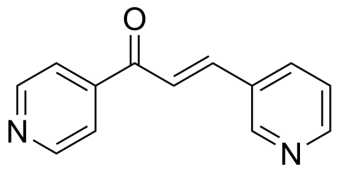
3PO

Cat. No.:	HY-19824
CAS No.:	18550-98-6
Molecular Formula:	$C_{13}H_{10}N_2O$
Molecular Weight:	210.23
Target:	Autophagy
Pathway:	Autophagy
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 60 mg/mL (285.40 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.7567 mL	23.7835 mL	47.5670 mL
	5 mM	0.9513 mL	4.7567 mL	9.5134 mL
	10 mM	0.4757 mL	2.3783 mL	4.7567 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: ≥ 3 mg/mL (14.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (14.27 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (14.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

3PO is an inhibitor of PFKFB3. 3PO attenuates the proliferation of several cancer cell lines with IC_{50}s of 1.4-24 μmol/L. 3PO suppresses glucose uptake and decreases the intracellular concentration of Fru-2,6-BP, lactate, ATP, NAD^+ and NADH. 3PO can be used for the research of cancer[1].

In Vitro

3PO (0-33 μM; 0-36 h) inhibits Jurkat cells proliferation by influence cell cycle[1].
3PO (10 μM/L; 0-36 h) suppresses glycolytic flux to lactate in Jurkat cells[1].
3PO (10 μM/L; 0-36 h) inhibits cellular proliferation of transformed tumor cell lines[1].

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

Cell Proliferation Assay[1]

Cell Line:	Jurkat cells
Concentration:	0.3, 1, 3, 10 and 33 μM
Incubation Time:	0, 4, 8, 16, 24 and 36 hours
Result:	Inhibited Jurkat T cells proliferation by G2-M phase cell cycle arrest.

Cell Viability Assay[1]

Cell Line:	Jurkat cells
Concentration:	0.3, 1, 3, 10 and 33 μM
Incubation Time:	0, 4, 8, 16, 24 and 36 hours
Result:	Decreased lactate secretion, NADH, NAD^+ and ATP in Jurkat cells.

Cell Proliferation Assay[1]

Cell Line:	Jurkat, K562, HL-60, MDA-MB231, HL-60, HeLa, Melanoma and Lewis Lung
Concentration:	0-20 μM
Incubation Time:	0, 4, 8, 16, 24 and 36 hours
Result:	Inhibited solid tumor and hematologic cell lines with IC_{50}s of 1.4-24 μM/L.

In Vivo

3PO (0.07 mg/g; i.p. once per day for 14 days) inhibits the growth of Lewis lung carcinoma xenografts in C57Bl/6 mice[1].
3PO (0.07 mg/g; i.p. three sequential daily injections followed by 3 off days for 14 days) inhibits the tumor growth in BALB/c athymic mice[1].
3PO (0.07 mg/g; i.p. two daily injections followed a 7-day rest for 14 days) inhibits the tumor growth in BALB/c athymic mice [1].
3PO (0.07 mg/g; i.p. once) inhibits Fru-2,6-BP and glucose uptake in C57Bl/6 mice[1].

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

Animal Model:	Female C57Bl/6 mice with Lewis lung carcinoma xenografts[1]
Dosage:	0.07 mg/g
Administration:	Intraperitoneal injection; 0.07 mg/g once a day; for 14 days
Result:	Suppressed the growth of Lewis lung carcinoma xenografts in C57Bl/6 mice compared to the control group.

Animal Model:	Female BALB/c athymic mice with MDA-MB231 human breast adenocarcinoma xenografts [1]
Dosage:	0.07 mg/g
Administration:	Intraperitoneal injection; 0.07 mg/g three sequential daily injections of either DMSO or 3PO followed by 3 off days; for 14 days

Result:	Suppressed xenograft tumorigenic growth of MDA-MB231 cells compared with the controls.

Animal Model:	Female BALB/c athymic mice with HL-60 leukemia xenografts[1]
Dosage:	0.07 mg/g
Administration:	Intraperitoneal injection; 0.07 mg/g two daily injections followed a 7-day rest; for 14 days
Result:	Suppressed xenograft tumorigenic growth of HL-60 leukemia compared with the controls, especially from the second administration cycle.

Animal Model:	Female C57Bl/6 mice with Lewis lung carcinoma xenografts[1]
Dosage:	0.07 mg/g
Administration:	Intraperitoneal injection; once
Result:	Decreased Fru-2,6-BP and glucose uptake in C57Bl/6 mice compared to control group.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2022 Sep 1;7(1):303.
- Nat Metab. 2022 Oct;4(10):1306-1321.
- Redox Biol. 2021 Jul 26;46:102082.
- Cell Prolif. 2021 May 30;e13058.
- Acta Pharmacol Sin. 2024 Sep 18.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Clem B, et al. Small-molecule inhibition of 6-phosphofructo-2-kinase activity suppresses glycolytic flux and tumor growth. Mol Cancer Ther. 2008 Jan;7(1):110-20.

[2]. Schoors S, et al. Partial and transient reduction of glycolysis by PFKFB3 blockade reduces pathological angiogenesis. Cell Metab. 2014 Jan 7;19(1):37-48.

[3]. Lea MA, Inhibition of Growth of Bladder Cancer Cells by 3-(3-Pyridinyl)-1-(4-pyridinyl)-2-propen-1-one in Combination with Other Compounds Affecting Glucose Metabolism. Anticancer Res. 2015 Nov;35(11):5889-99.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA