Capecitabine

Cat. No.: HY-B0016

CAS No.: 154361-50-9

Molecular Formula: $C_{15}H_{22}FN_3O_6$

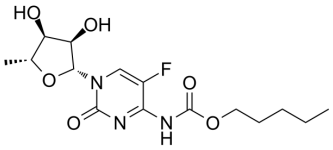
Molecular Weight: 359.35

Target: DNA/RNA Synthesis; Nucleoside Antimetabolite/Analog; Apoptosis

Pathway: Cell Cycle/DNA Damage; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (695.70 mM; Need ultrasonic)

H_2O : ≥ 33.33 mg/mL (92.75 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7828 mL	13.9140 mL	27.8280 mL
5 mM	0.5566 mL	2.7828 mL	5.5656 mL
10 mM	0.2783 mL	1.3914 mL	2.7828 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 25 mg/mL (69.57 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (5.79 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.79 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Capecitabine is an oral prodrug that is converted to its active metabolite, 5-FU, by thymidine phosphorylase.

IC_{50} & Target

DNA/RNA Synthesis[1]

In Vitro	Capecitabine is an anti-cancer chemotherapy drug. It is classified as an antimetabolite. Capecitabine is converted into 5′-deoxy-5-fluorocytidine (5′DFCR), 5′-deoxy-5-fluorouridine (5′DFUR) and 5-FU by carboxylesterases (CES1 and 2), cytidine deaminase (CDD), and thymidine phosphorylase (TP), in both liver and tumour. Capecitabine induces a significant cytotoxic effect in vitro only at high concentrations. Mean IC_{50} values vary from 860 μM in COLO205 cells to 6000 μM in HCT8 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	A pharmacokinetic/pharmacodynamic study is carried out in mice bearing HCT 116 xenografts receiving 0.52 and 2.1 mmol/kg/d of Capecitabine by oral gavage. Capecitabine administered at 0.52 mmol/kg/day induces partial control of HCT 116 xenografts tumour growth: growth rate =7.5±0.5 on day 21. Capecitabine 2.1 mmol/kg/day achieves complete control of tumor growth during the treatment period: growth rate =1±0.2 on day 21[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	HCT 116, HCT8, HCT15, HT29, SW620 and COLO205 human colon cancer cells are used. Cells are plated on day 1 in 96-well plates at a density of 2500 cells/well for HCT 116, 3500 cells/well for HCT8 and HT29, 5000 cells/well for HCT15, 6000 cells/well for SW620 and 7000 cells/wells for COLO205 in a volume of 150 μL/well. All cell lines are treated on day 2 with increasing concentrations of Capecitabine (0.1-10 mM), 5′DFCR (10 nM-100 μM), 5′DFUR (2.5-500 μM) or 5-FU (0.5-250 μM) for 24 h. After drug exposure, cells are washed once with cold PBS and placed in 200 μL of drug-free medium for 72 h after the end of drug exposure. The cells are then fixed with trichloroacetic acid and stained with sulforhodamine B. Optical densities are measured at 540 nm with a Biohit BP-800. The results are based on three independent experiments performed in triplicate[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Six-week-old C57/Bl6 Nu/Nu mice are used. Bilateral HCT 116 xenografts are obtained by subcutaneous injection of 10^7 cells/flank. Animals bearing HCT 116 xenografts are treated with vehicle or Capecitabine 0.52 or 2.1 mmol/kg (563 and 2250 mg/m^2, respectively) given once daily for 5 consecutive days/week by oral gavage for 3 weeks (days 0-4, 7-11, 14-18). Animals are culled on day 0 at 15, 30 min, 1, 2, 4, 8 and 24 h, and prior to planned treatment on days 7 and 14 after the start of treatment. Three animals per time-point are analysed. At the time of collection, blood is collected in heparin, and plasma isolated and stored at −80°C. The liver is removed immediately and stored in RNAlater solution. Tumours are macro-dissected to remove fibrotic tissue and blood vessels and snap-frozen in liquid nitrogen. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cancer Lett. 2020 Apr 28;476:67-74.
- J Adv Res. 12 June 2021.
- Acta Pharmacol Sin. 2021 Jan;42(1):108-114.
- Front Oncol. 2021 Jul 13;11:704042.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

[1]. PharmD CM, et al. Capecitabine: A review. Clinical Therapeutics. 2005 Jan; 27(1): 23-44.

[2]. Guichard SM, et al. Gene expression predicts differential capecitabine metabolism, impacting on both pharmacokinetics and antitumour activity. Eur J Cancer. 2008 Jan;44(2):310-7.

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