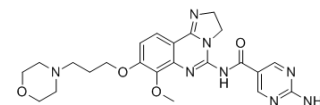


Copanisib

Cat. No.:	HY-15346		
CAS No.:	1032568-63-0		
Molecular Formula:	C ₂₃ H ₂₈ N ₈ O 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	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

1M HCl : 100 mg/mL (208.11 mM; Need ultrasonic)
 DMSO : 1 mg/mL (2.08 mM; Need ultrasonic)
 H₂O : 0.67 mg/mL (1.39 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		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.

BIOLOGICAL ACTIVITY

Description

Copanisib (BAY 80-6946) is a potent, selective and ATP-competitive pan-class I PI3K inhibitor, with IC₅₀s of 0.5 nM, 0.7 nM, 3.7 nM and 6.4 nM for PI3K α , PI3K δ , PI3K β and PI3K γ , respectively. Copanisib has more than 2,000-fold selectivity against other lipid and protein kinases, except for mTOR. Copanisib has superior antitumor activity^[1].

IC₅₀ & Target

IC ₅₀ & Target	PI3K α	PI3K δ	PI3K β	PI3K γ
	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

Copanisib (BAY 80-6946; 20-200 nM; 24 hours; BT20 breast cancer cells) treatment induces apoptosis in a subset of tumor cell lines that are resistant to Lapatinib and Trastuzumab^[1].
 Copanisib (BAY 80-6946; 0.5-500 nM; 2 hours; ELT3 cells) treatment 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 IC₅₀ 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 Ser15 and 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.
- Blood. 2019 Jan 3;133(1):70-80.
- Theranostics. 2020 Jan 1;10(4):1531-1543.
- Cell Biosci. 2020 Feb 12;10:16.
- Breast Cancer Res Treat. 2020 Jan;179(2):337-347.

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REFERENCES

[1]. Liu N, et al. BAY 80-6946 is a highly selective intravenous PI3K inhibitor with potent p110 α and p110 δ activities in tumor cell lines and xenograft models. Mol Cancer Ther. 2013 Nov;12(11):2319-30.

Caution: Product has not been fully validated for medical applications. For research use only.

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