Product Data Sheet

Product Name: ADH-1 trifluoroacetate
Cat. No.: GC34066

Chemical Properties

Cas. No.	1135237-88-5		
SMILES	O=C([C@@H](NC([C@H](C(C)C)NC([C@H](C)NC([C@H](CC1=CN=CN1)N2)=O)=O)=O)CSSC[C@H](NC(C)=O)C2=O)N.O=C(O)C(F)(F)F		
Formula	$C_{24}H_{35}F_3N_8O_8S_2$	M.Wt	684.71
Solubility	DMSO : ≥ 43 mg/mL (62.80 mM)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	ADH-1 trifluoroacetate		

Protocol

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Animal experiment:

Animals are anesthetized, and 40 μL of a single cell suspension containing 50,000 cells is injected into the pancreas. Mice are randomized into treatment groups 10 days after surgery. For treatment, mice are injected intraperitoneally once per day with ADH-1 at 50 mg/kg in 100 μL PBS (×1 per day, ×5 per week for 4 weeks). For in vivo bioluminescence, D-Luciferin is administered by intraperitoneal injection. Data are acquired 20 min after injection using the IVIS system. Tumor growth is monitored every 10 days from day 10 to day 50 after surgery. Luciferase activity is quantified using the IVIS system. Two months after surgery, the mice are killed, and the pancreas, liver, lung, and disseminated nodules are harvested, fixed in 10% buffered formalin, and embedded in paraffin. Serial 5-μM sections are cut, mounted on slides, and stained with H&E using standard procedures. Sections are also stained for TUNEL. Sections are examined using a Zeiss Axioscop 40 microscope equipped with an AxioCam MR digital camera and software.

References:

[1]. Shintani Y, et al. ADH-1 suppresses N-cadherin-dependent pancreatic cancer progression. Int J Cancer. 2008 Jan 1;122(1):71-7.
[2]. Li H, et al. ADH1, an N-cadherin inhibitor,

evaluated in preclinical models of angiogenesis and androgen-independent prostate cancer. Anticancer Drugs. 2007 Jun;18(5):563-8.
[3]. Turley RS, et al. Targeting N-cadherin increases vascular permeability and differentially activates AKT in melanoma. Ann Surg. 2015 Feb;261(2):368-77

Background

ADH-1 is a cyclic peptide antagonist of N-cadherin.[1] It inhibits neurite outgrowth in cerebellar neurons cultured on N-cadherin-expressing 3T3 cell monolayers (IC_{50} = 0.323 mM). ADH-1 (0.2 mg/ml) inhibits cell scattering and motility induced by collagen I in Capan-1 cells and wild-type and N-cadherin-overexpressing BxPC-3 cells.[2] It induces apoptosis in N-cadherin-overexpressing, but not knockdown, BxPC-3 cells when used at a concentration of 1 mg/ml. ADH-1 (50 mg/kg) reduces tumor growth in an N-cadherin-

overexpressing BxPC-3 mouse xenograft model.

1.Williams, E., Williams, G., Gour, B.J., et al.A novel family of cyclic peptide antagonists suggests that N-cadherin specificity is determined by amino acids that flank the HAV motifJ. Biol. Chem.275(6)4007-4012(2000) 2.Shintani, Y., Fukumoto, Y., Chaika, N., et al.ADH-1 suppresses N-cadherin-dependent pancreatic cancer progressionInt. J. Cancer122(1)71-77(2008)