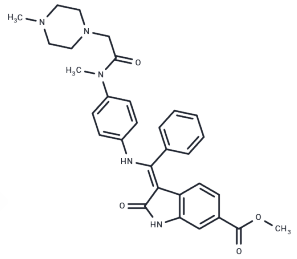
Data Sheet (Cat.No.T1777)

TargetMol

Nintedanib

Chemical Properties



CAS No. : 656247-17-5

Formula: C31H33N5O4

Molecular Weight: 539.62

Appearance: no data available

Storage: keep away from direct sunlight
Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Nintedanib (Intedanib) is a triple vascular kinase inhibitor that inhibits VEGFR1, VEGFR2, and VEGFR3 (IC50=34/13/13 nM), FGFR1, FGFR2, and FGFR3 (IC50=69/37/108 nM), PDGFRα, and PDGFRβ (IC50=59/65 nM). Nintedanib has antitumor activity and inhibits tumor growth by inhibiting angiogenesis.
Targets(IC50)	FGFR,FLT,PDGFR,Src,VEGFR
In vitro	**METHODS**: Human nasopharyngeal carcinoma cells CNE-2, HNE-1 and HONE-1 were treated with Nintedanib (0.078-10 μM) for 72 h, and cell viability was measured by CCK8 assay. **RESULTS**: Nintedanib significantly inhibited the growth of CNE-2, HNE-1 and HONE-1 cell lines in a dose-dependent manner with IC50 values of 4.16 μM, 5.62 μM and 6.32 μM, respectively. [1] **METHODS**: Human endothelial cells HUVEC, smooth muscle cells HUASMC and bovine pericytes were treated with Nintedanib (0.03-1 μM) for 2 h, and the expression levels of target proteins were detected by Western Blot. **RESULTS**: Nintedanib inhibited ligand-dependent phosphorylation of MAPK and Akt in HUVEC, HUASMC and bovine pericytes. [2]
In vivo	**METHODS**: To test the antitumor activity in vivo, Nintedanib (10-100 mg/kg) was administered by gavage to athymic NMRI-nu/nu mice bearing the human head and neck small cell carcinoma tumor FaDu or the human renal carcinoma tumor Caki-1 once daily for 23-35 days. **RESULTS**: Nintedanib inhibited tumor growth in FaDu and Caki-1. [2] **METHODS**: To test the antitumor activity in vivo, Nintedanib (40 mg/kg) and TFTD (150 mg/kg) were administered intraperitoneally to BALB/c nu/nu mice bearing tumors of DLD-1, DLD-1/5-FU, HT-29, or HCT116 twice daily for two weeks. **RESULTS**: At day 15, Nintedanib and TFTD monotherapy resulted in a significant reduction in tumor growth in vivo. The combination therapy exhibited greater anti-tumor activity than the two monotherapies. [3]
Kinase Assay	The cytoplasmic tyrosine kinase domain of VEGFR-2 (residues 797–1355 according to sequence deposited in databank SWISS-PROT P35968) was cloned into pFastBac fused to GST and extracted as described in supplementary methods. Enzyme activity was assayed using standard conditions using a random polymer (Glu/Tyr 4:1) and in the presence of 100 μmol/L ATP (for details, see supplementary methods). For all other kinase assays, the entire cytoplasmic domains of the receptors (from the end of the

	transmembrane to the COOH terminus) were cloned into pFastBac vector containing GST and assayed under standard conditions [1].
Cell Research	HUVEC, HUASMC, and BRP were cultured as described above. Two hours before the addition of ligands, BIBF 1120 was added to the cultures. Cell lysates were generated according to standard protocols. Western blotting was done using standard SDS-PAGE methods, loading 50 to 75 μg of protein per lane, with detection by enhanced chemiluminescence. Total and phosphorylated mitogen-activated protein kinase (MAPK) was analyzed using monoclonal antibodies M3807 and M8159. Total Akt was detected using the polyclonal antibody and phosphorylated Akt (Ser473) was analyzed with the monoclonal antibody. Cleaved caspase-3 was detected with the monoclonal antibody [1].
Animal Research	Five-week-old to 6-wk-old athymic NMRI-nu/nu female mice (21–31 g) were purchased from Harlan. After acclimatization, mice were inoculated with 1 to 5 × 10^6 (in 100 μL) FaDu, Caki-1, SKOV-3, H460, HT-29, or PAC-120 cells s.c. into the right flank of the animal. F344 Fischer rats after acclimatization were injected with 5 × 10^6 (in 100 μL) GS-9L cells s.c. into the right flank of the animal. For pharmacokinetic analysis, blood was isolated at indicated time points from the retroorbital plexus of mice and plasma was analyzed using high-performance liquid chromatography-mass spectrometry methodology [1].

Solubility Information

Solubility	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 0.6 mg/mL (1.11 mM),Solution. Ethanol: 3 mg/mL (5.56 mM),Sonication is recommended. DMSO: 10 mg/mL (18.53 mM),Sonication is recommended. H2O: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.8532 mL	9.2658 mL	18.5316 mL
5 mM	0.3706 mL	1.8532 mL	3.7063 mL
10 mM	0.1853 mL	0.9266 mL	1.8532 mL
50 mM	0.0371 mL	0.1853 mL	0.3706 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Xue C, et al. Efficacy of BIBF 1120 or BIBF 1120 plus chemotherapy on nasopharyngeal carcinoma in vitro and in vivo. Drug Des Devel Ther. 2016 Mar 15;10:1173-80.
Dong X, Wang L, Wang D, et al.Proteomic study on nintedanib in gastric cancer cells.PeerJ.2024, 12: e16771.
Hilberg F, et al. BIBF 1120: triple angiokinase inhibitor with sustained receptor blockade and good antitumor efficacy. Cancer Res. 2008 Jun 15;68(12):4774-82.
Ding X, Yue P, Li X, et al.Evaluation of nintedanib efficacy: Attenuating the lens fibrosis in vitro and vivo. International Immunopharmacology.2024, 136: 112334.
Suzuki N, et al. Effect of a novel oral chemotherapeutic agent containing a combination of trifluridine, tipiracil and the novel triple angiokinase inhibitor nintedanib, on human colorectal cancer xenografts. Oncol Rep. 2016 Dec;36 (6):3123-3130.
Dang Y, Zhao Z, Wang B, et al.Polymeric Polylactic Acid-Glycolic Acid-Based Nanoparticles Deliver Nintedanib Across the Blood-Brain Barrier to Inhibit Glioblastoma Growth.International Journal of Molecular Sciences.2025, 26 (2): 443.