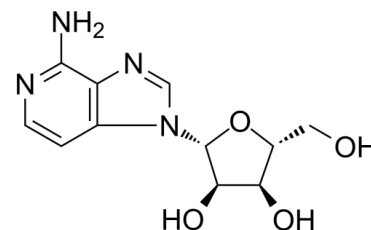


## Data Sheet

<b>Product Name:</b>	3-Deazaadenosine
<b>Cat. No.:</b>	CS-W014048
<b>CAS No.:</b>	6736-58-9
<b>Molecular Formula:</b>	C <sub>11</sub> H <sub>14</sub> N <sub>4</sub> O <sub>4</sub>
<b>Molecular Weight:</b>	266.25
<b>Target:</b>	HIV
<b>Pathway:</b>	Anti-infection
<b>Solubility:</b>	DMSO : 130 mg/mL (488.26 mM; Need ultrasonic)



### BIOLOGICAL ACTIVITY:

3-Deazaadenosine is an inhibitor of **S-adenosylhomocysteine hydrolase**, with a  $K_i$  of 3.9  $\mu\text{M}$ ; 3-Deazaadenosine has anti-inflammatory, anti-proliferative and anti-HIV activity. IC<sub>50</sub> & Target: IC<sub>50</sub>: 0.15 (HIV-1, A012 isolate), 0.20  $\mu\text{M}$  (HIV-1, A018 isolate)<sup>[1]</sup>  $K_i$ : 3.9  $\mu\text{M}$  (S-adenosylhomocysteine hydrolase)<sup>[1]</sup> **In Vitro**: 3-Deazaadenosine is an inhibitor of S-adenosylhomocysteine hydrolase, with a  $K_i$  of 3.9  $\mu\text{M}$ . 3-Deazaadenosine shows anti-HIV effect, and inhibits p24 antigen in peripheral blood mononuclear (PBMCs) cells infected with HIV-1 isolates (A012 and A018) with IC<sub>50</sub>s of 0.15 and 0.20  $\mu\text{M}$ , respectively<sup>[1]</sup>. 3-Deazaadenosine (1-100  $\mu\text{M}$ ) inhibits LPS-induced expression of TNF- $\alpha$  mRNA, increases DNA binding activity of NF- $\kappa\text{B}$ , and causes proteolytic degradation of I $\kappa\text{B}$   $\alpha$ , but Not I $\kappa\text{B}$   $\beta$  in RAW 264.7 cells. 3-Deazaadenosine (100  $\mu\text{M}$ ) enhances nuclear translocation of NF- $\kappa\text{B}$ , but blocks LPS-induced NF- $\kappa\text{B}$  transcriptional activity, and such inhibition is augmented by the addition of homocysteine<sup>[2]</sup>. 3-Deazaadenosine (50, 100  $\mu\text{M}$ ) dose-dependently inhibits the phosphorylation of Raf and ERK, protein-dependent kinase 1, protein kinase B (Akt), and forkhead transcription factor FoxO1a. 3-Deazaadenosine (50  $\mu\text{M}$ ) suppresses vascular smooth muscle cell (VSMC) proliferation via interfering with Ras signaling<sup>[3]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** <sup>[1]</sup>The HIV-1 strains **A012** and **A018** are used in the assay. Inhibition of p24 antigen is measured. Briefly, PHA-stimulated **peripheral blood mononuclear (PBMCs)** are incubated with either HIV-1 strain for 1 h at 37°C at 200-fold the 50% tissue culture infectious dose (TCID<sub>50</sub>) of the virus stock per **2 × 10<sup>5</sup> PBMC cells**. The TCID<sub>50</sub> is defined as the amount of virus stock at which 50% of the inoculated wells are positive. Cells are then grown in microtiter plates with different drug concentrations at 2 × 10<sup>5</sup> cells per well. On day 4, cells are resuspended and split 1:3 with fresh media and **3-Deazaadenosine**. Supernatant p24 antigen is determined on day 7 by ELISA<sup>[1]</sup>.

### References:

[1]. Gordon RK, et al. Anti-HIV-1 activity of 3-deaza-adenosine analogs. Inhibition of S-adenosylhomocysteine hydrolase and nucleotide congeners. Eur J Biochem. 2003 Sep;270(17):3507-17.

[2]. Jeong SY, et al. 3-deazaadenosine, a S-adenosylhomocysteine hydrolase inhibitor, has dual effects on NF- $\kappa\text{B}$  regulation. Inhibition of NF- $\kappa\text{B}$  transcriptional activity and promotion of I $\kappa\text{B}$   $\alpha$  degradation. J Biol Chem. 1999 Jul 2;274(27):18981-8.

[3]. Sedding DG, et al. 3-Deazaadenosine prevents smooth muscle cell proliferation and neointima formation by interfering with Ras signaling. Circ Res. 2009 May 22;104(10):1192-200.

**CAIndexNames:**

1H-Imidazo[4,5-c]pyridin-4-amine, 1-β-D-ribofuranosyl-

**SMILES:**

O[C@H]1[C@@H](O[C@H](CO)[C@H]1O)N2C3=C(C(N)=NC=C3)N=C2

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

Tel: 732-484-9848

Fax: 888-484-5008

E-mail: [sales@ChemScene.com](mailto:sales@ChemScene.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA