GNF-2

Cat. No.: HY-11007

CAS No.: 778270-11-4

Molecular Formula: $C_{18}H_{13}F_3N_4O_2$

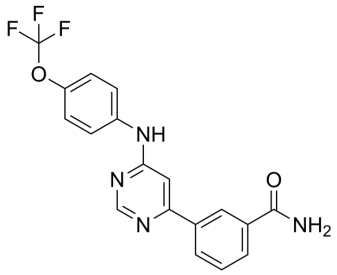
Molecular Weight: 374.32

Target: Bcr-Abl; SARS-CoV

Pathway: Protein Tyrosine Kinase/RTK; Anti-infection

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 : ≥ 100 mg/mL (267.15 mM)

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

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6715 mL	13.3577 mL	26.7154 mL
5 mM	0.5343 mL	2.6715 mL	5.3431 mL
10 mM	0.2672 mL	1.3358 mL	2.6715 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 (6.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.68 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GNF-2 is a highly selective, allosteric, non-ATP competitive inhibitor of Bcr-Abl. GNF-2 inhibits Ba/F3.p210 proliferation with an IC_{50} of 138 nM [1].

IC_{50} & Target

Bcr-Abl

In Vitro

GNF-2 selectively inhibits Bcr-abl-dependent cell proliferation. GNF-2 (0.005-10 μM; 48 hours) specifically inhibits the proliferation of the Bcr-abl-expressing cells with an IC_{50} of 138 nM and not show any cytotoxic effects on the nontransformed cells at concentrations of up to 10 μM. GNF-2 (0.005-10 μM; 48 hours) causes a dose-dependent growth inhibition of the Bcr-abl-positive cell lines with IC_{50} values of 273 nM (K562) and 268 nM (SUP-B15). GNF-2 (0.005-10 μM; 48 hours) inhibits E255V and Y253H mutant Bcr-abl cell growth (IC_{50} values of 268 and 194 nM, respectively)[1].
GNF-2 (1-10 μM; 48 hours) induces apoptosis of Bcr-abl-transformed cells[1].
GNF-2 (0.1-10 μM; 90 minutes) inhibits the cellular tyrosine phosphorylation of Bcr-abl in a dose-dependent manner with an IC_{50} of 267 nM[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	Ba/F3.p210, Ba/F3.p210^{E255V} and Ba/F3.p185^{Y253H} cells
Concentration:	0.005, 0.01, 0.1, 1, 10 μM
Incubation Time:	48 hours
Result:	Inhibited Bcr-abl-transformed cells proliferation.

Apoptosis Analysis[1]

Cell Line:	Ba/F3.p210 and Ba/F3.p210^{E255V} cells
Concentration:	1, 10 μM
Incubation Time:	48 hours
Result:	Increased number of Ba/F3.p210 cells undergoing apoptosis at 1 μM for 48 h. Ba/F3.p210^{E255V} underwent apoptotic death after 48 h incubation in the presence of 1 μM or higher concentration.

Western Blot Analysis[1]

Cell Line:	Ba/F3.p210 and Ba/F3.p210^{E255V} cells
Concentration:	0.1, 1, 10 μM
Incubation Time:	90 minutes
Result:	Decreased the autophosphorylation levels at a concentration of 1 μM and were barely detectable at 10 μM, whereas the level of total Bcr-abl remained unchanged. Induced a significant decrease in the levels of p-Stat5 (at Y694) at 1 μM in Ba/F3.p210 and Ba/F3.p210^{E255V} cells.

In Vivo

GNF-2 (10 mg/kg; i.p. for 8 days) protects LPS (5 mg/kg) induced bone erosion in mice. GNF-2 protects the LPS induced bone loss and abrogates the LPS-induced decreases of bone volume/tissue volume (BV/TV) of LPS-treated mice[2].
GNF-2 prevents the LPS-induced increases of N.Oc/B.Pm, the percentage of Oc.S/BS, and the percentage of ES/BS[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Eight-week-old C57/BL6 mice were administered i.p. injections of LPS (5 mg/kg)[2]
Dosage:	10 mg/kg
Administration:	I.p. injections for 8 days; 1 day before and every day after the LPS injection
Result:	Prevented inflammatory bone destruction in vivo.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2021 Jan 8;49(D1):D1113-D1121.
- Harvard Medical School LINCS LIBRARY

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REFERENCES

[1]. Adrián FJ, et al. Allosteric inhibitors of Bcr-abl-dependent cell proliferation. Nat Chem Biol. 2006 Feb;2(2):95-102.

[2]. Kim HJ, et al. The tyrosine kinase inhibitor GNF-2 suppresses osteoclast formation and activity. J Leukoc Biol. 2013 Oct 15.

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