Data Sheet (Cat.No.T2699)



BMS 777607

Chemical Properties

CAS No.: 1025720-94-8

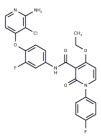
Formula: C25H19ClF2N4O4

Molecular Weight: 512.89

Appearance: no data available

Storage: Revider: 20% for 2 years Un solvent: 20% for 1 years

Powder: -20°C for 3 years | In solvent: -80°C for 1 year



Biological Description

Description	BMS 777607 (BMS 817378) is a Met-related inhibitor targeting c-Met, Axl, Ron, and Tyro3 with IC50 values of 3.9 nM, 1.1 nM, 1.8 nM, and 4.3 nM, respectively. BMS-777607 has been investigated in basic science research for malignant solid tumors.
Targets(IC50)	c-Met/HGFR,TAM Receptor
In vitro	BMS-777607 did not affect tumor cell growth much, it inhibited hepatocyte growth factor-induced cell dispersal in PC-3 and DU145 cells, it also dose-dependently inhibited cell migration and invasion (IC50<0.1 µM).BMS-777607 is a selective ATP-competitive Met kinase inhibitor, it has strong inhibition of c-Met autophosphorylation, the IC50 of GTL-16 cell lysates is 20 nM, and it inhibits Met-driven tumor cell lines such as GTL-16 cell line, H1993 and U87 cells. , with an IC50 of 20 nM for GTL-16 cell lysates and selective inhibition of proliferation of Met-driven tumor cell lines such as GTL-16 cell line, H1993 and U87 cells. In DU145 prostate cancer cells, BMS-777607 inhibited c-Met autophosphorylation induced by hepatocyte growth factor (HGF) (IC50<1 nM).BMS-777607 (1 µM) treatment for 24 h effectively inhibited KHT cell dispersal, motility, and invasion, which was associated with the inhibition of the MET gene, and had a certain effect on cell proliferation and colony formation. This is related to the inhibition of MET gene and has certain effects on cell proliferation and colony formation. In highly metastatic murine KHT cells, BMS-777607 (10 µM) treatment for 2 h effectively cleared the level of autophosphorylated c-Met (IC50: 10 nM) without affecting the whole c-Met, which dose-dependently inhibited downstream signaling molecules including ERK, Akt, p7056K and S6.
In vivo	BMS-777607 did not affect tumor cell growth much, it inhibited hepatocyte growth factor-induced cell dispersal in PC-3 and DU145 cells, it also dose-dependently inhibited cell migration and invasion (IC50<0.1 μ M).BMS-777607 is a selective ATP-competitive Met kinase inhibitor, it has strong inhibition of c-Met autophosphorylation, the IC50 of GTL-16 cell lysates is 20 nM, and it inhibits Met-driven tumor cell lines such as GTL-16 cell line, H1993 and U87 cells. , with an IC50 of 20 nM for GTL-16 cell lysates and selective inhibition of proliferation of Met-driven tumor cell lines such as GTL-16 cell line, H1993 and U87 cells. In DU145 prostate cancer cells, BMS-777607 inhibited c-Met autophosphorylation induced by hepatocyte growth factor (HGF) (IC50<1 nM).BMS-777607 (1 μ M) treatment for 24 h effectively inhibited KHT cell dispersal, motility, and invasion, which was associated with the inhibition of the MET gene, and had a certain

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effect on cell proliferation and colony formation. This is related to the inhibition of MET

	gene and has certain effects on cell proliferation and colony formation. In highly metastatic murine KHT cells, BMS-777607 (10 μ M) treatment for 2 h effectively cleared the level of autophosphorylated c-Met (IC50: 10 nM) without affecting the whole c-Met, which dose-dependently inhibited downstream signaling molecules including ERK, Akt, p70S6K and S6.
Kinase Assay	Met Kinase Assay: The kinase reaction consists of baculovirus expressed GST-Met, 3 μ g of poly(Glu/Tyr), 0.12 μ Ci 33P γ -ATP, 1 μ M ATP in 30 μ L of kinase buffer (20 mM Tris-Cl, 5 mM MnCl2, 0.1 mg/mL BSA, 0.5 mM DTT). Reactions are incubated for 1 hour at 30 °C and stopped by the addition of cold trichloroacetic acid (TCA) to a final concentration of 8%. TCA precipitates are collected onto GF/C unifilter plates using a Filtermate universal harvester, and the filters are quantitated using a TopCount 96-well liquid scintillation counter. Dose response curves are generated to determine the concentration required to inhibit 50% of substrate phosphorylation (IC50). BMS 777607 is dissolved at 10 mM in dimethylsulfoxide (DMSO) and evaluated at 10 concentrations, in duplicate.
Cell Research	KHT cells are exposed to serial dilution of BMS 777607 for 96 hours, then the MTT assay and trypan blue exclusion are used for the determination of cell proliferation and cell death, respectively. KHT cell colonies are incubated with BMS 777607 for 24 hours and then stained with crystal violet (0.1%) and photographed for the assessment of cell scattering. 2 mm scratch on the confluent KHT cell monolayer is made using a sterilized 1 ml pipette tip followed by treated with BMS-777607 for 24 hours, then the number of cells that have migrated into the denuded area is counted on 4 random fields for the evaluation of cell migration. For the examination of cell invasion, the commercial transwell inserts (8 µm pore membrane) pre-loaded with Matrigel are incubated with serum-free medium in the presence or absence of BMS 777607 at 37 °C for 2 hours to allow rehydration of Matrigel. Then cells suspended in serum-free medium are loaded onto the top chamber (5 × 103/insert) and complete medium (containing 10% FBS) is used in the lower chamber as a chemoattractant. After incubation for 24 hours, the Matrigel is removed and the inserts are stained with crystal violet. Invaded cells on the underside of the filter are photographed and counted. (Only for Reference)

Solubility Information

Solubility	Ethanol: < 1 mg/mL (insoluble or slightly soluble),	
	H2O: < 1 mg/mL (insoluble or slightly soluble),	
	DMSO: 44 mg/mL (85.79 mM), Sonication is recommended.	
	(< 1 mg/ml refers to the product slightly soluble or insoluble)	

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.9497 mL	9.7487 mL	19.4974 mL
5 mM	0.3899 mL	1.9497 mL	3.8995 mL
10 mM	0.195 mL	0.9749 mL	1.9497 mL
50 mM	0.039 mL	0.195 mL	0.3899 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.

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Reference

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Kim KS, et al. J Med Chem, 2008, 51(17), 5330-5341.

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