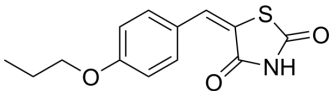
SMI-16a

Cat. No.:	HY-101947
CAS No.:	587852-28-6
Molecular Formula:	$C_{13}H_{13}NO_3S$
Molecular Weight:	263.31
Target:	Pim
Pathway:	JAK/STAT Signaling
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 : ≥ 100 mg/mL (379.78 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7978 mL	18.9890 mL	37.9781 mL
	5 mM	0.7596 mL	3.7978 mL	7.5956 mL
	10 mM	0.3798 mL	1.8989 mL	3.7978 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 (9.49 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.49 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	SMI-16a is a selective Pim kinase inhibitor with IC_{50} values of 0.15, 0.02 and 48 μM for Pim1, Pim2 and PC3 cells, respectively.	
IC_{50} & Target	PIM1	PIM2
In Vitro	SMI-16a has excellent potency for inhibition of both Pim-1 and Pim-2[1]. Treatment with Pim-2 short-interference RNA as well as the Pim inhibitor SMI-16a successfully restores osteoblastogenesis suppressed by all the above inhibitory factors and	

	MM cells. The SMI-16a treatment potentiates BMP-2-mediated anabolic signaling while suppressing TGF-β signaling[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Mice tolerate intraperitoneal dose of SMI-16a is 50 mg/kg daily for 5 days, while 100 mg/kg is overtly toxic. Treatment of the animals with SMI-16a for 5 days per week reduces the growth of tumors by approximately 50% and does not cause a loss of body weight. Subchronic dosing with SMI-16a does not affect the levels of red, white blood cells, including lymphocytes, monocytes, and granulocytes, indicating that the compound does not have myelosuppressive effects. SMI-16a does not have toxicity toward the liver as the albumin, alkaline phosphatase, and alanine aminotransferase levels are unchanged [1]. SMI-16a effectively prevents bone destruction while suppressing MM tumor growth in MM animal models[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Recombinant human Pim-1 (Upstate) is incubated with S6 kinase/Rsk-2 peptide 2 (KKRNRTLTK) as the substrate in the presence 100 μM of compounds from the screening library, 1 μM ATP and 10 mM MgCl2 for 1 h. The Kinase-Glo luciferase kit is used to measure residual ATP levels after the kinase reaction[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	Human prostate cancer PC3 cells are seeded in 96-well tissue culture dishes at approximately 10% confluency and allowed to attach and recover for 24 h. Varying concentrations of the test compounds (SMI-16a) are then added to each well, and the plates are incubated for an additional 48 h. The number of surviving cells is determined by the MTS assay. The percentage of cells killed is calculated as the percentage decrease in MTS metabolism compared with control cultures[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice: Female Balb/C mice are injected subcutaneously with JC cells suspended in PBS. After palpable tumor growth, animals are treated five days per week by intraperitoneal injection of vehicle alone or 50 mg/kg of SMI-16a.Whole body weights and tumor volume measurements are performed three times per week[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cancers (Basel). 2023, 15(1), 67.
- Research Square Print. October 24th, 2022.

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REFERENCES

[1]. Xia Z, et al. Synthesis and evaluation of novel inhibitors of Pim-1 and Pim-2 protein kinases. J Med Chem. 2009 Jan 8;52(1):74-86.

[2]. Hiasa M, et al. Pim-2 kinase is an important target of treatment for tumor progression and bone loss in myeloma. Leukemia. 2015 Jan;29(1):207-17.

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