Acyclovir sodium

Cat. No.: HY-17422A

CAS No.: 69657-51-8

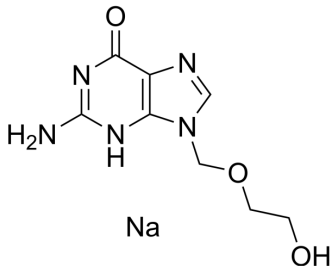
Molecular Formula: $C_8H_{11}N_5NaO_3$

Molecular Weight: 248.19

Target: Antibiotic; HSV; Apoptosis; Bacterial

Pathway: Anti-infection; Apoptosis

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

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

In Vitro

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

Acyclovir (Aciclovir) sodium (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) sodium (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[1]

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 (Aciclovir) sodium (20 mg/kg; p.o.; three times daily; for 10 d; BALB/c mice) suppresses the development of skin lesions and results in a dissociation between DTH response and antibody production[3]

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Animal Model:	Specific-pathogen-free BALB/c mice (7-week-old) infected with HSV-1[3]
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.
- Biomed Pharmacother. 2023 Mar 27;162:114595.
- Eur J Med Chem. 2023 Feb 4;250:115184.
- Front Microbiol. 2021 Jun 18;12:691008.
- Drug Des Devel Ther. 2022 Dec 20;16:4311-4323.

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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.

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

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