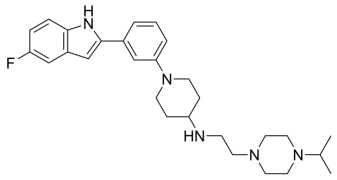
UPCDC-30245

Cat. No.:	HY-123636
CAS No.:	1883351-01-6
Molecular Formula:	$C_{28}H_{38}FN_5$
Molecular Weight:	463.63
Target:	p97
Pathway:	Cell Cycle/DNA Damage
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 : 200 mg/mL (431.38 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1569 mL	10.7845 mL	21.5689 mL
	5 mM	0.4314 mL	2.1569 mL	4.3138 mL
	10 mM	0.2157 mL	1.0784 mL	2.1569 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: ≥ 2.08 mg/mL (4.49 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.49 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

UPCDC-30245 is an allosteric p97 inhibitor with an IC_{50} of approximately 27 nM[1]. UPCDC-30245 inhibits the p97 mutant N660K similar to wild type (WT; IC_{50}=300 nM) and shows 3-fold resistance for p97 mutant T688A[2]. UPCDC-30245 can be used in the research of cancer[1][2][3].

IC_{50} & Target		
	allosteric p97 27 nM (IC_{50})	wild type p97 300 nM (IC_{50})

In Vitro	UPCDC-30245 binds at the junction between the D1 dan D2 domains of p97[1]. UPCDC-30245 inhibits cell proliferation in HeLa, A549, BxPC-3, PRMI8226, MM1S, HCT116 cells, and appears more potent in HT29 cells[2]. UPCDC-30245 (0.01-100 μM) shows anti-proliferative effects on parental and CB-5083 resistant HCT116 cell lines that harbor a new p97 double mutation (D649A/T688A)[2]. UPCDC-30245 (4 μm; 2, 6, 10, 18, 24 hours; functional enrichment analysis) is not linked to pathways typically impacted by p97 inhibition, such as protein processing in the ER, UPR, and asparagine N-linked glycosylation[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Banerjee S, et al. 2.3 Å resolution cryo-EM structure of human p97 and mechanism of allosteric inhibition. Science. 2016 Feb 19;351(6275):871-5.

[2]. Wang F, et al. Allosteric p97 Inhibitors Can Overcome Resistance to ATP-Competitive p97 Inhibitors for Potential Anticancer Therapy. ChemMedChem. 2020 Apr 20;15(8):685-694.

[3]. Wang F, et al. Temporal proteomics reveal specific cell cycle oncoprotein downregulation by p97/VCP inhibition. Cell Chem Biol. 2021 Nov 23:S2451-9456(21)00482-7.

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