CNDAC hydrochloride

Cat. No.: HY-16445B

CAS No.: 134665-72-8

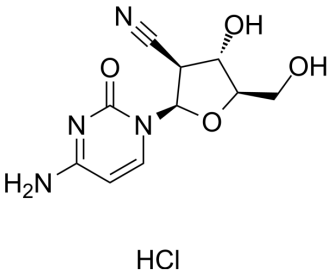
Molecular Formula: $C_{10}H_{13}ClN_4O_4$

Molecular Weight: 288.69

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

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

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (432.99 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4639 mL	17.3196 mL	34.6392 mL
	5 mM	0.6928 mL	3.4639 mL	6.9278 mL
	10 mM	0.3464 mL	1.7320 mL	3.4639 mL

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

BIOLOGICAL ACTIVITY

Description

CNDAC hydrochloride is a metabolite of the orally active agent Sapacitabine (HY-16445), and a nucleoside analog. CNDAC hydrochloride induces DNA damage and apoptosis[1][2].

In Vitro

CNDAC has a unique mechanism of action: after incorporation into DNA, it induces single-strand breaks (SSBs) that are converted into double-strand breaks (DSBs) when cells go through a second S phase[1].
Lack of Rad51D and XRCC3 sensitizes cells to CNDAC (0-1 μM; 24 h)[1].
CNDAC (0-100 μM; 3 days) inhibits proliferation of HL-60 and THP-1 cells[2].
CNDAC (0-10 μM; 3-6 days) induces apoptosis in HL-60 and THP-1 cells[2].
CNDAC (6 μM; 48 h) induces cell cycle arrest in the G2 phase following a delayed S phase in HCT116 cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Rad51D-deficient 51D1, Rad51D-complemented 51D1.3, wild-type AA8 and XRCC3-deficient irs1SF CHO cells
Concentration:	0-1 μM

Incubation Time:	24 h
Result:	Inhibited cell survival with IC_{50}s of 0.006, 0.32, 0.48 and 0.0053 μM against Rad51D-deficient 51D1, Rad51D-complemented 51D1.3, wild-type AA8 and XRCC3-deficient irs1SF cell lines, respectively.

Cell Proliferation Assay[2]

Cell Line:	HL-60 and THP-1 cells
Concentration:	0-100 μM
Incubation Time:	3 days
Result:	Inhibited proliferation with IC_{50}s of 1.5832 μM and 0.84 μM against HL-60 and THP-1 cells, respectively.

Apoptosis Analysis[2]

Cell Line:	HL-60 and THP-1 cells
Concentration:	0, 0.5, 1, 2, 3, 4, 5 and 10 μM
Incubation Time:	3, 4, 5, and 6 days
Result:	Induced apoptosis in both cells.

Cell Cycle Analysis[3]

Cell Line:	HCT116
Concentration:	6 μM
Incubation Time:	48 h
Result:	36 and 36% of cells were arrested in late-S and G2/M phases, respectively.

In Vivo

CNDAC (20mg/kg; i.p.; daily for 10 days) shows antitumor activity in mice[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	CDF1 mice, P388 tumor model[4]
Dosage:	20 mg/kg
Administration:	Intraperitoneal injection, daily for 10 days
Result:	Greatly increased the survival time and survival rate.

REFERENCES

[1]. Jagan S, et al. Bone Marrow and Peripheral Blood AML Cells Are Highly Sensitive to CNDAC, the Active Form of Sapacitabine. Adv Hematol. 2012;2012:727683.

[2]. Serova M, et al. Antiproliferative effects of sapacitabine (CYC682), a novel 2'-deoxycytidine-derivative, in human cancer cells. Br J Cancer. 2007 Sep 3;97(5):628-36.

[3]. Azuma A, et al. Nucleosides and nucleotides. 122. 2'-C-cyano-2'-deoxy-1-beta-D-arabinofuranosylcytosine and its derivatives. A new class of nucleoside with a broad antitumor spectrum. J Med Chem. 1993 Dec 24;36(26):4183-9.

[4]. Liu XJ, et al. Sapacitabine, the prodrug of CNDAC, is a nucleoside analog with a unique action mechanism of inducing DNA strand breaks. Chin J Cancer. 2012 Aug;31(8):373-80.

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