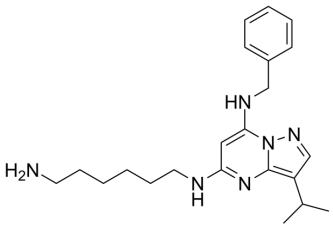
BS-181

Cat. No.:	HY-13266
CAS No.:	1092443-52-1
Molecular Formula:	$C_{22}H_{32}N_6$
Molecular Weight:	380.53
Target:	CDK; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Apoptosis
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 : ≥ 50 mg/mL (131.40 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6279 mL	13.1396 mL	26.2791 mL
5 mM	0.5256 mL	2.6279 mL	5.2558 mL
10 mM	0.2628 mL	1.3140 mL	2.6279 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: ≥ 3 mg/mL (7.88 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (7.88 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (7.88 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BS-181 is a potent and selective CDK7 inhibitor (IC_{50}=21 nM) than Seliciclib (HY-30237). BS-181 is also against CDK2, CDK5 and CDK9 with IC_{50} values of 880, 3000 and 4200 nM, respectively (fails to block CDK1, 4 and 6). BS-181 inhibits a panel of cancer cells growth (IC_{50}=11.5 μM-37.3 μM) and induces cell apoptosis. BS-181 has the potential for the research of cancer therapy[1][2].

IC_{50} & Target	CDK7/CycH/MAT1	CDK2/Cyc E	CDK5/p35NCK	CDK9/cycT

	0.021 μM (IC_{50})	0.88 μM (IC_{50})	3 μM (IC_{50})	4.2 μM (IC_{50})
	CDK1/cycB 8.1 μM (IC_{50})	CDK4/Cyc D1 33 μM (IC_{50})	CDK6/cycD1 47 μM (IC_{50})	

In Vitro

BS-181 (0-40 μM; 72 hours) inhibits cancer cells growth, it is against Breast cancer cell lines growth with IC_{50} values ranging from 15.1 μM to 20 μM, it is against Colorectal cancer cell lines growth with IC_{50} values ranging from 11.5 μM to15.3 μM and is against lung, osteosarcoma, prostate and liver cancer cell lines with IC_{50} values ranging from 11.5 μM to 37.3 μM, respectively[1].

BS-181 (0-50 μM; 4 hours) shows inhibition of phosphorylation of the RNA polymerase II C-terminal domain (CTD) at serine 5 (P-Ser5). It down-regulates CDK4 and cyclin D1 expression while does not effect other CDKs and cyclins[1].

BS-181 (0-50 μM; 24 hours) shows an increase in cells in G1, accompanied by a reduction in cell numbers in S and G2/M at low concentrations. At higher concentrations, however, cells accumulates in the sub-G1, indicative of apoptosis[1].

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

Cell Proliferation Assay[1]

Cell Line:	Breast cancer cell line: MCF-7, MDA-MB-231, T47D, ZR-75-1, etc Colorectal cancer cell line: COLO-205, HCT-116, HCT-116 ($p53^{-/-}$) Lung cancer cell line: A549, NCI-460 Osteosarcoma cancer cell line: U2OS, SaOS2 Prostate cancer cell line: PC3, LNCaP
Concentration:	0-50 μM
Incubation Time:	4 hours
Result:	Had anti-proliferative activities against a panel of cell lines, including breast, lung, prostate and colorectal cancer.

Western Blot Analysis[1]

Cell Line:	MCF-7 cells
Concentration:	0-40 μM
Incubation Time:	72 hours
Result:	Inhibited phosphorylation of CDK7 substrates.

Apoptosis Analysis[1]

Cell Line:	MCF-7 cells
Concentration:	0-50 μM
Incubation Time:	24 hours
Result:	Led cells to G1 arrest and apoptosis.

In Vivo

BS-181 (intraperitoneal injection; 5 mg/kg or 10 mg/kg twice daily; total daily doses of 10 mg/kg or 20 mg/kg; 14 days) inhibitstumor growth in a dose-dependent manner. Tumor growth exhibits 25% and 50% reduction compared with the control group, for 10 mg/kg/day and 20 mg/kg/day, respectively[1].

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

Animal Model:	7-week old female nu/nu-BALB/c athymic nude mice with MCF-7 cells[1]

Dosage:	5 mg/kg or 10 mg/kg; 10 mg/kg or 20 mg/kg
Administration:	Intraperitoneal injection; twice daily or once total daily; 14 days
Result:	Inhibited tumor growth significantly.

CUSTOMER VALIDATION

- Theranostics. 2017 Apr 20;7(7):1914-1927.
- Cell Rep. 2017 Dec 5;21(10):2796-2812.
- Biochem Biophys Res Commun. 2019 Jun 11;513(4):967-973.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Ali S et al. The development of a selective cyclin-dependent kinase inhibitor that shows antitumor activity. Cancer Res. 2009 Aug 1;69(15):6208-15.

[2]. Wang BY, et al. Selective CDK7 inhibition with BS-181 suppresses cell proliferation and induces cell cycle arrest and apoptosis in gastric cancer. Drug Des Devel Ther. 2016 Mar 16;10:1181-9.

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