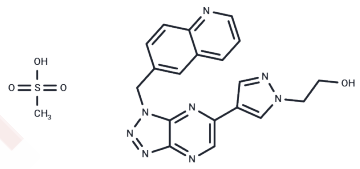
Data Sheet (Cat.No.T12417)

TargetMol

PF-04217903 methanesulfonate

Chemical Properties



CAS No. :	956906-93-7
Formula:	C20H20N8O4S
Molecular Weight:	468.49
Storage:	Powder: -20°C for 3 years | In solvent: -80°C for 1 year Actual storage temperature shall be subject to the COA.

Biological Description

Description	PF-04217903 methanesulfonate is a potent ATP-competitive inhibitor of c-Met kinase (Ki of 4.8 nM for human c-Met).
Targets(IC50)	c-Met/HGFR
In vitro	PF-04217903 induces apoptosis of GTL-16 cells (IC50=31 nM) [1]. PF-04217903 methanesulfonate also inhibits HGF-mediated cell migration and Matrigel invasion in several c-Met-overexpressing tumor cell lines such as human NCI-H441 lung carcinoma and HT29 colon carcinoma with IC50 values comparable with those for inhibition of c-Met phosphorylation in these cell lines (IC50= 7-12.5 nM)[1].
In vivo	PF-04217903 methanesulfonate shows a significant dose-dependent reduction of human IL-8 levels in both the U87MG and GTL-16 models and decreases human VEGFA levels in the GTL-16 model. PF-04217903 methanesulfonate strongly induces phospho-PDGFRβ levels in U87MG xenograft tumors[1].

Solubility Information

Solubility	DMSO: 50 mg/mL (106.73 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.1345 mL	10.6726 mL	21.3452 mL
5 mM	0.4269 mL	2.1345 mL	4.269 mL
10 mM	0.2135 mL	1.0673 mL	2.1345 mL
50 mM	0.0427 mL	0.2135 mL	0.4269 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Timofeevski SL, et al. Enzymatic characterization of c-Met receptor tyrosine kinase oncogenic mutants and kinetic studies with aminopyridine and triazolopyrazine inhibitors. Biochemistry, 2009, 48(23), 5339-5349.
Shojaei F, et al. HGF/c-Met acts as an alternative angiogenic pathway in sunitinib-resistant tumors. Cancer Res, 2010, 70(24), 10090-10100.
Krumbach R, et al. Primary resistance to cetuximab in a panel of patient-derived tumour xenograft models: activation of MET as one mechanism for drug resistance. Eur J Cancer, 2011, 47(8), 1231-1243.