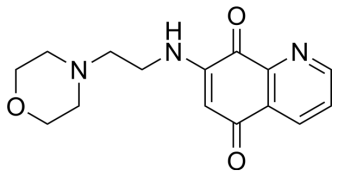
JUN-1111

Cat. No.:	HY-118529
CAS No.:	874351-38-9
Molecular Formula:	$C_{15}H_{17}N_3O_3$
Molecular Weight:	287.31
Target:	Phosphatase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (348.06 mM; ultrasonic and heat to 80°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4806 mL	17.4028 mL	34.8056 mL
	5 mM	0.6961 mL	3.4806 mL	6.9611 mL
	10 mM	0.3481 mL	1.7403 mL	3.4806 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 (8.70 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (8.70 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

JUN-1111 is an irreversible and selective Cdc25 phosphatase inhibitor with IC_{50} values of 0.38, 1.8, 0.66, 28, 37 μM for Cdc25A, Cdc25B, Cdc25C, VHR, PTP1B, respectively. JUN-1111 induces cell cycle arrest at G1 and G2/M phases. JUN-1111 decreases the expression of phosphoCdk1[1][2].

IC_{50} & Target

IC_{50}:0.38 μM (Cdc25A); 1.8 μM (Cdc25B); 0.66 μM (Cdc25C); 28 μM (VHR); 37 μM (PTP1B)[1]

In Vitro

JUN-1111 (10, 30 μM; 17 h) induces cell cycle arrest at G1 and G2/M phase in tsFT210 cells[1].
JUN-1111 (10, 30 μM; 1 h) decreases the expression of phosphoCdk1 in a dose-dependent manner in tsFT210 cells[1].
JUN-1111 (0-20 μM) induces the formation of reactive oxygen species in mammalian cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	tsFT210 cells
Concentration:	10, 30 μM
Incubation Time:	17 h
Result:	Induced cell cycle arrest at G1 and G2/M phases.

Western Blot Analysis[1]

Cell Line:	tsFT210 cells
Concentration:	10, 30 μM
Incubation Time:	1 h
Result:	Decreased the expression of phosphoCdk1 in a dose-dependent manner.

REFERENCES

[1]. Brisson M, et al. Redox regulation of Cdc25B by cell-active quinolinediones. Mol Pharmacol. 2005 Dec;68(6):1810-20.

[2]. Vogt A, et al. A cell-active inhibitor of mitogen-activated protein kinase phosphatases restores paclitaxel-induced apoptosis in dexamethasone-protected cancer cells. Mol Cancer Ther. 2008 Feb;7(2):330-40.

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