

Product Data Sheet

Product Name: Capecitabine

Cat. No.: GC15866

Chemical Properties

Cas. No. 154361-50-9

Chemical Name pentyl N-[1-[(2R,3R,4S,5R)-3,4-dihydroxy-5-methyloxolan-2-yl]-5-fluoro-2-oxypyrimidin-4-yl]carbamate

SMILES CCCCCOC(=O)NC1=NC(=O)N(C=C1F)C2C(C(C(O2)C)O)O

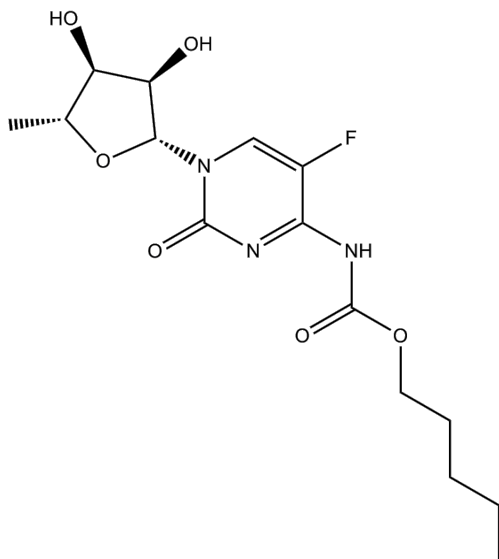
Formula $C_{15}H_{22}FN_3O_6$ M.Wt 359.35

Solubility $\geq 17.95\text{mg/mL}$ in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell experiment: 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 μ L/well. All cell lines are treated on day 2 with increasing concentrations of Capecitabine (0.1-10 mM), 5'DFCR (10 nM-100 μ M), 5'DFUR (2.5-500 μ M) or 5-FU (0.5-250 μ M) for 24 h. After drug exposure, cells are washed once with cold PBS and placed in 200 μ L 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].

Animal experiment: Mice[2] Six-week-old C57/Bl6 Nu/Nu mice are used. Bilateral HCT 116 xenografts are obtained by subcutaneous injection of 107 cells/flank. Animals bearing HCT 116 xenografts are treated with vehicle or Capecitabine 0.52 or 2.1 mmol/kg (563 and 2250 mg/m², 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.

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.

Background

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Background

Capecitabine (Xeloda) is an anti-cancer chemotherapy drug. Capecitabine (N4-pentyloxycarbonyl-5'-deoxy-5-fluorocytidine) is a novel fluoropyrimidinecarbamate that is converted to 5-fluorouracil (5-FUra) by three enzymes located in the liver and tumors [1].

In vitro: In antiproliferative assays, both LS174T WT and LS174T-c2 cells were more sensitive to capecitabine when cultivated in the same plates as HepG2 hepatoma. In LS174T WT alone and cultivated with HepG2, IC50 values of capecitabine were 890 ± 48 and 630 ± 14 μ m respectively. The IC50 fell from 330 ± 4 down to 89 ± 6 μ m in LS174T-C2 subline when cultivated in the same plates as hepatoma cells. In the LS174T-c2 subclone, whereas little cell death occurred in cells exposed to capecitabine, both early and late apoptosis were increased by 244 and 262%, respectively [1]. Furthermore, Capecitabine induces apoptosis in a Fas-dependent manner, and shows a 7-fold higher cytotoxicity and markedly stronger apoptotic potential in thymidine phosphorylase (TP)-transfected LS174T-c2 cells [2].

In vivo: Capecitabine was effective in a wider dose range in CXF280, HCT116, COLO205, and WiDr human colon cancer xenograft models [2]. In highly metastatic nude mice model, capecitabine inhibited tumor growth and metastatic recurrence after resection of HCC attributed to the high expression of PD-ECGF in tumors [3].

References:

- [1]. Ishikawa T, Utoh M, Sawada N, et al. Tumor selective delivery of 5-fluorouracil by capecitabine, a new oral fluoropyrimidine carbamate, in human cancer xenografts[J]. Biochemical pharmacology, 1998, 55(7): 1091-1097.
- [2]. Ishikawa T, Utoh M, Sawada N, et al. Tumor selective delivery of 5-fluorouracil by capecitabine, a new oral fluoropyrimidine carbamate, in human cancer xenografts[J]. Biochemical pharmacology, 1998, 55(7): 1091-1097.
- [3]. Zhou J, Tang Z Y, Fan J, et al. Capecitabine inhibits postoperative recurrence and metastasis after liver cancer resection in nude mice with relation to the expression of platelet-derived endothelial cell growth factor[J]. Clinical cancer research, 2003, 9(16): 6030-6037.

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