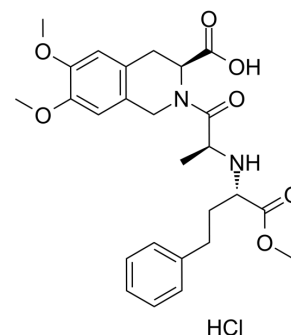


Moexipril hydrochloride

Cat. No.:	HY-B0378A
CAS No.:	82586-52-5
Molecular Formula:	C ₂₇ H ₃₅ ClN ₂ O 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)
H₂O : 100 mg/mL (186.91 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- Add each solvent one by one: PBS
Solubility: 50 mg/mL (93.45 mM); Clear solution; Need ultrasonic
- 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
- 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
- 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]}.

IC₅₀ & Target

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

<p>In Vitro</p>	<p>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 IC₅₀s 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 IC₅₀s 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.</p>																														
<p>In Vivo</p>	<p>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.</p> <table border="1" data-bbox="345 758 1515 1934"> <tbody> <tr> <td data-bbox="345 758 618 821">Animal Model:</td> <td data-bbox="618 758 1515 821">Spontaneously hypertensive rats^[3]</td> </tr> <tr> <td data-bbox="345 821 618 884">Dosage:</td> <td data-bbox="618 821 1515 884">30 mg/kg</td> </tr> <tr> <td data-bbox="345 884 618 947">Administration:</td> <td data-bbox="618 884 1515 947">Oral gavage; once daily; 5 days</td> </tr> <tr> <td data-bbox="345 947 618 1104">Result:</td> <td data-bbox="618 947 1515 1104"> <p>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.</p> <p>Dose-dependently decreased arterial blood pressure, and inhibited plasma and tissue ACE activity.</p> </td> </tr> <tr> <td data-bbox="345 1146 618 1209">Animal Model:</td> <td data-bbox="618 1146 1515 1209">Renal hypertensive rats^[3]</td> </tr> <tr> <td data-bbox="345 1209 618 1272">Dosage:</td> <td data-bbox="618 1209 1515 1272">0.03-10 mg/kg</td> </tr> <tr> <td data-bbox="345 1272 618 1335">Administration:</td> <td data-bbox="618 1272 1515 1335">Oral gavage; once daily; 5 days</td> </tr> <tr> <td data-bbox="345 1335 618 1409">Result:</td> <td data-bbox="618 1335 1515 1409"> <p>Caused a dose-dependent decrease in blood pressure with a threshold dose of 0.3 mg/kg.</p> <p>Lowered mean blood pressure by about 70 mmHg of 3 mg/kg.</p> </td> </tr> <tr> <td data-bbox="345 1451 618 1514">Animal Model:</td> <td data-bbox="618 1451 1515 1514">Perinephritic hypertensive dogs^[3]</td> </tr> <tr> <td data-bbox="345 1514 618 1577">Dosage:</td> <td data-bbox="618 1514 1515 1577">10 mg/kg</td> </tr> <tr> <td data-bbox="345 1577 618 1640">Administration:</td> <td data-bbox="618 1577 1515 1640">Oral gavage; once daily; 5 days</td> </tr> <tr> <td data-bbox="345 1640 618 1724">Result:</td> <td data-bbox="618 1640 1515 1724"> <p>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.</p> </td> </tr> <tr> <td data-bbox="345 1766 618 1829">Animal Model:</td> <td data-bbox="618 1766 1515 1829">NMRI mice (male, Permanent focal ischemia)^[4]</td> </tr> <tr> <td data-bbox="345 1829 618 1892">Dosage:</td> <td data-bbox="618 1829 1515 1892">0, 0.03, 0.3, and 3 mg/kg</td> </tr> <tr> <td data-bbox="345 1892 618 1934">Administration:</td> <td data-bbox="618 1892 1515 1934">Intraperitoneal injection (1 h before middle cerebral artery occlusion)</td> </tr> </tbody> </table>	Animal Model:	Spontaneously hypertensive rats ^[3]	Dosage:	30 mg/kg	Administration:	Oral gavage; once daily; 5 days	Result:	<p>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.</p> <p>Dose-dependently decreased arterial blood pressure, and inhibited plasma and tissue ACE activity.</p>	Animal Model:	Renal hypertensive rats ^[3]	Dosage:	0.03-10 mg/kg	Administration:	Oral gavage; once daily; 5 days	Result:	<p>Caused a dose-dependent decrease in blood pressure with a threshold dose of 0.3 mg/kg.</p> <p>Lowered mean blood pressure by about 70 mmHg of 3 mg/kg.</p>	Animal Model:	Perinephritic hypertensive dogs ^[3]	Dosage:	10 mg/kg	Administration:	Oral gavage; once daily; 5 days	Result:	<p>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.</p>	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)
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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.

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

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