



## 5-Fluorouracil

Catalog No: tcsc0993



## **Available Sizes**

Size: 1g

Size: 5g



## **Specifications**

**CAS No:** 

51-21-8

Formula:

 $C_4H_3FN_2O_2$ 

**Pathway:** 

Cell Cycle/DNA Damage

**Target:** 

Nucleoside Antimetabolite/Analog

**Purity / Grade:** 

>98%

**Solubility:** 

DMSO: 15 mg/mL (115.31 mM; Need ultrasonic and warming)

**Alternative Names:** 

5-FU

**Observed Molecular Weight:** 

130.08

## **Product Description**

5-Fluorouracil is a potent antitumor agent that affects pyrimidine synthesis by inhibiting **thymidylate synthetase** thus depleting intracellular dTTP pools.





In Vitro: 5-Fluorouracil (5-Fu) and doxorubicin (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<sup>[1]</sup>. 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<sup>[3]</sup>.

*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<sup>[2]</sup>.

All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!