Moexipril hydrochloride

Cat. No.: HY-B0378A

CAS No.: 82586-52-5

Molecular Formula: $C_{27}H_{35}ClN_2O_7$

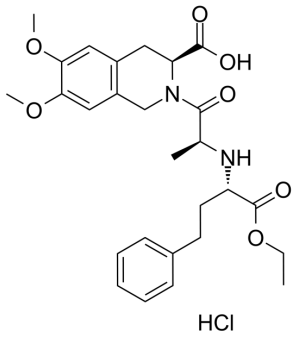
Molecular Weight: 535.03

Target: Angiotensin-converting Enzyme (ACE); Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (467.26 mM; Need ultrasonic)

H_2O : 100 mg/mL (186.91 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8691 mL	9.3453 mL	18.6905 mL
5 mM	0.3738 mL	1.8691 mL	3.7381 mL
10 mM	0.1869 mL	0.9345 mL	1.8691 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (93.45 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (3.89 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.89 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Moexipril hydrochloride (RS-10085) is an orally active inhibitor of angiotensin-converting enzyme (ACE), and becomes effective by being hydrolyzed to moexiprila (hydrochloride). Moexipril hydrochloride exhibits antihypertensive and neuroprotective effects[1]-[4].

IC_{50} & Target

IC50: 2.1 nM (purified ACE from rabbit lung), 1.75 nM (ACE in rat plasma)[3]

In Vitro

Moexipril hydrochloride is devoid of anti-inflammatory properties and has no effect on platelet function[2].
Moexipril hydrochloride hydrolyzes to Moexiprilat, and Moexiprilat inhibits ACE in guinea pig serum as well as on purified ACE from rabbit lung with IC_{50}s of 2.6 and 4.9 nM, respectively[2].
Moexipril hydrochloride (0.01 nM-0.1 mM) exhibits high potency against both plasma ACE and purified ACE from rabbit lung, with IC_{50}s of 2.7 mM and 0.165 mM, respectively[3].
Moexipril hydrochloride (0-100 μM, 24 h) significantly reduced the percentage of damaged neurons in a dose-dependent manner[4].
Moexipril hydrochloride (0-100 μM, 24 h) significantly attenuates $Fe^{2+/3+}$-induced neurotoxicity[4].
Moexipril hydrochloride dose not cause significant changes in the percentage of apoptotic neurons[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Moexipril hydrochloride can not cross the blood-brain barrier[1].
Moexipril hydrochloride (3 mg/kg, 30 mg/kg and 10 mg/kg, respectively; p.o.; once daily; 5 days) exhibits the a dose-dependent and antihypertensive effects in renal hypertensive rats, spontaneously hypertensive rats and perinephritic hypertensive dogs, respectively[3].
Moexipril hydrochloride (0.3 mg/kg, i.p.) significantly reduces the infarct area on the mouse brain surface in NMRI mice[4].
Moexipril hydrochloride (0.1 mg/kg, i.p.) significantly attenuates the cortical infarct volume in Long-Evans rats[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Spontaneously hypertensive rats[3]
Dosage:	30 mg/kg
Administration:	Oral gavage; once daily; 5 days
Result:	Caused a progressive lowering of mean blood pressure from pretreatment values of 180 +/- 7 mmHg to a trough on day 4 of 127 +/- 4 mmHg. Dose-dependently decreased arterial blood pressure, and inhibited plasma and tissue ACE activity.

Animal Model:	Renal hypertensive rats[3]
Dosage:	0.03-10 mg/kg
Administration:	Oral gavage; once daily; 5 days
Result:	Caused a dose-dependent decrease in blood pressure with a threshold dose of 0.3 mg/kg. Lowered mean blood pressure by about 70 mmHg of 3 mg/kg.

Animal Model:	Perinephritic hypertensive dogs[3]
Dosage:	10 mg/kg
Administration:	Oral gavage; once daily; 5 days
Result:	Caused a drop of mean blood pressure by 25 mmHg from pre-treatment control, which persisted for 24 h, by a rapid onset and a long duration of action.

Animal Model:	NMRI mice (male, Permanent focal ischemia)[4]
Dosage:	0, 0.03, 0.3, and 3 mg/kg
Administration:	Intraperitoneal injection (1 h before middle cerebral artery occlusion)

Result:	Significantly reduced the infarct area on the mouse brain surface with a dose of 0.3 mg/kg, and other doses were not effective.

Animal Model:	Long-Evans rats (male, Permanent focal ischemia)[4]
Dosage:	0, 0.01, 0.1 mg/kg
Administration:	Intraperitoneal injection (1 h before middle cerebral artery occlusion)
Result:	Significantly attenuated the cortical infarct volume from 114.4 to 98.2 mm as compared to non-treated animals in a dose of 0.01 mg/kg, without reducing the infarct volume of the rat brain at dosages >0.01 mg/kg.

REFERENCES

[1]. Friehe H, et al. Pharmacological and toxicological studies of the new angiotensin converting enzyme inhibitor moexipril hydrochloride. Arzneimittelforschung. 1997 Feb. 47(2):132-44.

[2]. Ravati A, et al. Enalapril and moexipril protect from free radical-induced neuronal damage in vitro and reduce ischemic brain injury in mice and rats. Eur J Pharmacol. 1999 May 28;373(1):21-33.

[3]. Chrysant, S.G. and G.S. Chrysant, Pharmacological and clinical profile of moexipril: a concise review. J Clin Pharmacol, 2004. 44(8): p. 827-36.

[4]. Edling, O., et al., Moexipril, a new angiotensin-converting enzyme (ACE) inhibitor: pharmacological characterization and comparison with enalapril. J Pharmacol Exp Ther, 1995. 275(2): p. 854-63.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA