

Product Data Sheet

Product Name: PP 1
Cat. No.: GC17990

Chemical Properties

Cas. No. 172889-26-8

Chemical Name 1-tert-butyl-3-(4-methylphenyl)pyrazolo[3,4-d]pyrimidin-4-amine

SMILES CC1=CC=C(C=C1)C2=NN(C3=C2C(=NC=N3)N)C(C)(C)C

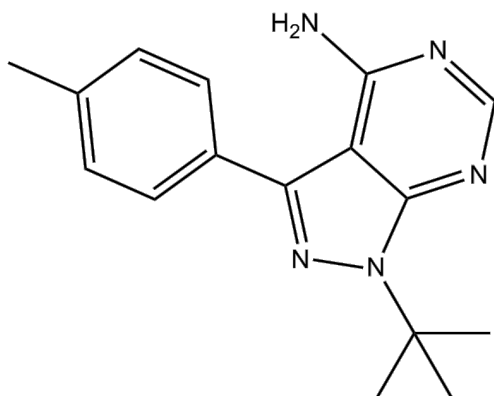
Formula $C_{16}H_{19}N_5$ M.Wt 281.36

Solubility $\geq 7.03\text{mg/mL}$ in DMSO, ≥ 20.6 mg/mL in EtOH with ultrasonic Storage Desiccate at 4°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines NMuMG/E9 cells (mouse mammary epithelial cell line)

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Preparation Method	NMuMG/E9 cells were maintained in Dulbecco's modified Eagle's medium (DMEM) supplemented with 10% fetal bovine serum, 4.5g/L glucose, and 10µg/mL insulin. NMuMG/E9 cells were pretreated with PP 1 (10µM) for 30 minutes, followed by stimulation with TGF-β1 (1-5ng/mL) for up to 2 days.
Reaction Conditions	10µM; 30min pretreatment + TGF-β1 (1-5ng/mL) stimulation for up to 2 days
Applications	PP 1 significantly prevented TGF-β1-mediated epithelial to mesenchymal transition (EMT) in NMuMG/E9 cells. PP 1 effectively inhibited TGF-β1-induced dissolution of cell-cell junctions and formation of stress fibers. Additionally, PP 1 suppressed the TGF-β1-induced expression of mesenchymal markers (such as N-cadherin and fibronectin) and the reduction of the epithelial marker ZO-1. PP 1 blocked the TGF-β1-induced transcriptional upregulation of N-cadherin, Snail, and SIP1, and inhibited the activation (tyrosine phosphorylation) of Src and its downstream substrates (including FAK, paxillin, and p120 catenin).
Animal experiment [2]:	
Animal models	Mice (subjected to unilateral ureteral obstruction - UUO)
Preparation Method	Mice were administered PP 1 (2mg/kg) via daily intraperitoneal injection for 7 or 14 days. Sham-operated and UUO vehicle-treated mice were used as controls. Mice were sacrificed at specified time points (days 3, 7, and 14) for tissue collection and analysis.
Dosage form	2mg/kg; i.p.; daily injection for 3-14 days

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Applications

PP 1 administration significantly reduced the deposition of extracellular matrix (ECM) proteins, as evidenced by Masson trichrome staining. PP 1 significantly inhibited the expression of fibronectin, collagen I, and α -smooth muscle actin (α -SMA), thereby attenuating UUO-induced renal fibrosis and activation of renal interstitial fibroblasts. PP 1 reduced the number of renal epithelial cells arrested at the G2/M phase of the cell cycle.

References:

- [1] Maeda M, Shintani Y, Wheelock MJ, et al. Src activation is not necessary for transforming growth factor (TGF)- β -mediated epithelial to mesenchymal transitions (EMT) in mammary epithelial cells. PP1 directly inhibits TGF- β receptors I and II. J Biol Chem. 2006 Jan 6;281(1):59-68.
- [2] Yan Y, Ma L, Zhou X, et al. Src inhibition blocks renal interstitial fibroblast activation and ameliorates renal fibrosis. Kidney Int. 2016 Jan;89(1):68-81.

Background

PP 1 is a novel, potent, and selective Src family tyrosine kinase inhibitor. PP 1 effectively inhibits the phosphorylation of Lck ($IC_{50}=5nM$) and Fyn ($IC_{50}=6nM$) to block T-cell receptor signaling pathways. PP 1 can be used in research related to cancers (such as small cell lung cancer and leukemia) as well as T-cell immune signal transduction^[1-4].

In vitro, pretreatment of NMuMG/E9 mouse mammary epithelial cells with PP 1 (10 μ M) for 30min, followed by stimulation with TGF- β 1 (1-5ng/mL) for 1-2 days. PP 1 significantly inhibited the occurrence of epithelial-mesenchymal transition (EMT), while preventing alterations in cell morphology, focal adhesion formation, and the activation of Smad2/3^[5]. Pretreatment of human coronary artery smooth muscle cells (HCASMCs) and Swiss 3T3 cells with PP 1 (0.1–10 μ M) for 15min–4h, followed by stimulation with PDGF-BB (10–15ng/mL) for 4–20h. PP 1 significantly inhibited cell migration and proliferation, while reducing the tyrosine phosphorylation level of PDGF β receptors^[6].

In vivo, a single intraperitoneal injection of PP 1 (1.5mg/kg) was administered to treat the transient middle cerebral artery occlusion (tMCAO) mouse model. PP 1 significantly rescued the FUNDC1-

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mediated neuronal mitophagy dysfunction caused by I/R injury^[7]. Daily intraperitoneal injection of PP 1 (2mg/kg) was administered to the unilateral ureteral obstruction (UUO)-induced renal fibrosis mouse model for 3-14 days. PP 1 significantly reduced the deposition of extracellular matrix and inhibited the activation of renal interstitial fibroblasts, thereby ameliorating the development of renal fibrosis^[8].

References:

- [1] Zan L, Zhang X, Xi Y, et al. Src regulates angiogenic factors and vascular permeability after focal cerebral ischemia-reperfusion. *Neuroscience*. 2014 Mar 14;262:118-28.
- [2] Warmuth M, Simon N, Mitina O, et al. Dual-specific Src and Abl kinase inhibitors, PP1 and CGP76030, inhibit growth and survival of cells expressing imatinib mesylate-resistant Bcr-Abl kinases. *Blood*. 2003 Jan 15;101(2):664-72.
- [3] Karni R, Mizrahi S, Reiss-Sklan E, et al. The pp60c-Src inhibitor PP1 is non-competitive against ATP. *FEBS Lett*. 2003 Feb 27;537(1-3):47-52.
- [4] Kantibekovna SL, Wang S, Kang H, et al. Selective antitumor and apoptosis-inducing effects of the Src inhibitor PP1 in human tongue squamous cell carcinoma cells. *Int J Oncol*. 2026 May;68(5):64.
- [5] Maeda M, Shintani Y, Wheelock MJ, et al. Src activation is not necessary for transforming growth factor (TGF)-beta-mediated epithelial to mesenchymal transitions (EMT) in mammary epithelial cells. PP1 directly inhibits TGF-beta receptors I and II. *J Biol Chem*. 2006 Jan 6;281(1):59-68.
- [6] Waltenberger J, Uecker A, Kroll J, et al. A dual inhibitor of platelet-derived growth factor beta-receptor and Src kinase activity potently interferes with mitogenic and mitogenic responses to PDGF in vascular smooth muscle cells. A novel candidate for prevention of vascular remodeling. *Circ Res*. 1999 Jul 9;85(1):12-22.
- [7] Tang T, Hu LB, Ding C, et al. Src inhibition rescues FUNDC1-mediated neuronal mitophagy in ischaemic stroke. *Stroke Vasc Neurol*. 2024 Aug 27;9(4):367-379.
- [8] Yan Y, Ma L, Zhou X, et al. Src inhibition blocks renal interstitial fibroblast activation and ameliorates renal fibrosis. *Kidney Int*. 2016 Jan;89(1):68-81.

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