

Product Data Sheet

Product Name: 3-Deazaadenosine

Cat. No.: GC14713

Chemical Properties

Cas. No. 6736-58-9

Chemical Name (2R,3R,4S,5R)-2-(4-amino-1H-imidazo[4,5-c]pyridin-1-yl)-5-(hydroxymethyl)tetrahydrofuran-3,4-diol

SMILES NC1=C2C(N(C=N2)[C@H]3[C@@H]([C@@H]([C@H](O3)CO)O)O)=CC=N1

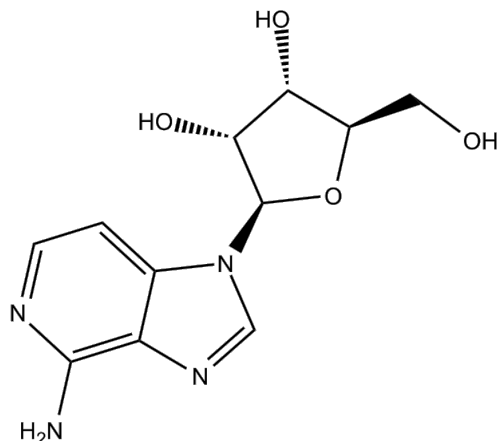
Formula $C_{11}H_{14}N_4O_4$ M.Wt 266.25

Solubility $\geq 26.6\text{mg/mL}$ in DMSO 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

HL-60 cells (human promyelocytic leukemia cell line)

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Preparation Method	HL-60 cells were grown in RPMI 1640 with 10% heat-inactivated horse serum at 37°C, 5% CO ₂ . HL-60 cells were treated with 3-Deazaadenosine at 50µM or 100µM for 4 hours, followed by analysis of cell morphology (light microscopy), DNA fragmentation (agarose gel electrophoresis) and cellular DNA/protein content (flow cytometry).
Reaction Conditions	50µM or 100µM; 4h
Applications	3-Deazaadenosine induced condensation and fragmentation of the nucleus, cytoplasmic blebs, formation of apoptotic bodies and endonucleolytic degradation of DNA into approximately 200bp multimers in HL-60 cells. 3-Deazaadenosine reduced early S-phase cells and produced particles with lower DNA and protein content than the G1 population.
Animal experiment [2]:	
Animal models	cystathionine β-synthase heterozygote ^(-/+) knockout (CBSKO) mice and wild-type (WT) mice
Preparation Method	CBSKO mice and WT mice were fed a rodent laboratory chow supplemented with 3-Deazaadenosine (0.04g/kg) for 32 days; mean aortic blood pressure and heart rate were monitored by radiotelemetry, aortic function and morphology were analyzed by M-mode and Doppler echocardiography, and thoracic aortas were collected for Masson trichrome staining, in situ zymography with DQ gelatin, RT-PCR and Western blot analyses.
Dosage form	0.04g/kg; p.o.; 32 days

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Applications	3-Deazaadenosine lowered plasma homocysteine levels, reduced mean arterial blood pressure and heart rate, decreased aortic wall thickness and ECM accumulation, improved aortic lumen-to-wall thickness ratio, reduced aortic resistance, attenuated in situ MMP gelatinolytic activity and MMP-2/MMP-9 mRNA expression in aorta, and diminished connexin 43 protein expression in CBSKO mice.
References: [1] Endresen PC, Eide TJ, Aarbakke J. Cell death initiated by 3-deazaadenosine in HL-60 cells is apoptosis and is partially inhibited by homocysteine. <i>Biochem Pharmacol.</i> 1993 Dec 1;46(11):1893-1901. [2] Ovechkin AV, Tyagi N, Sen U, et al. 3-Deazaadenosine mitigates arterial remodeling and hypertension in hyperhomocysteinemic mice. <i>Am J Physiol Lung Cell Mol Physiol.</i> 2006 Nov;291(5):L905-L911.	

Background

3-Deazaadenosine is an orally active S-adenosylhomocysteine (SAH) hydrolase inhibitor ($K_i=3.9\mu\text{M}$). 3-Deazaadenosine inhibits methylation reactions by inducing intracellular SAH accumulation. 3-Deazaadenosine has antiviral, anti-inflammatory and immunomodulatory activities. 3-Deazaadenosine can be used in research on viral infections such as HIV-1 and vascular restenosis and atherosclerosis^[1-4].

In vitro, treatment of BEAS-2B cells with $50\mu\text{M}$ 3-Deazaadenosine for 24 hours upregulated ZNRD1-AS1 expression in the cells^[5]. Treatment of RAW264 cells with 3-Deazaadenosine for 40 minutes inhibited chemotaxis, inhibited the synthesis of specific proteins, and reduced the incorporation of methionine methyl groups into phosphatidylcholine^[6]. Treatment of HL-60 cells with $50\text{-}100\mu\text{M}$ 3-Deazaadenosine for 4 hours induced apoptosis, promoted nuclear pyknosis and fragmentation, formation of apoptotic bodies and DNA fragmentation, and reduced early S-phase cells^[7].

In vivo, feeding cystathionine β -synthase knockout mice with 0.04g/kg 3-Deazaadenosine for 32 days

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reduced plasma homocysteine levels, attenuated aortic wall thickening and extracellular matrix accumulation, and decreased aortic MMP activity^[8]. Daily oral administration of 5mg/kg 3-Deazaadenosine to apoE^{-/-}/LDL^{-/-} double knockout mice from 6 weeks of age until 16, 18, and 20 weeks reduced descending aortic atherosclerotic lesion volume^[9]. Intraperitoneal injection of 10mg/kg 3-Deazaadenosine into aged C57BL/6 mice 3 days and 1 day before partial hepatectomy reduced the number of γH2AX-positive hepatocytes, increased the number of Ki67-positive hepatocytes, and alleviated liver pathological changes including diffuse glycogen swelling and oval cell hyperplasia^[10].

References:

- [1] Guerrero A, Gil J. 3-Deazaadenosine keeps senescence at bay. *Aging* (Albany NY). 2023 Mar 24;15(7):2369-2370.
- [2] Stopford CR, Wolberg G, Prus KL, et al. 3-Deazaadenosine-induced disorganization of macrophage microfilaments. *Proc Natl Acad Sci U S A*. 1985 Jun;82(12):4060-4.
- [3] Medzihradsky JL. Synergism of 3-deazaadenosine with some Fc receptor ligands in the inhibition of antibody-dependent phagocytosis. *Int J Immunopharmacol*. 1987;9(2):227-36.
- [4] Mayers DL, Mikovits JA, Joshi B, et al. Anti-human immunodeficiency virus 1 (HIV-1) activities of 3-deazaadenosine analogs: increased potency against 3'-azido-3'-deoxythymidine-resistant HIV-1 strains. *Proc Natl Acad Sci U S A*. 1995 Jan 3;92(1):215-9.
- [5] Wang J, Tan L, Yu X, et al. lncRNA ZNRD1-AS1 promotes malignant lung cell proliferation, migration, and angiogenesis via the miR-942/TNS1 axis and is positively regulated by the m6A reader YTHDC2. *Mol Cancer*. 2022 Dec 30;21(1):229.
- [6] Aksamit RR, Backlund PS Jr, Cantoni GL. Chemotaxis and the synthesis of specific proteins are inhibited by 3-deazaadenosine and other adenosine analogs in a mouse macrophage cell line. *J Biol Chem*. 1983 Jan 10;258(1):20-23.
- [7] Endresen PC, Eide TJ, Aarbakke J, et al. Cell death initiated by 3-deazaadenosine in HL-60 cells is apoptosis and is partially inhibited by homocysteine. *Biochem Pharmacol*. 1993 Dec 1;46(11):1893-1901.

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[9] Langheinrich A, Sedding D, Kampschulte M, et al. 3-Deazaadenosine inhibits vasa vasorum neovascularization in aortas of ApoE-/-/LDL-/- double knockout mice. *Atherosclerosis*. 2009 Jan;202(1):103-110.

[10] Guerrero A, Innes AJ, Roux PF, et al. 3-deazaadenosine(3DA) alleviates senescence to promote cellular fitness and cell therapy efficiency in mice. *Nat Aging*. 2022 Sep;2(9):851-866.

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