Tezacitabine

Cat. No.: HY-106014

CAS No.: 130306-02-4

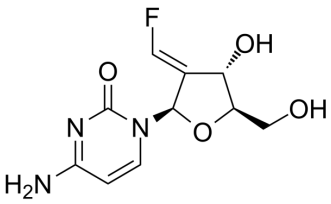
Molecular Formula: $C_{10}H_{12}FN_3O_4$

Molecular Weight: 257.22

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

Pathway: Cell Cycle/DNA Damage; Apoptosis

Storage: -80°C



SOLVENT & SOLUBILITY

In Vitro

DMSO : 200 mg/mL (777.54 mM; Need ultrasonic)

H_2O : 200 mg/mL (777.54 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	3.8877 mL	19.4386 mL	38.8772 mL
5 mM	0.7775 mL	3.8877 mL	7.7754 mL
10 mM	0.3888 mL	1.9439 mL	3.8877 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (388.77 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 5 mg/mL (19.44 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (19.44 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 5 mg/mL (19.44 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tezacitabine is a cytostatic and cytotoxic antimetabolite and a nucleoside analogue. Tezacitabine irreversibly inhibits the ribonucleotide reductase and interferes with DNA replication and repair. Tezacitabine effectively induces cells apoptotic. Tezacitabine has the potential for leukemias and solid tumors (carcinomas) treatment[1][2].

IC_{50} & Target

Ribonucleotide reductase[1]

In Vitro

Tezacitabine (0.01-10 μM; 24 hours; CCRF-SB, KG-1, Jurkat, COLO-205, MCF-7 and PC-3 cells) treatment induces the G1 and S-phase leaky block of the cell cycle[1].
Tezacitabine (0.01-10 μM; 24 hours; CCRF-SB, KG-1, Jurkat, COLO-205, MCF-7 and PC-3 cells) treatment apoptotic death of cells by the caspase 3/7 pathway in a concentration-dependent manner[1].
Tezacitabine has strong cytostatic and cytotoxic properties. Cytotoxic effect of Tezacitabine reveals not only as apoptosis, but also as a change in protein metabolism[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	CCRF-SB, KG-1, Jurkat, COLO-205, MCF-7 and PC-3 cells
Concentration:	0.01 μM, 0.1 μM, 1.0 μM, and 10 μM
Incubation Time:	24 hours
Result:	Induced the G1 (at concentrations higher than 10 nM) and S-phase (at low concentration) leaky block of the cell cycle.

Apoptosis Analysis[1]

Cell Line:	CCRF-SB, KG-1, Jurkat, COLO-205, MCF-7 and PC-3 cells
Concentration:	0.01 μM, 0.1 μM, 1.0 μM, and 10 μM
Incubation Time:	24 hours
Result:	Induced apoptotic death of cells by the caspase 3/7 pathway in a concentration-dependent manner.

In Vivo

Tezacitabine (100 mg/kg; intraperitoneal injection; daily; female nude mice) treatment inhibits tumor growth in HCT 116 tumor xenografts[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female nude mice (7-9-week-old) injected with HCT 116 cells[2]
Dosage:	100 mg/kg
Administration:	Intraperitoneal injection; daily; 14 days
Result:	Inhibited tumor growth in HCT 116 tumor xenografts.

REFERENCES

[1]. Janusz S Skierski, et al. Tezacitabine Blocks Tumor Cells in G1 and S Phases of the Cell Cycle and Induces Apoptotic Cell Death. Acta Pol Pharm. May-Jun 2005;62(3):195-205.

[2]. Pietro Taverna, et al. Tezacitabine Enhances the DNA-directed Effects of Fluoropyrimidines in Human Colon Cancer Cells and Tumor Xenografts. Biochem Pharmacol. 2007 Jan 1;73(1):44-55.

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