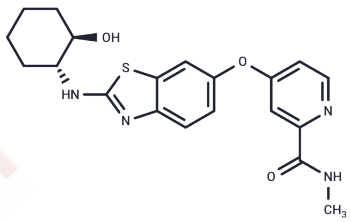
Data Sheet (Cat.No.T6119)

TargetMol

Sotuletinib

Chemical Properties



CAS No. : 953769-46-5

Formula: C20H22N4O3S

Molecular Weight: 398.48

Storage: Keep away from direct sunlight,Keep away from moisture,Store at low temperature
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Sotuletinib (BLZ945) is a CSF-1R inhibitor, a highly selective, brain-penetrant CSF-1R inhibitor (IC50 = 1 nM, with 1000-fold selectivity over other kinases), with oral activity, used for microglia depletion and tumor and neurological disease research.
Targets(IC50)	c-Fms,CSF-1R
In vitro	**Methods**: TGCT cell lines (Si-TGCT-1, -2, -3, -4) and control cells Bewo and MDA-MB-231 were incubated with 0-1000 μM Sotuletinib for 96 hours, and cell viability was detected by MTS assay. **Results**: Sotuletinib inhibited TGCT cell proliferation, induced apoptosis, and increased the BAX/BCL-2 ratio, with IC_{50} values varying with CSF1R expression levels. [1]
In vivo	**Methods**: In a male NSG mouse ccRCC model, Sotuletinib was administered orally by gavage daily at a dose of 200 mg/kg, dissolved in 20% DMSO, for three consecutive weeks.[2] **Results**: Compared to the control group, the Sotuletinib group showed significantly reduced tumor volume, decreased Ki67 expression in tumor tissue, and reduced proportion of M2 macrophages (CD68+CD163+). **Methods**: In a 4T1 tumor-bearing mouse model, Sotuletinib (BLZ-945) was administered by tail vein injection at a dose of 1.75 mg/kg every 3 days, with PBS as the vehicle, combined with 660 nm laser irradiation. **Results**:Tumor growth was significantly suppressed in this treatment group, with the proportion of M2 tumor-associated macrophages in tumor tissue reduced to approximately 19.86%, and the proportion of cytotoxic T lymphocytes in spleen reaching approximately 39.36%.[3] **Methods**: In male Wistar Han rats, Sotuletinib was administered orally by gavage daily at a dose of 150 mg/kg/day, with sodium acetate-methylcellulose solution as the vehicle, continuously for 16 days followed by drug discontinuation. **Results**: ALT elevation occurred during the administration period, which rapidly returned to control levels after drug discontinuation, without accompanying liver injury or miR-122 elevation.[4]
Kinase Assay	Inhibition of biochemical TrkA, TrkB and TrkC: TrkA and TrkC biochemical assays are carried out by HTRF method. The reaction mixtures contains 1 μM peptide substrate, 1 μM ATP, and either 1.8 nM TrkA or 34 nM TrkC in the reaction buffer (50 mM HEPES pH

Kinase Assay	7.1, 10 mM MgCl2, 2 mM MnCl2, 0.01% BSA, 2.5 mM DTT and 0.1 mM Na3VO4) at a final volume of 10 µL. All reactions are carried out at room temperature in white ProxiPlate? 384-well Plus plates and are quenched with 5 µL of 0.2 mM EDTA at 60 min. Five µL of the detection reagents (2.5 ng PT66K and 0.05 µg SAXL per well) are added, the plates are incubated at room temperature for 1 h and then read in EnVision reader. Compounds are diluted into assay mixture (final DMSO 0.5%), and IC50 values are determined by 12-point (from 50 to 0.000282 µM) inhibition curves in duplicate under the assay conditions. TrkB biochemical assay is carried out by caliper microfluidic method. The reaction mixtures contained 1 µM peptide substrate, 10 µM ATP, and 2 nM TrkB in a reaction buffer containing 100 mM HEPES, pH 7.5, 5 mM MgCl2, 0.01% Triton X-100, 0.1% BSA, 1 mM DTT, 10 µMNa3VO4, and 10 µMBeta-Glycerophosphate. The reactions are carried out at room temperature for 3 hrs, and the products are determined by Caliper EZ-reader. Compounds are diluted into assay mixture (final DMSO 1%), and IC50 values are determined by 12-point (from 50 to 0.000282 µM) inhibition curves in duplicate under the assay conditions.
Cell Research	Cell growth rate is determined using the MTT cell proliferation kit. Briefly, cells are plated in triplicate in 96-well plates: 1,000 cells per well for glioma cell lines, 5 x 1,000 cells per well for BMDM and CRL-2467, and 2.5 x 1,000 cells per well for HUVEC and HBMEC cell lines. For all experiments, media is changed every 48 h. Cells are grown in the presence or absence of 6.7-6,700 nM of BLZ945, or 8 µg/mL of CSF-1R neutralizing antibody. BMDM and CRL-2467 cells were supplemented with 10 ng/mL and 30 ng/ mL recombinant mouse CSF-1, respectively. Reduction of the MTT substrate is detected by colorimetric analysis using a plate reader as per the manufacturer's protocol, and measured at 595 nm and 750 nm on a spectraMax 340pc plate reader.(Only for Reference)

Solubility Information

Solubility	DMSO: 257 mg/mL (644.95 mM),Sonication is recommended. Ethanol: 3 mg/mL (7.53 mM),Heating is recommended. H2O: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 5 mg/mL (12.55 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.5095 mL	12.5477 mL	25.0954 mL
5 mM	0.5019 mL	2.5095 mL	5.0191 mL
10 mM	0.251 mL	1.2548 mL	2.5095 mL
50 mM	0.0502 mL	0.251 mL	0.5019 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Thongchot, Suyanee et al. Novel CSF1R-positive tenosynovial giant cell tumor cell lines and their pexidartinib (PLX3397) and sotuletinib (BLZ945)-induced apoptosis. Human cell vol. 36,1 (2023): 456-467.
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Huang C, Ou R, Chen X, et al. Tumor cell-derived SPON2 promotes M2-polarized tumor-associated macrophage infiltration and cancer progression by activating PYK2 in CRC. Journal of Experimental & Clinical Cancer Research. 2021, 40(1): 1-17.
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