Sacubitril/Valsartan

Cat. No.: HY-18204A

CAS No.: 936623-90-4

Molecular Formula: $C_{48}H_{55}N_6Na_3O_8 \cdot 2.5H_2O$

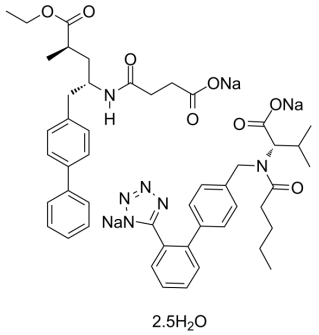
Molecular Weight: 957.99

Target: Angiotensin Receptor; Neprilysin; Apoptosis

Pathway: GPCR/G Protein; Metabolic Enzyme/Protease; Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (104.39 mM)

H_2O : ≥ 50 mg/mL (52.19 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.0439 mL	5.2193 mL	10.4385 mL
	5 mM	0.2088 mL	1.0439 mL	2.0877 mL
	10 mM	0.1044 mL	0.5219 mL	1.0439 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (104.39 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 (2.61 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.61 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.61 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sacubitril/Valsartan (LCZ696), comprised Valsartan and Sacubitril (AHU377) in 1:1 molar ratio, is a first-in-class, orally bioavailable, and dual-acting angiotensin receptor-neprilysin (ARN) inhibitor for hypertension and heart failure[1][2][3]. Sacubitril/Valsartan ameliorates diabetic cardiomyopathy by inhibiting inflammation, oxidative stress and apoptosis[4].

IC_{50} & Target

Angiotensin receptor-neprilysin[1]

In Vitro

Sacubitril/Valsartan (LCZ696; 1-30 μM; 0.5 hours) inhibits HG-treated H9C2 cells apoptosis in an experimental model of Diabetic cardiomyopathy (DCM)[4].
Sacubitril/Valsartan (1-30 μM; 0.5 hours) increases the expression level of cleaved caspase-3 and the ratio of Bax/Bcl-2 in HG-treated H9C2 cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[4]

Cell Line:	HG-treated H9C2 cells
Concentration:	1, 10, or 30 μM
Incubation Time:	0.5 hours
Result:	Inhibited HG-treated H9C2 cells apoptosis.

Western Blot Analysis[4]

Cell Line:	HG-treated H9C2 cells
Concentration:	1, 10, or 30 μM
Incubation Time:	0.5 hours
Result:	Increased the expression level of cleaved caspase-3 and the ratio of Bax/Bcl-2.

In Vivo

Sacubitril/Valsartan (LCZ696; perorally; 68 mg/kg for 4 weeks) significantly exhibits small weights and reduces interstitial fibrosis both in the noninfarct zone and peri-infarct zone[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult 6- to 8-week-old male Sprague-Dawley rats (220-250 g body weight) [2]
Dosage:	68 mg/kg
Administration:	Perorally; for 4 weeks
Result:	Exhibited small weights and reduced interstitial fibrosis both in the noninfarct zone and peri-infarct zone.

CUSTOMER VALIDATION

- Oxid Med Cell Longev. 2020 Sep 17;2020:9815039.
- ESC Heart Fail. 2022 Oct 17.
- Front Pharmacol. 2021 Sep 2;12:724147.
- Exp Biol Med (Maywood). 2019 Sep;244(12):1028-1039.
- Clin Exp Pharmacol Physiol. 2022 May 20.

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REFERENCES

[1]. Gu J, et al. Pharmacokinetics and pharmacodynamics of LCZ696, a novel dual-acting angiotensin receptor-neprilysin inhibitor (ARNi). J Clin Pharmacol. 2010

Apr;50(4):401-14.

[2]. von Lueder TG, et al. Angiotensin receptor neprilysin inhibitor LCZ696 attenuates cardiac remodeling and dysfunction after myocardial infarction by reducing cardiac fibrosis and hypertrophy. Circ Heart Fail. 2015 Jan;8(1):71-8.

[3]. Huo H, et al. Erastin Disrupts Mitochondrial Permeability Transition Pore (mPTP) and Induces Apoptotic Death of Colorectal Cancer Cells. PLoS One. 2016 May 12;11(5):e0154605.

[4]. Ge Q, et al. Feature article: LCZ696, an angiotensin receptor-neprilysin inhibitor, ameliorates diabeticcardiomyopathy by inhibiting inflammation, oxidative stress and apoptosis. Exp Biol Med (Maywood). 2019 Sep;244(12):1028-1039.

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