Candesartan

Cat. No.: HY-B0205

CAS No.: 139481-59-7

Molecular Formula: $C_{24}H_{20}N_6O_3$

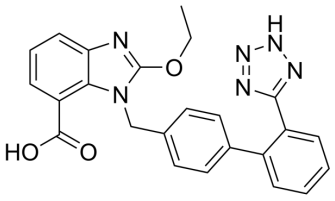
Molecular Weight: 440

Target: Angiotensin Receptor; PPAR

Pathway: GPCR/G Protein; Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (227.27 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent, Mass	1 mg	5 mg	10 mg
1 mM	2.2727 mL	11.3636 mL	22.7273 mL
5 mM	0.4545 mL	2.2727 mL	4.5455 mL
10 mM	0.2273 mL	1.1364 mL	2.2727 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Candesartan (CV 11974) is an orally active angiotensin II AT1-Receptor blocker and PPAR-γ agonist. Candesartan has potent and long-lasting antihypertensive effects. Candesartan can be used for the research of hypertension, chronic heart failure (CHF) and Traumatic brain injury (TBI)[1][2][3].

IC_{50} & Target		
	PPAR-γ	PPAR-γ

In Vivo

Candesartan (i.p.; 1?mg/kg/day; continuously for 1, 3 or 28 dpi) has neuroprotective effect, reducing neuronal injury, decreasing lesion volume and microglial activation, protecting CBF and improving functional behavior in a mouse model of

TBI[3].

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

Animal Model:	C57BL/6 mice (nine-week-old, male, 22–28 g)[3]
Dosage:	1 mg/kg
Administration:	i.p.; 1 mg/kg/day; continuously for 1, 3 or 28 dpi.
Result:	Reduced the lesion volume after CCI injury by approximately 50%, decreased the number of dying neurons, lessened the number of activated microglial cells, protected cerebral blood flow (CBF), and reduced the expression of the cytokine TGFß1 while increasing expression of TGFB3. Showed good motor skills on the rotarod 3 days after injury, and improved performance in the Morris water maze 4 weeks after injury.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2022 Nov 30.
- Free Radic Biol Med. 2023 Mar 1;S0891-5849(23)00094-1.
- Biomedicines. 2022, 10(12), 3131.
- Mol Med Rep. 2022 Oct;26(4):318.

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REFERENCES

[1]. Sonia Villapol, et al. Candesartan, an angiotensin II AT_1-receptor blocker and PPAR-γ agonist, reduces lesion volume and improves motor and memory function after traumatic brain injury in mice. Neuropsychopharmacology. 2012 Dec;37(13):2817-29.

[2]. Pfeffer MA, et al. Effects of candesartan on mortality and morbidity in patients with chronic heart failure: the CHARM-Overall programme. Lancet. 2003 Sep 6;362(9386):759-66.

[3]. Nishimura Y, et al. Chronic peripheral administration of the angiotensin II AT(1) receptor antagonist candesartan blocks brain AT(1) receptors. Brain Res. 2000 Jul 14;871(1):29-38.

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