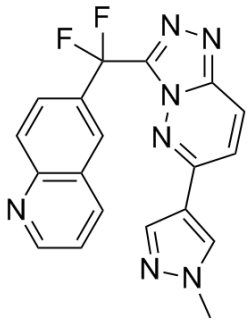
JNJ-38877605

Cat. No.:	HY-50683
CAS No.:	943540-75-8
Molecular Formula:	$C_{19}H_{13}F_2N_7$
Molecular Weight:	377.35
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 : ≥ 30 mg/mL (79.50 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6501 mL	13.2503 mL	26.5006 mL
	5 mM	0.5300 mL	2.6501 mL	5.3001 mL
	10 mM	0.2650 mL	1.3250 mL	2.6501 mL

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

In Vivo

1. Add each solvent one by one: **10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline**
   Solubility: ≥ 2.08 mg/mL (5.51 mM); Clear solution
2. Add each solvent one by one: **10% DMSO >> 90% (20% SBE-β-CD in saline)**
   Solubility: ≥ 2.08 mg/mL (5.51 mM); Clear solution
3. Add each solvent one by one: **10% DMSO >> 90% corn oil**
   Solubility: ≥ 2.08 mg/mL (5.51 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

JNJ-38877605 is an ATP-competitive inhibitor of c-Met with IC50 of 4 nM, 600-fold selective for c-Met than 200 other tyrosine and serine-threonine kinases.IC50 value: 4 nM [1]Target: c-Metin vitro: JNJ-38877605 shows more than 600-fold selectivity for c-Met compared with more than 200 other diverse tyrosine and serine-threonine kinases and also potently inhibits HGF-stimulated and constitutively activated c-Met phosphorylation in vitro. [1] In EBC1, GTL16, NCI-H1993, and MKN45 cells, JNJ-38877605 (500 nM) leads to a significant reduction of phosphorylation of Met and

RON, another key player in invasive growth [2]. A recent study shows that JNJ-38877605 is involved in modulating secretion of IL-8, GROa, uPAR and IL-6 in GTL16 cells [3]. in vivo: In mice bearing established GTL16 xenografts, JNJ-38877605, dosed orally with 40 mg/kg/day for 72 hours, results in a statistically significant decrease in the plasma levels of human IL-8 (from 0.150 ng/mL to 0.050 ng/mL) and GROα (from 0.080 ng/mL to 0.030 ng/mL). While concentrations of uPAR in the blood become reduced to more than 50% at the same dose [3].

CUSTOMER VALIDATION

- **Sci Transl Med**. 2018 Jul 18;10(450). pii: eaaq1093.
- **Harvard Medical School LINCS LIBRARY**

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REFERENCES

[1]. Perera T, et al. JNJ-38877605: a selective Met kinase inhibitor inducing regression of Met-driven tumor models. Presented at the 99th AACR Annual Meeting; 2008 Apr 12-16

[2]. De Bacco F, et al. Induction of MET by ionizing radiation and its role in radioresistance and invasive growth of cancer. J Natl Cancer Inst. 2011 Apr, 103(8), 645-661.

[3]. Torti D, et al. A preclinical algorithm of soluble surrogate biomarkers that correlate with therapeutic inhibition of the MET oncogene in gastric tumors. Int J Cancer. 2012, 130(6), 1357-1366.

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