Cladribine

Cat. No.: HY-13599

CAS No.: 4291-63-8

Molecular Formula: $C_{10}H_{12}ClN_5O_3$

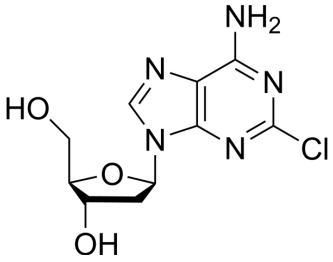
Molecular Weight: 285.69

Target: Adenosine Deaminase; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: 4°C, protect from light

* In solvent : -80°C, 1 years; -20°C, 6 months (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 30 mg/mL (105.01 mM)

H_2O : 10 mg/mL (35.00 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	3.5003 mL	17.5015 mL	35.0030 mL
5 mM	0.7001 mL	3.5003 mL	7.0006 mL
10 mM	0.3500 mL	1.7501 mL	3.5003 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 25 mg/mL (87.51 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Cladribine (2-Chloro-2′-deoxyadenosine), a purine nucleoside analog, is an orally active adenosine deaminase inhibitor. Cladribine functions as an inhibitor of DNA synthesis to block the repair of the damaged DNA. Cladribine can inhibit DNA methylation. Cladribine has anti-lymphoma activity. Cladribine can be used for the research of several hematologic malignancies and multiple sclerosis[1][2].

IC_{50} & Target

Adenosine deaminase[2]

In Vitro

Cladribine (0.25-4 μM; 24-48 hours) inhibits human DLBCL cells proliferation[1].

?Cladribine (1-4 μM; 24 hours) induces G1 phase arrest via decreasing the expressions of Cyclin D1 and Cyclin E, and increasing the expressions of p21 and p27 in DLBCL cells[1].

?Cladribine (1-4 μM; 24 hours) induces apoptosis and activates extrinsic and intrinsic signaling pathways in human DLBCL cells[1].

?Cladribine (1-4 μM; 24 hours) activates endoplasmic reticulum stress[1].
?Cladribine inhibits cell proliferation of multiple myeloma (MM) cells in a dose-dependent manner; with IC_{50}s of approximately 2.43 μM, 0.75 μM and 0.18 μM for U266, RPMI8226 and MM1.S cells, respectively[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	The human DLBCL cell lines (U2932, OCI-LY10, SUDHL2, WSU-DLCL2, DB)
Concentration:	0 μM, 0.25 μM, 0.5 μM, 1 μM, 2 μM, 4 μM
Incubation Time:	24 hours, 48 hours
Result:	Exhibited notable suppression of cell proliferation in five DLBCL cells.

Western Blot Analysis[1]

Cell Line:	U2932 cells, WSU-DLCL2 cells
Concentration:	0 μM, 1 μM, 2 μM, 4 μM
Incubation Time:	24 hours
Result:	Decreased the expressions of Cyclin D1 and Cyclin E, and increased the expressions of p21 and p27.

Apoptosis Analysis[1]

Cell Line:	U2932 cells, WSU-DLCL2 cells
Concentration:	0 μM, 1 μM, 2 μM, 4 μM
Incubation Time:	24 hours
Result:	Induced apoptosis and activates exogenous and endogenous apoptotic signaling pathways.

Cell Cycle Analysis[1]

Cell Line:	U2932 cells, WSU-DLCL2 cells
Concentration:	0 μM, 1 μM, 2 μM, 4 μM
Incubation Time:	24 hours
Result:	Caused G1 phase arrest.

RT-PCR[1]

Cell Line:	U2932 cells, WSU-DLCL2 cells
Concentration:	0 μM, 1 μM, 2 μM, 4 μM
Incubation Time:	24 hours
Result:	Activated ER stress.

In Vivo

Cladribine (10 μg/kg; i.p.; 3 times/week; for 2 weeks) may have benefits in the treatment of ischemia/reperfusion injury to the biochemical and histopathologic parameters[3].
?Cladribine (0.5 mg/kg; i.p.; daily; for 60 days) increases amyloid beta peptide generation and plaque burden in APdE9 mice

[4].

?Cladribine exhibits C_{max} (rat 4.9 ng/mL) following intra-arterial injection[5].

?Cladribine exhibits C_{max} (rat 1.1 ng/mL) following subcutaneous injection[5].

?Cladribine exhibits elimination half-lives (rat 3.5 h) and plasma clearance (rat 2.8 L/h/kg) following intra-arterial injection[5].

?Cladribine exhibits elimination half-lives (rat 4.5 h) and plasma clearance (rat 2.3 L/h/kg) following subcutaneous injection [5].

?Cladribine administration with s.c. injection may produce more favourable pharmacokinetic profiles than i.a. injection following a single dose[5].

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

Animal Model:	Male Sprague-Dawley rats, ischemic injury model[3]
Dosage:	10 μg/kg
Administration:	Intraperitoneal injection, 3 times/week, for 2 weeks
Result:	Might increase expression of Sphk1 and consecutively SphK1 suppressed HIF-1α.

Animal Model:	Male Sprague Dawley rats (350-450 g)[5]
Dosage:	2 mg/kg for s.c., 1 mg/kg for i.a. (Pharmacokinetic Analysis)
Administration:	Subcutaneous injection, intra-arterial
Result:	C_{max} (4.9 ng/mL i.a.; 1.1 ng/mL s.c.), $T_{1/2}$ β (3.5 h i.a.; 4.5 s.c.).

CUSTOMER VALIDATION

- Molecules. 2018 Mar 23;23(4). pii: E736.
- Chem Pharm Bull (Tokyo). 2017 Aug 1;65(8):768-775.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Linyan Xu, et al. Cladribine Induces ATF4 Mediated Apoptosis and Synergizes with SAHA in Diffuse Large B-Cell Lymphoma Cells. Int J Med Sci. 2020; 17(10): 1375–1384.

[2]. Jian Ma, et al. Therapeutic potential of cladribine in combination with STAT3 inhibitor against multiple myeloma. BMC Cancer. 2011 Jun 16;11:255.

[3]. Young Seop Chang, et al. MP28-10 COMBINED EFFECTS OF MELATONIN AND CLADRIBINE ON APOPTOSIS IN ISCHEMIA/REPERFUSION INJURY IN RAT BLADDER. THE JOURNAL OF UROLOGY. April 2016. 195 (4S): e375.

[4]. Crystal D Hayes, et al. Chronic cladribine administration increases amyloid beta peptide generation and plaque burden in mice. PLoS One. 2012;7(10):e45841.

[5]. Yeung, P. K. F., et al. Pharmacokinetics of Cladribine in a Rat Model Following Subcutaneous and Intra-arterial Injections. Drug Metabol Drug Interact. 2008;23(3-4):291-8.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA