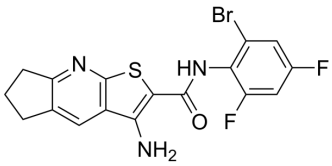
DC_AC50

Cat. No.:	HY-107636
CAS No.:	497061-48-0
Molecular Formula:	$C_{17}H_{12}BrF_2N_3OS$
Molecular Weight:	424.26
Target:	Apoptosis
Pathway:	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 : 50 mg/mL (117.85 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3570 mL	11.7852 mL	23.5705 mL
	5 mM	0.4714 mL	2.3570 mL	4.7141 mL
	10 mM	0.2357 mL	1.1785 mL	2.3570 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.89 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DC_AC50 is a dual inhibitor of Atox1 and CCS (copper chaperones). Inhibiting intracellular copper chaperones as a means of reducing/preventing acquired chemotherapy resistance[1].

In Vitro

DC_AC50 exhibits IC_{50} values of 9.88 μM, 12.57 μM, 5.96 μM and 6.68 μM in Canine Abrams, Canine D1, human HOS and human MG63) cells, respectively[1].
?DC_AC50 (0-10 μM)-treated cells are significantly less mitotically active, as demonstrated by decreased expression of phospho-histone H3 and cell cycle analysis[1].
?DC_AC50 (10 μM) potentiates carboplatin-induced apoptosis in OSA cells and decreasesclonogenic survival[1].
?DC_AC50 induces cell cycle arrest at both the 3 and 10 μM doses and? DC_AC50 induces increase S phase cells dose-independently[1].

?DC_AC50 (3 μM) inhibits the migration and of canine and human OSA cells[1].
?DC_AC50 (2.5-10 μM) is highly efficient at inhibiting cancer cell proliferation (human lung cancer H1299 cells, leukaemia cancer K562 cells, breast cancer MDA-MB-231 cells and head and neck cancer 212LN cells) in a dose-dependent manner. DC_AC50 fails to exhibit any notable inhibition of the cell proliferation of human normal epithelial lung BEAS-2B cells or breast MCF-10A cells as control cells[2].

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

Cell Viability Assay[1]

Cell Line:	Canine OSA (Abrams, D1 and human OSA (HOS, MG63) cells.
Concentration:	0-10 μM.
Incubation Time:	72 h.
Result:	Dose-dependently decreased viability of OSA cells.

Apoptosis Analysis[1]

Cell Line:	Abrams and HOS cells.
Concentration:	1, 3 and 10 μM (10 μM Carboplatin).
Incubation Time:	24 h.
Result:	Potentiated carboplatin-induced apoptosis.

CUSTOMER VALIDATION

- Cancer Lett. 2022 Jun 28;536:215651.

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REFERENCES

[1]. Jordon M Inkol, et al. Inhibition of copper chaperones sensitizes human and canine osteosarcoma cells to carboplatin chemotherapy. Vet Comp Oncol. 2020 Dec;18(4):559-569.

[2]. Jing Wang, et al. Inhibition of human copper trafficking by a small molecule significantly attenuates cancer cell proliferation. Nat Chem. 2015 Dec;7(12):968-79.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA