Captopril

Cat. No.: HY-B0368

CAS No.: 62571-86-2

Molecular Formula: $C_9H_{15}NO_3S$

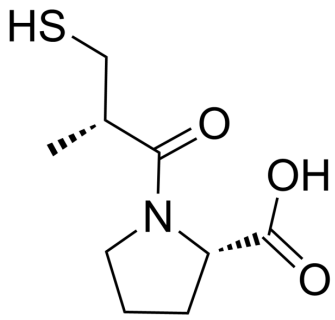
Molecular Weight: 217.29

Target: Angiotensin-converting Enzyme (ACE)

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (230.11 mM)

H_2O : ≥ 50 mg/mL (230.11 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.6021 mL	23.0107 mL	46.0214 mL
5 mM	0.9204 mL	4.6021 mL	9.2043 mL
10 mM	0.4602 mL	2.3011 mL	4.6021 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 32.5 mg/mL (149.57 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (11.51 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.51 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.51 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Captopril (SQ 14225), antihypertensive agent, is a thiol-containing competitive, orally active angiotensin-converting enzyme (ACE) inhibitor (IC_{50}=0.025 μM) and has been widely used for research of hypertension and congestive heart failure. Captopril is also a New Delhi metallo-β-lactamase-1 (NDM-1) inhibitor with an IC_{50} of 7.9 μM[1][2][3].

IC_{50} & Target	ACE[1].
In Vitro	Captopril (SQ 14225) has been shown to have similar morbidity and mortality benefits to those of diuretics and beta-blockers in hypertensive patients. Captopril has been shown to delay the progression of diabetic nephropathy, and enalapril and lisinopril prevent the development of nephropathy in normoalbuminuric patients with diabetes[4]. An equimolar ratio of the cis and trans states of Captopril exists in solution and that the enzyme selects only the trans state of the inhibitor that presents architectural and stereoelectronic complementarity with its substrate binding groove[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2023 May 2;14(1):2523.
- Phytomedicine. 2024 Feb 19, 155467.
- Phytomedicine. 2023 Sep 23, 155118.
- Eur J Med Chem. 13 January 2022, 114121.
- Mar Drugs. 2023 Sep 29, 21(10), 522.

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REFERENCES

[1]. Tzakos, A.G., et al., The molecular basis for the selection of captopril cis and trans conformations by angiotensin I converting enzyme. Bioorg Med Chem Lett, 2006. 16(19): p. 5084-7.

[2]. Song, J.C. and C.M. White, Clinical pharmacokinetics and selective pharmacodynamics of new angiotensin converting enzyme inhibitors: an update. Clin Pharmacokinet, 2002. 41(3): p. 207-24.

[3]. Afrin S, et al. Eritadenine from Edible Mushrooms Inhibits Activity of Angiotensin Converting Enzyme in Vitro. J Agric Food Chem. 2016;64(11):2263-2268.

[4]. Esmaeili S, et al. Captopril/enalapril inhibit promiscuous esterase activity of carbonic anhydrase at micromolar concentrations: An in vitro study. Chem Biol Interact. 2017;265:24-35.

[5]. Li N, et al. Simplified captopril analogues as NDM-1 inhibitors. Bioorg Med Chem Lett. 2014;24(1):386-389.

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