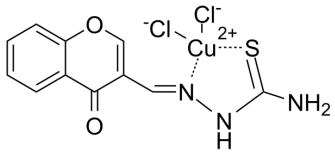
FPA-124

Cat. No.:	HY-15369
CAS No.:	902779-59-3
Molecular Formula:	$C_{11}H_9Cl_2CuN_3O_2S$
Molecular Weight:	381.73
Target:	Akt; Apoptosis
Pathway:	PI3K/Akt/mTOR; Apoptosis
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (13.10 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6197 mL	13.0983 mL	26.1965 mL
5 mM	0.5239 mL	2.6197 mL	5.2393 mL
10 mM	0.2620 mL	1.3098 mL	2.6197 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: ≥ 0.67 mg/mL (1.76 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 0.67 mg/mL (1.76 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

FPA-124, a cell-permeable copper complex, is a selective Akt inhibitor with an IC_{50} of 0.1 μM. FPA-124 interacts with both the pleckstrin homology (PH) and the kinase domains of Akt. FPA-124 induces apoptosis[1][2].

In Vitro

FPA-124 exhibits dose-dependent growth inhibitory effects with IC_{50}s of 7, 10, 34, and 55 μM in BT20, PC-3, COLO 357 and BxPC-3 cancer cell lines, respectively[1].

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

In Vivo

FPA-124 exhibits PKB (Akt protein) inhibitory activities and causes NF-κB inactivation in a well-established orthotopic pancreatic tumor model using COLO 357 cells[1].

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

REFERENCES

[1]. Barve V, et al. Synthesis, molecular characterization, and biological activity of novel synthetic derivatives of chromen-4-one in human cancer cells. J Med Chem. 2006 Jun 29;49(13):3800-8.

[2]. Biscetti F, et al. Pioglitazone enhances collateral blood flow in ischemic hindlimb of diabetic mice through an Akt-dependent VEGF-mediated mechanism, regardless of PPARgamma stimulation. Cardiovasc Diabetol. 2009 Sep 8;8:49.

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