Nilotinib

Cat. No.: HY-10159

CAS No.: 641571-10-0

Molecular Formula: $C_{28}H_{22}F_3N_7O$

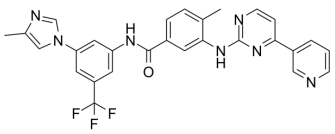
Molecular Weight: 529.52

Target: Bcr-Abl; Autophagy

Pathway: Protein Tyrosine Kinase/RTK; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 12.5 mg/mL (23.61 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8885 mL	9.4425 mL	18.8850 mL
5 mM	0.3777 mL	1.8885 mL	3.7770 mL
10 mM	0.1889 mL	0.9443 mL	1.8885 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 (0.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Nilotinib is an orally available Bcr-Abl tyrosine kinase inhibitor with antineoplastic activity.

IC_{50} & Target

Bcr-Abl[1]

In Vitro

Nilotinib (AMN107), selective Abl inhibitor, is designed to interact with the ATP-binding site of BCR-ABL with a higher affinity than imatinib while being significantly more potent compared with imatinib (IC_{50}<30 nM), also maintains activity against most of the BCR-ABL point mutants that confer Imatinib resistance[1].

Nilotinib demonstrates significant antitumor efficacy against GIST xenograft lines and imatinib-resistant GIST cell lines which parent cell lines GK1C and GK3C shows imatinib sensitivity with IC_{50} of 4.59±0.97 μM and 11.15±1.48 μM, respectively, imatinib-resistant cell lines GK1C-IR and GK3C-IR shows Imatinib resistance with IC_{50} values of 11.74±0.17 μM (P<0.001) and 41.37±1.07 μM (P<0.001), respectively[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Nilotinib (oral gavage, 40 mg/kg, daily, 4 weeks) shows equivalent or higher antitumor effects in BALB/cSLc-nu/nu mice with GIST xenograft[2].

Nilotinib has a significant healing effect on the macroscopic and microscopic pathologic scores and ensures considerable mucosal healing in the indomethacin-induced enterocolitis rat model while decreases the PDGFR α and β levels and apoptotic scores in the colon[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/cSLc-nu/nu mice with GIST xenograft (GK1X, GK2X and GK3X)[2]
Dosage:	40 mg/kg
Administration:	Oral gavage; daily; 4 weeks
Result:	Inhibited tumor growth by 69.6% in GK1X, 85.3% in GK2X and 47.5% in GK3X xenograft line.

CUSTOMER VALIDATION

- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Biomaterials. 16 September 2022.
- Cell Syst. 2018 Apr 25;6(4):424-443.e7.
- Cell Death Dis. 2021 Sep 25;12(10):875.
- Br J Cancer. 2021 Nov 24.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Weisberg E, et al. Beneficial effects of combining nilotinib and imatinib in preclinical models of BCR-ABL+ leukemias. Blood. 2007 Mar 1;109(5):2112-20.

[2]. Sako H, et al. Antitumor effect of the tyrosine kinase inhibitor Nilotinib on gastrointestinal stromal tumor (GIST) and Imatinib-resistant GIST cells. PLoS One. 2014 Sep 15;9(9):e107613.

[3]. Dervis Hakim G, et al. Mucosal healing effect of nilotinib in indomethacin-induced enterocolitis: A rat model. World J Gastroenterol. 2015 Nov 28;21(44):12576-85.

[4]. Fujita KI, et al. Involvement of the Transporters P-Glycoprotein and Breast Cancer Resistance Protein in Dermal Distribution of the Multikinase Inhibitor Regorafenib and Its Active Metabolites. J Pharm Sci. 2017 Sep;106(9):2632-2641.

[5]. Meirson T, et al. Targeting invadopodia-mediated breast cancer metastasis by using ABL kinase inhibitors. Oncotarget. 2018 Apr 24;9(31):22158-22183.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA