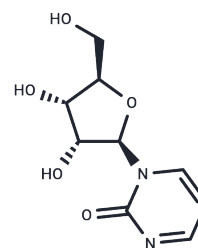


## Zebularine

## Chemical Properties

CAS No. :	3690-10-6
Formula:	C <sub>9</sub> H <sub>12</sub> N <sub>2</sub> O <sub>5</sub>
Molecular Weight:	228.2
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	Zebularine (4-Deoxyuridine) is a DNA methylation inhibitor. Acts as a transition state analog inhibitor of cytidine deaminase by binding to the active site as covalent hydrates. It also inhibits cytidine deaminase (K <sub>i</sub> : 2 μM, in a cell-free assay).
Targets(IC50)	Autophagy, DNA Methyltransferase
In vitro	Zebularine forms a tight covalent complex with bacterial methyltransferases. In <i>N. crassa</i> , Zebularine inhibited DNA methylation and reactivated genes silenced by methylation. In T24 bladder cancer cells, Zebularine reactivated the silenced p16 gene and demethylated its promoter region. Zebularine was only slightly cytotoxic to T24 cells. Zebularine was preferentially incorporated into the DNA of cancer cell lines and inhibited cell growth and gene expression to a greater extent than in normal fibroblasts. In addition, Zebularine preferentially inhibited DNA methyltransferase 1 and induced the expression of cancer-associated antigenic genes in cancer cells compared to normal fibroblasts.
In vivo	Zebularine forms a tight covalent complex with bacterial methyltransferases. In <i>N. crassa</i> , Zebularine inhibited DNA methylation and reactivated genes silenced by methylation. In T24 bladder cancer cells, Zebularine reactivated the silenced p16 gene and demethylated its promoter region. Zebularine was only slightly cytotoxic to T24 cells. Zebularine was preferentially incorporated into the DNA of cancer cell lines and inhibited cell growth and gene expression to a greater extent than in normal fibroblasts. In addition, Zebularine preferentially inhibited DNA methyltransferase 1 and induced the expression of cancer-associated antigenic genes in cancer cells compared to normal fibroblasts.
Cell Research	For methylation analysis, 10T1/2 cells and T24 cells are treated with the various concentrations of zebularine. For 10T1/2 cells, the medium is changed 24 hours after the initial drug treatment, whereas for T24 cells, the medium is changed 24 hours or 48 hours after the initial drug treatment. DNA and RNA are harvested from 10T1/2 cells 72 hours after initial drug treatment and from T24 cells 96 hours after initial drug treatment. The methylation status of the indicated DNA regions is measured in two separate and independent experiments, both of which are done in duplicate[2].

## Solubility Information

## A DRUG SCREENING EXPERT

Solubility	DMSO: 250 mg/mL (1095.53 mM),Sonication is recommended. H2O: 22.8 mg/mL (99.91 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+90% Saline: 10 mg/mL (43.82 mM),Solution. 10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (8.76 mM),Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. 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>

### Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	4.3821 mL	21.9106 mL	43.8212 mL
5 mM	0.8764 mL	4.3821 mL	8.7642 mL
10 mM	0.4382 mL	2.1911 mL	4.3821 mL
50 mM	0.0876 mL	0.4382 mL	0.8764 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

- Zhou L, et al. J Mol Biol, 2002, 321(4), 591-599.  
Lemaire M, et al. Cancer Chemother Pharmacol, 2009, 63(3), 411-416.  
Cheng JC, et al. J Natl Cancer Inst, 2003, 95(5), 399-409.  
Cheng JC, et al. Cancer Cell, 2004, 6(2), 151-158.

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