Tenofovir Disoproxil fumarate

Cat. No.: HY-13782

CAS No.: 202138-50-9

Molecular Formula: $C_{23}H_{34}N_5O_{14}P$

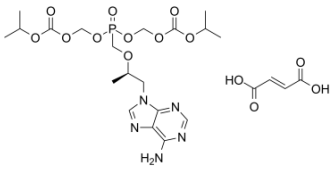
Molecular Weight: 635.51

Target: HIV; Reverse Transcriptase; HBV

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 (78.68 mM)

H_2O : 16.67 mg/mL (26.23 mM; Need ultrasonic and warming)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.5735 mL	7.8677 mL	15.7354 mL
5 mM	0.3147 mL	1.5735 mL	3.1471 mL
10 mM	0.1574 mL	0.7868 mL	1.5735 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 20 mg/mL (31.47 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (3.93 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.93 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.93 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tenofovir Disoproxil fumarate is a nucleotide reverse transcriptase inhibitor used to treat HIV and chronic Hepatitis B.

In Vitro

Tenofovir shows cytotoxic effects on cell viability in HK-2 cells, with IC_{50} values of 9.21 and 2.77 μM at 48 and 72 h in MTT

	assay, respectively. Tenofovir diminishes ATP levels in HK-2 cells. Tenofovir (3.0 to 28.8 μM) increases oxidative stress and protein carbonylation in HK-2 cells. Furthermore, Tenofovir induces apoptosis in HK-2 cells, and that apoptosis is induced via mitochondrial damage[1]. Tenofovir and M48U1 formulated in 0.25% HEC each inhibits the replication of both R5-tropic HIV-1_{BaL} and X4-tropic HIV-1_{IIIb} in activated PBMCs, and inhibits several laboratory strains and patient-derived HIV-1 isolates. The combined formulation of M48U1 and tenofovir in 0.25% HEC exhibits synergistic antiretroviral activity against infection with R5-tropic HIV-1_{BaL}, and is not toxic to PBMCs[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Tenofovir Disoproxil fumarate (20, 50, 140, or 300 mg/kg) administered to BLT mice, shows dose dependent activity during vaginal HIV challenge in BLT humanized mice. Tenofovir Disoproxil fumarate (50, 140, 300 mg/kg) significantly reduces HIV transmission in BLT mice[3]. Tenofovir Disoproxil fumarate (0.5, 1.5, or 5.0 mg/kg/day, p.o.) induces a dose-dependent decline in serum viremia in woodchucks chronically infected with WHV. Tenofovir Disoproxil fumarate administration is safe and effective in the woodchuck model of chronic HBV infection[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cells are plated into 48-well tissue culture plates (39,000 cells/mL) and allowed to grow for 48 h followed by treatment with vehicle or Tenofovir. Following the treatment period, cell viability is assessed using the MTT assay. The MTT assay relies on the conversion of tetrazolium dye 3-(4,5-dimethlthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) to formazan by NAD(P)H-dependent oxidoreductases. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Twenty adult chronic WHV carrier woodchucks are stratified equally by age, sex, body weight, and serum GGT activity into five treatment groups consisting of four animals each: (i) Tenofovir Disoproxil fumarate at 15.0 mg/kg once per day, (ii) Tenofovir Disoproxil fumarate at 5.0 mg/kg/day, (iii) Tenofovir Disoproxil fumarate at 1.5 mg/kg/day, (iv) Tenofovir Disoproxil fumarate at 0.5 mg/kg/day, and (v) a placebo control. The woodchucks are treated daily for 4 weeks and observed for an additional 12 weeks following cessation of drug treatment. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Gastroenterol. 2020 Nov 19.
- Int J Antimicrob Agents. 2019 Dec;54(6):814-819.
- J Neuroimmune Pharmacol. 2019 Jul 23;10.1007/s11481-019-09862-1.
- J Neuroimmune Pharmacol. 2017 Dec;12(4):682-692.
- Sci Rep. 2019 Nov 20;9(1):17158.

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REFERENCES

[1]. Murphy RA, et al. Establishment of HK-2 Cells as a Relevant Model to Study Tenofovir-Induced Cytotoxicity. Int J Mol Sci. 2017 Mar 1;18(3)

[2]. Musumeci G, et al. M48U1 and Tenofovir combination synergistically inhibits HIV infection in activated PBMCs and human cervicovaginal histocultures. Sci Rep. 2017 Feb 1;7:41018

[3]. Wahl A, et al. Predicting HIV Pre-exposure Prophylaxis Efficacy for Women using a Preclinical Pharmacokinetic-Pharmacodynamic In Vivo Model. Sci Rep. 2017 Feb

1;7:41098

[4]. Menne S, Cote PJ, Korba BE, Antiviral effect of oral administration of tenofovir disoproxil fumarate in woodchucks with chronic woodchuck hepatitis virus infection. Antimicrob Agents Chemother. 2005 Jul;49(7):2720-8.

[5]. Xu P, et al. Combined Medication of Antiretroviral Drugs Tenofovir Disoproxil Fumarate, Emtricitabine, and Raltegravir Reduces Neural Progenitor Cell Proliferation In Vivo and In Vitro. J Neuroimmune Pharmacol. 2017 Dec;12(4):682-692.

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