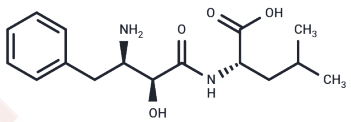
Data Sheet (Cat.No.T1257)

TargetMol

Bestatin

Chemical Properties



CAS No. :	58970-76-6
Formula:	C16H24N2O4
Molecular Weight:	308.37
Appearance:	no data available
Storage:	Pure form: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Bestatin (Ubenimex) competitively inhibits many aminopeptidases, including B, N and leucine aminopeptidases. Ubenimex is a microbial metabolite and dipeptide with potential immunomodulatory and antitumor activities. Aminopeptidases has been implicated in the process of cell adhesion and invasion of tumor cells. Therefore, inhibiting aminopeptidases may partially attribute to the antitumor effect of ubenimex. This agent also activates T lymphocyte, macrophage, and bone marrow stem cell as well as stimulates the release of interleukin-1 and -2, thus further enhances its antitumor activity.
Targets(IC50)	Aminopeptidase,Antibacterial,Antibiotic,LTR
In vitro	In mice bearing B16-BL6 melanoma tumors on their dorsolateral sides, Bestatin reduced the number of blood vessels in established primary tumor masses. Additionally, Bestatin significantly inhibited angiogenesis induced by melanoma cells in the dorsal air sac assay in mice. In EGDA rats, Bestatin decreased the incidence of EAC from 57.7% to 26.1%. Moreover, Bestatin demonstrated statistically significant inhibition of leukotriene B4 biosynthesis in the esophageal tissues of EGDA rats.
In vivo	Bestatin exhibits a concentration-dependent inhibition of SN12M cell invasion into reconstituted basement membrane (matrigel). It also concentration-dependently inhibits the degradation of type IV collagen by tumor cells in a non-neoplastic condition medium. In U937 cells, bestatin enhances the activity of caspase-3, inducing DNA laddering and fragmentation. Additionally, in SN12M, bestatin inhibits the hydrolytic activity of aminopeptidase substrates. Furthermore, bestatin inhibits tube formation in human umbilical vein endothelial cells. Through binding to leucine aminopeptidase fixed on the cell surface, bestatin directly stimulates lymphocytes (and monocytes), whereas it indirectly does so by impeding the degradation metabolism of the phagocytosis stimulating hormones through the inhibition of aminopeptidase B.
Kinase Assay	Cells are harvested, washed, and lysed in NP-40 lysis buffer (50 mM Tris-HCl [pH 7.5], 150 mM NaCl, 0.5% NP-40). Total cell protein is quantified using the Bradford assay and 1-mg/mL protein aliquots are made. Ten microliters of total cell protein is mixed with 290 µL of substrate solution (0.1 mg/mL dithiothreitol [DTT], 0.1 mg/mL albumin, and 1 mM alanine-β-naphthylamide). Fluorometric measurements (340 nm excitation, 400 nm emission) are made after 15 and 30 min. The slope of the line between the 15- and 30-min measurements is used to represent aminopeptidase activity. Total cell protein is preincubated with bestatin, amastatin, puromycin, EDTA, and/or ZnCl2 for 20 min

	before the fluorometric aminopeptidase assay.
Cell Research	Growing cells (1×106 to 2×106 cells/mL) are diluted to 1.0×103 cells/mL and transferred (3 mL) into a well in a 12-well multiwell plate (2.5-cm diameter/well). Cells are treated with 0, 10, 50, 100, 300, or 600 μM Bestatin and allowed to grow at 21°C shaking at 180 rpm for 48 h. A hemocytometer is used to measure cell density after 0, 24, and 48 h.

Solubility Information

Solubility	DMSO: 10 mg/mL (32.43 mM),Sonication is recommended. 1eq. NaOH: 15.4 mg/mL (49.94 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.2429 mL	16.2143 mL	32.4286 mL
5 mM	0.6486 mL	3.2429 mL	6.4857 mL
10 mM	0.3243 mL	1.6214 mL	3.2429 mL
50 mM	0.0649 mL	0.3243 mL	0.6486 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Sekine K, et al. Leukemia, 1999, 13(5), 729-734.
Jiang T Y, Pan Y F, Wan Z H, et al. PTEN status determines chemosensitivity to proteasome inhibition in cholangiocarcinoma. Science Translational Medicine. 2020, 12(562).
Jiang T Y, Feng X F, Fang Z, et al. PTEN deficiency facilitates the therapeutic vulnerability to proteasome inhibitor bortezomib in gallbladder cancer. Cancer Letters. 2021, 501: 187-199.
Yoneda J, et al. Clin Exp Metastasis, 1992, 10(1), 49-59.
Aozuka Y, et al. Cancer Lett, 2004, 216(1), 35-42.
Mathé G, et al. Biomed Pharmacother, 1991, 45(2-3), 49-54.
Chen X, et al. J Natl Cancer Inst, 2003, 95(14), 1053-1061.
Jiang T Y, Feng X F, Fang Z, et al. PTEN deficiency facilitates the therapeutic vulnerability to proteasome inhibitor bortezomib in gallbladder cancer[J]. Cancer Letters. 2020
Jiang T Y, Pan Y F, Wan Z H, et al. PTEN status determines chemosensitivity to proteasome inhibition in cholangiocarcinoma[J]. Science Translational Medicine. 2020, 12(562).