Tenofovir exalidex

Cat. No.: HY-109014

CAS No.: 911208-73-6

Molecular Formula: $C_{28}H_{52}N_5O_5P$

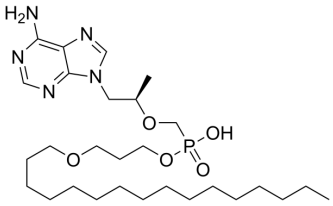
Molecular Weight: 569.72

Target: HIV; HBV; Nucleoside Antimetabolite/Analog

Pathway: Anti-infection; Cell Cycle/DNA Damage

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 : 100 mg/mL (175.52 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7552 mL	8.7762 mL	17.5525 mL
	5 mM	0.3510 mL	1.7552 mL	3.5105 mL
	10 mM	0.1755 mL	0.8776 mL	1.7552 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (3.51 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tenofovir exalidex (CMX157) is a lipid conjugate of the acyclic nucleotide analog Tenofovir with activity against both wild-type and antiretroviral drug-resistant HIV strains, including multidrug nucleoside/nucleotide analog-resistant viruses. Tenofovir exalidex is active against all major subtypes of HIV-1 and HIV-2 in fresh human PBMCs and against all HIV-1 strains evaluated in monocyte-derived macrophages, with EC_{50}s ranging between 0.2 and 7.2 nM. CMX157 is orally available and has no apparent toxicity. Tenofovir exalidex also shows antiviral activity against HBV[1][2][3].

IC_{50} & Target	HIV-1	HIV-2

In Vitro

Tenofovir exalidex is consistently >300-fold more active than Tenofovir against multiple viruses in several different cell systems. Tenofovir exalidex will be effective against MNR mutants, including those that are unresponsive to all currently available NRTIs. Notably, the average EC_{50} in PBMCs for CMX157 against a panel of 27 wild-type HIV-1 isolates representing group M subtypes A to G and group O was 2.6 nM (range, 0.2 to 7.2 nM)[1].

Tenofovir exalidex exerts its therapeutic actions by inhibiting HBV polymerase-mediated HBV DNA elongation, but there is no known binding of cyclophilins to HBV polymerase nor participation of cyclophilins in DNA elongation. The combinational effect of CRV431 (host-targeting) and Tenofovir exalidex (direct-acting) on HBV DNA production is more consistent with the two compounds acting on distinct steps of the HBV life cycle[3].

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

In Vivo

Tenofovir exalidex (Sprague-Dawley rats) is orally available and has no apparent toxicity when given orally to rats for 7 days at doses of 10, 30, or 100 mg/kg/day[2].Tenofovir exalidex (5-10 mg/kg; oral gavage; daily for a period of 16 days) decreases liver HBV DNA levels dose-dependently[3].

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

Animal Model:	Female transgenic mice HBV transgenic Tg05 mice (C57BL/6)[1]
Dosage:	5 mg/kg, 10 mg/kg
Administration:	Oral gavage; daily for a period of 16 days
Result:	The reductions in HBV DNA were 55% and 97% for low-dose (5 mg/kg/day) and high-dose (10 mg/kg/day), respectively.

REFERENCES

[1]. Lanier ER, et al. Development of hexadecyloxypropyl tenofovir (CMX157) for treatment of infection caused by wild-type and nucleoside/nucleotide-resistant HIV. Antimicrob Agents Chemother. 2010;54(7):2901-2909.

[2]. Gallay P, et al. The cyclophilin inhibitor CRV431 inhibits liver HBV DNA and HBsAg in transgenic mice. PLoS One. 2019;14(6):e0217433. Published 2019 Jun 10.

[3]. Painter GR, et al. Evaluation of hexadecyloxypropyl-9-R-[2-(Phosphonomethoxy)propyl]- adenine, CMX157, as a potential treatment for human immunodeficiency virus type 1 and hepatitis B virus infections [published correction appears in Antimicrob Agents Chemother. 2007 Dec;51(12):4538]. Antimicrob Agents Chemother. 2007;51(10):3505-3509.

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