Rifampicin

Cat. No.: HY-B0272

CAS No.: 13292-46-1

Molecular Formula: $C_{43}H_{58}N_4O_{12}$

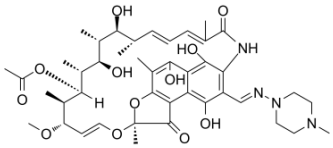
Molecular Weight: 822.94

Target: Bacterial; Influenza Virus; Antibiotic

Pathway: Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (60.76 mM; Need ultrasonic)

H_2O : 1 mg/mL (1.22 mM; ultrasonic and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2152 mL	6.0758 mL	12.1516 mL
5 mM	0.2430 mL	1.2152 mL	2.4303 mL
10 mM	0.1215 mL	0.6076 mL	1.2152 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 (3.04 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.04 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rifampicin is a potent and broad spectrum antibiotic against bacterial pathogens. Rifampicin has anti-influenza virus activities.

In Vitro

Rifampicin (100 mg/mL) can block the functional activity of P-glycoprotein. Rifampicin is not a substract for P-glycoprotein. The mechanism of rifampicin resistance is unassociated with the functional activity of P-glycoprotein[3].

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

In Vivo

Rifampicin (200, 400 mg/kg) can induce fatty liver at high concentration[1]. Rifampicin (30 mg/kg, i.p.) treatment of S464P biofilms in vivo results in a slight decline, but earlier rebinds in bioluminescence from these catheters compared with the

parental signal, whereas rifampicin has no affect on bioluminescence in mice infected with mutant H481Y[2].

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PROTOCOL

Animal Administration [2]

Briefly, 1 cm Teflon catheter (14-gauge) carrying 104 cfu S. aureus, either the parental strain Xen 29 or the RifR mutants S464P or H481Y, are implanted subcutaneously in groups of nine mice per strain. One catheter segment is inserted on each side of each animal. Six days after the implantation of the catheters, five mice from each group are treated with rifampicin at 30 mg/kg intraperitoneally in 0.1 mL saline, twice daily for four consecutive days. The remaining four mice in each group are left untreated as controls. At various time points during the infection, the mice are anaesthetized using a constant flow of 1.5% isoflurane from the IVIS® manifold, and imaged using an IVIS® Image System 100 Series. The bioluminescent signals (photons/s) emitted from the mice are analysed using LivingImage® software and plotted over the course of infection. The mice are sacrificed 20 days after infection (11 days after final rifampicin treatment). The catheters are surgically removed and the bacteria are detached by sonication for determination of bacterial burdens on the catheters.

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

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2020 Jul 21;7(17):2001374.
- Phytomedicine. 2019 Mar 15;56:175-182.
- Onco Targets Ther. 2018 Sep 17;11:5885-5894.
- RSC Adv. 2019, 9(28):16136-16146.
- Neurotox Res. 2020 Dec;38(4):859-870.

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REFERENCES

[1]. Piriou A, et al. Fatty liver induced by high doses of rifampicin in the rat: possible relation with an inhibition of RNA polymerases in eukariotic cells. Arch Toxicol Suppl. 1979;(2):333-7.

[2]. Yu J, et al. Monitoring in vivo fitness of rifampicin-resistant Staphylococcus aureus mutants in a mouse biofilm infection model. J Antimicrob Chemother. 2005 Apr;55(4):528-34. Epub 2005 Mar 2.

[3]. Erokhina MV, et al. [In vitro development of rifampicin resistance in the epithelial cells]. Probl Tuberk Bolezn Legk. 2006;(8):58-61.

[4]. Hamzehei M, et al. Inhibition of influenza A virus replication by rifampicin and selenocystamine. J Med Virol. 1980;6(2):169-74.

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