# Rifamycin S

**Cat. No.:** HY-125365

**CAS No.:** 13553-79-2

**Molecular Formula:** $C_{37}H_{45}NO_{12}$

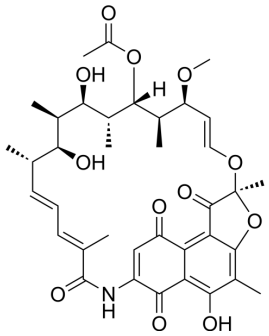
**Molecular Weight:** 695.75

**Target:** Bacterial; Reactive Oxygen Species; Antibiotic

**Pathway:** Anti-infection; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

**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 : 100 mg/mL (143.73 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 1.4373 mL | 7.1865 mL | 14.3730 mL |
| | 5 mM | 0.2875 mL | 1.4373 mL | 2.8746 mL |
| | 10 mM | 0.1437 mL | 0.7186 mL | 1.4373 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: 2.5 mg/mL (3.59 mM); Suspended solution; Need ultrasonic

## BIOLOGICAL ACTIVITY

**Description**

Rifamycin S, a quinone, is an antibiotic against Gram-positive bacteria (including MRSA). Rifamycin S is the oxidized forms of a reversible oxidation-reduction system involving two electrons. Rifamycin S generates reactive oxygen species (ROS) and inhibits microsomal lipid peroxidation. Rifamycin S can be used for tuberculosis and leprosy[1][2][3].

**$IC_{50}$ & Target**

Gram-positive bacteria[3]
Reactive oxygen species (ROS)[1]

**In Vitro**

The inhibition of bacterial growth by Rifamycin SV is due to the production of active species of oxygen resulting from the oxidation-reduction cycle of Rifamycin SV in the cells. The aerobic oxidation of Rifamycin SV to Rifamycin S is induced by metal ions, such as $Mn^{2+}$, $Cu^{2+}$, and $Co^{2+}$. The most effective metal ion is $Mn^{2+}$[2].

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

| | |
|---|---|
| **In Vivo** | Rat liver sub-mitochondrial particles also generated hydroxyl radical in the presence of NADH and Rifamycin S. NADH dehydrogenase (complex I) as the major component involved in the reduction of Rifamycin S. Compared to NADPH, NADH is almost as effective (Rifamycin S) in catalyzing the interactions of these antibiotics with rat liver microsomes. Rifamycin S is shown to be readily reduced to Rifamycin SV, the corresponding hydroquinone by Fe(II). Rifamycin S forms a detectable Fe(II)-(Rifamycin S)3 complex. The Fe:ATP induced lipid peroxidation is completely inhibited by Rifamycin S. Rifamycin S can interact with rat liver microsomes to undergo redox-cycling, with the subsequent production of hydroxyl radicals when iron complexes are present[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2023 Mar 7;120(10):e2217804120.

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## REFERENCES

[1]. Rao DN, et al. A comparative study of the redox-cycling of a quinone (rifamycin S) and a quinonimine (rifabutin) antibiotic by rat liver microsomes. Free Radic Biol Med. 1997;22(3):439-46.

[2]. Kono Y. Oxygen Enhancement of bactericidal activity of rifamycin SV on Escherichia coli and aerobic oxidation of rifamycin SV to rifamycin S catalyzed by manganous ions: the role of superoxide. J Biochem. 1982 Jan;91(1):381-95.

[3]. Huang H, et al. Rifamycin S and its geometric isomer produced by a newly found actinomycete, Micromonospora rifamycinica. Antonie Van Leeuwenhoek. 2009 Feb;95(2):143-8.



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