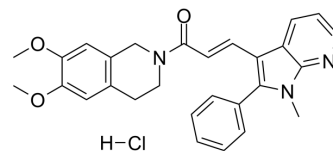


SIS3

Cat. No.:	HY-13013
CAS No.:	521984-48-5
Molecular Formula:	C ₂₈ H ₂₈ ClN ₃ O ₃
Molecular Weight:	489.99
Target:	TGF-beta/Smad
Pathway:	Stem Cell/Wnt; TGF-beta/Smad
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 125 mg/mL (255.11 mM; Need ultrasonic)					
	H ₂ O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)					
	Preparing Stock Solutions	Solvent	Mass	1 mg	5 mg	10 mg
		Concentration				
		1 mM		2.0409 mL	10.2043 mL	20.4086 mL
5 mM			0.4082 mL	2.0409 mL	4.0817 mL	
	10 mM		0.2041 mL	1.0204 mL	2.0409 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: ≥ 2.08 mg/mL (4.24 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	(E)-SIS3 is a potent and selective inhibitor of Smad3 with an IC ₅₀ of 3 μM for Smad3 phosphorylation. (E)-SIS3 inhibits the myofibroblast differentiation of fibroblasts by TGF-β1 ^[1] .
IC₅₀ & Target	IC ₅₀ : 3 μM (Smad3 phosphorylation) ^[1]
In Vitro	(E)-SIS3 (0.3-10 μM; for 1 hour) attenuates the TGF-beta1-induced phosphorylation of Smad3 and interaction of Smad3 with Smad4 ^[1] . (E)-SIS3 (0.1, 10, 50 μM; 30 min) significantly suppresses the expression of FN and α-SMA, but not that of Sphk2 provoked by TGF-β1 ^[2] . (E)-SIS3 (10 μM; 24 hours) significantly reduces both α-SMA and palladin expression that is enhanced by TWEAK in Primary human dermal fibroblasts ^[3] . (E)-SIS3 significantly inhibits L4 development at five concentrations from as low as 2 μM to 50 μM (5 μM, 10 μM, 20 μM and 50 μM) in a dose-dependent manner ^[4] .

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

Western Blot Analysis^[1]

Cell Line:	Human dermal fibroblasts
Concentration:	0.3, 1, 3, 10 μ M
Incubation Time:	For 1 hour
Result:	Attenuated the TGF-beta1-induced phosphorylation of Smad3 and interaction of Smad3 with Smad4.

CUSTOMER VALIDATION

- Brain Behav Immun. 2021 Mar 15;S0889-1591(21)00115-X.
- Nucleic Acids Res. 2023 Feb 2;gkad043.
- Theranostics. 2021 May 13;11(14):7110-7125.
- Cell Death Differ. 2021 Mar;28(3):1001-1012.
- Redox Biol. 2023 Jun, 102709.

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REFERENCES

- [1]. Jinnin M et al. Characterization of SIS3, a novel specific inhibitor of Smad3, and its effect on transforming growth factor-beta1-induced extracellular matrix expression. Mol Pharmacol. 2006 Feb;69(2):597-607.
- [2]. Zhu X, et al. Sphingosine kinase 2 cooperating with Fyn promotes kidney fibroblast activation and fibrosis via STAT3 and AKT. Biochim Biophys Acta Mol Basis Dis. 2018 Nov;1864(11):3824-3836.
- [3]. Liu J, et al. Topical TWEAK Accelerates Healing of Experimental Burn Wounds in Mice. Front Pharmacol. 2018 Jun 21;9:660.
- [4]. Li FF, et al. Identification and characterization of an R-Smad homologue (Hco-DAF-8) from Haemonchus contortus. Parasit Vectors. 2020 Apr 3;13(1):164.

Caution: Product has not been fully validated for medical applications. For research use only.

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