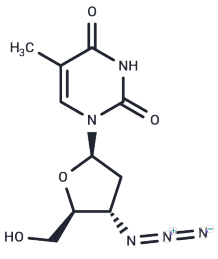
Data Sheet (Cat.No.T1416)

TargetMol

Zidovudine

Chemical Properties



CAS No. : 30516-87-1

Formula: C10H13N5O4

Molecular Weight: 267.24

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Zidovudine (ZDV) is a synthetic dideoxynucleoside. After intracellular phosphorylation to its active metabolite, zidovudine inhibits DNA polymerase, resulting in the inhibition of DNA replication and cell death. This agent also decreases levels of available pyrimidines.
Targets(IC50)	HIV Protease,Reverse Transcriptase,CRISPR/Cas9,Telomerase
In vivo	Intravitreal injection of Nucleoside Reverse Transcriptase Inhibitors (NRTIs) Lamivudine (3TC), Zidovudine (AZT), or Abacavir (ABC) significantly mitigates laser-induced choroidal neovascularization (CNV) in wild-type mice when compared to the PBS vehicle treatment. Additionally, these NRTIs lead to a notable reduction in the mean VEGF-A levels within the RPE/choroid area, which typically reach their highest concentration on day 3 post-laser injury, in treated eyes relative to control eyes in wild-type mice. This effect, however, does not extend to P2rx7 knockout (P2rx7-/-) mice[6].
Cell Research	Zidovudine is prepared in DMSO (10 mM) and stored, and then diluted with appropriate medium before use[1]. Assays are performed in all cell types in the presence of titrating concentrations of ARV. 5,000 SVG, 2,500 PFA, 200,000 PBMC, or 50,000 MDM cells/well are seeded into triplicate wells of 96-well plates. Twenty-four hours later, the culture medium is removed and replaced with medium containing the ARV or DMSO (0.5% vol/vol), and equivalent TCID50 infectious units of luciferase reporter virus are added to the cells. After a 16 h incubation at 37°C, the initial viral inoculum is removed and replaced with culture medium containing the same antiretroviral drug (ARV) or DMSO (0.5% vol/vol) concentrations. At 72 h post infection, the medium is aspirated, the cells are lysed and HIV-1 infection measured using the Luciferase Assay System. Luminescence is measured using a FLUOStar Optima microplate reader. Inhibition curves and the 50% (EC50) and 90% (EC90) effective concentrations are determined by nonlinear regression analysis, using GraphPad Prism software[1].

Solubility Information

Solubility	H2O: 13.4 mg/mL (50.14 mM),Sonication is recommended. DMSO: 242.5 mg/mL (907.42 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (7.48 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.742 mL	18.7098 mL	37.4195 mL
5 mM	0.7484 mL	3.742 mL	7.4839 mL
10 mM	0.3742 mL	1.871 mL	3.742 mL
50 mM	0.0748 mL	0.3742 mL	0.7484 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Chiang G, et al. Clin Ther,1996, 18(6), 1080-1092.
Tan S, Li J Q, Cheng H, et al. The anti-parasitic drug suramin potently inhibits formation of seminal amyloid fibrils and their interaction with HIV-1. Journal of Biological Chemistry. 2019: jbc. RA118. 006797
Tan S, Li J Q, Cheng H, et al. The anti-parasitic drug suramin potently inhibits formation of seminal amyloid fibrils and their interaction with HIV-1[J]. Journal of Biological Chemistry. 2019: jbc. RA118. 006797.
Tan S, Li W, Li Z, et al. A Novel CXCR4 Targeting Protein SDF-1/54 as an HIV-1 Entry Inhibitor. Viruses. 2019, 11(9): 874
Panther LA, et al. J Med Virol,1999, 58(2), 165-173.
Wang H, et al. Biochim Biophys Acta,1996, 1316(1), 51-59.
Wu Y, Chu L, Yang H, et al. Simultaneous Determination of 6 Antiretroviral Drugs in Human Hair Using an LC-ESI+-MS/MS Method: Application to Adherence Assessment. Therapeutic Drug Monitoring. 2021, 43(6): 756-765.
Fical L, Khalikova M, Kočová Vlčková H, et al. Determination of Antiviral Drugs and Their Metabolites Using Micro-Solid Phase Extraction and UHPLC-MS/MS in Reversed-Phase and Hydrophilic Interaction Chromatography Modes. Molecules. 2021, 26(8): 2123.
Chitnis S, et al. Life Sci,2002, 71(8), 967-978.
Gogu SR, et al. Biochem Pharmacol,1995, 50(3), 413-419.
Xu H, You H, Gong J, et al.Discovery of Zidovudine as a cardiomyocyte protectant for doxorubicin-induced toxicity through high-throughput phenotypic drug screening.Fundamental Research.2023
Hou P, et al. Genome editing of CXCR4 by CRISPR/cas9 confers cells resistant to HIV-1 infection. Sci Rep. 2015 Oct 20;5:15577.
Mizutani T, et al. Nucleoside Reverse Transcriptase Inhibitors Suppress Laser-Induced Choroidal Neovascularization in Mice. Invest Ophthalmol Vis Sci. 2015 Nov;56(12):7122-9.
Fical L, Khalikova M, Kočová Vlčková H, et al. Determination of Antiviral Drugs and Their Metabolites Using Micro-Solid Phase Extraction and UHPLC-MS/MS in Reversed-Phase and Hydrophilic Interaction Chromatography Modes [J]. Molecules. 2021, 26(8): 2123.
Tan S, Li W, Li Z, et al. A Novel CXCR4 Targeting Protein SDF-1/54 as an HIV-1 Entry Inhibitor. Viruses. 2019, 11(9): 874.

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