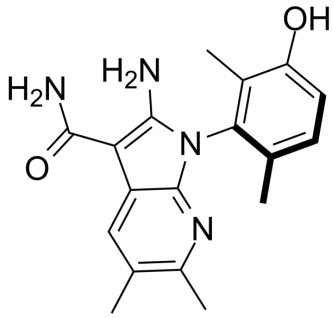
RP-6306

Cat. No.:	HY-145817A
CAS No.:	2719793-90-3
Molecular Formula:	$C_{18}H_{20}N_4O_2$
Molecular Weight:	324.38
Target:	Wee1
Pathway:	Cell Cycle/DNA Damage
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 : 50 mg/mL (154.14 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0828 mL	15.4140 mL	30.8280 mL
	5 mM	0.6166 mL	3.0828 mL	6.1656 mL
	10 mM	0.3083 mL	1.5414 mL	3.0828 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: 5 mg/mL (15.41 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.71 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

RP-6306 ((S)-RP-6306) is a potent, selective and orally active PKMYT1 inhibitor with an IC_{50} of 14 nM. RP-6306 shows a high degree of selectivity over other kinases in cellular binding assays. RP-6306 shows anticancer effects[1].

IC_{50} & Target

IC50: 14 nM (PKMYT1)[1]

In Vitro

RP-6306 (500 nM; for 24 h) treatment induces pan-γH2AX in an HCC1569 breast cancer cell line, indicating that tumour-derived CCNE1 amplification also renders cells vulnerable to DNA damage induction following PKMYT1 inhibition[2].

RP-6306 treatment causes unscheduled activation of CDK1 selectively in CCNE1-overexpressing cells, promoting early mitosis in cells undergoing DNA synthesis[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

RP-6306 (15, 50, and 300 ppm; oral; daily; for 21 days) results in a statistically significant and dose-dependent reduction in OVCAR3 tumor growth in CCNE1-amplified ovarian xenograft model (OVCAR3)[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	OVCAR3-bearing mice[1]
Dosage:	15, 50, and 300 ppm (equivalent to approximately 3, 10, and 60 mg/kg/day)
Administration:	Oral; daily; for 21 days
Result:	Resulted in a statistically significant and dose-dependent reduction in OVCAR3 tumor growth.

REFERENCES

[1]. Janek Szychowski, et al. Discovery of an Orally Bioavailable and Selective PKMYT1 Inhibitor, RP-6306. J Med Chem. 2022 Aug 11;65(15):10251-10284.

[2]. David Gallo, et al. CCNE1 amplification is synthetic lethal with PKMYT1 kinase inhibition. Nature. 2022 Apr;604(7907):749-756.

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