Product Datasheet

Nilotinib monohydrochloride monohydrate

Catalog No. **BP-02104A** · CAS **923288-90-8** · Form **salt · hydrochloride** · Physical state **other** · Color **white to light yellow** · Version **1.0** · Revision **2026-09-14**

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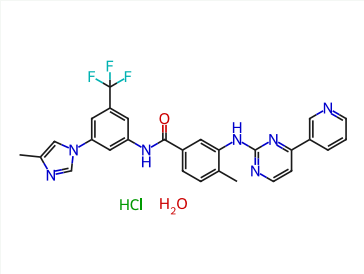
Target	Research area
Bcr-Abl; Autophagy	**Cancer**

Molecular formula	$C_{28}H_{25}ClF_3N_7O_2$
Molecular weight	**583.9920**

STRUCTURE



DESCRIPTION

Nilotinib monohydrochloride monohydrate is a second generation tyrosine kinase inhibitor (TKI), is significantly potent against BCR-ABL, and is active against many BCR-ABL mutants.

BIOLOGICAL ACTIVITY

In vitro: Nilotinib (AMN107) is significantly more potent against BCR-ABL than imatinib, and is active against many imatinib-resistant BCR-ABL mutants. Nilotinib inhibits proliferation, migration, and actin filament formation, as well as the expression of α-SMA and collagen in activated HSCs. Nilotinib induces apoptosis of HSCs, which is correlated with reduced bcl-2 expression, increases p53 expression, cleavage of PARP, as well as increases expression of PPARγ and TRAIL-R. Nilotinib also induces cell cycle arrest, accompanied by increased expression of p27 and downregulation of cyclin D1. Interestingly, Nilotinib not only inhibits activation of PDGFR, but also TGFRII through Src. Nilotinib significantly inhibits PDGF and TGFβ-simulated phosphorylation of ERK and Akt. Furthermore, PDGF- and TGFβ-activated phosphorylated form(s) of Abl in human HSCs are inhibited by Nilotinib. In vivo: exposure of a variety of BCR-ABL+ cell lines to imatinib and nilotinib results in additive or synergistic cytotoxicity, including testing of a large panel of cells expressing BCR-ABL point mutations causing resistance to imatinib in patients. Nilotinib reduces collagen deposition and α-SMA expression in CCl4 and BDL-induced fibrosis. Nilotinib could induce HSC undergoing apoptosis, which is correlated with downregulation of bcl-2. AMN107 prolongs survival of mice injected with Bcr-Abl-transformed hematopoietic cell lines or primary marrow cells, and prolongs survival in imatinib-resistant CML mouse models.

SOLUBILITY

Soluble in DMSO; H2O

In vitro	DMSO : 33.33 mg/mL H2O : 12.9 mg/mL

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.

Tel: +86 20 31130026 · Fax: +86 20 36822386 · tech@biochemprobe.com · www.biochemprobe.com