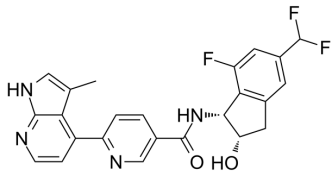
BLU2864

Cat. No.:	HY-150076
CAS No.:	2810747-89-6
Molecular Formula:	$C_{24}H_{19}F_3N_4O_2$
Molecular Weight:	452.43
Target:	Others
Pathway:	Others

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 : 100 mg/mL (221.03 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2103 mL	11.0514 mL	22.1029 mL
	5 mM	0.4421 mL	2.2103 mL	4.4206 mL
	10 mM	0.2210 mL	1.1051 mL	2.2103 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 (5.53 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.53 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (5.53 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

BLU2864 is an orally active, highly selective, ATP-competitive PRKACA inhibitor (IC_{50}=0.3 nM). BLU2864 shows anti-tumor activity. BLU2864 can be used in cancer and polycystic kidney disease research[1][2].

IC_{50} & Target

IC50: 0.3 nM (PRKACA)[2]

In Vitro

BLU2864 (40 nM and 200 nM; 5 d) inhibits forskolin (HY-15371)-induced in vitro cystogenesis[1].

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

Cell Viability Assay[1]

Cell Line:	mIMCD3 cells
Concentration:	40 nM and 200 nM
Incubation Time:	5 days
Result:	Inhibited forskolin induced in vitro cystogenesis of mIMCD3 cells cultured in Matrigel by 72% and 100% at 40 and 200 nM concentrations, respectively, relative to control.

In Vivo

BLU2864 (oral gavage; 45 mg/kg; once daily; 5 d) inhibits renal PKA activity in Pkd1$^{RC/RC}$ mice[1].
BLU2864 (oral gavage; 30 mg/kg; once daily; 5 d) inhibits PKA activity and ameliorates PKD in Pkd1$^{RC/RC}$ mice[1].
BLU2864 (oral gavage; 30 mg/kg and 75 mg/kg; once daily; 34 d) reduces FLC tumor growth in vivo[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Pkd1$^{RC/RC}$ mice[1]
Dosage:	45 mg/kg
Administration:	Oral gavage; 45 mg/kg; once daily; 5 days
Result:	Suppressed kidney basal and total PKA activities by 74% and 87% at 3 hours and by 46% and 56% at 15 hours, respectively, in the BLU2864-treated mice compared with controls.

Animal Model:	Pkd1$^{RC/RC}$ mice[1]
Dosage:	30 mg/kg
Administration:	Oral gavage; 30 mg/kg; once daily; 5 days
Result:	Showed higher urine outputs at 15 weeks in the BLU2864-treated mice than in the controls. Showed lower kidney weights, kidney volumes as percent of body weights, and cyst indices. Showed renal basal and total PKA activities by 69% and 84% lower in the BLU2864-treated mice compared with controls.

Animal Model:	Mice harboring FLC PDX tumors[2]
Dosage:	30 mg/kg and 75 mg/kg
Administration:	Oral gavage; 30 mg/kg and 75 mg/kg; once daily; 34 days
Result:	Inhibited tumor growth by 48.5% (P=0.003) and by 45.3% (P=0.0005), respectively, at day 34.

REFERENCES

[1]. Xiaofang Wang, et al. Protein Kinase A Downregulation Delays the Development and Progression of Polycystic Kidney Disease. J Am Soc Nephrol. 2022 Jun;33(6):1087-1104.

[2]. Stefanie S. Schalm, et al. Evaluation of PRKACA as a Therapeutic Target for Fibrolamellar Carcinoma. bioRxiv 2022.01.31.477690.

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

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