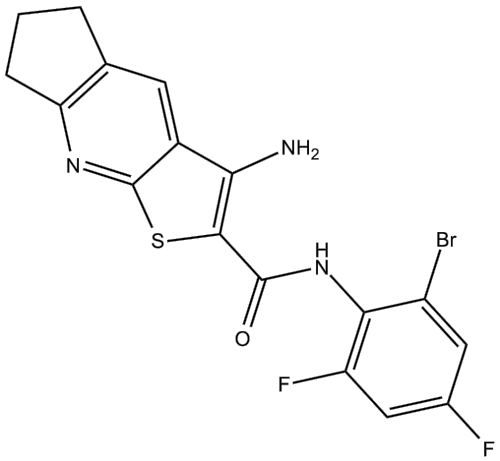
Product Data Sheet

Product Name: DC_AC50
Cat. No.: GC15206

Chemical Properties

Cas. No.	497061-48-0		
Chemical Name	3-amino-N-(2-bromo-4,6-difluorophenyl)-6,7-dihydro-5H-cyclopenta[b]thieno[3,2-e]pyridine-2-carboxamide		
SMILES	NC1=C(C(NC2=C(C=C(F)C=C2Br)F)=O)SC3=NC4=C(CCC4)C=C31		
Formula	$C_{17}H_{12}BrF_2N_3OS$	M.Wt	424.26
Solubility	DMSO: 10 mg/ml	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

Cell experiment [1]:

Cell lines Abrams cells

Product Data Sheet

Preparation Method	Abrams cells were cultured in Dulbecco's Modified Eagle Medium (DMEM) supplemented with 10% fetal bovine serum (FBS) and 100U/ml penicillin/streptomycin. Cells were cultured in a humidified incubator with 5% CO_2 at 37°C. Cells were seeded in 96-well plates at a density of 2×10^3 cells/well and incubated for 16 hours. Each cell line was treated with increasing concentrations of DC_AC50 (0.1, 1, 10, and 100μM) for 72 hours. Cells were then fixed with 4% paraformaldehyde for 10min, rinsed with water, and then fixed with 0.5% crystal violet in 20% methanol. The dye was dried overnight, then eluted with 10% acetic acid, and the absorbance was read at 590nm.
Reaction Conditions	0.1, 1, 10, and 100μM; 72h
Applications	DC_AC50 treatment significantly inhibited the viability of Abrams cells in a dose-dependent manner.

Animal experiment [2]:

Animal models	Female Foxn1nu/nu mice
Preparation Method	Eight-week-old female Foxn1nu/nu mice were anesthetized by inhalation of 2% vaporized isoflurane, and 4×10^6 MDA-MB-468 cells (100μl) were injected unilaterally into the fourth inguinal mammary gland at the base of the nipple. Seven days after cell implantation, mice were randomly and blindly assigned to treatment groups (6-8 mice per group) and given an intraperitoneal injection of either DC_AC50 or DMSO (vehicle control) at a dose of 50mg/kg/day. Mice were monitored twice weekly for tumor growth and body weight. The tumor was measured using a caliper, and the tumor volume was calculated using equation $1/2(\text{length}\times\text{width}^2)$. The entire experiment was continued for 5 weeks after DC_AC50 administration.

Product Data Sheet

Dosage form	50mg/kg/day for 5 weeks; i.p.
Applications	DC_AC50 treatment inhibited tumor growth in mice with the MDA-MB-468 xenograft model.

References:
[1] Inkol J M, Poon A C, Mutsaers A J. Inhibition of copper chaperones sensitizes human and canine osteosarcoma cells to carboplatin chemotherapy[J]. Veterinary and comparative oncology, 2020, 18(4): 559-569.
[2] Karginova O, Weekley C M, Raoul A, et al. Inhibition of copper transport induces apoptosis in triple-negative breast cancer cells and suppresses tumor angiogenesis[J]. Molecular cancer therapeutics, 2019, 18(5): 873-885.

Background

DC_AC50 is a small molecule that inhibits the copper-trafficking proteins ATOX-1 and CCS, with K_d values of 6.8±1.7μM and 8.2±2.7μM, respectively[1]. DC_AC50 is a self-fluorescing compound with excitations at 290nm and 355nm, and emission at 494nm[1]. DC_AC50 can block the copper transport interface of the two major copper chaperones ATOX-1 and CCS to increase intracellular copper levels and lead to enhanced oxidative stress in the cells[2]. DC_AC50 has been widely used to enhance intracellular reactive oxygen species (ROS) levels and inhibit tumor cell growth[3].

In vitro, DC_AC50 treatment for 72 hours significantly inhibited the viability of Abrams and D17 cells with IC_{50} values of 9.88μM and 12.57μM, respectively[4]. Treatment with 25μM DC_AC50 for 7 days can effectively inhibit the proliferation of BRAF-mutated melanoma cells and weaken the activation of the MAPK pathway[5]. Treatment with 10μM DC_AC50 for 24 hours significantly inhibited the growth of HepG2 cells, while promoting the accumulation of ROS and cell apoptosis[6]. 20μM of DC_AC50 pretreatment for 24 hours significantly inhibited the production of ROS and the M1 polarization of mouse macrophages induced by $CuCl_2$[7].

Product Data Sheet

In vivo, DC_AC50 treatment via intraperitoneal injection at a dose of 50mg/kg/day for 5 weeks inhibited tumor growth and suppressed angiogenesis in mice of MDA-MB-468 xenograft model[8]. Intraperitoneal injection of 20mg/kg/day dose of DC_AC50 for 21 consecutive days markedly reduced tumor growth in the xenograft mouse model of A549 cells, and suppressed the expression of ATOX1 in the tumors[9].

References:

[1] Wang J, Luo C, Shan C, et al. Inhibition of human copper trafficking by a small molecule significantly attenuates cancer cell proliferation[J]. Nature chemistry, 2015, 7(12): 968-979.
[2] Karginova O, Weekley C, Raoul A, et al. Selective inhibition of copper metabolism as a novel approach to treat triple-negative breast cancer[J]. Cancer Research, 2017, 77(13_Supplement): 3552-3552.
[3] Li W Y, Kan J L, Wan J J, et al. A reactive oxygen species-responsive covalent organic framework for tumor combination therapy[J]. Chemical Communications, 2023, 59(36): 5423-5426.
[4] Inkol J M, Poon A C, Mutsaers A J. Inhibition of copper chaperones sensitizes human and canine osteosarcoma cells to carboplatin chemotherapy[J]. Veterinary and comparative oncology, 2020, 18(4): 559-569.
[5] Kim Y J, Bond G J, Tsang T, et al. Copper chaperone ATOX1 is required for MAPK signaling and growth in BRAF mutation-positive melanoma[J]. Metallomics, 2019, 11(8): 1430-1440.
[6] Ouyang Q, Jia S, Zhu Q, et al. ATOX1 Promotes Hepatocellular Carcinoma Carcinogenesis via Activation of the c-Myb/PI3K/AKT Signaling Pathway[J]. Journal of Clinical and Translational Hepatology, 2025, 13(8): 630.
[7] Chen M X, Chen Y, Fu R, et al. Atox1 regulates macrophage polarization in intestinal inflammation via ROS-NLRP3 inflammasome pathway[J]. Journal of Translational Medicine, 2024, 22(1): 497.
[8] Karginova O, Weekley C M, Raoul A, et al. Inhibition of copper transport induces apoptosis in triple-negative breast cancer cells and suppresses tumor angiogenesis[J]. Molecular cancer therapeutics, 2019, 18(5): 873-885.
[9] Qin X, Wang P, Liang H, et al. Curcumin suppresses copper accumulation in non-small cell lung cancer by binding ATOX1[J]. BMC Pharmacology and Toxicology, 2024, 25(1): 54.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA