

Product Name: Rapamycin (Sirolimus) Revision Date: 01/10/2021



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Rapamycin (Sirolimus)

Cat. No.:	A8167
CAS No.:	53123-88-9
Formula:	C51H79NO13
M.Wt:	914.18
Synonyms:	Sirolimus,(-)-Rapamycin, AY-22989,
	WY-090217, Antibiotic AY22989
Target:	PI3K/Akt/mTOR Signaling
Pathway:	mTOR
Storage:	Desiccate at -20°C
	PERMIT

Solvent & Solubility

	\geqslant 45.7 mg/mL in DM	MSO; insoluble in H2O; \geq 58.9 mg/mL in EtOH with ultrasonic			
Preparing In Vitro Stock Solutions		Mass Solvent Concentration	1mg	5mg	10mg
	1 mM	1.0939 mL	5.4694 mL	10.9388 mL	
	DEP	5 mM	0.2188 mL	1.0939 mL	2.1878 mL
	A Contraction	10 mM	0.1094 mL	0.5469 mL	1.0939 mL

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

Biological Activity

Shortsummary	Original antifungal antibiotic			
IC ₅₀ & Target	~0.1 nM (mTOR)			
	Cell Viability Assay			
In Vitro	Cell Line:	Hepatocyte growth factor (HGF)-induced lens epithelial cells (LECs)		
	Preparation method:	The solubility of this compound in DMSO is >10 mM. General tips for obtaining		
		a higher concentration: Please warm the tube at 37 °C for 10 minutes and/or		
		shake it in the ultrasonic bath for a while.Stock solution can be stored below		
		-20°C for several months.		

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	Reacting conditions:	10 ng/ml, 72h		
	Applications:	Using cell proliferation, cell viability and flow cytometric apoptosis assays, we		
		found that rapamycin potently not only suppressed proliferation but also		
		induced the apoptosis of LECs in a dose-dependent manner under HGF		
		administration. Further investigation of the underlying mechanism using siRNA		
	210	transfection revealed that rapamycin could promote apoptosis of LECs via		
	E	inhibiting HGF-induced phosphorylation of AKT/mTOR, ERK and JAK2/STAT3		
	all an an and a second	signaling molecules. Moreover, the forced expression of AKT, ERK and STAT3		
		could induce a significant suppression of apoptosis in these cells after		
		treatment of rapamycin.		
	Animal experiment			
	Animal models:	Ndufs4(−/−) mice		
	Dosage form:	8 mg/kg every other day, intraperitoneal injection		
	Applications:	Rapamycin, a specific inhibitor of the mechanistic target of rapamycin (mTOR)		
	610	signaling pathway, robustly enhances survival and attenuates disease		
	OE contract	progression in a mouse model of Leigh syndrome. Administration of rapamycin		
	Provide State	to these mice, which are deficient in the mitochondrial respiratory chain subunit		
		Ndufs4 [NADH dehydrogenase (ubiquinone) Fe-S protein 4], delays onset of		
In Vivo		neurological symptoms, reduces neuroinflammation, and prevents brain		
		lesions. Although the precise mechanism of rescue remains to be determined,		
		rapamycin induces a metabolic shift toward amino acid catabolism and away		
		from glycolysis, alleviating the buildup of glycolytic intermediates. This		
		therapeutic strategy may prove relevant for a broad range of mitochondrial		
	-10	diseases.		
	Other notes:	Please test the solubility of all compounds indoor, and the actual solubility may		
	and A Provint St.	slightly differ with the theoretical value. This is caused by an experimental		
	and the second	system error and it is normal.		

Product Citations

1. Tang RH, Qi RQ, et al. "Interleukin-4 affects microglial autophagic flux." Neural Regen Res. 2019 Sep;14(9):1594-1602.PMID:31089059

2. Yang D, Zhang B, et al. "COPS5 negatively regulates goat endometrial function via the ERN1 and mTOR-autophagy pathways during early pregnancy." J Cell Physiol. 2019 Aug;234(10):18666-18678.PMID:30927262

3. Zhang M, Liu F, et al. "The MTOR signaling pathway regulates macrophage differentiation from mouse myeloid progenitors by inhibiting autophagy." Autophagy. 2019 Feb 27:1-13.PMID:30724690

4. Rossi A, Pakhomova ON, et al. "Mechanisms and immunogenicity of nsPEF-induced cell death in B16F10 melanoma tumors." Sci Rep. 2019 Jan 23;9(1):431.PMID:30674926

5. Benjamin Cook. "Investigating Autophagy, Extracellular Vesicles, and Glycobiology." Loyola University Chicago.2018.

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References

1. Tian F, Dong L, Zhou Y et al. Rapamycin-Induced Apoptosis in HGF-Stimulated Lens Epithelial Cells by AKT/mTOR, ERK and JAK2/STAT3 Pathways. Int J Mol Sci. 2014 Aug 11;15(8):13833-48.

2. Johnson SC1, Yanos ME, Kayser EB et al. mTOR inhibition alleviates mitochondrial disease in a mouse model of Leigh syndrome. Science. 2013 Dec 20;342(6165):1524-8.

Caution

FOR RESEARCH PURPOSES ONLY.

NOT FOR HUMAN, VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

Specific storage and handling information for each product is indicated on the product datasheet. Most APExBIO products are stable under the recommended conditions. Products are sometimes shipped at a temperature that differs from the recommended storage temperature. Shortterm storage of many products are stable in the short-term at temperatures that differ from that required for long-term storage. We ensure that the product is shipped under conditions that will maintain the quality of the reagents. Upon receipt of the product, follow the storage recommendations on the product data sheet.

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