Azilsartan

Cat. No.: HY-14914

CAS No.: 147403-03-0

Molecular Formula: $C_{25}H_{20}N_4O_5$

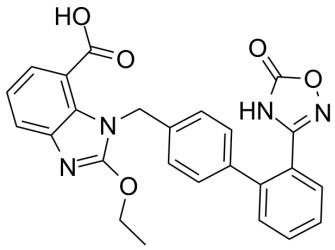
Molecular Weight: 456.45

Target: Angiotensin Receptor; Reactive Oxygen Species; Apoptosis

Pathway: GPCR/G Protein; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis

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 : 25 mg/mL (54.77 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1908 mL	10.9541 mL	21.9082 mL
5 mM	0.4382 mL	2.1908 mL	4.3816 mL
10 mM	0.2191 mL	1.0954 mL	2.1908 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 (5.48 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.48 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Azilsartan (TAK-536) is an orally active, potent, selective and specific angiotensin II type 1 receptor (AT1) antagonist. Azilsartan induces ROS formation and apoptosis in HepG2 cells. Azilsartan shows neuroprotective and anticancer activity. Azilsartan can be used for hypertension and stroke research[1][2][3][4][5].

IC_{50} & Target

AT1 Receptor

In Vitro

Azilsartan (0-200 μM, 0-72 h) decreases the viability of HepG2 cells[5].
Azilsartan (100 μM, 24 h) induces apoptosis in HepG2 cells[5].
Azilsartan inhibits the specific binding of ^{125}I-Sar^1-Ile^8-AII to human angiotensin type 1 receptors with an IC_{50} of 2.6 nM[3].

Azilsartan potently inhibits aortic endothelial and vascular cell proliferation in the absence of exogenous Ang II supplementation[5].
Azilsartan enhances adipogenesis and exerted greater effects than Valsartan (HY-18204) on expression of genes encoding peroxisome proliferator-activated receptor-α (PPARα), PPARδ, leptin, adipsin, and adiponectin[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[5]

Cell Line:	HepG2 and KDR cells
Concentration:	5, 25, 50, 100 and 200 μM
Incubation Time:	24, 48, and 72 h
Result:	Gradually decreased the viability of HepG2 cells by increasing the incubation time and dose, the inhibitory concentration of Azilsartan (IC 50%) against HepG2 cells was 100 μM for 24 h treatment time point while in KDR epithelial normal cells no significant cytotoxic effect was observed during the similar treatment conditions.

Apoptosis Analysis[5]

Cell Line:	HepG2 cells
Concentration:	100 μM
Incubation Time:	24 h
Result:	Induced 57.2% early and 0.52% late apoptosis respectively after 24 h.

In Vivo

Azilsartan (0-3 mg/kg, Oral gavage, once daily for 5 days) decreases SBP (systolic blood pressure) in obese Koletsky rats at 2 mg/kg[2].
Azilsartan (0-2 mg/kg, Oral gavage, once daily for 21 days) lowers blood pressure and basal plasma insulin concentration[2].
Azilsartan (2 and 4 mg/kg; PO, daily for 9 days) offers protection against ischemia induced secondary brain injury[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar-Kyoto (WKY) rats, obese Koletsky rats (n=6 per group)[2]
Dosage:	0, 1, 2 and 3 mg/kg
Administration:	Oral gavage, once daily (9:00-10:00 hours) for 5 days
Result:	Decreased SBP (systolic blood pressure) in obese Koletsky rats to that of normal rats at 2 mg/kg, whereas the 3 mg/kg dose elicited hypotension.

Animal Model:	Obese Koletsky rats (16, n = 8 per group)[2]
Dosage:	0 and 2 mg/kg
Administration:	Oral gavage, once daily (9:00-10:00 hours) for 21 days
Result:	Lowered blood pressure, basal plasma insulin concentration and the homeostasis model assessment of insulin resistance index, and inhibited over-increase of plasma glucose and insulin concentrations during oral glucose tolerance test.

Animal Model:	Male Wistar Rats (240–280 g)[4]

Dosage:	0, 2, and 4 mg/kg
Administration:	Orally, daily for 9 days, starting 7 days before the day of surgery
Result:	Individual treatments with Azilsartan (2 & 4 mg/kg) and Coenzyme Q10 (HY-N0111) (20 & 40 mg/kg) significantly attenuated the reduction in locomotor activity. Further, combination treatment with azilsartan (2 mg/kg) and Coenzyme Q10 (20 mg/kg) significantly improved the locomotor activity of animals as compared to their effects per se in BCCAO treated animals.

CUSTOMER VALIDATION

- EMBO Rep. 2022 Apr 11;e53932.
- J Lumin. 2018 Nov; 203;616-628.
- bioRxiv. 2020 Jun.
- Oncotarget. 2017 Apr 11;8(15):24099-24109.

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REFERENCES

[1]. Kajiya T, et al. Molecular and cellular effects of azilsartan: a new generation angiotensin II receptor blocker. J Hypertens. 2011 Dec;29(12):2476-83.

[2]. Zhao M, et al. Azilsartan treatment improves insulin sensitivity in obese spontaneously hypertensive Koletsky rats. Diabetes Obes Metab. 2011 Dec;13(12):1123-9.

[3]. Ojima M, et al. In vitro antagonistic properties of a new angiotensin type 1 receptor blocker, azilsartan, in receptor binding and function studies. J Pharmacol Exp Ther. 2011 Mar;336(3):801-8.

[4]. Gupta V, et al. Neuroprotective potential of azilsartan against cerebral ischemic injury: Possible involvement of mitochondrial mechanisms. Neurochem Int. 2020 Jan;132:104604.

[5]. Ahmadian E, et al. Novel angiotensin receptor blocker, azilsartan induces oxidative stress and NFkB-mediated apoptosis in hepatocellular carcinoma cell line HepG2. Biomed Pharmacother. 2018 Mar;99:939-946.

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