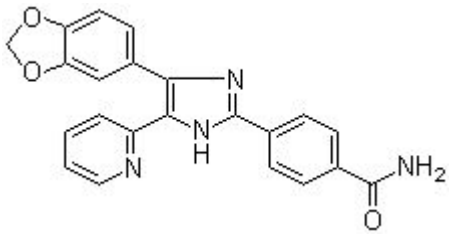
Product Introduction

SB431542

SB431542 is a potent and selective inhibitor of **ALK5** with **IC50** of 94 nM, 100-fold more selective for ALK5 than p38 MAPK and other kinases.

Technical Data:

Molecular Weight (MW):	384.39	
Formula:	C22H16N4O3	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 77 mg/mL	
	Water <1 mg/mL	
	Ethanol 3 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	301836-41-9	

Biological Activity

SB 431542 inhibits the activin type I receptor ALK4 and the nodal type I receptor ALK7, which are responsible for the phosphorylation of Smad2. SB 431542 has little effect on ALK1, ALK2, ALK3, and ALK6, which show phosphorylation of Smad1. SB 431542 is a selective inhibitor of endogenous activin but has no apparent effect on BMP signaling. SB 431542 could induce both Smad2/Smad4- and Smad3/Smad4-dependent transcription. [2] In A498 cells, SB 431542 inhibits both TGF-β1-induced collagen Iα1 and PAI-1 mRNA with IC50 of 60 nM and 50 nM, respectively. In addition, SB 431542 inhibits

production of TGF-β1-induced fibronectin mRNA and protein with IC50 of 62 nM and 22 nM, respectively. [3] SB 431542 blocks the TGF-β-mediated growth factors, including PDGF-A, FGF-2 and HB-EGF, leading to an increase in proliferation of MG63 cells. SB 431542 also inhibits TGF-β-induced c-Myc and p21 WAF1/CIP1. [4] SB 431542 significantly suppresses TGF-β-induced G1 arrest, leading to accumulation of cells in the S phase of the cell cycle in FET, RIE, and Mv1Lu cells. SB 431542 also inhibits TGF-β-induced epithelial to mesenchymal transition (EMT) in NMuMG and PANC-1 cells. [5] SB 431542 significantly elevates the expression of CD86 in BM-DCs and that of CD83 within CD11c+ cells suppressed by TGF-β. SB 431542 is able to induce NK activity through functional maturation and IL-12 production of human DCs. [6]

SB 431542 triggers cytotoxic T lymphocyte (CTL) activities in the colon-26 carcinoma models and is most likely to produce antitumor immunological outcomes through alteration of DC function suppressed by TGF-β. [6]

References

[1] Callahan JF, et al. J Med Chem, 2002, 45(5), 999-1001.

[2] Inman GJ, et al. Mol Pharmacol, 2002, 62(1), 65-74.

[3] Laping NJ, et al. Mol Pharmacol, 2002, 62(1), 58-64.

[4] Matsuyama S, et al. Cancer Res, 2003, 63(22), 7791-7798.

[5] Halder SK, et al. Neoplasia, 2005, 7(5), 509-521.

[6] Tanaka H, et al. Oncol Rep, 2010, 24(6), 1637-1643.

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