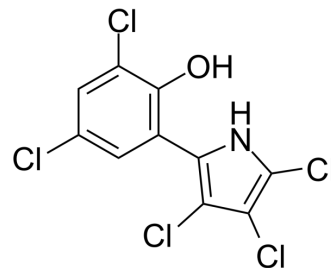


Pentachloropseudilin

Cat. No.:	HY-115669									
CAS No.:	69640-38-6									
Molecular Formula:	C ₁₀ H ₄ Cl ₅ NO									
Molecular Weight:	331.41									
Target:	Myosin; TGF-β Receptor									
Pathway:	Cytoskeleton; TGF-beta/Smad									
Storage:	<table border="0"> <tr> <td>Powder</td> <td>-20°C</td> <td>3 years</td> </tr> <tr> <td>In solvent</td> <td>-80°C</td> <td>6 months</td> </tr> <tr> <td></td> <td>-20°C</td> <td>1 month</td> </tr> </table>	Powder	-20°C	3 years	In solvent	-80°C	6 months		-20°C	1 month
Powder	-20°C	3 years								
In solvent	-80°C	6 months								
	-20°C	1 month								



BIOLOGICAL ACTIVITY

Description	<p>Pentachloropseudilin (Antibiotic A 15104 Y; PCIP) is a reversible and allosteric potent inhibitor of Myo1s (class 1 myosins) with IC₅₀s range from 1 to 5 μM for mammalian class-1 myosins and greater than 90 μM for class-2 and class-5 myosins. Pentachloropseudilin is a potent inhibitor of transforming growth factor-β (TGF-β)-stimulated signaling, with an IC₅₀ of 0.1 to 0.2 μM for TGF-β^{[1][2]}.</p>
In Vitro	<p>Pentachloropseudilin (PCIP) inhibits TGF-β-stimulated Smad2/3 phosphorylation and plasminogen activator inhibitor-1 (PAI-1) promoter activation with an IC₅₀ of 0.1 μM in target cells (A549, HepG2, and Mv1Lu cells)^[1]. Pentachloropseudilin attenuates TGF-β-stimulated expression of vimentin, N-cadherin, and fibronectin and, thus, blocks TGF-β-induced epithelial to mesenchymal transition (EMT) in these cells. Pentachloropseudilin (0.05 to 1 μM; 0-6 hours) pretreatment inhibits TGF-β-mediated (50 or 100 pM) increases in p-Smad2/3 expression to 47% (Mv1Lu) and 79% (A549), respectively^[1].</p> <p>Pentachloropseudilin (0.2 μM) suppresses TGF-β-stimulated cellular responses by attenuating cell-surface expression of the type II TGF-β receptor through accelerating caveolae-mediated internalization followed by primarily lysosome-dependent degradation of the receptor, as demonstrated by sucrose density gradient analysis and immune fluorescence staining^[1]. Pentachloropseudilin (200 μM; 24 hours) exhibits and altered cell viability in HUVECs^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

REFERENCES

- [1]. Chinthalapudi K, et al. Mechanism and specificity of pentachloropseudilin-mediated inhibition of myosin motor activity. *J Biol Chem.* 2011;286(34):29700-29708.
- [2]. Chung CL, et al. Pentachloropseudilin Inhibits Transforming Growth Factor-β (TGF-β) Activity by Accelerating Cell-Surface Type II TGF-β Receptor Turnover in Target Cells. *ChemBiochem.* 2018;19(8):851-864.
- [3]. Cota Teixeira S, et al. Pentachloropseudilin Impairs Angiogenesis by Disrupting the Actin Cytoskeleton, Integrin Trafficking and the Cell Cycle. *ChemBiochem.* 2019;20(18):2390-2401.

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

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