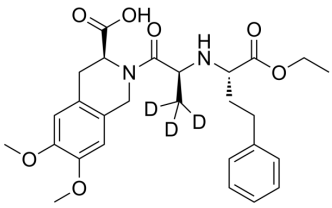
Moexipril-d_3

Cat. No.:	HY-117281S1
Molecular Formula:	$C_{27}H_{31}D_3N_2O_7$
Molecular Weight:	501.59
Target:	Apoptosis; Angiotensin-converting Enzyme (ACE); Isotope-Labeled Compounds
Pathway:	Apoptosis; Metabolic Enzyme/Protease; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Moexipril-d_3 is deuterated labeled Moexipril (HY-117281). 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].
In Vitro	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1]. Moexipril is devoid of anti-inflammatory properties and has no effect on platelet function[3]. 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[3]. 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[4]. Moexipril (0-100 μM, 24 h) significantly reduced the percentage of damaged neurons in a dose-dependent manner[5]. Moexipril (0-100 μM, 24 h) significantly attenuates $Fe^{2+/3+}$-induced neurotoxicity[5]. Moexipril dose not cause significant changes in the percentage of apoptotic neurons[5]. 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[2]. 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[4]. Moexipril (0.3 mg/kg, i.p.) significantly reduces the infarct area on the mouse brain surface in NMRI mice[5]. Moexipril (0.1 mg/kg, i.p.) significantly attenuates the cortical infarct volume in Long-Evans rats[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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.

[5]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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