal Administration: Ilomastat (GM6001) is prepared in placebo.[3] To assess the effects of MMP inhibition, animals are given daily injections of either vehicle ("placebo group") or Ilomastat (GM6001) (100 mg/kg per day as subcutaneous suspension), beginning one day before the second injury until seven days after the procedure. Ilomastat (GM6001) is a nonspecific hydroxamic acid-based MMPI with potent inhibitory activity against collagenase, gelatinases and stromelysin. Animals are euthanized at either 1 week or 10 weeks after the second injury.

References:

[1]. Grobelny D, et al. Inhibition of human skin fibroblast collagenase, thermolysin, and Pseudomonas aeruginosa elastase by peptide hydroxamic acids. Biochemistry. 1992 Aug 11;31(31):7152-4.

[2]. Schultz GS, et al. Treatment of alkali-injured rabbit corneas with a synthetic inhibitor of matrix metalloproteinases. Invest Ophthalmol Vis Sci. 1992 Nov;33(12):3325-31.

[3]. Li C, et al. Arterial repair after stenting and the effects of GM6001, a matrix metalloproteinase inhibitor. J Am Coll Cardiol. 2002 Jun 5;39(11):1852-8.

[4]. Leppert D, et al. T cell gelatinases mediate basement membrane transmigration in vitro. J Immunol. 1995 May 1;154(9):4379-89.

[5]. Yamamoto M, et al. Inhibition of membrane-type 1 matrix metalloproteinase by hydroxamate inhibitors: an examination of the subsite pocket. J Med Chem. 1998 Apr 9;41(8):1209-17.

CAIndexNames:

Butanediamide, N4-hydroxy-N1-[(1S)-1-(1H-indol-3-ylmethyl)-2-(methylamino)-2-oxoethyl]-2-(2-methylpropyl)-, (2R)-

SMILES:

O=C(N[C@@H](CC1=CNC2=C1C=CC=C2)C(NC)=O)[C@H](CC(C)C)CC(NO)=O

Caution: Product has not been fully validated for medical applications. For research use only.

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