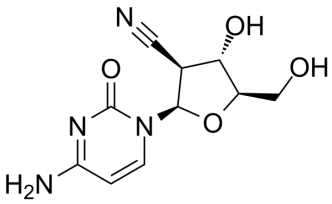
CNDAC

Cat. No.:	HY-16445A
CAS No.:	135598-68-4
Molecular Formula:	$C_{10}H_{12}N_4O_4$
Molecular Weight:	252.23
Target:	Nucleoside Antimetabolite/Analog; Drug Metabolite; Apoptosis; DNA/RNA Synthesis
Pathway:	Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

CNDAC is a metabolite of the orally active agent Sapacitabine (HY-16445), and a nucleoside analog. CNDAC 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].

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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.

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REFERENCES

[1]. 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.

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

[3]. 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.

[4]. 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.