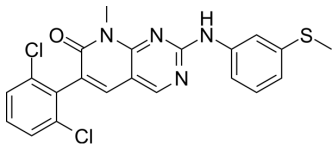
PD173955

Cat. No.:	HY-10395
CAS No.:	260415-63-2
Molecular Formula:	$C_{21}H_{16}Cl_2N_4OS$
Molecular Weight:	443.35
Target:	Bcr-Abl; Src
Pathway:	Protein Tyrosine Kinase/RTK
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 : 10 mg/mL (22.56 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2556 mL	11.2778 mL	22.5555 mL
	5 mM	0.4511 mL	2.2556 mL	4.5111 mL
	10 mM	0.2256 mL	1.1278 mL	2.2556 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 (5.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (2.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PD173955 is an orally active inhibitor of Src (IC_{50}= 22 nM), Yes, Abl, ATP and MAP kinases. PD173955 can effectively prevent the mitotic process and has anticancer activity[1][2][3][4].

IC_{50} & Target

IC50: 22 nM (Src)[1].

In Vitro

PD173955 inhibits cell growth in MDA-MB-468 and MCF-7 breast cancer cells with IC_{50} values of 500 nM and 1 μM, respectively[1].

PD173955 (100 nM; 10 min) significantly inhibits the activity of src and yes kinases in MDA-MB-468 breast cancer cells[1].

PD173955 (5 μM; 24 h) can block the cells in the G_2-M phase and has anti-mitotic activity in MDA-MB-468 breast cancer cells [1].

PD166326 (0.1-1000 nM; 42 h) can inhibit cell proliferation in R10(-) cells with IC_{50} 0.2 nM, and can inhibit stem cell factor (SCF) -dependent proliferation in parental MO7e cells with IC_{50} 12 nM[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	MDA-MB-468
Concentration:	5 μM
Incubation Time:	24 h
Result:	Blocked 95% of the cells in the G_2-M phase.

Cell Proliferation Assay[4]

Cell Line:	MO7e, R10(-)(a cell line derived from P210-MO7e)
Concentration:	0.1 nM, 1 nM, 10 nM, 100 nM, 1000 nM
Incubation Time:	48 h
Result:	Completely blocked the growth of R10 cells at 2.5 nM. Inhibited stem cell factor (SCF) -dependent proliferation at 5-10 nM and completely inhibited SCF-dependent growth at 50 nM.

In Vivo

PD166326 (50 mg/kg; Oral gavage; Twice daily) has anti-leukemia activity in CML mice[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Balb/c mice model of chronic myeloid leukemia (CML)[4]
Dosage:	50 mg/kg
Administration:	Oral gavage (p.o.), Twice daily for 4 days
Result:	Significantly inhibited constitutive tyrosine phosphorylation of numerous proteins (including Lyn) in primary Bcr/abl expressing leukemia cells. Reduced the number of mice with splenomegaly and inhibited splenomegaly in at least half of the mice compared to the control group. Was effective in reducing the peripheral blood granulocytosis of the murine CML-like disease.

CUSTOMER VALIDATION

- Technical University of Munich. 24.01.2018.

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REFERENCES

[1]. Moasser MM, et al. Inhibition of Src kinases by a selective tyrosine kinase inhibitor causes mitotic arrest. Cancer Res. 1999 Dec 15;59(24):6145-52. PMID: 10626805.

[2]. Martinelli G, et al. Dual tyrosine kinase inhibitors in chronic myeloid leukemia. Leukemia. 2005 Nov;19(11):1872-9.

[3]. Netzer WJ, et al. Gleevec shifts APP processing from a β-cleavage to a nonamyloidogenic cleavage. Proc Natl Acad Sci U S A. 2017 Feb 7;114(6):1389-1394.

[4]. Wolff NC, et al. PD166326, a novel tyrosine kinase inhibitor, has greater antileukemic activity than imatinib mesylate in a murine model of chronic myeloid leukemia. Blood. 2005 May 15;105(10):3995-4003.

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