# Rilpivirine hydrochloride

**Cat. No.:** HY-10574A

**CAS No.:** 700361-47-3

**Molecular Formula:** $C_{22}H_{19}ClN_6$

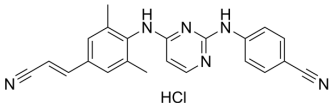
**Molecular Weight:** 402.88

**Target:** SARS-CoV; MMP

**Pathway:** Anti-infection; Metabolic Enzyme/Protease

**Storage:** 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)




## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 125 mg/mL (310.27 mM; Need ultrasonic)

**Preparing Stock Solutions**

| Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 2.4821 mL | 12.4106 mL | 24.8213 mL |
| 5 mM | 0.4964 mL | 2.4821 mL | 4.9643 mL |
| 10 mM | 0.2482 mL | 1.2411 mL | 2.4821 mL |

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

## BIOLOGICAL ACTIVITY

**Description**

Rilpivirine (R278474) hydrochloride is a potent and specific diarylpyrimidine (DAPY) non-nucleoside reverse transcriptase inhibitor (NNRTI). Rilpivirine hydrochloride has high antiviral activity against wild-type HIV ($EC_{50}$=0.4 nM) and mutant viruses ($EC_{50}$=0.1-2.0 nM). Rilpivirine hydrochloride has a high genetic barrier to resistance development of HIV[1][2].

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

IC50: 20±10 μM (MMP)[1]Ki: 1.5±0.27 nM (MMPs)[1]

**In Vitro**

R278474 is active against wild-type HIV-1 ($EC_{50}$=0.4 nM) and all single and double mutants tested ($EC_{50}$=0.1-2.0 nM)[1].
R278474 (10-5000 nM; 30 d) does not observe the sign of wild-type HIV-1 breakthrough at 1 μM within 30 days[1].
R278474 inhibits 81% of clinical isolates (about 1200 recombinant clinical isolates) at a 50% inhibitory concentration ($EC_{50}$) less than 1 nM, and inhibits 94% at $EC_{50}$ less than 10 nM[1].
TMC278 shows subnanomolar $EC_{50}$s against wild-type HIV-1 group M isolates (0.07-1.01 nM)[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**In Vivo**

R278474 (10-160 mg/kg; p.o. for 1 month) does not produce abnormal effects in rat, apart from liver weight increase and species-specific thyroid hypertrophy, both at the higher dose levels[1].
R278474 (i.v.) exhibits elimination half-life ranges from 4.4 h in rat to 31 h in dog, and exposure ($AUC_{inf}$) amounts to 3.1 μg h/mL (4 mg/kg) in rat, 8.7 μg h/mL (1.25 mg/kg) in dog, 1.4 μg h/mL (1.25 mg/ kg) in monkey, and 44 μg h/mL (1.25 mg/kg) in

rabbit[1].

R278474 (p.o.) exhibits half-life ranges between 2.8 h in rat and 39 h in dog, and oral bioavailability of 32% and 31% in rat and dog[1].

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

## CUSTOMER VALIDATION

- Int J Antimicrob Agents. 2019 Dec;54(6):814-819.
- Br J Cancer. 2023 Jan 30.
- Pharmaceuticals. 2022, 15(10), 1186.
- Sci Rep. 2015 Oct 29;5:15806.
- PLoS One. 2021 Mar 10;16(3):e0248139.

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## REFERENCES

[1]. Shiori Haga, et al. TACE Antagonists Blocking ACE2 Shedding Caused by the Spike Protein of SARS-CoV Are Candidate Antiviral Compounds. Antiviral Res. 2010 Mar;85(3):551-5.

[2]. Kruse MN, et al. Human meprin alpha and beta homo-oligomers: cleavage of basement membrane proteins and sensitivity to metalloprotease inhibitors. Biochem J. 2004 Mar 1;378(Pt 2):383-9.

[3]. Wang R, et al. A Disintegrin and Metalloproteinase Domain 17 Regulates Colorectal Cancer Stem Cells and Chemosensitivity Via Notch1 Signaling. Stem Cells Transl Med. 2016 Mar;5(3):331-8.



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