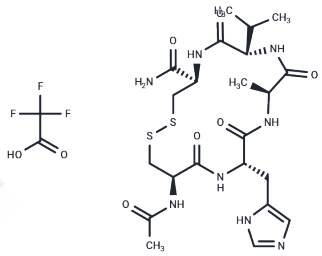
Data Sheet (Cat.No.T1608)

TargetMol

ADH-1 trifluoroacetate

Chemical Properties



CAS No. :	1135237-88-5
Formula:	C24H35F3N8O8S2
Molecular Weight:	684.71
Appearance:	no data available
Storage:	keep away from moisture Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	ADH-1 trifluoroacetate (Exherin trifluoroacetate) is a cyclic pentapeptide vascular-targeting agent with potential antineoplastic and antiangiogenic activities. ADH-1 selectively and competitively binds to and blocks N-cadherin, which may result in disruption of tumor vasculature, inhibition of tumor cell growth, and the induction of tumor cell and endothelial cell apoptosis.
Targets(IC50)	Dehydrogenase
In vitro	In pancreatic cancer cells, Exherin (0.2 mg/mL) blocks collagen I-mediated changes and is highly potent at preventing cell motility induced by expression of N-cadherin. Exherin (0-1.0 mg/mL) dose-dependently induces apoptosis in a N-cadherin-dependent manner.
In vivo	In a mouse model for pancreatic cancer, ADH-1 (50 mg/kg) markedly inhibits tumor growth and metastasis [1]. In a rat aortic ring assay or antitumor potential in a PC3 subcutaneous xenograft tumor model, ADH-1 does not display either antiangiogenic activity [2]. The augmentation of melanoma tumor growth mediated by ADH-1 is not altered by regionally infused temozolomide. In A375, but not DM443 xenografts, ADH-1 can increase phosphorylation of AKT at serine 473. ADH-1 slightly diminishes N-cadherin expression in both xenografts[3].
Animal Research	Exherin is prepared in PBS.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 Exherin 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.

Solubility Information

Solubility	DMSO: 43 mg/mL (62.8 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.4605 mL	7.3024 mL	14.6047 mL
5 mM	0.2921 mL	1.4605 mL	2.9209 mL
10 mM	0.146 mL	0.7302 mL	1.4605 mL
50 mM	0.0292 mL	0.146 mL	0.2921 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Shintani Y, et al. ADH-1 suppresses N-cadherin-dependent pancreatic cancer progression. Int J Cancer. 2008 Jan 1; 122(1):71-7.

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.

Turley RS, et al. Targeting N-cadherin increases vascular permeability and differentially activates AKT in melanoma. Ann Surg. 2015 Feb;261(2):368-77.