Rapamycin

Cat. No.: HY-10219

CAS No.: 53123-88-9

Molecular Formula: $C_{51}H_{79}NO_{13}$

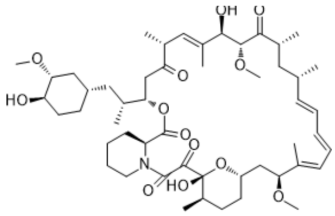
Molecular Weight: 914.17

Target: mTOR; FKBP; Autophagy; Endogenous Metabolite; Fungal; Antibiotic; Bacterial

Pathway: PI3K/Akt/mTOR; Apoptosis; Autophagy; Immunology/Inflammation; Metabolic Enzyme/Protease; Anti-infection

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 : 125 mg/mL (136.74 mM; Need ultrasonic)
Ethanol : 50 mg/mL (54.69 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.0939 mL	5.4694 mL	10.9389 mL
	5 mM	0.2188 mL	1.0939 mL	2.1878 mL
	10 mM	0.1094 mL	0.5469 mL	1.0939 mL

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

In Vivo

1. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (2.73 mM); Suspended solution
2. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (2.73 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.73 mM); Suspended solution
4. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (2.28 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (2.28 mM); Suspended solution; Need ultrasonic
6. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (2.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Rapamycin (Sirolimus; AY 22989) is a potent and specific mTOR inhibitor with an IC_{50} of 0.1 nM in HEK293 cells. Rapamycin binds to FKBP12 and specifically acts as an allosteric inhibitor of mTORC1[1]. Rapamycin is an autophagy activator, an immunosuppressant[2].

IC_{50} & Target				
	mTOR 0.1 nM (IC_{50}, in HEK293 cells)	Microbial Metabolite	Autophagy	Human Endogenous Metabolite

In Vitro

Rapamycin (12.5-100 nM; 24 hours) treatment exerts modest inhibitory effect on lung cancer cell proliferation in a dose-dependent manner in all cell lines (A549, SPC-A-1, 95D and NCI-H446 cells) tested, achieving about 30-40% reduction in cell proliferation at 100 nM vs. ~10% reduction at 12.5 nM[3].

Lung cancer cell line 95D cells are exposed to Rapamycin (10 nM, 20 nM) and RP-56976 (1 nM, 10 nM) alone or in combination (Rapamycin 20 nM+ RP-56976 10 nM). After 24 hours exposure to Rapamycin or RP-56976 alone does not significantly alter the level of expression or phosphorylation of ERK1/2, whereas cells treated with the combination of Rapamycin with RP-56976 exhibit a marked reduction in the phosphorylation levels of ERK1/2[3].

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

Cell Viability Assay[3]

Cell Line:	Lung cancer cell lines A549, SPC-A-1, 95D and NCI-H446
Concentration:	12.5 nM, 25 nM, 50 nM, 100 nM
Incubation Time:	24 hours
Result:	Treatment exerted modest inhibitory effect on lung cancer cell proliferation in a dose-dependent manner in all cell lines.

Western Blot Analysis[3]

Cell Line:	95D cells
Concentration:	10 nM and 20 nM
Incubation Time:	24 hours
Result:	Combination treatment with RP-56976 decreased phosphorylation of ERK.

In Vivo

Rapamycin (2.0 mg/kg; intraperitoneal injection; every other day; 28 days) alone has a moderate inhibitory effect. However, the combination of Metformin and Rapamycin exerts a significantly increased inhibition of tumor growth compared with the control group, the Rapamycin monotherapy group and the Metformin monotherapy group[4].

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

Animal Model:	24 male nu/nu mice aged 4-5 week old (15-20 g)[4]
Dosage:	2.0 mg/kg
Administration:	Intraperitoneal injection; every other day; 28 days
Result:	Had a moderate inhibitory effect in monotherapy group. The combination with Metformin exerted a significantly increased inhibition of tumor growth.

CUSTOMER VALIDATION

- Nature. 2021 Jun;594(7862):271-276.
- Nature. 2018 Jun;558(7711):540-546.
- Nature. 2016 Dec 1;540(7631):119-123.
- Cell. 2023 Jun 22;186(13):2802-2822.e22.
- Cancer Cell. 2021 Mar 8;39(3):380-393.e8.

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REFERENCES

[1]. Edwards SR, et al. The rapamycin-binding domain of the protein kinase mammalian target of rapamycin is a destabilizing domain. J Biol Chem, 2007, 282(18), 13395-13401.

[2]. Rangaraju S, et al. Rapamycin activates autophagy and improves myelination in explant cultures from neuropathicmice. J Neurosci. 2010 Aug 25;30(34):11388-97.

[3]. Niu H, et al. Rapamycin potentiates cytotoxicity by RP-56976 possibly through downregulation of Survivin in lung cancer cells. J Exp Clin Cancer Res. 2011 Mar 10;30:28.

[4]. Zhang JW, et al. Metformin synergizes with rapamycin to inhibit the growth of pancreatic cancer in vitro and in vivo. Oncol Lett. 2018 Feb;15(2):1811-1816.

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