Product Data Sheet

Product Name: Enalapril (MK-421)
Cat. No.: GC32499

Chemical Properties

Cas. No.	75847-73-3		
SMILES	O=C(O)[C@H]1N(C([C@H](C)N[C@H](C(OCC)=O)CCC2=CC=CC=C2)=O)CCC1		
Formula	$C_{20}H_{28}N_2O_5$	M.Wt	376.45
Solubility	Soluble in DMSO	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	Enalapril (MK-421)		

Protocol

Cell experiment [1]:

Cell lines	Human Umbilical Vein Endothelial Cells (HUVECs)
Preparation Method	HUVECs were cultured in endothelial basal medium (EBM) supplemented with 10% fetal calf serum (FCS). HUVECs were treated with Enalapril at a concentration of 50μM.
Reaction Conditions	50μM; 24 hours (pretreatment) prior to exposure to serum from Alzheimer's disease (AD) patients.

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Applications	Enalapril pretreatment significantly suppressed the induction of apoptosis by AD patient serum in HUVECs, reducing the apoptosis compared to the AD serum-only group. Enalapril also significantly decreased the elevated levels of nitrite (NO_2), a nitric oxide metabolite, in the culture media from 1.03μm/L to 0.07μm/L.

Animal experiment [2]:

Animal models	C57BL/6 mice
Preparation Method	Mice were intraperitoneally administered Enalapril (5mg/kg/day) for 14 days, starting 1 hour after endotracheal instillation of Bleomycin (4U/kg). Pulmonary hemodynamics and tissue analyses were performed 14 days post-bleomycin exposure.
Dosage form	5mg/kg/day; i.p.; Daily for 14 days.
Applications	Enalapril treatment significantly attenuated Bleomycin-induced pulmonary hypertension, reducing mean pulmonary arterial pressure and pulmonary vascular resistance. Enalapril also inhibited right ventricular hypertrophy, suppressed lung collagen deposition, and ameliorated vascular remodeling.

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References:
[1] Meamar R, Dehghani L, Ghasemi M, et al. Enalapril protects endothelial cells against induced apoptosis in Alzheimer's disease. J Res Med Sci. 2013 Mar;18(Suppl 1):S1-5.
[2] Ortiz LA, Champion HC, Lasky JA, et al. Enalapril protects mice from pulmonary hypertension by inhibiting TNF-mediated activation of NF-kappaB and AP-1. Am J Physiol Lung Cell Mol Physiol. 2002 Jun;282(6):L1209-21.

Background

Enalapril (MK-421) is an orally active angiotensin-converting enzyme (ACE) inhibitor that is rapidly hydrolyzed in vivo to its active metabolite, Enalaprilat. Enalaprilat effectively dilates blood vessels, lowers blood pressure, and reduces cardiac load by inhibiting the production of angiotensin II[1-2]. Enalapril is applicable in studies of conditions such as hypertension and congestive heart failure[3-4].

In vitro, pretreatment of human umbilical vein endothelial cells (HUVECs) with Enalapril (50μM) for 24h, followed by stimulation with serum from Alzheimer’s disease (AD) patients for 24h, Enalapril significantly suppressed the expression of pro-apoptotic factors and reduced the apoptosis rate[5]. Pretreatment of human colorectal cancer cells (HCT116, SW620) and primary human colorectal cancer cells (P1–P4) with Enalapril (100μM) for 24–72h, followed by stimulation with 5-FU (10μM) for 48–72h, significantly inhibited cell proliferation and NF-κB/STAT3 signaling pathway activity, while also reducing the expression of angiogenesis- and epithelial-mesenchymal transition (EMT)-related proteins[6].

In vivo, daily intraperitoneal injection of Enalapril (5mg/kg) for 14 days in a

bleomycin-induced (4U/kg) C57BL/6 mouse model of lung injury significantly suppressed the development of bleomycin-induced pulmonary hypertension, while also reducing pulmonary collagen deposition and inflammatory responses[7]. Oral administration of Enalapril (30mg/kg/day) via drinking water, starting at 12 months of age and continuing until 15 months of age in C57BL/6 mice, significantly improved age-related decline in multiple organ functions and cognitive-behavioral performance[8].

References:

[1] Gomez HJ, Cirillo VJ, Smith SG 3rd. Enalapril: benefit-to-risk ratio in hypertensive patients. J Cardiovasc Pharmacol. 1990;15 Suppl 3:S26-9.
[2] McRaith J, Fitz A. Enalapril: a new angiotensin-converting enzyme inhibitor. Iowa Med. 1986 Oct;76(10):482, 484, 486-8.
[3] Cleary JD, Taylor JW. Enalapril: a new angiotensin converting enzyme inhibitor. Drug Intell Clin Pharm. 1986 Mar;20(3):177-86.
[4] Vlasses PH, Larijani GE, Conner DP, et al. Enalapril, a nonsulfhydryl angiotensin-converting enzyme inhibitor. Clin Pharm. 1985 Jan-Feb;4(1):27-40.
[5] Meamar R, Dehghani L, Ghasemi M, et al. Enalapril protects endothelial cells against induced apoptosis in Alzheimer's disease. J Res Med Sci. 2013 Mar;18(Suppl 1):S1-5.
[6] Yang Y, Ma L, Xu Y, et al. Enalapril overcomes chemoresistance and potentiates antitumor efficacy of 5-FU in colorectal cancer by suppressing proliferation, angiogenesis, and NF-κB/STAT3-regulated proteins. Cell Death Dis. 2020 Jun 24;11(6):477.
[7] Ortiz LA, Champion HC, Lasky JA, et al. Enalapril protects mice from pulmonary hypertension by inhibiting TNF-mediated activation of NF-kappaB and AP-1. Am J Physiol Lung Cell Mol Physiol. 2002 Jun;282(6):L1209-21.
[8] Lyu W, Wang H, Du Z, et al. Enalapril mitigates senescence and aging-related phenotypes in human cells and mice via pSmad1/5/9-driven antioxidative genes. Elife. 2025 Aug 28;14:RP104774.

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