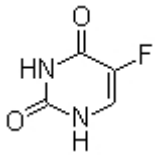
Product Introduction

Fluorouracil (5-Fluoracil, 5-FU)

Fluorouracil (5-Fluoracil, 5-FU) is an DNA/RNA synthesis inhibitor, which interrupts nucleotide synthetic by inhibiting thymidylate synthase (TS).

Technical Data:

Molecular Weight (MW):	130.08	
Formula:	$C_4H_3FN_2O_2$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 26 mg/mL	
	Water <1 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	51-21-8	

Biological Activity

Adrucil is an analogue of uracil with a fluorine atom at the C-5 position in place of hydrogen. It rapidly enters the cell using the same facilitated transport mechanism as uracil. Adrucil is converted intracellularly to several active metabolites: fluorodeoxyuridine monophosphate (FdUMP), fluorodeoxyuridine triphosphate (FdUTP) and fluorouridine triphosphate (FUTP). The Adrucil metabolite FdUMP binds to the nucleotide-binding site of TS, forming a stable ternary complex with the enzyme and CH2THF, thereby blocking binding of the normal substrate dUMP and inhibiting dTMP synthesis. Metabolite of Adrucil also can be misincorporated into DNA, leading to DNA strand breaks and cell death. The pro-apoptosis effects of Adrucil may be related to its activation of tumor suppressor p53. Loss of p53 function reduces cellular

sensitivity to Adrucil. [1] Adrucil is able to inhibit the survival and induce apoptosis of a board range of cancer cells. Adrucil suppresses viabilities of the nasopharyngeal carcinoma cell line CNE2 and HONE1 [2], pancreatic cancer cell lines Capan-1 [3], and human colon carcinoma cell line HT-29 [4] with IC50 of 9 μg/mL, 3 μg/mL, 0.22 μM, 2.5 μM, respectively

Adrucil is widely used in the treatment of a range of cancers, including colorectal and breast cancers. [1] 100mg/kg Adrucil significantly suppresses tumor growth of murine colon carcinomas Colon 38 with tumor-doubling time (TD), growth-delay factor (GDF), and T/C of 26.5 days, 4.4, and 14%. [5]

References

[1] Longley DB, et al. Nat Rev Cancer, 2003, 3(5), 330-338.

[2] Qin L, et al. Biochem Biophys Res Commun, 2008, 371(3), 531-535.

[3] Shi X, et al. Oncology, 2002, 62(4), 354-362.

[4] Schwartz EL. J Biol Chem, 1995, 270(32), 19073-19077.

[5] Van Laar JA, et al. Cancer Chemother Pharmacol, 1996, 39(1-2), 79-89.

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