# Rifaximin

**Cat. No.:** HY-13234

**CAS No.:** 80621-81-4

**Molecular Formula:** $C_{43}H_{51}N_3O_{11}$

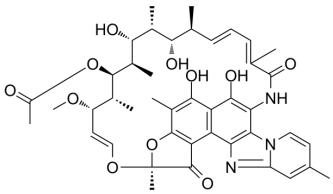
**Molecular Weight:** 785.88

**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.

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 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**

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3 mg/mL (3.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (3.82 mM); Clear solution
3. 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 μM) 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 μM) downregulates Akt/mTOR and p38MAPK/NF-κB 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 μM |
| 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 μM |
| Incubation Time: | 24 hours |
| Result: | Reduced Akt, mTOR, p38 MAPK and HIF-1α expression in a concentration-dependent manner.Inhibited NF-κB 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.



Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA