Ridaforolimus

Cat. No.: HY-50908

CAS No.: 572924-54-0

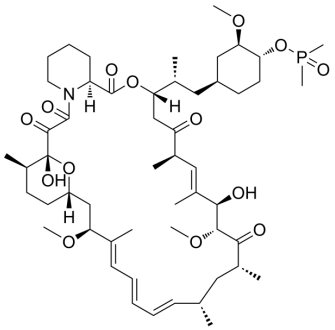
Molecular Formula: $C_{53}H_{84}NO_{14}P$

Molecular Weight: 990.21

Target: mTOR; Autophagy; Bacterial

Pathway: PI3K/Akt/mTOR; Autophagy; Anti-infection

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.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	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

1. 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
2. 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 IC_{50} of 0.2 nM in HT-1080 cells[1].

IC_{50} & 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 IC_{50}s of 0.2 and 5.6 nM, respectively, and EC_{50}s of 0.2 and 1.0 nM, respectively. In HT-1080 cells, the EC_{50} for inhibition of cell proliferation (0.5 nM) is similar to the EC_{50}s 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.
- Mol Syst Biol. 2023 Dec 18.
- Molecules. 2020 Apr 23;25(8):1980.
- J Cell Sci. 2019 May 20;132(10):jcs227777.

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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.

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