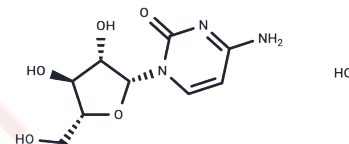


## Cytarabine hydrochloride

### Chemical Properties

CAS No. : 69-74-9  
 Formula: C<sub>9</sub>H<sub>14</sub>ClN<sub>3</sub>O<sub>5</sub>  
 Molecular Weight: 279.68  
 Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year  
*Actual storage temperature shall be subject to the COA.*



### Biological Description

Description	Cytarabine hydrochloride (Ara-C hydrochloride) is a nucleoside analog that causes S phase cell cycle arrest and inhibits DNA polymerase. Cytarabine inhibits DNA synthesis with an IC <sub>50</sub> of 16 nM. Cytarabine hydrochloride has antiviral effects against HSV.
Targets(IC <sub>50</sub> )	Nucleoside Antimetabolite/Analog, Autophagy, HSV, DNA/RNA Synthesis
In vitro	Cytarabine is converted into its active form, Ara-CTP (tri-phosphate), by the enzyme deoxycytidine kinase (dCK). It then competes with the natural nucleotide dCTP for incorporation into DNA, ultimately inhibiting DNA synthesis by disrupting DNA and RNA polymerases' functions. This compound exhibits potent growth inhibitory effects on wild-type CCRF-CEM cells, with an IC <sub>50</sub> of 16 nM, showcasing higher efficacy compared to other acute myelogenous leukemia (AML) cells. Moreover, Cytarabine is capable of inducing apoptosis in rat sympathetic neurons at a concentration of 10 μM, with the highest observed toxicity at 100 μM, resulting in over 80% cell death within 84 hours. This process involves the release of mitochondrial cytochrome-c and activation of caspase-3, with noted attenuation of toxicity via p53 knockdown and a delay through bax deletion.
In vivo	Cytarabine (250 mg/kg) induces placental growth retardation and elevates apoptosis of trophoblastic cells in the placental labyrinth zone in pregnant Slc:Wistar rats. This effect begins 3 hours post-treatment, peaks at 6 hours, and reverts to baseline at 48 hours. It notably increases p53 protein levels and the expression of p53 transcriptional target genes, such as p21, cyclinG1, fas, and activates caspase-3 [3]. Additionally, Cytarabine effectively combats acute leukemias by causing a characteristic G1/S phase block and cell synchronization, thereby extending the survival of leukemic Brown Norway rats. Its effectiveness shows a weak dose-related pattern, suggesting that higher doses do not enhance its anti-leukemic effect in humans [4].

### Solubility Information

Solubility	DMSO: 50 mg/mL (178.78 mM), Sonication is recommended. ( < 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (7.15 mM), Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one.</i>

In vivo Formulation	<i>Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>
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### Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.5755 mL	17.8776 mL	35.7551 mL
5 mM	0.7151 mL	3.5755 mL	7.151 mL
10 mM	0.3576 mL	1.7878 mL	3.5755 mL
50 mM	0.0715 mL	0.3576 mL	0.7151 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

### Reference

- Tobias, S.C. and R.F. Borch, Synthesis and biological evaluation of a cytarabine phosphoramidate prodrug. *Mol Pharm*, 2004. 1(2): p. 112-6.
- Shepshelovich D, et al. Pharmacodynamics of cytarabine induced leucopenia: a retrospective cohort study. *Br J Clin Pharmacol*. 2015 Apr;79(4):685-91.
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- 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.
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