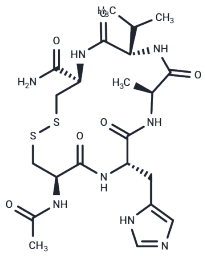
Data Sheet (Cat.No.T2637)

TargetMol

ADH-1

Chemical Properties



CAS No. : 229971-81-7

Formula: C22H34N8O6S2

Molecular Weight: 570.69

Appearance: no data available

Storage: keep away from moisture,keep away from direct sunlight,store at low temperature
Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	ADH-1 (Exherin) is an N-cadherin antagonist, inhibits N-cadherin mediated cell adhesion with potential antineoplastic and antiangiogenic activities.
Targets(IC50)	Dehydrogenase
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].
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]. ADH-1 (10 mL/kg, i.p.) augmentation of melanoma tumor growth is overcome through its ability to make regionally infused melphalan more effective. ADH-1 mediated augmentation of melanoma tumor growth is not altered by regionally infused temozolomide. 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].
Kinase Assay	Kinase Activity Assays: The effect of VX-509 on JAK3 activity is assessed by measuring the residual kinase activity of the recombinantly expressed JAK3 kinase domain using a radiometric assay. The final concentrations of the components in the assay are as follows: 100 mM HEPES (pH 7.5), 10 mM MgCl2, 1 mM dithiothreitol (DTT), 0.01% BSA, 0.25 nM JAK3, 0.25 mg/ml polyE4Y, and 5 μM 33P-γ-ATP (200 μCi/μMol). A 10 mM stock solution of VX-509 is prepared in DMSO, from which additional dilutions are prepared. A substrate mixture (100 mM HEPES, 10 mM MgCl2, 0.5 mg/ml polyE4Y, and 10 μM 33P-γ-ATP) is added and mixed with VX-509 stock solution. The reaction is initiated by the addition of an enzyme mixture [100 mM HEPES (pH 7.5), 10 mM MgCl2, 2 mM DTT, 0.02% BSA, 0.5 nM JAK3]. After 15 minutes, the reaction was quenched with 20% trichloroacetic acid (TCA). The quenched reaction was transferred to the GF/B filter plates and washed three times with 5% TCA. Following the addition of Ultimate Gold scintillant (50 μl), the samples were counted in a Packard TopCount gamma counter (PerkinElmer). In this

procedure, the radioactivity trapped is a measure of the residual JAK3 kinase activity. From the activity versus concentration of VX-509 titration curve, the Ki value was determined by fitting the data to an equation for competitive tight binding inhibition kinetics using Prism software.

Solubility Information

Solubility	DMSO: 10 mM,Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.7523 mL	8.7613 mL	17.5226 mL
5 mM	0.3505 mL	1.7523 mL	3.5045 mL
10 mM	0.1752 mL	0.8761 mL	1.7523 mL
50 mM	0.035 mL	0.1752 mL	0.3505 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.

Jiang D, Christ S, Correa-Gallegos D, et al. Injury triggers fascia fibroblast collective cell migration to drive scar formation through N-cadherin. Nature communications. 2020 Nov 6;11(1):5653. doi: 10.1038/s41467-020-19425-1.

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.

Jiang D, Christ S, Correa-Gallegos D, et al. Injury triggers fascia fibroblast collective cell migration to drive scar formation through N-cadherin[J]. Nature communications. 2020, 11(1): 1-13.