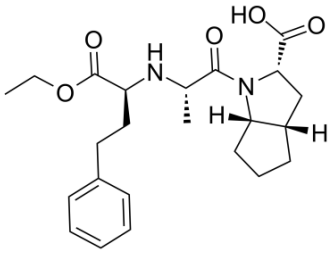
Ramipril

Cat. No.:	HY-B0279
CAS No.:	87333-19-5
Molecular Formula:	$C_{23}H_{32}N_2O_5$
Molecular Weight:	416.51
Target:	Angiotensin-converting Enzyme (ACE); Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (240.09 mM)
H_2O : 1 mg/mL (2.40 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4009 mL	12.0045 mL	24.0090 mL
5 mM	0.4802 mL	2.4009 mL	4.8018 mL
10 mM	0.2401 mL	1.2005 mL	2.4009 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: **10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline**
   Solubility: ≥ 3.25 mg/mL (7.80 mM); Clear solution
2. Add each solvent one by one: **10% DMSO >> 90% (20% SBE-β-CD in saline)**
   Solubility: ≥ 3.25 mg/mL (7.80 mM); Clear solution
3. Add each solvent one by one: **10% DMSO >> 90% corn oil**
   Solubility: ≥ 3.25 mg/mL (7.80 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Ramipril (HOE-498) is an angiotensin-converting enzyme (ACE) inhibitor with IC_{50} of 5 nM.
IC_{50} & Target	ACE[1].

In Vitro	Ramipril (HOE-498) is an angiotensin-converting enzyme (ACE) inhibitor with IC_{50} of 5 nM[1]. Ramipril (HOE-498) enhances the activity of ACE-associated CK2 and the phosphorylation of ACE Ser1270 in cultured endothelial cells, but is unable to activate JNK or stimulate the nuclear accumulation of c-Jun in endothelial cells expressing a S1270A ACE mutant or in ACE-deficient cells. Prolonged Ramipril treatment increases ACE expression in primary cultures of human endothelial cells and in vivo (mouse lung), which can be prevented by pretreatment with the JNK inhibitor SP600125[2].
In Vivo	Chronic in vivo administration of Ramipril (HOE-498) to rats at a dosage that has similar hypotensive effects in vitro HUVECs significantly reduces the rate of LPS-induced apoptosis compared to the other ACE inhibitors, which contrasts with the apoptosis effect in vitro[3]. Ramipril (HOE-498) inhibits systolic blood pressure (SBP) with IC_{50} of 1.97 mg/kg in spontaneously hypertensive rats (SHR). When in combination with AT1-receptor blockade by candesartan-cilexetil increases SBP reduction synergistically rather than additively[4].

REFERENCES

[1]. Raasch, W., et al., Combined blockade of AT1-receptors and ACE synergistically potentiates antihypertensive effects in SHR. J Hypertens, 2004. 22(3): p. 611-8.

[2]. Stevens, B.R., M.I. Phillips, and A. Fernandez, Ramipril inhibition of rabbit (Oryctolagus cuniculus) small intestinal brush border membrane angiotensin converting enzyme. Comp Biochem Physiol C, 1988. 91(2): p. 493-7.

[3]. Kohlstedt, K., et al., Angiotensin-converting enzyme is involved in outside-in signaling in endothelial cells. Circ Res, 2004. 94(1): p. 60-7.

[4]. Ceconi, C., et al., Differences in the effect of angiotensin-converting enzyme inhibitors on the rate of endothelial cell apoptosis: in vitro and in vivo studies. Cardiovasc Drugs Ther, 2007. 21(6): p. 423-9.

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