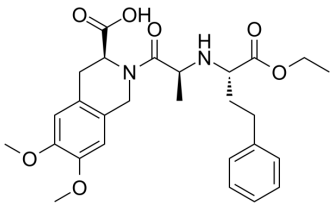
Moexipril

Cat. No.:	HY-117281
CAS No.:	103775-10-6
Molecular Formula:	$C_{27}H_{34}N_2O_7$
Molecular Weight:	498.57
Target:	Angiotensin-converting Enzyme (ACE); Apoptosis
Pathway:	Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Moexipril is an orally active inhibitor of angiotensin-converting enzyme (ACE), and becomes effective by being hydrolyzed to moexiprila (hydrochloride). Moexipril 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 is devoid of anti-inflammatory properties and has no effect on platelet function[2]. Moexipril 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 nM and 4.9 nM, respectively[2]. Moexipril (0.01 nM-0.1 mM) exhibits high potency against both ACE in rats plasma and purified ACE from rabbit lung, with IC_{50}s of 1.75 nM and 2.1 nM, respectively[3]. Moexipril (0-100 μM, 24 h) significantly reduced the percentage of damaged neurons in a dose-dependent manner[4]. Moexipril (0-100 μM, 24 h) significantly attenuates $Fe^{2+/3+}$-induced neurotoxicity[4]. Moexipril 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 can not cross the blood-brain barrier[1]. Moexipril (3 mg/kg, 30 mg/kg and 10 mg/kg; p.o.; once daily; 5 days) exhibits a dose-dependent and antihypertensive effects in renal hypertensive rats, spontaneously hypertensive rats and perinephritic hypertensive dogs, respectively[3]. Moexipril (0.3 mg/kg, i.p.) significantly reduces the infarct area on the mouse brain surface in NMRI mice[4]. Moexipril (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]. 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.

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

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

[4]. 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.

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

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