

Product Data Sheet

Product Name: Zidovudine

Cat. No.: GC10637

Chemical Properties

Cas. No. 30516-87-1

Chemical Name 1-[(2R,4S,5S)-4-azido-5-(hydroxymethyl)oxolan-2-yl]-5-methylpyrimidine-2,4-dione

SMILES CC1=CN(C(=O)NC1=O)C2CC(C(O2)CO)N=[N+]=[N-]

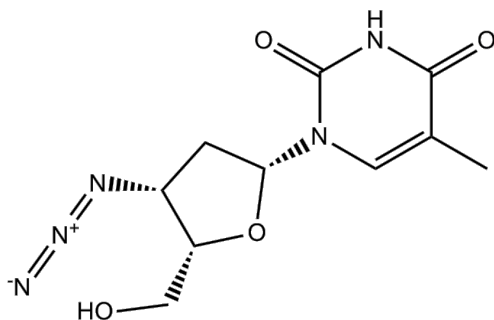
Formula $C_{10}H_{13}N_5O_4$ M.Wt 267.24

Solubility DMF: 30 mg/ml, DMSO: 30 mg/ml, Ethanol: 10 mg/ml, PBS (pH 7.2): 10 mg/ml
Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines HeLa cells

Caution: Product has not been fully validated for medical applications. For research use only.

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Preparation Method HeLa cells (1×10^6) were cultured in minimum essential medium Eagle supplemented with 10% fetal bovine serum. Zidovudine was dissolved in phosphate-buffered saline and the final concentration was determined by spectrophotometry at 266nm for Zidovudine. Single drug treatments were: 125, 250, and 500 μ M Zidovudine. After incubating the cells for 24h, the cells were harvested for cell cycle analysis by flow cytometry.

Reaction Conditions 125, 250, and 500 μ M; 24h

Applications Zidovudine treatment at 500 μ M significantly caused an arrest of HeLa cells in S phase.

Animal experiment []:

Animal models DMD^{mdx} mouse

Preparation Method The DMD^{mdx} mice were raised under standard conditions. Starting from 4 weeks of age, male DMD^{mdx} mice were intraperitoneally injected twice daily for 4 consecutive weeks with 50mg/kg dose of Zidovudine. Age-matched control mice received the same volume of phosphate-buffered saline (pH 7.4). After the treatment, the mice were euthanized by inhalation of carbon dioxide; anterior tibial muscle (TA), gastrocnemius muscle (GC), and cardiac muscle were collected for evaluation.

Dosage form 50mg/kg twice daily; for 4 weeks; i.p.

Applications Zidovudine treatment significantly resulted in a significant reduction in the mRNA levels of various inflammatory markers in the muscles of mice.

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References:

- [1] Olivero O A, Tejera A M, Fernandez J J, et al. Zidovudine induces S-phase arrest and cell cycle gene expression changes in human cells[J]. *Mutagenesis*, 2005, 20(2): 139-146.
- [2] Al-Khalidi R, Panicucci C, Cox P, et al. Zidovudine ameliorates pathology in the mouse model of Duchenne muscular dystrophy via P2RX7 purinoceptor antagonism[J]. *Acta Neuropathologica Communications*, 2018, 6(1): 27.

Background

Zidovudine is a synthetic nucleoside analog that blocks replication of the human immunodeficiency virus (HIV) by inhibiting reverse transcriptase^[1]. Zidovudine has been used as a model compound to develop a series of related derivatives for viral inhibition studies^[2].

In vitro, Zidovudine treatment for 48 hours significantly inhibited HIV-1 activity in primary human fetal astrocytes (PFA) with an EC₅₀ value of 1.311±0.130μM^[3]. Treatment with 500μM Zidovudine for 24h induced upregulation of cyclin D1 in HeLa cells and caused a cell arrest in S phase^[4]. Treatment of C2C12 cells with 30μM Zidovudine for 8 days resulted in the accumulation of dysfunctional mitochondria and increased reactive oxygen species (ROS) production^[5]. Treatment with 1000μM of Zidovudine for 5 weeks resulted in a decrease in checkpoint kinase 1 (Chk1) and checkpoint kinase 2 (Chk2) in NIH 3T3 cells and interference with cell cycle progression^[6].

In vivo, Zidovudine treatment (50mg/kg twice daily; i.p.) for 4 weeks significantly resulted in a significant reduction in the levels of various inflammatory markers in the muscles of DMD^{mdx} mouse model^[7]. Male OF1 mice were treated with drinking water containing Zidovudine (10mg/kg; p.o.) for 35 days, causing oxidative damage to mitochondrial DNA in the liver of mice^[8].

References:

- [1] Sperling R. Zidovudine[J]. *Infectious diseases in obstetrics and gynecology*, 1998, 6(5): 197.
- [2] Bianco M C A D, Inacio Leite D, Silva Castelo Branco F, et al. The use of Zidovudine pharmacophore in multi-target-directed ligands for AIDS therapy[J]. *Molecules*, 2022, 27(23): 8502.
- [3] Gray L R, Tachedjian G, Ellett A M, et al. The NRTIs lamivudine, stavudine and zidovudine have reduced HIV-1 inhibitory activity in astrocytes[J]. *PloS one*, 2013, 8(4): e62196.

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- [4] Olivero O A, Tejera A M, Fernandez J J, et al. Zidovudine induces S-phase arrest and cell cycle gene expression changes in human cells[J]. *Mutagenesis*, 2005, 20(2): 139-146.
- [5] Lin H, Stankov M V, Hegermann J, et al. Zidovudine-mediated autophagy inhibition enhances mitochondrial toxicity in muscle cells[J]. *Antimicrobial Agents and Chemotherapy*, 2019, 63(1): 10.1128/aac.01443-18.
- [6] Fang J L, McGarrity L J, Beland F A. Interference of cell cycle progression by zidovudine and lamivudine in NIH 3T3 cells[J]. *Mutagenesis*, 2009, 24(2): 133-141.
- [7] Al-Khalidi R, Panicucci C, Cox P, et al. Zidovudine ameliorates pathology in the mouse model of Duchenne muscular dystrophy via P2RX7 purinoceptor antagonism[J]. *Acta Neuropathologica Communications*, 2018, 6(1): 27.
- [8] de la Asunción J G, del Olmo M L, Sastre J, et al. Zidovudine (AZT) causes an oxidation of mitochondrial DNA in mouse liver[J]. *Hepatology*, 1999, 29(3): 985-987.

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