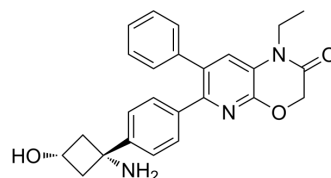


ALM301

Cat. No.:	HY-151504		
CAS No.:	1313439-71-2		
Molecular Formula:	C ₂₅ H ₂₅ N ₃ O ₃		
Molecular Weight:	415.48		
Target:	Akt		
Pathway:	PI3K/Akt/mTOR		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (120.34 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.4069 mL	12.0343 mL	24.0685 mL
		5 mM	0.4814 mL	2.4069 mL	4.8137 mL
10 mM		0.2407 mL	1.2034 mL	2.4069 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: ≥ 2.5 mg/mL (6.02 mM); Clear solution 2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (6.02 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	ALM301 is an orally active highly specific AKT inhibitor with IC ₅₀ values of 0.13 μM, 0.09 μM and 2.75 μM for AKT1, AKT2 and AKT3, respectively. ALM301 inhibits AKT phosphorylation and modulates downstream signalling in vitro. ALM301 can inhibit cancer cell proliferation and tumor growth ^[1] .		
IC₅₀ & Target	AKT1 0.13 μM (IC ₅₀)	Akt2 0.09 μM (IC ₅₀)	Akt3 2.75 μM (IC ₅₀)
In Vitro	ALM301 (0.001-10 μM; 72 h) inhibits the proliferation of cancer cells, and PI3KCA-mutant MCF-7 cells is the most sensitive; increases sub-G0 population in a dose-dependent manner ^[1] . ALM301 (1 μM; 1 h) inhibits AKT phosphorylation in MCF-7 and sustains up to 48 h, with an EC ₅₀ value of 0.47 μM ^[1] .		

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

Cell Proliferation Assay^[1]

Cell Line:	MCF-7, T47D, NCI-H460, HCT116 and other cancer cells
Concentration:	0.001-10 μ M
Incubation Time:	72 h
Result:	Inhibited the proliferation of cancer cells, and PI3KCA-mutant MCF-7 cells was the most sensitive with an IC ₅₀ of 2.25 μ M.

Western Blot Analysis^[1]

Cell Line:	MCF-7
Concentration:	0.001-10 μ M
Incubation Time:	1, 4, 24 and 48 h
Result:	Inhibited pAKT and pGSK3 β at various concentrations and timepoints up to 48 h.

In Vivo

ALM301 (10, 30 and 100 mg/kg; p.o.; single dosage) inhibits pAKT^{S473} in tumors and suppresses tumor growth^[1].
ALM301 (3 or 10 mg/kg; p.o.; q.d. for 49 days) shows better tumor inhibition ability when combined with [Tamoxifen](#) (HY-13757A)^[1].

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

Animal Model:	BALB/c mice (bearing A549 xenografts) ^[1]
Dosage:	10, 30 and 100 mg/kg
Administration:	p.o.; single dosage
Result:	Increased total plasma concentrations dose-dependently that resulted in almost total abrogation of measurable pAKT ^{S473} in tumors at all timepoints over 24 h. Exhibited the tumour growth inhibition (TGI) of 23, 31 and 41% at 10, 30 and 100 mg/kg, respectively.

Animal Model:	BALB/c mice (bearing PIK3CA-mutant MCF-7 xenografts) ^[1]
Dosage:	3 or 10 mg/kg
Administration:	p.o.; q.d. for 49 days
Result:	Showed significant tumour regressions of 57% and 50% at 3 and 10 mg/kg, respectively, when combined with Tamoxifen (HY-13757A) (5 mg/kg; q.d.).

REFERENCES

[1]. Page N, et al. Identification and development of a subtype-selective allosteric AKT inhibitor suitable for clinical development. Sci Rep. 2022 Sep 20;12(1):15715.

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

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