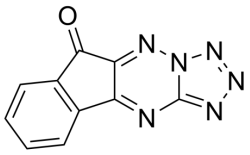
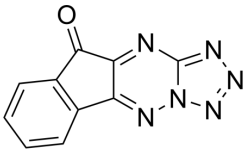
KP372-1





Cat. No.:	HY-15673
CAS No.:	1374996-60-7
Molecular Formula:	$C_{20}H_8N_{12}O_2$
Molecular Weight:	448.36
Target:	Akt; Reactive Oxygen Species; Apoptosis
Pathway:	PI3K/Akt/mTOR; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis
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 : 17.86 mg/mL (39.83 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2304 mL	11.1518 mL	22.3035 mL
	5 mM	0.4461 mL	2.2304 mL	4.4607 mL
	10 mM	0.2230 mL	1.1152 mL	2.2304 mL

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

BIOLOGICAL ACTIVITY

Description

KP372-1 is an Akt inhibitor that inhibits proliferation and induces apoptosis and anoikis. KP372-1 is also an NQO1 redox cycling agent that causes DNA damage (including DNA breakage) by generating ROS. KP372-1 can be used in cancer research (such as head and neck squamous cell carcinoma (HNSCC) and pancreatic cancer)[1][2][3].

In Vitro

KP372-1 (0.0625, 0.125, 0.25, 0.5, 1.0 μM; 48 h) inhibits growth of JMARc42 and Tu167c2 cells with IC_{50}s of 200 and 100 nM, respectively[1].

KP372-1 (125 nM; 24 h) induces Tu167c2 cells apoptosis and induces anoikis in the JMARc42 cells[1].

KP372-1 (125 nM; 30 min) blocks Akt, thereby decreasing the phosphorylation of the S6 ribosomal protein in both Tu167 and JMAR cells[1].

KP372-1 (0.250, 0.5, 1.0 μM; 30 min) inhibits Akt kinase activity with an IC_{50} of 250 nM in JMAR cells[1].

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

Cell Proliferation Assay[1]

Cell Line:	JMARc42 and Tu167c2 cells
Concentration:	0.0625, 0.125, 0.25, 0.5, 1.0 μM
Incubation Time:	48 h
Result:	Showed antiproliferative activity.

Apoptosis Analysis[1]

Cell Line:	Tu167c2 and JMARc42 cells
Concentration:	125 nM
Incubation Time:	24 h
Result:	Induced approximately 90% of cells apoptosis.

Western Blot Analysis[1]

Cell Line:	Tu167 and JMAR cells
Concentration:	125 nM
Incubation Time:	30 min
Result:	Induced a small but consistent decrease in Akt phosphorylation with a concomitant marked decrease in S6 phosphorylation. Inhibited the EGF induced phosphorylation of Aktser473 in Tu167 and AktThr308 in JMAR.

Western Blot Analysis[1]

Cell Line:	JMAR cells
Concentration:	0.250, 0.5, 1.0 μM
Incubation Time:	30 min
Result:	Significantly blocked Akt kinase activity in a dose-dependent fashion, with an IC_{50} of 250 nM.

In Vivo

KP372-1(10, 20 mg/kg; i.v.; single daily for 33 days) induces NADH oxidation and impairs tumor growth in vivo without apparent toxicity[2].

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

Animal Model:	Nude mice (H1299 xenografts model)[2].
Dosage:	10, 20 mg/kg
Administration:	Tailvein injection; single daily for 33 days
Result:	Affected tumor metabolism and suppressed tumor growth.

CUSTOMER VALIDATION

- J Control Release. 2022 May 31;347:632-648.

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REFERENCES

[1]. Mandal M, et al. The Akt inhibitor KP372-1 inhibits proliferation and induces apoptosis and anoikis in squamous cell carcinoma of the head and neck. Oral Oncol. 2006 Apr;42(4):430-9.

[2]. Zhao Y, et al. SoNar, a Highly Responsive NAD+/NADH Sensor, Allows High-Throughput Metabolic Screening of Anti-tumor Agents. Cell Metab. 2015 May 5;21(5):777-89.

[3]. Viera T, et al. DNA damage induced by KP372-1 hyperactivates PARP1 and enhances lethality of pancreatic cancer cells with PARP inhibition. Sci Rep. 2020 Nov 19;10(1):20210.

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