# Product data sheet

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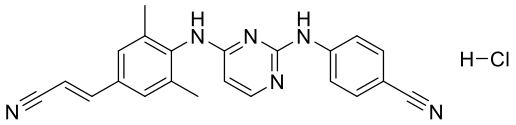
|---|---|
| MedKoo Cat#: 329466<br>Name: Rilpivirine HCl<br>CAS#: 700361-47-3 (HCl)<br>Chemical Formula: $C_{22}H_{19}ClN_6$<br>Molecular Weight: 402.886 |  |
| Product supplied as: | Powder |
| Purity (by HPLC): | ≥ 98% |
| Shipping conditions | Ambient temperature |
| Storage conditions: | Powder: -20°C 3 years; 4°C 2 years. |
| | In solvent: -80°C 3 months; -20°C 2 weeks. |

**1. Product description:**
Rilpivirine, also known as TMC278, is a second-generation non-nucleoside reverse transcriptase inhibitor (NNRTI) with higher potency, longer half-life and reduced side-effect profile compared with older NNRTIs, such as efavirenz. Rilpivirine was approved for use in the United States in May 2011.

**2. CoA, QC data, SDS, and handling instruction**
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

**3. Solubility data**

| Solvent | Max Conc. mg/mL | Max Conc. mM |
|---|---|---|
| DMSO | 50 | 136.46 |

**4. Stock solution preparation table:**

| Concentration / Solvent Volume / Mass | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 2.48 mL | 12.41 mL | 24.82 mL |
| 5 mM | 0.50 mL | 2.48 mL | 4.96 mL |
| 10 mM | 0.25 mL | 1.24 mL | 2.48 mL |
| 50 mM | 0.05 mL | 0.25 mL | 0.50 mL |

**5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator**
Please refer the product web page under section of "Calculator"

**6. Recommended literature which reported protocols for in vitro and in vivo study**
In vitro study

1. Azijn H, Tirry I, Vingerhoets J, de Béthune MP, Kraus G, Boven K, Jochmans D, Van Craenenbroeck E, Picchio G, Rimsky LT. TMC278, a next-generation nonnucleoside reverse transcriptase inhibitor (NNRTI), active against wild-type and NNRTI-resistant HIV-1. Antimicrob Agents Chemother. 2010 Feb;54(2):718-27. doi: 10.1128/AAC.00986-09. Epub 2009 Nov 23. PMID: 19933797; PMCID: PMC2812151.

2. Martí-Rodrigo A, Alegre F, Moragrega ÁB, García-García F, Martí-Rodrigo P, Fernández-Iglesias A, Gracia-Sancho J, Apostolova N, Esplugues JV, Blas-García A. Rilpivirine attenuates liver fibrosis through selective STAT1-mediated apoptosis in hepatic stellate cells. Gut. 2020 May;69(5):920-932. doi: 10.1136/gutjnl-2019-318372. Epub 2019 Sep 17. PMID: 31530714.

In vivo study

1. Martí-Rodrigo A, Alegre F, Moragrega ÁB, García-García F, Martí-Rodrigo P, Fernández-Iglesias A, Gracia-Sancho J, Apostolova N, Esplugues JV, Blas-García A. Rilpivirine attenuates liver fibrosis through selective STAT1-mediated apoptosis in hepatic stellate cells. Gut. 2020 May;69(5):920-932. doi: 10.1136/gutjnl-2019-318372. Epub 2019 Sep 17. PMID: 31530714.

**7. Bioactivity**
Biological target:

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Rilpivirine (R278474, TMC27, DB08864) is a non-nucleoside reverse transcriptase inhibitor (NNRTI), and used to treat HIV-1 infection.

## In vitro activity

In vitro, considerable cross-resistance between TMC278 and etravirine was observed. Sensitivity to TMC278 was observed for 62% of efavirenz- and/or nevirapine-resistant HIV-1 recombinant clinical isolates. TMC278 inhibited viral replication at concentrations at which first-generation NNRTIs could not suppress replication. The rates of selection of TMC278-resistant strains were comparable among HIV-1 group M subtypes. NNRTI RAMs emerging in HIV-1 under selective pressure from TMC278 included combinations of V90I, L100I, K101E, V106A/I, V108I, E138G/K/Q/R, V179F/I, Y181C/I, V189I, G190E, H221Y, F227C, and M230I/L. E138R was identified as a new NNRTI RAM. These in vitro analyses demonstrate that TMC278 is a potent next-generation NNRTI, with a high genetic barrier to resistance development.

Reference: Antimicrob Agents Chemother. 2010 Feb;54(2):718-27. https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/19933797/

## In vivo activity

Transcriptomic analysis of livers from HFD-fed mice treated with RPV showed significant differences in biological functions associated with lipid metabolism, inflammation, the immune system, cell cycle and apoptosis when compared with Veh-administered HFD animals (online supplementary figure 1). Data analysis revealed a conjunction of enhanced pro-proliferative and antiproliferative signalling pathways that may be associated with differential responses exerted by liver cell populations (online supplementary figure 1C and online supplementary tables 5–9). To clarify this point, we analysed cell proliferation and apoptosis in liver sections using Ki67 staining and TUNEL assay, respectively. Livers from RPV-treated mice presented a lower number of proliferating non-parenchymal cells and rise in that of parenchymal cells with respect to Veh-treated HFD mice (figure 2A). Conversely, apoptosis was clearly increased in non-parenchymal cells and diminished in hepatocytes (figure 2B). RPV-mediated alterations of cell proliferation were confirmed in whole-liver samples by qRT-PCR (figure 2C), with significant gene expression of important markers.

Reference: Gut. 2020 May;69(5):920-932. https://gut.bmj.com/lookup/pmidlookup?view=long&pmid=31530714