Acyclovir

Cat. No.: HY-17422

CAS No.: 59277-89-3

Molecular Formula: $C_8H_{11}N_5O_3$

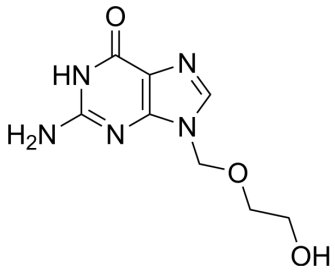
Molecular Weight: 225.2

Target: HSV; Antibiotic; Bacterial; Apoptosis

Pathway: Anti-infection; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (222.02 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.4405 mL	22.2025 mL	44.4050 mL
	5 mM	0.8881 mL	4.4405 mL	8.8810 mL
	10 mM	0.4440 mL	2.2202 mL	4.4405 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 0.5% CMC/saline water
   Solubility: 20 mg/mL (88.81 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 20 mg/mL (88.81 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (11.10 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Acyclovir (Aciclovir) is a potent, orally active antiviral agent. Acyclovir has antiherpetic activity with IC_{50} values of 0.85 μM and 0.86 μM for HSV-1 and HSV-2, respectively. Acyclovir induces cell cycle perturbation and apoptosis. Acyclovir prevents bacterial infections during induction therapy for acute leukaemia[1][2][3][4].

IC_{50} & Target	HSV-1 0.85 μM (IC_{50})	HSV-2 0.86 μM (IC_{50})

In Vitro

Acyclovir (Aciclovir, 3-100 μM; 24-72 hours; Jurkat, U937, and K562 leukemia cells) reduces cell viability in a dose- and time-dependent[1].

?Acyclovir (Aciclovir, 10-100 μM; 24-72 hours; Jurkat cells) blocks DNA synthesis, thereby arresting the cell cycle in G2/M and S phases and increasing the sub-G1 hypodiploid peak in a dose-dependent manner[1].

?Acyclovir (Aciclovir, 10-100 μM; 24-72 hours; Jurkat cells) induces apoptosis through activates caspase-3 and presences nuclear DNA fragmentation[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Jurkat, U937 and K562 leukemia cells
Concentration:	3, 10, 30 and 100 μM
Incubation Time:	24, 48 and 72 hours
Result:	Showed a dose- and time-dependent reduction of cell viability.

Apoptosis Analysis[3]

Cell Line:	Jurkat cells
Concentration:	10 and 100 μM
Incubation Time:	24, 48 and 72 hours
Result:	Increased of caspase-3 activity and cleavaged the internucleosomal DNA.

Cell Cycle Analysis[1]

Cell Line:	Jurkat cells
Concentration:	10 and 100 μM
Incubation Time:	24, 48 and 72 hours
Result:	Revealed a dose-dependent accumulation of cells in S phase after 24 and 48 h. Showed a dose-dependent increase of the sub-G1 hypodiploid peak after 72 h.

In Vivo

Acyclovir (20 mg/kg; p.o.; three times daily; for 10 days; BALB/c mice) treatment in infected mice suppresses the development of skin lesions and results in a dissociation between DTH response and antibody production[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Specific-pathogen-free BALB/c mice (7-week-old) infected with HSV-1[1]
Dosage:	20 mg/kg
Administration:	Oral administration; three times daily; for 10 days
Result:	Suppressed the development of skin lesions and resulted in a dissociation between DTH response and antibody production.

CUSTOMER VALIDATION

- J Med Virol. 2022 Oct 17.
- Antiviral Res. 2023 Dec 23, 105787.
- Biomed Pharmacother. 2023 Mar 27;162:114595.
- Eur J Med Chem. 2023 Feb 4;250:115184.
- Front Microbiol. 2021 Jun 18;12:691008.

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REFERENCES

[1]. Benedetti S, et, al. Acyclovir induces cell cycle perturbation and apoptosis in Jurkat leukemia cells, and enhances chemotherapeutic drug cytotoxicity. Life Sci. 2018 Dec 15;215:80-85.

[2]. Suzuki M, et, al. Synergistic antiviral activity of acyclovir and vidarabine against herpes simplex virus types 1 and 2 and varicella-zoster virus. Antiviral Res. 2006 Nov;72(2):157-61.

[3]. Li Z, et, al. Acyclovir treatment of skin lesions results in immune deviation in mice infected cutaneously with herpes simplex virus. Antivir Chem Chemother. 1999 Sep;10(5):251-7.

[4]. Lönnqvist B, et, al. Oral acyclovir as prophylaxis for bacterial infections during induction therapy for acute leukaemia in adults. The Leukemia Group of Middle Sweden. Support Care Cancer. 1993 May;1(3):139-44.

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