Roginolisib

Cat. No.: HY-135827

CAS No.: 1305267-37-1

Molecular Formula: $C_{26}H_{27}FN_4O_5S$

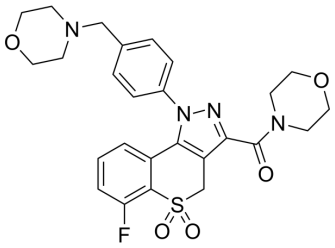
Molecular Weight: 526.58

Target: PI3K

Pathway: PI3K/Akt/mTOR

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 : 100 mg/mL (189.90 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8990 mL	9.4952 mL	18.9905 mL
	5 mM	0.3798 mL	1.8990 mL	3.7981 mL
	10 mM	0.1899 mL	0.9495 mL	1.8990 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: ≥ 10 mg/mL (18.99 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 10 mg/mL (18.99 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 10 mg/mL (18.99 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Roginolisib (MSC2360844; IOA-244) is a potent, orally active and selective PI3Kδ inhibitor, with an IC_{50} of 145 nM. Roginolisib shows highly selective against a panel of 278 additional kinases[1].

IC_{50} & Target

PI3Kδ	PI3Kα	PI3Kβ
145 nM (IC_{50})	18500 nM (IC_{50})	2850 nM (IC_{50})

In Vitro

Roginolisib (0-10 μM; 1 hours) completely abolished BCR-induced pAkt in Ramos B cells in a concentration-dependent

manner with IC_{50} values of 280 nM[1].
Roginolisib inhibits B cell proliferation in a concentration-dependent manner with an IC_{50} of 48 nM. Roginolisib blocks BCR- and TCR-mediated responses in lymphocytes and TLR-induced IFNα by pDC in human primary cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	B cells
Concentration:	0-10 μM
Incubation Time:	1 hour
Result:	Inhibited B cell proliferation in a concentration-dependent manner with an IC_{50} of 48 nM.

In Vivo

Roginolisib (6.6-66 mg/kg; daily from week 2 to 10) ameliorates disease manifestations in a murine SLE model[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	NZB/W F1 female mice[1]
Dosage:	6.6, 22, or 66 mg/kg
Administration:	Oral; starting at week 2 post ADV-IFNα delivery, once daily at 10 weeks
Result:	Significantly reduced proteinuria incidence and severity in a dose-dependent manner.

REFERENCES

[1]. Haselmayer P, et al. Characterization of Novel PI3Kδ Inhibitors as Potential Therapeutics for SLE and Lupus Nephritis in Pre-Clinical Studies. Front Immunol. 2014 May 22;5:233.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA