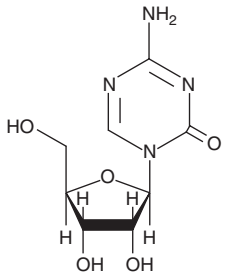
PRODUCT INFORMATION

5-Azacytidine

Item No. 11164

CAS Registry No.:	320-67-2
Formal Name:	4-amino-1-β-D-ribofuranosyl-1,3,5-triazin-2(1H)-one
Synonyms:	Antibiotic U 18496, 5-AzaC, Ladakamycin, Mylosar, NSC 102816, NSC 103-627, U 18496, WR 183027
MF:	$C_8H_{12}N_4O_5$
FW:	244.2
Purity:	≥95%
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

5-Azacytidine is supplied as a crystalline solid. A stock solution may be made by dissolving the 5-azacytidine in the solvent of choice, which should be purged with an inert gas. 5-Azacytidine is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of 5-azacytidine in these solvents is approximately 30 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of 5-azacytidine can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of 5-azacytidine in PBS (pH 7.2) is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

5-Azacytidine, a chemical analogue of the DNA and RNA nucleoside cytidine, is an inhibitor of DNA methyltransferases, potentially serving to reverse epigenetic changes.[1] It reduces hypermethylation associated with certain diseases, including myelodysplastic syndromes (IC_{50}s = 2.4 and 2.6 µM for *in vitro* anti-myeloma activity) and cancer (IC_{50}s ~ 0.4 µM for inhibiting proliferation of various cancer cell lines).[2-4] 5-Azacytidine has a reported half-life of 17 hours and is considerably cytotoxic; it must be incorporated into DNA to covalently trap DNA methyltransferases.[1]

References

1. Brueckner, B., Boy, R.G., Siedlecki, P., *et al.* Epigenetic reactivation of tumor suppressor genes by a novel small-molecule inhibitor of human DNA methyltransferases. *Cancer Res.* **65(14)**, 6305-6311 (2005).
2. Stresemann, C., Brueckner, B., Musch, T., *et al.* Functional diversity of DNA methyltransferase inhibitors in human cancer cell lines. *Cancer Res.* **66(5)**, 2794-2800 (2006).
3. Esteller, M. Epigenetics in cancer. *N. Engl. J. Med.* **358(11)**, 1148-1159 (2008).
4. Lyko, F. and Brown, R. DNA methyltransferase inhibitors and the development of epigenetic cancer therapies. *J. Natl. Cancer Inst.* **97(20)**, 1498-1506 (2005).

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