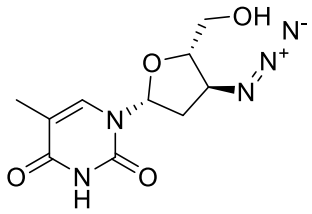
Product data sheet

MedKoo Cat#: 318994 Name: Zidovudine CAS#: 30516-87-1 Chemical Formula: $C_{10}H_{13}N_5O_4$ Exact Mass: 267.09675 Molecular Weight: 267.245	
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:

Zidovudine, also known as azidothymidine, is an antiretroviral medication used to prevent and treat HIV/AIDS. It is of the nucleoside analog reverse-transcriptase inhibitor (NRTI) class. Zidovudine inhibits the enzyme (reverse transcriptase) that HIV uses to synthesize DNA, thus preventing viral DNA from forming. Zidovudine was the first breakthrough in AIDS therapy, significantly reducing the replication of the virus and leading to clinical and immunologic improvements. It can also be used to prevent HIV transmission, such as from mother to child during the period of birth or after a needle stick injury. Used by itself in HIV-infected patients, AZT slows HIV replication in patients, but does not stop it entirely.

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	61.0	228.25

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.74 mL	18.71 mL	37.42 mL
5 mM	0.75 mL	3.74 mL	7.48 mL
10 mM	0.37 mL	1.87 mL	3.74 mL
50 mM	0.07 mL	0.37 mL	0.75 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. Carnicelli V, Di Giulio A, Bozzi A, Strom R, Oratore A. Zidovudine inhibits protein kinase C activity in human chronic myeloid (K562) cells. Basic Clin Pharmacol Toxicol. 2006 Oct;99(4):317-22. doi: 10.1111/j.1742-7843.2006.pto_486.x. PMID: 17040218.
2. Lin H, Stankov MV, Hegermann J, Budida R, Panayotova-Dimitrova D, Schmidt RE, Behrens GMN. Zidovudine-Mediated Autophagy Inhibition Enhances Mitochondrial Toxicity in Muscle Cells. Antimicrob Agents Chemother. 2018 Dec 21;63(1):e01443-18. doi: 10.1128/AAC.01443-18. PMID: 30373793; PMCID: PMC6325205.

In vivo study

1. Drzazga Z, Ciszek W, Binek M. Prenatal Zidovudine Treatment Modifies Early Development of Rat Osteoid - Confocal Microspectroscopy Analysis. J Fluoresc. 2019 Sep;29(5):1257-1263. doi: 10.1007/s10895-019-02429-6. Epub 2019 Oct 16. PMID: 31620936; PMCID: PMC6853851.
2. Al-Khalidi R, Panicucci C, Cox P, Chira N, Róg J, Young CNJ, McGeehan RE, Ambati K, Ambati J, Zabłocki K, Gazzerro E, Arkle S, Bruno C, Górecki DC. Zidovudine ameliorates pathology in the mouse model of Duchenne muscular dystrophy via P2RX7 purinoceptor antagonism. Acta Neuropathol Commun. 2018 Apr 11;6(1):27. doi: 10.1186/s40478-018-0530-4. PMID: 29642926; PMCID: PMC5896059.

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7. Bioactivity

Biological target:

Zidovudine is a nucleoside reverse transcriptase inhibitor (NRTI), widely used to treat HIV infection and increases CRISPR/Cas9-mediated editing frequency.

In vitro activity

The effect of defective autophagy on total cellular mitochondrial abundance (MitoTracker Red [MTR Red] staining) and ROS production [5-(and-6)-chloromethyl-2′,7′-dichlorodihydrofluorescein diacetate, acetyl ester (CM-H2DCFDA)] in C2C12 cells incubated in the presence or absence of different concentrations of AZT (Zidovudine) (6, 30, 180 μM) for up to 8 days or with 3MA (5 mM) or nocodazole-vinblastine (50 μM) for 24 h was analyzed. Consistent with autophagy inhibition, incubation with AZT produced profound myocyte mitochondrial accumulation (Fig. 3A). AZT treatment was associated with mitochondrial membrane hyperpolarization, as revealed by an AZT-mediated increase in the ratio of potential-dependent MitoTracker Deep Red (MTR Deep Red) and potential-independent MitoTracker Green (MTR Green) staining (34) (see Fig. S1A and B in the supplemental material) as well as by increased mitochondrial potential-specific MitoProbe TMRM staining. At the same time, AZT had no major effect on the enzymatic activity of mitochondrial respiratory chain complexes (complexes I to V) (Fig. S2). Collectively, these data suggest that AZT-mediated autophagy inhibition prevents the removal of dysfunctional mitochondria produced in relation to its mitochondrial toxicity.

Reference:Antimicrob Agents Chemother. 2019 Jan; 63(1): e01443-18. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6325205/

In vivo activity

AZT treatment resulted in significantly improved sarcolemma integrity: An analysis of membrane permeability using IgG influx into TA muscle revealed a significant reduction in IgG-positive fibers already after 2 weeks of treatment (IgG pixel intensity PBS-mdx = 14.7 AU, AZT-mdx = 5.14 AU) (Fig. 7a). After 4 weeks of AZT treatment there was a significant, 60% reduction in serum CK level (Fig.7b), indicative of less sarcolemma damage and therefore less leakage of this intracellular muscle enzyme (serum CK levels: PBS-mdx = 864.37 UI/l, AZT-mdx = 351.72 UI/l; p = 0.004). The serum levels of miR-206, dystroMir, in mdx mice have been found to be less affected by movement compared to CK and therefore this dystroMir has been proposed as a stable molecular marker of muscle damage. Here, the serum levels of Mir-206 were significantly decreased in the AZT-treated mdx mice after 2 weeks of treatment (Fig.7c). The revertant fiber reduction was not statistically significant but no increase indicated that AZT treatment is not accelerating muscle damage (Additional file 5: Figure S4).

Reference: Acta Neuropathol Commun. 2018; 6: 27. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5896059/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.