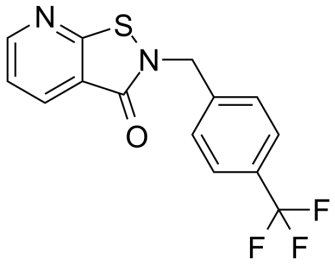
PU141

Cat. No.:	HY-120290
CAS No.:	168334-34-7
Molecular Formula:	$C_{14}H_9F_3N_2OS$
Molecular Weight:	310.29
Target:	Histone Acetyltransferase
Pathway:	Epigenetics
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 (322.28 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2228 mL	16.1140 mL	32.2279 mL
	5 mM	0.6446 mL	3.2228 mL	6.4456 mL
	10 mM	0.3223 mL	1.6114 mL	3.2228 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: 5 mg/mL (16.11 mM); Clear solution; Need ultrasonic and warming and heat to 80°C
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 5 mg/mL (16.11 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

PU141 is a selected pyridoisothiazolone HAT inhibitor. PU141 is selective toward CBP and p300. PU141 induces cellular histone hypoacetylation and inhibits growth of several neoplastic cell lines originating from different tissues. Anticancer activity[1].

IC_{50} & Target

CBP/p300[1]

In Vitro

PU141 inhibits cell growth at micromolar concentrations in A431 (epidemoid carcinoma), A549 (alveolar basal epithelial adenocarcinoma), A2780 (ovarian carcinoma), HCT116 (epithelial colon carcinoma), HepG2 (hepatocellular carcinoma), MCF7 (breast carcinoma), SK-N-SH (neuroblastoma), SW480 (colon adenocarcinoma) and U-87MG (epithelial-like glioblastoma-astrocytoma)[1].

PU141 causes both histone hypoacetylation and growth inhibition in vitro. PU141 (25 μM) leads to a decrease in SAHA-induced H3K14 and H4K8 hyperacetylation, whereas H3K9 and H4K16 acetylation levels remaine stable after co-treatment of HDAC and HAT inhibitor. The impact on histone acetylation is similar in both SK-N-SH and HCT116 cells[1].

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

Cell Viability Assay[1]

Cell Line:	A431 (epidemoid carcinoma), A549 (alveolar basal epithelial adenocarcinoma), A2780 (ovarian carcinoma), HCT116 (epithelial colon carcinoma), HepG2 (hepatocellular carcinoma), MCF7 (breast carcinoma), SK-N-SH (neuroblastoma), SW480 (colon adenocarcinoma) and U-87MG (epithelial-like glioblastoma-astrocytoma)
Concentration:	0, 10, 20, 30, 40, 50, and 60 μM
Incubation Time:	
Result:	Inhibited cell growth at micromolar concentrations in all screened cell lines. The highest cellular antiproliferative activity was detected for the neuroblastoma SK-N-SH cell line.

Western Blot Analysis[1]

Cell Line:	SK-N-SH neuroblastoma and HCT116 colon carcinoma cells
Concentration:	25 μM
Incubation Time:	3 hours
Result:	Led to a decrease in SAHA-induced H3K14 and H4K8 hyperacetylation.

In Vivo

PU141 (25 mg/kg; administered once intraperitoneally for 24 days) exhibits a significant antitumor effects against neuroblastoma xenografts in vivo[1].

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

Animal Model:	Male NMRI:nu/nu mice bearing a xenograft model[1]
Dosage:	12.5 and 25 mg/kg
Administration:	Administered once intraperitoneally (i.p.) as a detergent containing saline microemulsion; for 24 days
Result:	Led to significant tumor volume reduction (19%) at 25 mg/kg.

REFERENCES

[1]. M Gajer,et al. Histone acetyltransferase inhibitors block neuroblastoma cell growth in vivo. Oncogenesis.2015 Feb 9;4(2):e137.

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