Product Data Sheet

Product Name: Rapamycin (Sirolimus)
Cat. No.: GC15031

Chemical Properties

Cas. No.	53123-88-9		
SMILES	O[C@H]1[C@H](OC)C[C@H](C[C@@H](C)[C@H](CC([C@H](C)/C=C(C)/[C@H]([C@@H](OC)C([C@@H](C[C@@H](/C=C/C=C/C=C(C)/[C@@H](OC)C[C@@H]2CC[C@@H](C)[C@@](C(C(N3[C@H]4CCCC3)=O)=O)(O)O2)C)C)=O)O)=O)OC4=O)CC1		
Formula	$C_{51}H_{79}NO_{13}$	M.Wt	914.18
Solubility	≥ 45.709mg/mL in DMSO, ≥ 58.9 mg/mL in EtOH with ultrasonic	Storage	Desiccate at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

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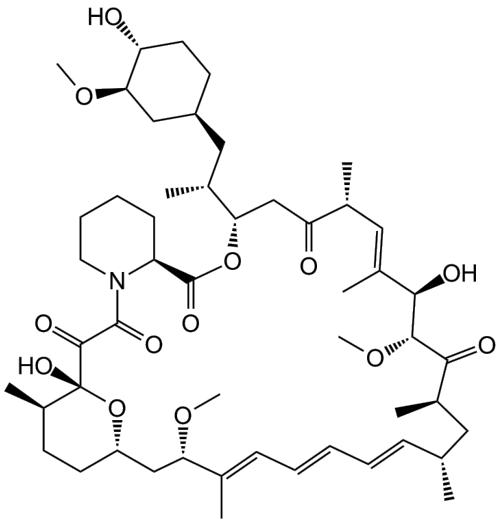
Structure



Protocol

Cell experiment [1]:

Cell lines	Jurkat cells (human T-cell leukemia cell line)
Preparation Method	Jurkat cells were cultured in RPMI-1640 medium supplemented with 10% fetal bovine serum at 37°C, 5% CO_2. Cells were treated with Rapamycin at indicated concentrations.
Reaction Conditions	0.1-100nM; 24-72h

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Applications	Rapamycin inhibited cell proliferation in a dose- and time-dependent manner, induced G1 phase arrest, upregulated p21 and p27 protein levels, downregulated cyclin D3, phospho-p70S6K(Thr389), and phospho-S6(Ser235/236), slightly downregulated phospho-Akt(Ser473), reduced telomerase activity and hTERT mRNA and protein expression, and did not induce apoptosis.

Animal experiment [2]:

Animal models	Male C57BL/6 mice (10 weeks old)
Preparation Method	Cecal ligation and puncture (CLP) was performed to establish septic mouse model, sham-operated mice were set as control. Rapamycin (5mg/kg) was intraperitoneally administered at 9h before CLP or 1h after CLP. Mice were sacrificed at 8h or 24h post CLP for tissue collection, and survival was monitored up to 80h post CLP.
Dosage form	5mg/kg; i.p.; single injection
Applications	Rapamycin upregulated LC3-II expression and promoted p62 degradation in lung tissue of CLP mice, downregulated pro-caspase-1 and pro-IL-1β expression to inhibit inflammasome activation, alleviated acute-phase pathological injuries in lung, liver and spleen, and reduced 80h mortality of CLP mice.

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References:

[1] Zhao YM, Zhou Q, Xu Y, et al. Antiproliferative effect of rapamycin on human T-cell leukemia cell line Jurkat by cell cycle arrest and telomerase inhibition. Acta Pharmacol Sin. 2008 Apr;29(4):481-488.

[2] Wang Z, Li Y, Yang X, et al. Protective effects of rapamycin induced autophagy on CLP septic mice. Comp Immunol Microbiol Infect Dis. 2019 Mar;64:47-52.

Background

Rapamycin (Sirolimus) is a macrolide mTOR inhibitor (IC_{50}=0.1nM). Rapamycin binds FKBP12 to form a complex that specifically inhibits mTORC1 signaling. Rapamycin exerts immunosuppressive effects by blocking IL-2-mediated T cell proliferation. Rapamycin can be used in research related to organ transplant rejection, anti-tumor, and anti-aging[1-4].

In vitro, Rapamycin (0.1–100nM) treated human T-cell leukemia Jurkat cells for 24–72h. Rapamycin inhibited cell proliferation in a dose- and time-dependent manner and induced G1 phase arrest. Rapamycin upregulated p21 and p27 protein levels and downregulated cyclin D3, phosphorylated p70S6K (Thr389), and phosphorylated S6 (Ser235/236) expression[5]. Rapamycin (5μM) treated human acute monocytic leukemia SHI-1 cells, KG-1α cells, HL-60 cells, and primary acute myeloid leukemia cells for 24h. Rapamycin inhibited proliferation of SHI-1 and other cells and promoted apoptosis. Rapamycin inhibited intracellular mTORC1 downstream p-4EBP1 and p-S6K1 expression and upregulated Bax, p53, and autophagy-related proteins Beclin-1, LC3B-II, and ATG5[6].

In vivo, Rapamycin (5mg/kg; single injection) was intraperitoneally injected into CLP sepsis model C57BL/6 mice at 9h before or 1h after cecal ligation and puncture. Rapamycin upregulated LC3-II expression in lung tissue, promoted p62 degradation, and reduced inflammasome activation. Rapamycin alleviated acute-phase pathological injury in lung, liver, and spleen tissues and reduced mortality of CLP septic mice[7]. Rapamycin (2.24mg/kg) was fed to female BALB/C mice for 4 months. Rapamycin delayed brain aging in mice[8].

References:

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[1] Niu H, Wang J, Li H, et al. Rapamycin potentiates cytotoxicity by docetaxel possibly through downregulation of Survivin in lung cancer cells. J Exp Clin Cancer Res. 2011 Mar 10;30(1):28.

[2] Edwards SR, Wandless TJ. The rapamycin-binding domain of the protein kinase mammalian target of rapamycin is a destabilizing domain. J Biol Chem. 2007 May 4;282(18):13395-401.

[3]Rangaraju S, Verrier JD, Madorsky I, et al. Rapamycin activates autophagy and improves myelination in explant cultures from neuropathic mice. J Neurosci. 2010 Aug 25;30(34):11388-97.

[4] Selvarani R, Mohammed S, Richardson A. Effect of rapamycin on aging and age-related diseases-past and future. Geroscience. 2021 Jun;43(3):1135-1158.

[5] Zhao YM, Zhou Q, Xu Y, et al. Antiproliferative effect of rapamycin on human T-cell leukemia cell line Jurkat by cell cycle arrest and telomerase inhibition. Acta Pharmacol Sin. 2008 Apr;29(4):481-488.

[6] Xu J, Zong S, Sheng T, et al. Rapamycin increases leukemia cell sensitivity to chemotherapy by regulating mTORC1 pathway-mediated apoptosis and autophagy. Int J Hematol. 2024;119(5):541-551.

[7] Wang Z, Li Y, Yang X, et al. Protective effects of rapamycin induced autophagy on CLP septic mice. Comp Immunol Microbiol Infect Dis. 2019 Mar;64:47-52.

[8] Yin Z, Guo X, Qi Y, et al. Dietary Restriction and Rapamycin Affect Brain Aging in Mice by Attenuating Age-Related DNA Methylation Changes. Genes. 2022 Apr;13(4):699.

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