

Product Data Sheet

Product Name: Acyclovir
Cat. No.: GC17186

Chemical Properties

Cas. No. 59277-89-3

Chemical Name 2-amino-9-(2-hydroxyethoxymethyl)-3H-purin-6-one

SMILES C1=NC2=C(N1COCCO)NC(=NC2=O)N

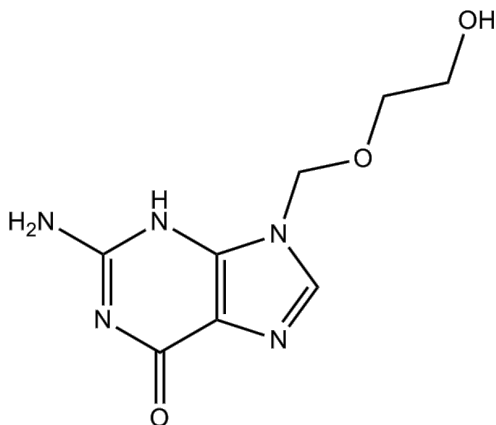
Formula $C_8H_{11}N_5O_3$ M.Wt 225.21

Solubility $\geq 11.4\text{mg/mL}$ in DMSO Storage Store at RT

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 Vero cells

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

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Preparation Method Vero cells were infected with HSV-1 for 1h and then treated with NPRE (0.1, 0.2, 0.4, 0.6, 0.8mg/mL). The DMSO was used in the control samples and Acyclovir (20μM) was used as positive control.

Reaction Conditions 20μM; 3d.

Applications Treatment with 20μM Acyclovir showed 100% plaque inhibition.

Animal experiment [2]:

Model Nephrotoxicity Model

Preparation Method ICR mice were randomly divided into three groups to receive saline, 150mg/kg Acyclovir, or 600mg/kg Acyclovir once-daily for 9 days. Mice were kept at room temperature and 70±10% humidity, with a light-dark cycle. Food and water were available ad libitum. Acyclovir was administered intraperitoneally (ip) diluted in 20mL/kg of 0.9% sodium chloride injection solution. The saline group received an equal volume of the 0.9% sodium chloride solution.

Dosage form 150,600mg/kg; ip; 9d

Applications After administration of Acyclovir, levels of serum creatinine and urea nitrogen increased, mouse kidneys exhibited histopathological changes and reduced expression levels of vascular endothelial growth factor (VEGF) and its receptor VEGFR2.

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References:

- [1]. Musarra-Pizzo M, Pennisi R, Ben-Amor I, et al. In vitro anti-HSV-1 activity of polyphenol-rich extracts and pure polyphenol compounds derived from pistachios kernels (*Pistacia vera* L.)[J]. *Plants*, 2020, 9(2): 267.
- [2]. Lu H, Han Y J, Xu J D, et al. Proteomic characterization of acyclovir-induced nephrotoxicity in a mouse model[J]. *PLoS One*, 2014, 9(7): e103185.

Background

Acyclovir is a potent oral antiviral drug. Acyclovir has anti-herpetic activity with IC₅₀ values of 0.85 μ M and 0.86 μ M for HSV-1 and HSV-2, respectively. Acyclovir induces cell cycle disruption and apoptosis^{[1] [2]}.

In the plaque reduction assay (PRA) of Vero cells, Acyclovir (20 μ M; 3d) treatment showed 100% plaque inhibition^[1]. Acyclovir (10-100 μ M; 24-72h) blocks DNA synthesis, thereby arresting the cell cycle at G2/M and S phases and increasing the G1 hypodiploid peak in a dose-dependent manner. Acyclovir (10-100 μ M; 24-72h) induces apoptosis through activation of caspase-3 and the presence of nuclear DNA fragmentation^[2].

Treatment of infected mice with Acyclovir (20mg/kg; po; three times daily; 10d) inhibited the development of skin lesions and led to a dissociation between the DTH response and antibody production^[3]. In the Acyclovir-induced nephrotoxicity model, after mice were given Acyclovir (150, 600 mg/kg; ip; 9d), serum creatinine and urea nitrogen levels increased, histopathological changes occurred in the kidneys of mice, and the expression levels of vascular endothelial growth factor (VEGF) and its receptor VEGFR2 were reduced^[4].

References:

- [1]. Musarra-Pizzo M, Pennisi R, Ben-Amor I, et al. In vitro anti-HSV-1 activity of polyphenol-rich extracts and pure polyphenol compounds derived from pistachios kernels (*Pistacia vera* L.)[J]. *Plants*, 2020, 9(2): 267.
- [2]. Suzuki M, Okuda T, Shiraki K. Synergistic antiviral activity of Acyclovir and vidarabine against herpes simplex virus types 1 and 2 and varicella-zoster virus[J]. *Antiviral research*, 2006, 72(2): 157-161.

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[3]. Benedetti S, Catalani S, Palma F, et al. Acyclovir induces cell cycle perturbation and apoptosis in Jurkat leukemia cells, and enhances chemotherapeutic drug cytotoxicity[J]. Life sciences, 2018, 215: 80-85.

[4]. Lu H, Han Y J, Xu J D, et al. Proteomic characterization of acyclovir-induced nephrotoxicity in a mouse model[J]. PLoS One, 2014, 9(7): e103185.

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