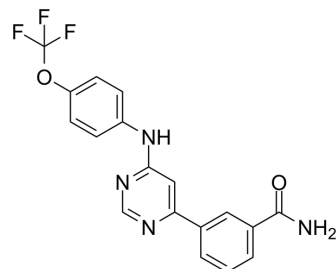


GNF-2

Cat. No.:	HY-11007		
CAS No.:	778270-11-4		
Molecular Formula:	C ₁₈ H ₁₃ F ₃ N ₄ O 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	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (267.15 mM)
 H₂O : < 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	13.3576 mL	26.7151 mL
	5 mM		0.5343 mL	2.6715 mL	5.3430 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

- 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
- 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
- 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 IC₅₀ of 138 nM ^[1].

IC₅₀ & 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.
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Caution: Product has not been fully validated for medical applications. For research use only.

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