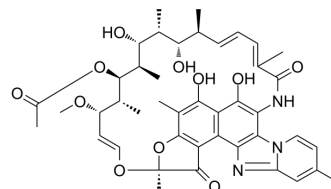


Rifaximin

Cat. No.:	HY-13234
CAS No.:	80621-81-4
Molecular Formula:	C ₄₃ H ₅₁ N ₃ O ₁₁
Molecular Weight:	785.88
Target:	Bacterial; Antibiotic; DNA/RNA Synthesis
Pathway:	Anti-infection; Cell Cycle/DNA Damage
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (63.62 mM)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		1.2725 mL	6.3623 mL	12.7246 mL
	5 mM		0.2545 mL	1.2725 mL	2.5449 mL
	10 mM		0.1272 mL	0.6362 mL	1.2725 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: ≥ 3 mg/mL (3.82 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 3 mg/mL (3.82 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: 2.08 mg/mL (2.65 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Rifaximin, a gastrointestinal-selective antibiotic, binds the β-subunit of bacterial DNA-dependent RNA polymerase, resulting in inhibition of bacterial RNA synthesis. Rifaximin susceptibility is higher against Gram-positive strains (MIC: 0.03-5 mg/ml) compared to Gram-negative bacteria (MIC: 8-50 mg/mL)^{[1][2]}.

In Vitro

Rifaximin has a good inhibitory activity against Staphylococcus, Streptococcus, Enterococcus, Escherichia coli, Shigella, Salmonella, Bacillus cereus, Moraxella catarrhalis, Haemophilus influenzae, Haemophilus ducreyi, Bacteroides bivius-

disiens, Gardnerella vaginalis, Lactobacillus spp., Mobiluncus spp., Neisseria gonorrhoeae, Pseudomonas and Acinetobacter . Rifaximin rarely causes side effects^[1].
 Rifaximin (0.1, 1.0 and 10.0 μ M) causes significant and concentration-dependent reduction of cell proliferation, cell migration and PCNA expression in the Caco-2 cells vs. untreated cells^[2].
 Rifaximin (0.1-10 μ M) downregulates Akt/mTOR and p38MAPK/NF- κ B pathways through a PXR-dependent mechanism^[2].
 MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay^[2]

Cell Line:	Caco-2 cells
Concentration:	0.1, 1.0 and 10.0 μ M
Incubation Time:	48 hours
Result:	Caused a significant and concentration-dependent reduction in cell proliferation. Reduced the expression of PCNA in a concentration-dependent manner.

Western Blot Analysis^[2]

Cell Line:	Caco-2 cells
Concentration:	0.1, 1.0 and 10.0 μ M
Incubation Time:	24 hours
Result:	Reduced Akt, mTOR, p38 MAPK and HIF-1 α expression in a concentration-dependent manner. Inhibited NF- κ B nuclear activation and p70S6K.

In Vivo

Rifaximin administration (30 or 50 mg/kg/day) increases survival rates of colitic mice and reduces colitis severity by improvement of wasting syndrome, histologic scores, decrease in colon IL-2, IL-12, IFN-gamma and TNF-alpha (protein and mRNA) levels, and diminishes colon myeloperoxidase (MPO) activity^[3].
 MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Balb/c mice (6–8 weeks old) bearing 2,4,6-trinitrobenzene sulfonic acid (TNBS)-induced colitis ^[3]
Dosage:	10, 30 and 50 mg/kg/day
Administration:	Orally, p.o. daily for 7 days
Result:	Significantly reduced TNBS induced colitis at the dose of 30 and 50 mg/kg, but not 10 mg/kg. A 7-day course of 30 and 50 mg/kg resulted in an almost complete tissue protection.

CUSTOMER VALIDATION

- Mol Pharm. 2022 Oct 21.

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REFERENCES

[1]. Veronica Ojetti, et al. Rifaximin pharmacology and clinical implications. *Expert Opin Drug Metab Toxicol.* 2009 Jun;5(6):675-82.

[2]. Giuseppe Esposito, et al. Rifaximin, a non-absorbable antibiotic, inhibits the release of pro-angiogenic mediators in colon cancer cells through a pregnane X receptor-dependent pathway. *Int J Oncol.* 2016 Aug;49(2):639-45.

[3]. Stefano Fiorucci, et al. Inhibition of intestinal bacterial translocation with rifaximin modulates lamina propria monocytic cells reactivity and protects against inflammation in a rodent model of colitis. *Digestion.* 2002;66(4):246-56.

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

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