Copanlisib

Cat. No.: HY-15346

CAS No.: 1032568-63-0

Molecular Formula: $C_{23}H_{28}N_8O_4$

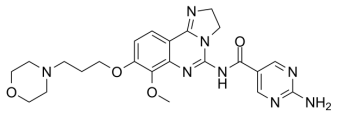
Molecular Weight: 480.52

Target: PI3K; Apoptosis

Pathway: PI3K/Akt/mTOR; 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

1M HCl : 100 mg/mL (208.11 mM; Need ultrasonic)

1M HCl : 100 mg/mL (208.11 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0811 mL	10.4054 mL	20.8108 mL
	5 mM	0.4162 mL	2.0811 mL	4.1622 mL
	10 mM	0.2081 mL	1.0405 mL	2.0811 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 5 mg/mL (10.41 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Copanlisib (BAY 80-6946) is a potent, selective and ATP-competitive pan-class I PI3K inhibitor, with IC_{50}s of 0.5 nM, 0.7 nM, 3.7 nM and 6.4 nM for PI3Kα, PI3Kδ, PI3Kβ and PI3Kγ, respectively. Copanlisib has more than 2,000-fold selectivity against other lipid and protein kinases, except for mTOR. Copanlisib has superior antitumor activity[1].

IC_{50} & Target

PI3Kα	PI3Kδ	PI3Kβ	PI3Kγ
0.5 nM (IC_{50})	0.7 nM (IC_{50})	3.7 nM (IC_{50})	6.4 nM (IC_{50})

mTOR
45 nM (IC_{50})

In Vitro

Copanlisib (BAY 80-6946; 20-200 nM; 24 hours; BT20 breast cancer cells) treatmemnt induces apoptosis in a subset of tumor

cell lines that are resistant to Lapatinib and Trastuzumab[1].
Copanlisib (BAY 80-6946; 0.5-500 nM; 2 hours; ELT3 cells) shows complete inhibition of PI3K-mediated AKT phosphorylation in ELT3 cells[1].
Copanlisib potently inhibits cell proliferation in a panel of human tumor cell lines. Copanlisib has mean IC_{50} values of 19 nM against cell lines with PIK3CA-activating mutations and 17 nM against HER2-positive cell lines, whereas the activity in PIK3CA wild-type and HER2-negative cells is about 40-fold less potent[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	BT20 breast cancer cells
Concentration:	20 nM and 62 nM, 200 nM
Incubation Time:	24 hours
Result:	Significantly increased caspase9 activities. Also increased levels of phosphorylated p53 at Ser15and cleaved PARP. Induced caspase-9 activation with an EC_{50} of 340 nM.

Western Blot Analysis[1]

Cell Line:	ELT3 cells
Concentration:	0.5 nM, 5 nM, 50 nM, 500 nM
Incubation Time:	2 hours
Result:	Complete inhibition of PI3K-mediated AKT phosphorylation was clearly shown at a concentration of 5 nM.

In Vivo

Copanlisib (BAY 80-6946; 0.5-6 mg/kg; intravenous injection; every second day, every third day; for 60 days; athymic nude rats) treatment displays robust antitumor activity in the rat KPL4 tumor xenograft model[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Athymic nude rats injected with KPL4 tumor cells[1]
Dosage:	0.5 mg/kg, 1 mg/kg, 3 mg/kg or 6 mg/kg
Administration:	Intravenous injection; every second day, every third day; for 60 days
Result:	On day 25, tumor growth inhibition (TGI) rates of 77%, 84%, 99%, and 100% were observed at doses of 0.5, 1, 3, and 6 mg/kg, respectively. All rats remained tumor free at the termination of the study on day 73.

CUSTOMER VALIDATION

- Science. 2017 Dec 1;358(6367):eaan4368.
- Mol Cancer. 2023 Mar 30;22(1):64.
- Blood. 2019 Jan 3;133(1):70-80.
- J Clin Invest. 2021 Dec 15;131(24):e140436.
- Theranostics. 2020 Jan 1;10(4):1531-1543.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Liu N, et al. BAY 80-6946 is a highly selective intravenous PI3K inhibitor with potent p110α and p110δ activities in tumor cell lines and xenograft models. Mol Cancer Ther. 2013 Nov;12(11):2319-30.

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