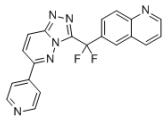
JNJ-38877618

Cat. No.:	HY-111050
CAS No.:	943540-74-7
Molecular Formula:	$C_{20}H_{12}F_2N_6$
Molecular Weight:	374.35
Target:	c-Met/HGFR
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



Solvent & Solubility

In Vitro

DMSO : 5 mg/mL (13.36 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6713 mL	13.3565 mL	26.7130 mL
	5 mM	0.5343 mL	2.6713 mL	5.3426 mL
	10 mM	0.2671 mL	1.3356 mL	2.6713 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: **10% DMSO >> 90% corn oil**
   Solubility: ≥ 0.5 mg/mL (1.34 mM); Clear solution
2. Add each solvent one by one: **10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline**
   Solubility: ≥ 0.5 mg/mL (1.34 mM); Clear solution
3. Add each solvent one by one: **10% DMSO >> 90% (20% SBE-β-CD in saline)**
   Solubility: ≥ 0.5 mg/mL (1.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

JNJ-38877618 is a potent, highly selective, orally bioavailable **Met** kinase inhibitor with **IC_{50}**s of 2 and 3 nM for wild type and mutant Met, respectively.

IC_{50} & Target

IC50: 2 nM (wt Met), 2 nM (mutant Met)[1]

In Vitro

OMO-1 (formerly JNJ-38877618), is a potent, highly selective, orally bioavailable Met kinase inhibitor with nM binding

	affinity (K_d=1.4 nM) and enzyme inhibitory activity against wt and M1268T mutant Met (2 and 3 nM IC_{50}). Met inhibitory effects are assessed in proliferation, colony formation and motility assays. JNJ-38877618 displays nM potency against Met Ampl/mutant and therapy resistant models[1].
In Vivo	JNJ-38877618 induces complete inhibition of tumor growth in 3 models: the SNU5 Met amp gastric, U87-MG HGF autocrine glioblastoma and Hs746T Met exon 14 skipping mutant gastric cancer. JNJ-38877618 induces regression of large Met amplified EBC-1 SqNSCLC where JNJ-38877618 leads to dose- and time-dependent inhibition of Met kinase activation, with the duration of target shut down considerably exceeding plasma exposure times. Combination treatments are well tolerated and improved EGFR targeted therapy[1].

REFERENCES

[1]. Libouban M, et al. OMO-1, a potent, highly selective, orally bioavailable, Met kinase inhibitor with a favorable preclinical toxicity profile, shows both monotherapy activity, against Met pathway-driven tumors, and EGFR TKI combination activity in acquired resistance models [abstract]. In: Proceedings of the American Association for Cancer Research Annual Meeting 2018; 2018 Apr 14-18; Chicago, IL. Philadelphia (PA): AACR; Cancer Res 2018;78(13 Suppl):Abstract nr 4791.

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