5-Fluorouracil

Cat. No.: HY-90006

CAS No.: 51-21-8

Molecular Formula: $C_4H_3FN_2O_2$

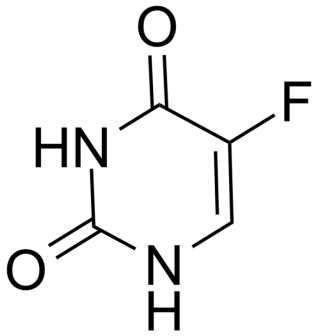
Molecular Weight: 130.08

Target: Nucleoside Antimetabolite/Analog; HIV; Apoptosis; Endogenous Metabolite

Pathway: Cell Cycle/DNA Damage; Anti-infection; Apoptosis; Metabolic Enzyme/Protease

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (384.38 mM; Need ultrasonic)

H_2O : 10 mg/mL (76.88 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	7.6876 mL	38.4379 mL	76.8758 mL
5 mM	1.5375 mL	7.6876 mL	15.3752 mL
10 mM	0.7688 mL	3.8438 mL	7.6876 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 (19.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (19.22 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (19.22 mM); Clear solution
4. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 2.5 mg/mL (19.22 mM); Clear solution
5. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (19.22 mM); Clear solution
6. Add each solvent one by one: 5% DMSO >> 95% saline
   Solubility: 2.5 mg/mL (19.22 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

5-Fluorouracil (5-FU) is an analogue of uracil and a potent antitumor agent. 5-Fluorouracil affects pyrimidine synthesis by

	inhibiting thymidylate synthetase thus depleting intracellular dTTP pools. 5-Fluorouracil induces apoptosis and can be used as a chemical sensitizer[1][2]. 5-Fluorouracil also inhibits HIV[3].
IC_{50} & Target	HIV
In Vitro	5-Fluorouracil (5-Fu) and NSC 123127 (Dox) show synergistic anticancer efficacy. The IC_{50} value of 5-Fu/Dox-DNM toward human breast cancer (MDA-MB-231) cells is 0.25 μg/mL, presenting an 11.2-fold and 6.1-fold increase in cytotoxicity compared to Dox-DNM and 5-Fu-DNM, respectively[1]. In 5-fluorouracil (5-FU) and CDDP treated NFBD1-inhibited NPC cells, the NFBD1 expression in NPC CNE1 cell lines is depleted using lentivirus-mediated short hairpin RNA, and the sensitivity of these cells is elevated. NFBD1 knockdown leads to an obvious induction of apoptosis in CDDP- or 5-FU-treated CNE1 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	5-Fluorouracil (23 mg/kg, 3 times/week) for 14 days, induces accelerated gastrointestinal transit associated with acute intestinal inflammation at day 3 after the start of treatment, which may have led to persistent changes in the ENS observed after days 7 and 14 of treatment contributing to delayed gastrointestinal transit and colonic dysmotility[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]	Mice receive intraperitoneal injections of 5-FU (23 mg/kg), 3 times a week via a 26 gauge needle. 5-FU is dissolved in 100% dimethyl sulfoxide (DMSO) to make 1 M/L stock solution refrigerated at −20°C. The stock is then defrosted and diluted with sterile water to make 0.1 M/L (10% DMSO) solutions for intraperitoneal injections. The dose of 5-FU is calculated to be equivalent to standard human dose per body surface area. The low doses of 5-FU (10-40 mg/kg) have been shown to have antitumor efficacy in mouse models of cancer. Sham-treated mice received 10% DMSO in sterile water via intraperitoneal injection three times a week via a 26 gauge needle. The injected volumes are calculated to the body weight; the maximum volume does not exceed 200 μL per injection. Mice are euthanized via cervical dislocation at 3 (2 treatments), 7 (3 treatments), and 14 (6 treatments) days after the first injection and colon is collected for in vitro experiments. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Med. 2024 Jan 29.
- Mol Cancer. 2024 Jan 24;23(1):23.
- Immunity. 2024 Feb 13;57(2):364-378.e9.
- Gastroenterology. 2021 Nov;161(5):1601-1614.e23.
- Adv Mater. 2021 May;33(18):e2100949.

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REFERENCES

[1]. Han R, et al. Amphiphilic dendritic nanomicelle-mediated co-delivery of 5-fluorouracil and NSC 123127 for enhanced therapeutic efficacy. J Drug Target. 2016 Jun 29:1-28. [Epub ahead of print]

[2]. McQuade RM, et al. Gastrointestinal dysfunction and enteric neurotoxicity following treatment with anticancer chemotherapeutic agent 5-fluorouracil. Neurogastroenterol Motil. 2016 Jun 28.

[3]. Zeng Q, et al. Knockdown of NFBD1/MDC1 enhances chemosensitivity to NSC 119875 or 5-fluorouracil in nasopharyngeal carcinoma CNE1 cells. Mol Cell Biochem. 2016

Jul;418(1-2):137-46.

[4]. Yin L, et al. Antitumor effects of oncolytic herpes simplex virus type 2 against colorectal cancer in vitro and in vivo. Ther Clin Risk Manag. 2017 Feb 7;13:117-130.

[5]. Jones DH, et al. Ten-Year and Beyond Follow-up After Treatment With Highly Purified Liquid-Injectable Silicone for HIV-Associated Facial Lipoatrophy: A Report of 164 Patients. Dermatol Surg. 2019 Jul;45(7):941-948.

[6]. Snyder SM, et al. Initial Experience with Topical Fluorouracil for Treatment of HIV-Associated Anal Intraepithelial Neoplasia. J Int Assoc Physicians AIDS Care (Chic). 2011;10(2):83-88.

[7]. Pek Yee Lum, et al. Discovering modes of action for therapeutic compounds using a genome-wide screen of yeast heterozygotes. Cell. 2004 Jan 9;116(1):121-37.

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