ADH-1

Cat. No.: HY-13541

CAS No.: 229971-81-7

Molecular Formula: $C_{22}H_{34}N_8O_6S_2$

Molecular Weight: 571

Target: Others

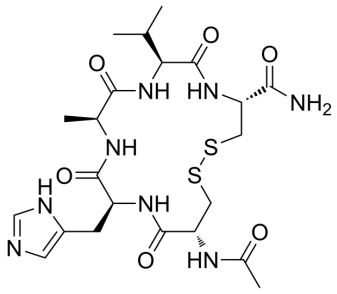
Pathway: Others

Storage: Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (437.83 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7513 mL	8.7566 mL	17.5131 mL
	5 mM	0.3503 mL	1.7513 mL	3.5026 mL
	10 mM	0.1751 mL	0.8757 mL	1.7513 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: ≥ 2.08 mg/mL (3.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.64 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ADH-1, an N-cadherin antagonist, inhibits N-cadherin mediated cell adhesion.

In Vitro

ADH-1 (0.2 mg/mL) blocks collagen I-mediated changes in pancreatic cancer cells, and is highly effective at preventing cell motility that is induced by expression of N-cadherin. ADH-1 (0, 0.1, 0.2, 0.5 and 1.0 mg/mL) induces apoptosis in a dose-dependent and N-cadherin-dependent manner[1].

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

In Vivo	ADH-1 (50 mg/kg) significantly prevents tumor growth and metastasis in a mouse model for pancreatic cancer. ADH-1 prevents tumor cell invasion and metastasis in an orthotopic model for pancreatic cancer using N-cadherin overexpressing BxPC-3 cells[1]. ADH-1, at the dosages evaluated, does not display either antiangiogenic activity in a rat aortic ring assay or antitumor potential in a PC3 subcutaneous xenograft tumor model[2]. In A375, but not DM443 xenografts, ADH-1 treatment increases phosphorylation of AKT at serine 473. ADH-1 slightly diminishes N-cadherin expression in both xenografts[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	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. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Death Dis. 2022 Jun 20;13(6):557.
- Oncogene. 2022 Oct 12.
- J Cell Mol Med. 2022 Feb 27.
- Sci Rep. 2019 Feb 6;9(1):1517.
- Biochem Biophys Res Commun. 2019 Oct 5. pii: S0006-291X(19)31851-0.

See more customer validations on www.MedChemExpress.com

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

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