GNF-5

Cat. No.: HY-15738

CAS No.: 778277-15-9

Molecular Formula: $C_{20}H_{17}F_3N_4O_3$

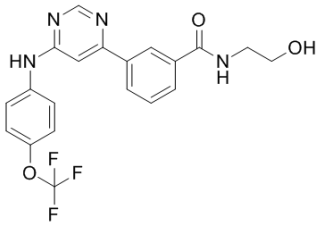
Molecular Weight: 418.37

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 : ≥ 49 mg/mL (117.12 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3902 mL	11.9511 mL	23.9023 mL
	5 mM	0.4780 mL	2.3902 mL	4.7805 mL
	10 mM	0.2390 mL	1.1951 mL	2.3902 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 (5.98 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GNF-5, an analogue of GNF-2 with improved pharmacokinetic properties, is a selective non-ATP competitive inhibitor of Bcr-Abl with an IC50 value of 0.22±0.1 uM (Wild type Abl).IC50 Value: 0.22±0.1 uM (Wild type Abl) [1]Target: Abl GNF-5 is a cell-permeable GNF-2 N-hydroxyethyl carboxamide analog that exhibits in vivo efficacy in suppressing the proliferation of Bcr-abl-expressing Ba/F3 (93% and 83% of no-treatment control, respectively, on days 5 and 7 post treatment; 100 mg/kg b.i.d.) and bone marrow cells (~75% of no-treatment control in both WBC counts and spleen weight on day 7 post treatment; 50 mg/kg b.i.d.) in murine xenograft models of leukemia. Similar to GNF-2, GNF-5 exerts its effect via an allosteric mechanism

(IC50 = 0.22 M against wild-type Abl) by targeting the myristate-binding pocket near the c-terminus of Abl kinase domain and thereby altering the conformational dynamics of the ATP-binding pocket. GNF-5 is ineffective toward the myristate-binding site mutant E505K and the ATP-binding site 'gatekeeper' mutant T315I.

CUSTOMER VALIDATION

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

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Zhang J, et al. Targeting Bcr-Abl by combining allosteric with ATP-binding-site inhibitors. Nature. 2010 Jan 28;463(7280):501-6.

[2]. 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