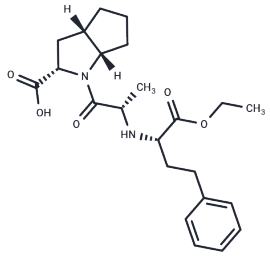
Data Sheet (Cat.No.T1645)

TargetMol

Ramipril

Chemical Properties



CAS No. : 87333-19-5

Formula: C23H32N2O5

Molecular Weight: 416.51

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Ramipril (Altace) is a long-acting angiotensin-converting enzyme inhibitor. It is a prodrug that is transformed in the liver to its active metabolite ramiprilat.
Targets(IC50)	Apoptosis,RAAS,Angiotensin-converting Enzyme (ACE)
In vitro	In spontaneously hypertensive rats, Ramipril significantly inhibits both aortic and pulmonary ACE (ACE Angiotensin Converting Enzyme) activity with an IC50 of approximately 5 mg/kg, without affecting cerebral ACE. When administered slowly, Ramipril lowers blood pressure similar to its effect in vitro on HUVECs (Human Umbilical Vein Endothelial Cells), and compared to other ACE inhibitors, it notably reduces the apoptosis rate induced by lipopolysaccharide. In spontaneously hypertensive rats, Ramipril suppresses systolic blood pressure (SBP) with an IC50 of 1.97 mg/kg. When used in conjunction with AT1 receptor blockers, it synergistically enhances the reduction in SBP. Furthermore, in rats treated with bone morphogenetic protein, Ramipril inhibits beta-cell dysfunction by decreasing monocyte/macrophage permeability, apoptosis, and fibrosis, while also reducing the expression of growth factor genes, the Renin-Angiotensin System (RAS), and inflammatory molecules.
In vivo	Long-term treatment of primary human endothelial cells with Ramipril increases ACE expression, an effect that is blocked by the JNK inhibitor SP600125. In serum-starved endothelial cells in vitro, Ramipril does not promote apoptosis. When applied to cultured endothelial cells, Ramipril enhances the activity of CK2 associated with ACE and increases phosphorylation at the ACE Ser1270 site. However, in cells lacking ACE or possessing the S1270A ACE mutation, Ramipril fails to stimulate nuclear accumulation of c-Jun or activate JNK.
Cell Research	The HUVECs are pretreated with the active metabolites of Ramipril for 24 hours. A serum deprivation method is used to induce apoptosis in the presence of Ramipril for an additional 24 hours. The rate of apoptosis is then determined using flow cytometry with two makers annexinV fluorescein isothiocyanate (FITC+) and propidium iodide (PI).(Only for Reference)

Solubility Information

Solubility	DMSO: 50 mg/mL (120.05 mM),Sonication is recommended. Ethanol: 41.7 mg/mL (100.12 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2.5 mg/mL (6 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.4009 mL	12.0045 mL	24.009 mL
5 mM	0.4802 mL	2.4009 mL	4.8018 mL
10 mM	0.2401 mL	1.2005 mL	2.4009 mL
50 mM	0.048 mL	0.2401 mL	0.4802 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Stevens BR, et al. Comp Biochem Physiol C, 1988, 91(2), 493-497.
Kohlstedt K, et al. Circ Res, 2004, 94(1), 60-67.
Ceconi C, et al. Cardiovasc Drugs Ther, 2007, 21(6), 423-429.
Raasch W, et al. J Hypertens, 2004, 22(3), 611-618.
Cushman DW, et al. Br J Clin Pharmacol, 1989, 28, 115S-131S.