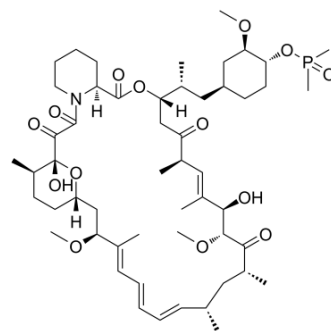


Ridaforolimus

Cat. No.:	HY-50908
CAS No.:	572924-54-0
Molecular Formula:	C ₅₃ H ₈₄ NO ₁₄ P
Molecular Weight:	990.21
Target:	mTOR; Autophagy
Pathway:	PI3K/Akt/mTOR; Autophagy
Storage:	Powder -20°C 3 years 4°C 2 years



* The compound is unstable in solutions, freshly prepared is recommended.

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 44 mg/mL (44.44 mM)
* "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	1.0099 mL	5.0494 mL	10.0989 mL
	5 mM	0.2020 mL	1.0099 mL	2.0198 mL
	10 mM	0.1010 mL	0.5049 mL	1.0099 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (2.52 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (2.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ridaforolimus (MK-8669) is a potent and selective mTOR inhibitor; inhibits ribosomal protein S6 phosphorylation with an IC₅₀ of 0.2 nM in HT-1080 cells^[1].

IC₅₀ & Target

mTOR

In Vitro

Treatment of HT-1080 fibrosarcoma cells with Ridaforolimus results in a dose-dependent inhibition of phosphorylation of both S6 and 4E-BP1, with IC₅₀s of 0.2 and 5.6 nM, respectively, and EC₅₀s of 0.2 and 1.0 nM, respectively. In HT-1080 cells, the EC₅₀ for inhibition of cell proliferation (0.5 nM) is similar to the EC₅₀s for inhibition of S6 and 4E-BP1 phosphorylation. Exposure to Ridaforolimus reduces the proliferation of cell lines representing a variety of tumor types. Administration of Ridaforolimus to tumor cells in vitro elicit dose-dependent inhibition of mTOR activity with concomitant effects on cell

growth and division. Ridaforolimus exhibits a predominantly cytostatic mode of action, consistent with the findings for other mTOR inhibitors. Potent inhibitory effects on vascular endothelial growth factor secretion, endothelial cell growth, and glucose metabolism^[1].

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

In Vivo

Ridaforolimus inhibits tumor growth in mice bearing PC-3 (prostate), HCT-116 (colon), MCF7 (breast), PANC-1 (pancreas), or A549 (lung) xenografts. Ridaforolimus inhibits tumor growth in a dose-dependent manner, with 0.3 mg/kg being the lowest dose that inhibits tumor growth significantly and 3 and 10 mg/kg doses achieving maximum inhibition^[1].

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

PROTOCOL

Cell Assay ^[1]

Cells are treated with 10-fold serial dilutions of Ridaforolimus (1,000 to 0.0001 nM) or vehicle (ethanol). Following 72 hours culture at 37°C, the plates are aspirated and stored at -80°C for proliferation analysis^[1].

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

Animal Administration ^[1]

Mice: Animals selected with tumors in the proper size range are assigned to various treatment groups. Ridaforolimus, at dosages of 3 and 10 mg/kg, is administered i.p. on 2 different treatment schedules: (a) daily, 5 continuous days every other week and (b) once weekly. The control group is untreated^[1].

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

CUSTOMER VALIDATION

- Nat Nanotechnol. 2019 Oct;14(10):988-993.
- Cell Metab. 2018 Jan 9;27(1):118-135.e8.
- J Cell Sci. 2019 May 20;132(10):jcs227777.
- Molecules. 2020 Apr 23;25(8):1980.
- Patent. US20160128986A1.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Rivera VM, et al. Deforolimus (AP23573; MK-8669), a potent mTOR inhibitor, has broad antitumor activity and can be optimally administered using intermittent dosing regimens. Mol Cancer Ther. 2011 Jun;10(6):1059-71.

[2]. Brandt M, et al. mTORC1 Inactivation Promotes Colitis-Induced Colorectal Cancer but Protects from APC Loss-Dependent Tumorigenesis. Cell Metab. 2018 Jan 9;27(1):118-135.e8.

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

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