Perindoprilat

Cat. No.: HY-B1433

CAS No.: 95153-31-4

Molecular Formula: $C_{17}H_{28}N_2O_5$

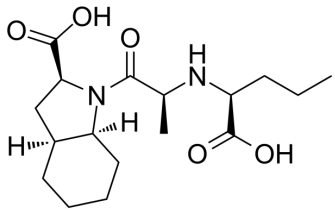
Molecular Weight: 340.41

Target: Angiotensin-converting Enzyme (ACE)

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 250 mg/mL (734.41 mM; Need ultrasonic)

DMSO : 100 mg/mL (293.76 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Concentration / Solvent / Mass	1 mg	5 mg	10 mg
1 mM	2.9376 mL	14.6882 mL	29.3763 mL
5 mM	0.5875 mL	2.9376 mL	5.8753 mL
10 mM	0.2938 mL	1.4688 mL	2.9376 mL

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

BIOLOGICAL ACTIVITY

Description

Perindoprilat (S 9780) is an angiotensin-converting enzyme (ACE) inhibitor with the IC_{50} value ranging from 1.5 to 3.2 nM. Perindoprilat can be used in hypertension research[1][2].

In Vitro

Perindoprilat (1 μM, 10 days) treatment suppresses the angiotensin II production in HNSCC cells[2].Perindoprilat (40 μM, 3 days) treatment attenuates mesangial cell fibronectin level[3].

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

Cell Viability Assay[2]

Cell Line:	HNSCC cells
Concentration:	1 μM
Incubation Time:	10 days
Result:	Suppressed the angiotensin II production in HNSCC cells (P=0.028).

Cell Viability Assay[3]

Cell Line:	Human mesangial cells
Concentration:	40 μM
Incubation Time:	3 days
Result:	Resulted in decreases in MPCM-stimulated fibronectin levels of 19.4±0.6% (P<0.001) and 21.7±1.0% (P<0.001) for secreted and cell-associated fibronectin levels, respectively.

In Vivo

Perindoprilat (oral gavage; 1.5 mg/kg; once daily; 7 d) treatment improves cardiac function in mice with acute myocardial infarction and reduces the number of apoptotic myocardial cells[4].
Perindoprilat (oral gavage; 1.5 mg/kg; once daily; 7 d) treatment reduces the expression levels of myocardial Bax and Bcl-2 in infarction zones of mice with acute myocardial infarction[4].
Perindoprilat (oral gavage; 1.5 mg/kg; once daily; 7 d) treatment lowers the expression of myocardial TLR4/NF-κB in infarction zones in mice with acute myocardial infarction[4].

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

Animal Model:	C57BL/6J mice underwent coronary ligation[4]
Dosage:	1.5 mg/kg
Administration:	Oral gavage; 1.5 mg/kg; once daily; 7 days
Result:	Exhibited markedly lowered the number of apoptotic myocardial cells in comparison with the acute myocardial infarction group (p<0.05).

Animal Model:	C57BL/6J mice underwent coronary ligation[4]
Dosage:	1.5 mg/kg
Administration:	Oral gavage; 1.5 mg/kg; once daily; 7 days
Result:	Reduced the gene and protein expression levels of Bax (a myocardial apoptosis gene) in infarction zones in mice with acute myocardial infarction.

Animal Model:	C57BL/6J mice underwent coronary ligation[4]
Dosage:	1.5 mg/kg
Administration:	Oral gavage; 1.5 mg/kg; once daily; 7 days
Result:	Declined the number of stained NF-κB p50 protein in the nucleus in infarction zones (p<0.05), compared to the acute myocardial infarction group.

REFERENCES

[1]. Perindopril. Expert Opin Pharmacother. 2006 Jan;7(1):63-71.

[2]. Angiotensin-converting enzyme (ACE) inhibitors have different selectivity for bradykinin binding sites of human somatic ACE. Eur J Pharmacol. 2007 Dec 22;577(1-3):1-6.

[3]. Izabella Z A Pawluczyk, et al. The role of bradykinin in the antifibrotic actions of perindoprilat on human mesangial cells. Kidney Int. 2004 Apr;65(4):1240-51.

[4]. X-Z Wang, et al. Perindopril inhibits myocardial apoptosis in mice with acute myocardial infarction through TLR4/NF-κB pathway. Eur Rev Med Pharmacol Sci. 2019

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