Losartan potassium

Cat. No.: HY-17512A

CAS No.: 124750-99-8

Molecular Formula: $C_{22}H_{22}ClKN_6O$

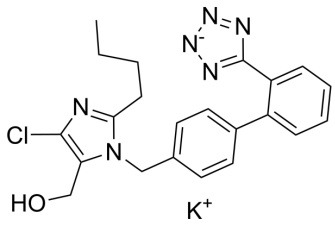
Molecular Weight: 461

Target: Angiotensin Receptor

Pathway: GPCR/G Protein

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

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 110 mg/mL (238.61 mM)

H_2O : 50 mg/mL (108.46 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1692 mL	10.8460 mL	21.6920 mL
	5 mM	0.4338 mL	2.1692 mL	4.3384 mL
	10 mM	0.2169 mL	1.0846 mL	2.1692 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 (216.92 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Losartan potassium (DuP-753 potassium) is an angiotensin II receptor type 1 (AT1) antagonist, competing with the binding of angiotensin II to AT1 with an IC_{50} of 20 nM.

IC_{50} & Target

AT1 Receptor

In Vitro

Losartan competes with the binding of angiotensin II to AT1 receptors. The concentration that inhibits 50% of the binding of angiotensin II (IC_{50}) is 20 nM[1]. Losartan (40 μM) affects I_{SC} but prevents the effect of ANGII on I_{SC}[2]. Losartan significantly reduces Ang II-mediated cell proliferation in endometrial cancer cells. The combination of losartan and anti-miR-155 has a significantly greater antiproliferative effect compared to each drug alone[3].

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

In Vivo

Losartan (0.6 g/L, p.o.) -treated $Fbn1^{C1039G/+}$ mice show a reduction in distal airspace caliber relative to placebo-treated

	$Fbn1^{C1039G/+}$ animals. The doses of losartan and propranolol are titrated to achieve comparable hemodynamic effects. Analysis of pSmad2 nuclear staining reveals that losartan antagonizes TGF-β signaling in the aortic wall of $Fbn1^{C1039G/+}$ mice. Losartan can improve disease manifestations in the lungs, an event that cannot plausibly relate to improved hemodynamics[4]. Losartan (10 mg/kg, intraarterial injection) increases blood angiotensin levels four- to sixfold. Losartan (10 mg/kg, i.p.) increases plasma renin levels 100-fold; plasma angiotensinogen levels decreases to 24% of control; and plasma aldosterone levels are unchanged[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	An MTT assay is used to measure cell proliferation and viability. For the assay, 5000 cells in 200 μL media per well are seeded in a 96 well plate. After overnight incubation to allow for cell attachment, the medium is removed by suction. MTT at 1 mg/mL concentration in serum-free medium is added and then incubated for 4 h at 37°C. After removal of MTT solution, 100 μL of DMSO is added to dissolve formazan crystals. Absorbance at 570 nm and at 600 nm as a reference is then measured using a microplate reader. The difference in absorbance is thus relative to the extent of cell survival. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Female $Fbn1^{C1039G/+}$ mice undergo timed matings with wild-type male mice. At 14.5d post-coitum, pregnant female $Fbn1^{C1039G/+}$ mice are treated with oral losartan (0.6 g/L in drinking water; n=10), propranolol (0.5 g/L; n=6) or placebo (n=12). Therapy is continued throughout lactation and after weaning until 10 months of age. Mice are sacrificed and examined using the techniques described above. Propranolol is used for comparison with losartan because β-adrenergic receptor blockade is the current albeit controversial standard of care to modulate abnormal growth of the aortic root in MFS. Beginning at 7 weeks of age, wild-type and $Fbn1^{C1039G/+}$ mice are treated with oral losartan (0.6 g/L in drinking water; n=5), propranolol (0.5 g/L; n=7) or placebo (n=10). Mice are continued on oral therapy for 6 months and then sacrificed. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Clin Transl Med. 2023 Mar;13(3):e1213.
- Cell Death Dis. 2020 May 22;11(5):390.
- Oncogene. 2023 Feb 23.
- Int J Nanomedicine. 2018 Nov 13;13:7409-7426.
- Phytomedicine. 2023 Feb 4;112:154700.

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REFERENCES

[1]. Burnier, M. Angiotensin II type 1 receptor blockers. Circulation, 2001. 103(6): p. 904-12.

[2]. Ashry, O., et al. Evidence for expression and function of angiotensin II receptor type 1 in pulmonary epithelial cells. Respir Physiol Neurobiol, 2014.

[3]. Choi, C.H., et al. Angiotensin II type I receptor and miR-155 in endometrial cancers: synergistic antiproliferative effects of anti-miR-155 and losartan on endometrial cancer cells. Gynecol Oncol, 2012. 126(1): p. 124-31.

[4]. Habashi, J.P., et al. Losartan, an AT1 antagonist, prevents aortic aneurysm in a mouse model of Marfan syndrome. Science, 2006. 312(5770): p. 117-21.

[5]. Campbell, D.J., et al. Effects of losartan on angiotensin and bradykinin peptides and angiotensin-converting enzyme. J Cardiovasc Pharmacol, 1995. 26(2): p. 233-40.

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

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