

Product Data Sheet

Product Name: Ramipril
Cat. No.: GC15760

Chemical Properties

Cas. No. 87333-19-5

Chemical Name (2S,3aS,6aS)-1-[(2S)-2-[(2S)-1-ethoxy-1-oxo-4-phenylbutan-2-yl]amino]propanoyl]-3,3a,4,5,6,6a-hexahydro-2H-cyclopenta[b]pyrrole-2-carboxylic acid

SMILES CCOC(=O)C(CCC1=CC=CC=C1)NC(C)C(=O)N2C3CCCC3CC2C(=O)O

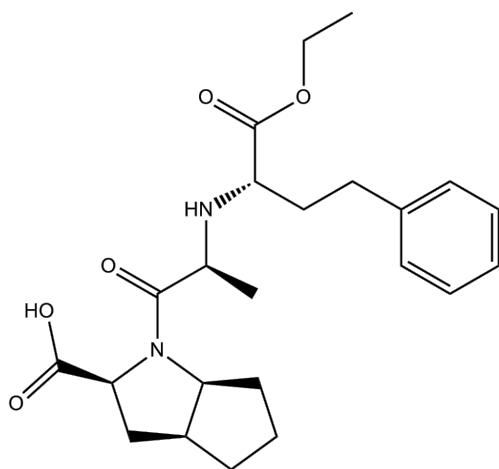
Formula C₂₃H₃₂N₂O₅ M.Wt 416.51

Solubility ≥ 17.55mg/mL in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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



Background

Ramipril (HOE-498) is an angiotensin-converting enzyme (ACE) inhibitor with IC₅₀ of 5 nM.

Ramipril (HOE-498) is an angiotensin-converting enzyme (ACE) inhibitor with IC₅₀ of 5 nM[1].

Caution: Product has not been fully validated for medical applications. For research use only.

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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].

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 IC₅₀ 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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