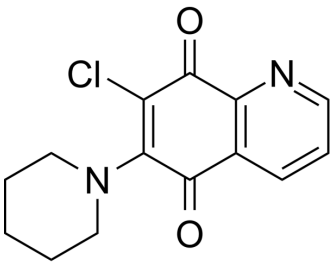
PT-262

Cat. No.:	HY-100035
CAS No.:	86811-36-1
Molecular Formula:	$C_{14}H_{13}ClN_2O_2$
Molecular Weight:	276.72
Target:	ROCK; ERK; CDK; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Cytoskeleton; Stem Cell/Wnt; TGF-beta/Smad; MAPK/ERK Pathway; Apoptosis
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (361.38 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.6138 mL	18.0688 mL	36.1376 mL
	5 mM	0.7228 mL	3.6138 mL	7.2275 mL
	10 mM	0.3614 mL	1.8069 mL	3.6138 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.03 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PT-262 is a potent ROCK inhibitor with an IC_{50} value of around 5 μM. PT-262 induces the loss of mitochondrial membrane potential and elevates the caspase-3 activation and apoptosis. PT-262 inhibits the ERK and CDC2 phosphorylation via a p53-independent pathway. PT-262 blocks cytoskeleton function and cell migration. PT-262 has anti-cancer activity[1][2].

IC_{50} & Target			
	ROCK 5 μM (IC_{50})	ERK	CDK2

In Vitro

PT-262 (5-40 μM; 24 h) induces cytotoxicity and proliferation inhibition in human lung cancer cells[1].
PT-262 (2-20 μM; 4-24 h) induces caspase-3 activation, mitochondrial dysfunction and apoptosis in lung cancer cells[1].
PT-262 (10-20 μM; 24 h) induces the accumulation of G2/M phases in both the p53-wild type and p53-null lung cancer cells, and inhibits the phosphorylation of CDC2 proteins[1].
PT-262 (0-10 μM; 24 h) represses ERK phosphorylation in lung cancer cells[1].

PT-262 (2 μM; 24 h) induces the cytoskeleton alteration and cell elongation in lung carcinoma A549 cells[2].
PT-262 (2-10 μM; 6 h) significantly blocks the cell migration in a concentration-dependent manner[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	A549 cells
Concentration:	5-40 μM
Incubation Time:	24 h
Result:	Reduced the cell viability via a concentration-dependent manner in A549 cells. The IC_{50} value toward human normal lung fibroblast was >20 μM.

Apoptosis Analysis[1]

Cell Line:	A549 cells
Concentration:	2-20 μM
Incubation Time:	4-24 h
Result:	The apoptotic cells were increased after treatment at 10 μM for 8-24 h. The active forms of caspase-3 (12 and 17 kD) were induced following treatment with 2-20 μM for 24 h.

Cell Cycle Analysis[1]

Cell Line:	A549 and H1299 cells
Concentration:	10-20 μM
Incubation Time:	24 h
Result:	Significantly decreased the G1 fractions while increased the G2/M fractions in both A549 and H1299 cells with 10 μM for 24 h. Decreased the protein levels of cyclin B1 and phospho-CDC2 at Thr14, Tyr15, and Thr161 via a concentration-dependent manner in A549 cells.

Western Blot Analysis[1]

Cell Line:	A549 cells
Concentration:	0-10 μM
Incubation Time:	24 h
Result:	Significantly inhibited the phosphorylation of ERK.

REFERENCES

[1]. Tzu-Sheng Hsu, et al. 7-Chloro-6-piperidin-1-yl-quinoline-5,8-dione (PT-262), a novel synthetic compound induces lung carcinoma cell death associated with inhibiting ERK and CDC2 phosphorylation via a p53-independent pathway. Cancer Chemother Pharmacol. 2008 Oct;62(5):799-808.

[2]. Chih-Chien Tsai, et al. 7-Chloro-6-piperidin-1-yl-quinoline-5,8-dione (PT-262), a novel ROCK inhibitor blocks cytoskeleton function and cell migration. Biochem Pharmacol. 2011 Apr 1;81(7):856-65.

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

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