

Product Data Sheet

Product Name: Captopril

Cat. No.: GC16223

Chemical Properties

Cas. No. 62571-86-2

Chemical Name (2S)-1-[(2S)-2-methyl-3-sulfanylpropanoyl]pyrrolidine-2-carboxylic acid

SMILES CC(CS)C(=O)N1CCCC1C(=O)[O-]

Formula $C_9H_{15}NO_3S$ M.Wt 217.3

Solubility ≥ 21.7 mg/mL in DMSO, ≥ 105.2 mg/mL in EtOH with ultrasonic, ≥ 48.6 mg/mL in Water with ultrasonic
Storage Store at $-20^{\circ}C$

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

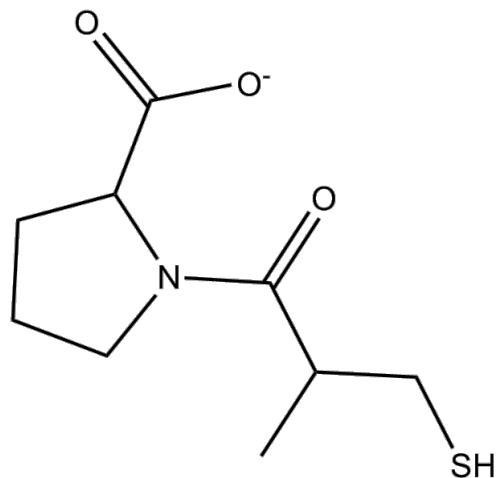
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines BV2 microglia

Preparation Method BV2 microglia were incubated in 24-well plates in the presence of 7ng/ml LPS with Captopril (0.1, 0.3, 1 or 3mM) for 24h. Thereafter, supernatants were analyzed for nitrite levels using the Griess reaction. Nitrite levels were normalized to cell counts.

Reaction 0.1, 0.3, 1, and 3mM; 24h
Conditions

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Applications Treatment of BV2 cells with 7ng/ml LPS and low doses of Captopril (0.3 and 1mM) resulted in 50% and 45% increased NO production, respectively, as compared to cells induced with LPS alone. Higher dose of Captopril (3mM) reduced LPS-induced NO levels in the BV2 microglial cell line by more than 40%.

Animal experiment [2]:

Animal models BALB/c mice

Preparation Method Mice were pretreated with Captopril (25 or 50mg/kg; i.p.) for 7 days, then received PTZ (60mg/kg, i.p.) 30min after the last dose. The animals were sacrificed 4h later, and brain sections were stained with Toluidine blue to assess dark neurons in hippocampal regions.

Dosage form 25, 50mg/kg; 7 days; i.p.

Applications Both the 25 mg/kg and 50 mg/kg doses of Captopril significantly prevented dark neuron formation in CA1, CA2, and CA3 regions of the hippocampus and DG after PTZ induced seizures.

References:

- [1] Asraf K, Torika N, Apte R N, et al. Microglial activation is modulated by captopril: in vitro and in vivo studies[J]. *Frontiers in Cellular Neuroscience*, 2018, 12: 116.
- [2] Tastemur Y, Gumus E, Ergul M, et al. Positive effects of angiotensin-converting enzyme (ACE) inhibitor, captopril, on pentylene-tetrazole-induced epileptic seizures in mice[J]. *Tropical Journal of Pharmaceutical Research*, 2020, 19(3): 637-643.

Background

Captopril is an orally active angiotensin-converting enzyme (ACE) inhibitor with an IC₅₀ value of 0.025μM^[1]. Captopril exerts its effects by blocking the conversion of angiotensin I to angiotensin II, thereby achieving vasodilation, lowering blood pressure, and reducing cardiac workload^[2]. Captopril

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is commonly used in the treatment and research of hypertension, heart failure, and diabetic nephropathy^[3,4].

In vitro, BV2 microglial cells were incubated with Captopril (0.1, 0.3, 1, and 3mM) and lipopolysaccharide (LPS, 7ng/mL) for 24h. Low concentrations of Captopril (0.3, 1mM) enhanced LPS-induced NO production, whereas a high concentration of Captopril (3mM) inhibited LPS-induced NO production^[5].

In vivo, normal ICR mice were treated with Captopril (10mg/kg) by gavage for 8 weeks, resulting in decreased mean arterial pressure and serum calcium levels, as well as increased urinary calcium excretion^[6]. BALB-c mice were pretreated with Captopril (25, 50mg/kg) via intraperitoneal injection for 7 days. On day 7, 30min after the last dose, pentylenetetrazole (PTZ, 60mg/kg) was injected to induce epileptic seizures. Captopril treatment significantly reduced the formation of dark neurons in the hippocampal CA1, CA2, CA3 regions and the dentate gyrus (DG)^[7].

References:

- [1] Afrin S, Rakib M A, Kim B H, et al. Eritadenine from edible mushrooms inhibits activity of angiotensin converting enzyme in vitro[J]. Journal of Agricultural and Food Chemistry, 2016, 64(11): 2263-2268.
- [2] Bolterman R J, Manriquez M C, Ruiz M C O, et al. Effects of captopril on the renin angiotensin system, oxidative stress, and endothelin in normal and hypertensive rats[J]. Hypertension, 2005, 46(4): 943-947.
- [3] Atkinson A B, Robertson J I S. Captopril in the treatment of clinical hypertension and cardiac failure[J]. The Lancet, 1979, 314(8147): 836-839.
- [4] Heel R C, Brogden R N, Speight T M, et al. Captopril: a preliminary review of its pharmacological properties and therapeutic efficacy[J]. Drugs, 1980, 20(6): 409-452.
- [5] Asraf K, Torika N, Apte R N, et al. Microglial activation is modulated by captopril: in vitro and in vivo studies[J]. Frontiers in Cellular Neuroscience, 2018, 12: 116.
- [6] Yang M, Xia C, Song Y, et al. Impairing effects of angiotensin-converting enzyme inhibitor captopril on bone of normal mice[J]. European Journal of Pharmacology, 2016, 771: 40-47.
- [7] Tastemur Y, Gumus E, Ergul M, et al. Positive effects of angiotensin-converting enzyme (ACE) inhibitor, captopril, on pentylenetetrazole-induced epileptic seizures in mice[J]. Tropical Journal of Pharmaceutical Research, 2020, 19(3): 637-643.

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