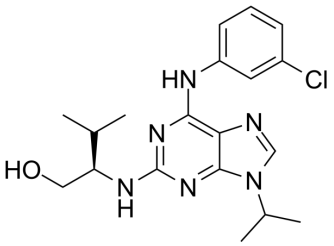
Purvalanol A

Cat. No.:	HY-18299A
CAS No.:	212844-53-6
Molecular Formula:	$C_{19}H_{25}ClN_6O$
Molecular Weight:	388.89
Target:	CDK; Autophagy; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Autophagy; 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 : ≥ 50 mg/mL (128.57 mM)
Ethanol : 10 mg/mL (25.71 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5714 mL	12.8571 mL	25.7142 mL
	5 mM	0.5143 mL	2.5714 mL	5.1428 mL
	10 mM	0.2571 mL	1.2857 mL	2.5714 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 (6.43 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.43 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Purvalanol A is a potent CDK inhibitor, which inhibits cdc2-cyclin B, cdk2-cyclin A, cdk2-cyclin E, cdk4-cyclin D1, and cdk5-p35 with IC_{50}s of 4, 70, 35, 850, 75 nM, resepctively.

IC_{50} & Target

cdc2-cyclin B 4 nM (IC_{50})	cdk2-cyclin E 35 nM (IC_{50})	cdk2-cyclin A 70 nM (IC_{50})	cdk4-cyclin D1 850 nM (IC_{50})
cdk5-p35 75 nM (IC_{50})	erk1 9000 nM (IC_{50})		

In Vitro	Purvalanol A inhibits cdc28 (S. cerevisiae) and erk1 with IC_{50}s of 80 and 9000 nM. Purvalanol A shows inhibitory activities against the NCI panel of 60 human tumor cell lines, with average GI50 of 2 μM; two cell lines show an -20-fold increase in sensitivity to purvalanol A: the KM12 colon cancer cell line with a GI_{50} of 76 nM and the NCI-H522 non–small cell lung cancer cell line with a GI_{50} of 347 nM[1]. Purvalanol A is a 2.5-fold more potent inhibitor of CDK2, but also inhibits DYRK1A potently and a number of other protein kinases in the low micromolar range. Purvalanol A inhibits MKK1, MAPK2/ERK2, JNK/SAPK1c with IC_{50}s of 80, 26, 84 μM[2]. Purvalanol A selectively inhibits the phosphorylation of cellular proteins. Purvalanol A prevents the increases of the contents of cyclins D and E during serum-induced G1 phase progression. Purvalanol A does not inhibit transcription under cell-free conditions[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- EMBO Rep. 2022 Apr 11;e53932.
- iScience. 2019 May 31;15:291-306.

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REFERENCES

[1]. Gray NS, et al. Exploiting chemical libraries, structure, and genomics in the search for kinase inhibitors. Science. 1998 Jul 24;281(5376):533-8.

[2]. Bain J, et al. The specificities of protein kinase inhibitors: an update. Biochem J. 2003 Apr 1;371(Pt 1):199-204.

[3]. Villerbu N, et al. Cellular effects of purvalanol A: a specific inhibitor of cyclin-dependent kinase activities. Int J Cancer. 2002 Feb 20;97(6):761-9.

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

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