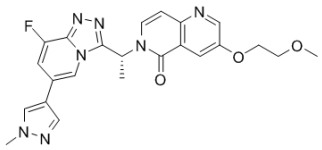
AMG-337

Cat. No.:	HY-18696
CAS No.:	1173699-31-4
Molecular Formula:	$C_{23}H_{22}FN_7O_3$
Molecular Weight:	463.46
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 : 100 mg/mL (215.77 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1577 mL	10.7884 mL	21.5768 mL
	5 mM	0.4315 mL	2.1577 mL	4.3154 mL
	10 mM	0.2158 mL	1.0788 mL	2.1577 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: ≥ 2.5 mg/mL (5.39 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.39 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.39 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AMG-337 is a potent and highly selective small molecule ATP-competitive MET kinase inhibitor. AMG 337 inhibits MET kinase activity with an IC50 of < 5nM in enzymatic assays.IC50 value: < 5nM [1]Target: METin vitro: AMG-337 demonstrates exquisite selectivity for MET when profiled against a diverse panel of over 400 protein and lipid kinases in a competitive binding assay. In cellular assays, AMG 337 inhibits HGF-dependent MET phosphorylation with an IC50 of < 10 nM. [1] AMG 337 is a selective inhibitor of Met, which inhibits multiple mechanisms of Met activation. [2]in vivo: AMG-337 demonstrates robust activity in MET-dependent cancer models. Oral administration of AMG 337 results in robust dose-dependent anti-tumor efficacy in MET amplified gastric cancer xenograft models, with inhibition of tumor growth consistent with the pharmacodynamic

	modulation of MET signaling.[1]

REFERENCES

[1]. Paul E. Hughes, et al. AMG 337, a novel, potent and selective MET kinase inhibitor, has robust growth inhibitory activity in MET-dependent cancer models. Cancer Res October 1, 2014 74; 728

[2]. Cecchi F, et al. Targeting the HGF/Met signaling pathway in cancer therapy. Expert Opin Ther Targets. 2012 Jun;16(6):553-572.

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