Valsartan

Cat. No.: HY-18204

CAS No.: 137862-53-4

Molecular Formula: $C_{24}H_{29}N_5O_3$

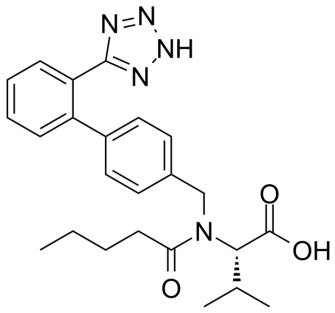
Molecular Weight: 435.52

Target: Angiotensin Receptor

Pathway: GPCR/G Protein

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 : 50 mg/mL (114.81 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2961 mL	11.4805 mL	22.9611 mL
5 mM	0.4592 mL	2.2961 mL	4.5922 mL
10 mM	0.2296 mL	1.1481 mL	2.2961 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 10 mg/mL (22.96 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 (5.74 mM); Suspended solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.74 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Valsartan (CGP 48933) is an angiotensin II receptor antagonist and has the potential for high blood pressure and heart failure research[1].

In Vitro

Valsartan (CGP 48933) is a synthetic non-peptide angiotensin II type 1 receptor antagonist that dilates blood vessels and reduces blood pressure by blocking the action of angiotensin. Valsartan significantly decreases the expression of AT1R in ageing aorta endothelial cells[1].

	The pretreatment of valsartan results in an inhibition of TLR2 signaling and proinflammatory cytokines. The expression of AGTR1 is up-regulated after alcohol exposure, and is blocked by valsartan pretreatment[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Valsartan (CGP 48933) significantly attenuates the expression of TGF-β/Smad, Hif-1α and fibrosis-related protein in rats after MI. Heart function, infarcted size, wall thickness as well as myocardial vascularization of ischaemic hearts are also significantly improved by valsartan compared with saline and hydralazine[3]. Valsartan partially reverses the effects of high-salt diet on hypertension, cardiac injuries such as fibrosis and inflammatory cell infiltration, and inhibition of aquaporin 1 and angiogenic factors; valsartan alone does not exert such effects[4]. Valsartan is an effective antidepressant/antianxiety reagent and can promote the hippocampal neurogenesis and expression of BDNF. Chronic administration of valsartan (5-40 mg/kg/d, p.o.) increases the time spent in the center of the field in OFT and the latency to eat in NSF, reduces the immobility time in both TST and FST, and increases the sucrose preference in SPT[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [4]	Rats: Rats are randomly divided into two groups: (i) valsartan-treated group that is given intravenously 3 mg/kg/day valsartan in 0.5 mL normal saline via the vein daily for 1 week; (ii) hydralazine-treated group receiving 0.2 mg/kg/day hydralazine injection in saline; and (iii) control group that receives saline injection in the same way (n=15 for each group)[4]. Mice: Valsartan is dissolved in water containing 0.5% methylcellulose solution. Valsartan (5-40 mg/kg/d) is administered by oral (p.o.) route in a volume of 10 mL/kg body weight using the gavage technique. Potential alteration in blood pressure in response to chronic treatment with valsartan is assessed with a commercial blood pressure analysis systemdesigned. The mice are trained for at least 2 consecutive days to adapt to the apparatus before the study is initiated. To record the blood pressure, the mice are placed on a heated pad (35°C) and measured with a programmable tail-cuff sphygmomanometer in steady state. The average of 10 readings from each mouse is recorded[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2023 Sep 21;14(1):5891.
- Clin Transl Med. 2023 Mar;13(3):e1213.
- Phytomedicine. 2023 Mar 5.
- Int J Mol Sci. 2023 Feb 3;24(3):2960.
- Front Pharmacol. 2021 Sep 2;12:724147.

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REFERENCES

[1]. Shan H, et al. Valsartan ameliorates ageing-induced aorta degeneration via angiotensin II type 1 receptor-mediated ERK activity. J Cell Mol Med. 2014 Jun;18(6):1071-80.

[2]. Wang Y, et al. Valsartan blocked alcohol-induced, Toll-like receptor 2 signaling-mediated inflammation in human vascular endothelial cells. Alcohol Clin Exp Res. 2014 Oct;38(10):2529-40.

[3]. Sui X, et al. Novel mechanism of cardiac protection by valsartan: synergetic roles of TGF-β1 and HIF-1α in Ang II-mediated fibrosis after myocardial infarction. J Cell Mol Med. 2015 Aug;19(8):1773-82.

[4]. Jiang Y, et al. Cardioprotective effect of valsartan in mice with short-term high-salt diet by regulating cardiac aquaporin 1 and angiogenic factor expression. Cardiovasc Pathol. 2015 Jul-Aug;24(4):224-9.

[5]. Ping G, et al. Valsartan reverses depressive/anxiety-like behavior and induces hippocampal neurogenesis and expression of BDNF protein in unpredictable chronic mild stress mice. Pharmacol Biochem Behav. 2014 Sep;124:5-12.

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