Cytarabine hydrochloride

Cat. No.: HY-13605A

CAS No.: 69-74-9

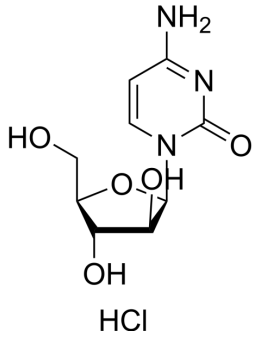
Molecular Formula: $C_9H_{14}ClN_3O_5$

Molecular Weight: 279.68

Target: DNA/RNA Synthesis; Nucleoside Antimetabolite/Analog; Autophagy; HSV

Pathway: Cell Cycle/DNA Damage; Autophagy; Anti-infection

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (178.78 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5755 mL	17.8776 mL	35.7551 mL
	5 mM	0.7151 mL	3.5755 mL	7.1510 mL
	10 mM	0.3576 mL	1.7878 mL	3.5755 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (8.94 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.94 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cytarabine hydrochloride, a nucleoside analog, causes S phase cell cycle arrest and inhibits DNA polymerase. Cytarabine inhibits DNA synthesis with an IC_{50} of 16 nM. Cytarabine hydrochloride has antiviral effects against HSV.

IC_{50} & Target

IC50: 16 nM (DNA synthesis)

In Vitro

Cytarabine is phosphorylated into a triphosphate form (Ara-CTP) involving deoxycytidine kinase (dCK), which competes with dCTP for incorporation into DNA, and then blocks DNA synthesis by inhibiting the function of DNA and RNA polymerases. Cytarabine displays a higher growth inhibitory activity towards wild-type CCRF-CEM cells compared to other acute myelogenous leukemia (AML) cells with IC_{50} of 16 nM[1]. Cytarabine apparently induces apoptosis of rat sympathetic

	neurons at 10 μM, of which 100 μM shows the highest toxicity and kills over 80% of the neurons by 84 hours, involving the release of mitochondrial cytochrome-c and the activation of caspase-3, and the toxicity can be attenuated by p53 knockdown and delayed by bax deletion[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Cytarabine (250 mg/kg) also causes placental growth retardation and increases placental trophoblastic cells apoptosis in the placental labyrinth zone of the pregnant Slc:Wistar rats, which increases from 3 hour after the treatment and peaks at 6 hour before returning to control levels at 48 hour, with remarkably enhanced p53 protein, p53 trancriptional target genes such as p21, cyclinG1 and fas and caspase-3 activity[3]. Cytarabine is highly effective against acute leukaemias, which causes the chCytarabineteristic G1/S blockage and synchronization, and increases the survival time for leukaemic Brown Norway rats in a weak dose-related fashion indicating that the use of higher dosages of Cytarabine does not contribute to its antileukaemic effectiveness in man[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3]	Pregnant rats are injected intraperitoneally (i.p.) with 250 mg/kg of Cytarabine on Day 13 of gestation (GD13). Under the conditions of this experiment, congenital anomalies and growth retardation are detected at a high rate in perinatal fetuses, although the incidence of fetal death is not markedly increased. At 1, 3, 6, 9, 12, 24, and 48 h after the treatment, six dams each are killed by heart puncture under ether anesthesia, and the placentas are collected. As controls, six pregnant rats are injected i.p. with an equivalent volume of PBS on GD13 and killed at the same time point as Cytarabine-treated groups. Of the six dams obtained at each time point, three are used for histopathological analyses and three for reverse transcription-polymerase chain reaction (RT-PCR) analysis. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell. 2018 Sep 20;175(1):171-185.e25.
- Cell Discov. 2023 Mar 7;9(1):26.
- Cell Death Differ. 2022 Mar 28.
- ACS Appl Mater Interfaces. 2022 Dec 20.
- Leukemia. 2023 Mar 28.

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REFERENCES

[1]. Tobias, S.C. and R.F. Borch, Synthesis and biological evaluation of a cytarabine phosphoramidate prodrug. Mol Pharm, 2004. 1(2): p. 112-6.

[2]. Besirli, C.G., et al. Cytosine arabinoside rapidly activates Bax-dependent apoptosis and a delayed Bax-independent death pathway in sympathetic neurons. Cell Death Differ, 2003. 10(9): p. 1045-58.

[3]. Yamauchi, H., et al., Involvement of p53 in 1-beta-D-arabinofuranosylcytosine-induced trophoblastic cell apoptosis and impaired proliferation in rat placenta. Biol Reprod, 2004. 70(6): p. 1762-7.

[4]. Richel, D.J., et al., Comparison of the antileukaemic activity of 5 aza-2-deoxycytidine and arabinofuranosyl-cytosine in rats with myelocytic leukaemia. Br J Cancer, 1988. 58(6): p. 730-3.

[5]. Shepshelovich D, et al. Pharmacodynamics of cytarabine induced leucopenia: a retrospective cohort study. Br J Clin Pharmacol. 2015 Apr;79(4):685-91.

[6]. Renis HE. Antiviral activity of cytarabine in herpesvirus-infected rats. Antimicrob Agents Chemother. 1973 Oct;4(4):439-44.

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