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产品名称: **Exherin (trifluoroacetate)**
产品别名: **ADH-1 trifluoroacetate**

生物活性:

Description	ADH-1 trifluoroacetate is an N-cadherin antagonist, which 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].				
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].				
Solvent&Solubility	In Vitro: DMSO : ≥ 43 mg/mL (62.80 mM) * "≥" means soluble, but saturation unknown.				
	Preparing Stock Solutions	<div><div>Solvent Concentration</div><div>Mass</div></div>	1 mg	5 mg	10 mg
		1 mM	1.4605 mL	7.3024 mL	14.6047 mL
		5 mM	0.2921 mL	1.4605 mL	2.9209 mL
		10 mM	0.1460 mL	0.7302 mL	1.4605 mL
	*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液; 一旦配成溶液, 请分装保存, 避免反复冻融造成的产品失效。 储备液的保存方式和期限: -80°C, 6 months; -20°C, 1 month。 -80°C 储存时, 请在 6 个月内使用, -20°C 储存时, 请在 1 个月内使用。 In Vivo: 请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 In Vitro 方式配制澄清的储备液, 再依次添加助溶剂: ——为保证实验结果的可靠性, 澄清的储备液可以根据储存条件, 适当保存; 体内实验的工作液, 建议您现用现配, 当天使用; 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比; 如在配制过程中出现沉淀、析出现象, 可以通过加热和/或超声的方式助溶 1.请依序添加每种溶剂: 10% DMSO→40% PEG300 →5% Tween-80 → 45% saline Solubility: ≥ 2.5 mg/mL (3.65 mM); Clear solution 此方案可获得 ≥ 2.5 mg/mL (3.65 mM, 饱和度未知) 的澄清溶液。 以 1 mL 工作液为例, 取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 400 μL PEG300 中, 混合均匀; 向上述体系中加入 50 μL Tween-80, 混合均匀; 然后继续加入 450 μL 生理盐水定容至 1 mL。				



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	<p>2.请依序添加每种溶剂: 10% DMSO→ 90% (20% SBE-β-CD in saline)</p> <p>Solubility: ≥ 2.5 mg/mL (3.65 mM); Clear solution</p> <p>此方案可获得 ≥ 2.5 mg/mL (3.65 mM, 饱和度未知) 的澄清溶液。</p> <p>以 1 mL 工作液为例, 取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 20% 的 SBE-β-CD 生理盐水溶液中, 混合均匀。</p> <p>3.请依序添加每种溶剂: 10% DMSO →90% corn oil</p> <p>Solubility: ≥ 2.5 mg/mL (3.65 mM); Clear solution</p> <p>此方案可获得 ≥ 2.5 mg/mL (3.65 mM, 饱和度未知) 的澄清溶液, 此方案不适用于实验周期在半个月以上的实验。</p> <p>以 1 mL 工作液为例, 取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中, 混合均匀。</p>
References	<p>[1]. Shintani Y, et al. ADH-1 suppresses N-cadherin-dependent pancreatic cancer progression. Int J Cancer. 2008 Jan 1;122(1):71-7.</p> <p>[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.</p> <p>[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</p>
实验参考:	
Animal Administration	<p>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. [1]</p>
References	<p>[1]. Shintani Y, et al. ADH-1 suppresses N-cadherin-dependent pancreatic cancer progression. Int J Cancer. 2008 Jan 1;122(1):71-7.</p> <p>[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.</p> <p>[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</p>