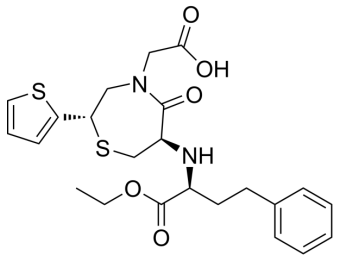
Temocapril

Cat. No.:	HY-100713
CAS No.:	111902-57-9
Molecular Formula:	$C_{23}H_{28}N_2O_5S_2$
Molecular Weight:	476.61
Target:	Angiotensin-converting Enzyme (ACE)
Pathway:	Metabolic Enzyme/Protease
Storage:	-20°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



BIOLOGICAL ACTIVITY

Description	Temocapril is an orally active angiotensin-converting enzyme (ACE) inhibitor. Temocapril can be used for the research of hypertension, congestive heart failure, acute myocardial infarction, insulin resistance, and renal diseases[1][2].
IC_{50} & Target	Angiotensin-converting Enzyme (ACE)[1]

In Vitro

Temocapril hydrochloride is a prodrug of the ACE inhibitor, Temocaprilat. Temocapril hydrochloride can be readily uptaken via the small intestine, and then be converted to its active metabolite (temocaprilat) by CES1 (human carboxylesterase 1) in the liver[1].

Temocapril hydrochloride (500 nM) reduces the inhibitory effects of RS (N-acetyltetradecapeptide renin substrate) and AngI (angiotensin) on neurogenic vasodilation in the spontaneously hypertensive rats (SHR)[2].

Temocapril hydrochloride (0.1-10 μM; 24 h) shows inductive effects on redox proteins thioredoxin (TRX) while no effect on antioxidant enzymes Cu/ZnSOD and Mn-SOD expressions[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	Cultured neonatal rat cardiomyocytes
Concentration:	0.1 μM, 1 μM, 10 μM
Incubation Time:	24 hours
Result:	Enhanced redox proteins thioredoxin (TRX) expression 1.9-fold at 10 μM without affecting TRX2, Cu/Zn-SOD or Mn-SOD protein expression.

In Vivo

Temocapril (10 mg/kg; p.o.; 21 d) enhances cardiomyocyte thioredoxin expression and ameliorates autoimmune myocarditis[3].

Temocapril (30 mg/kg; p.o.; daily; for 4 weeks) suppresses Angiotensin I-induced hypertension, plasma and renal ACE activity, but fails to reduce the level of Ang II in the kidney[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Experimental autoimmune myocarditis (EAM) rat model[3]
Dosage:	10 mg/kg

Administration:	Oral gavage; administration by water; 21 days
Result:	Ameliorated EAM and prevented cellular proteins from oxidation. Enhanced cardiomyocyte redox regulatory protein TRX expression.

Animal Model:	Male Sprague Dawley rats[4]
Dosage:	30 mg/kg
Administration:	Oral gavage, daily, for 4 weeks
Result:	Suppressed the blood pressure elevation induced by Ang I.

REFERENCES

[1]. Fukami T, et al. In vitro evaluation of inhibitory effects of antidiabetic and antihyperlipidemic drugs on human carboxylesterase activities. Drug Metab Dispos. 2010 Dec;38(12):2173-8.

[2]. Kawasaki H, et al. Angiotensin inhibits neurotransmission of calcitonin gene-related peptide-containing vasodilator nerves in mesenteric artery of spontaneously hypertensive rats. J Pharmacol Exp Ther. 1998 Feb;284(2):508-15.

[3]. Yuan Z, et al. Temocapril treatment ameliorates autoimmune myocarditis associated with enhanced cardiomyocyte thioredoxin expression. Cardiovasc Res. 2002 Aug 1;55(2):320-8.

[4]. Ohnishi K, et al. Angiotensin-converting enzyme inhibitor does not suppress renal angiotensin II levels in angiotensin I-infused rats. J Pharmacol Sci. 2013;122(2):103-8.

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