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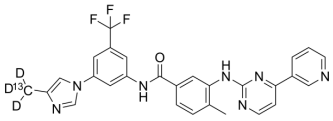
Nilotinib-$^{13}C,d_3$

Cat. No.:	HY-10159S1
CAS No.:	1261398-62-2
Molecular Formula:	$C_{27}{}^{13}CH_{19}D_3F_3N_7O$
Molecular Weight:	533.53
Target:	Bcr-Abl; Autophagy; Isotope-Labeled Compounds
Pathway:	Protein Tyrosine Kinase/RTK; Autophagy; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Nilotinib-13C,d3 is a deuterated labeled Nilotinib[1]. Nilotinib is an orally available Bcr-Abl tyrosine kinase inhibitor with antineoplastic activity.
In Vitro	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1]. 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[2]. 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[3]. 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[3]. 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[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

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

[2]. 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.

[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]. 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.

[6]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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