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Zuschläge

- Mindermengenzuschlag
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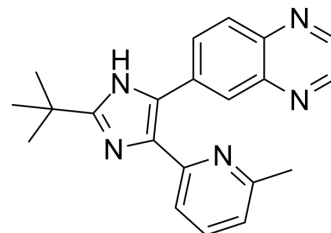
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SB 525334

Cat. No.:	HY-12043
CAS No.:	356559-20-1
Molecular Formula:	C ₂₁ H ₂₁ N ₅
Molecular Weight:	343.42
Target:	TGF-β Receptor
Pathway:	TGF-beta/Smad
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 (291.19 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		2.9119 mL	14.5594 mL	29.1189 mL
		5 mM		0.5824 mL	2.9119 mL	5.8238 mL
		10 mM		0.2912 mL	1.4559 mL	2.9119 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 (7.28 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (7.28 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	SB 525334 is a potent and selective transforming growth factor β1 receptor (ALK5) inhibitor with an IC ₅₀ of 14.3 nM.
IC ₅₀ & Target	IC ₅₀ : 14.3 nM (ALK5) ^[1]
In Vitro	SB525334 (1 μM; for 15 minutes before stimulating with 0.625 ng/ml of TGF-β1, assesses after 6 days) inhibits TGF-β1-mediated proliferation of familial idiopathic pulmonary arterial hypertension (iPAH) pulmonary artery smooth muscle cells (PASCs) at an IC₅₀ of 295 nM ^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Cell Proliferation Assay^[2]

Cell Line:	PASMC cells
Concentration:	1 μ M
Incubation Time:	Pre-incubated for 15 minutes (before stimulating with 0.625 ng/ml of TGF- β 1), assessed after 6 days
Result:	Inhibited TGF- β 1-mediated proliferation of familial iPAH PASMCs at an IC ₅₀ of 295 nM.

In Vivo

SB525334 (3-30 mg/kg; p.o.; daily from days 17 to 35) significantly reverses pulmonary arterial pressure in a rat model of pulmonary arterial hypertension (PAH)^[2].

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

Animal Model:	Adult male Sprague-Dawley rats (MCT rat model of pulmonary hypertension) ^[2]
Dosage:	3, 30 mg/kg
Administration:	Oral administration; daily from days 17 to 35
Result:	Reduced the proportion of fully muscularized vessels to 28% at 3 mg/kg and returned fully muscularized vessel distribution beyond that seen at day 17 and approaching the phenotype observed in saline-exposed controls at 30 mg/kg.

CUSTOMER VALIDATION

- J Control Release. 2021 Jun 10;S0168-3659(21)00299-6.
- Cell Commun Signal. 2018 Dec 7;16(1):97.
- Biomed Pharmacother. 2023 Jul 28;165:115225.
- Glia. 2014 Feb;62(2):185-98.
- Mol Med Rep. 2022 Nov;26(5):330.

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REFERENCES

- [1]. Grygielko ET, et al. Inhibition of gene markers of fibrosis with a novel inhibitor of transforming growth factor-beta type I receptor kinase in puromycin-induced nephritis. J Pharmacol Exp Ther, 2005, 313(3), 943-951.
- [2]. Thomas M, et al. ALK5 mediates abnormal proliferation of vascular smooth muscle cells from patients with familial pulmonary arterial hypertension and is involved in the progression of experimental pulmonary arterial hypertension induced by monocrotaline.
- [3]. Laping NJ, et al. Tumor-specific efficacy of transforming growth factor-beta RI inhibition in Eker rats. Clin Cancer Res, 2007, 13(10), 3087-3899.

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

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