# Telmisartan

**Cat. No.:** HY-13955

**CAS No.:** 144701-48-4

**Molecular Formula:** $C_{33}H_{30}N_4O_2$

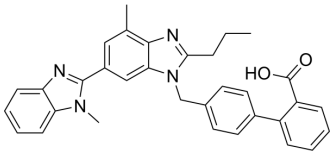
**Molecular Weight:** 514.62

**Target:** Angiotensin Receptor; Autophagy

**Pathway:** GPCR/G Protein; Autophagy

**Storage:**

| | | |
|---|---|---|
| Powder | -20°C | 3 years |
| | 4°C | 2 years |
| In solvent | -80°C | 1 year |
| | -20°C | 6 months |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 6.67 mg/mL (12.96 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 1.9432 mL | 9.7159 mL | 19.4318 mL |
| | 5 mM | 0.3886 mL | 1.9432 mL | 3.8864 mL |
| | 10 mM | 0.1943 mL | 0.9716 mL | 1.9432 mL |

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

**In Vivo**

1. Add each solvent one by one: 17% Polyethylene glycol 12-hydroxystearate in saline
   Solubility: 3.33 mg/mL (6.47 mM); Suspended solution; Need ultrasonic and warming
2. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 3 mg/mL (5.83 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.67 mg/mL (1.30 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.67 mg/mL (1.30 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

Telmisartan is a potent, long lasting antagonist of angiotensin II type 1 receptor (AT1), selectively inhibiting the binding of $^{125}$I-AngII to AT1 receptors with $IC_{50}$ of 9.2 nM.

**$IC_{50}$ & Target**

AT1 Receptor

| | |
|---|---|
| **In Vitro** | In intact RVSMC cells and in membrane preparations, telmisartan inhibits the binding of $^{125}$I-AngII to AT1 receptors in a concentration-dependent manner, with an $IC_{50}$ of 9.2 ± 0.8 nM. In the same experimental conditions, angiotensin II displaces $^{125}$I-AngII with an $IC_{50}$ value of 2.9 ± 0.5 nM. The specific binding of [$^3$H]telmisartan to SMC membranes is displaced by unlabeled telmisartan with an $IC_{50}$ of 7.7 ± 1.8 nM and by cold AngII with an $IC_{50}$ of 32.7 ± 5.7 nM [1]. Telmisartan treatment (100 μM) reduces the proliferation of three EAC cell lines (OE19, OE33, and SKGT-4), induces cell cycle arrest in G0/G1 phase and regulates cell cycle-related proteins in EAC cells, and induces the phosphorylation of AMPK and regulates cell cycle-related proteins via the AMPK/mTOR pathway in EAC cells. Telmisartan inhibits the activation of RTKs, downstream effectors and cell cycle-related proteins[5].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | In the telmisartan (0.1, 0.3, and 1 mg/kg)-treated rats, the specific binding of [$^3$H]telmisartan to the surface of living RVSMC is saturable and increases quickly to reach equilibrium within 1 h. Telmisartan dissociates very slowly from the receptor with a dissociation half-life (t1/2) of 75 min, which is comparable with candesartan and almost 5 times slower than angiotensin II (AngII). In vivo, telmisartan blunts the blood pressure response to exogenous AngII dose dependently[1]. Telmisartan (10 mg/kg/d) administration effectively suppresses aneurysm pathogenesis after PPE infusion as well, regardless of whether treatment is initiated before or after aneurysm creation or continues for a limited or extended time. Telmisartan treatment is associated with reduced messenger RNA levels for CCL5 and matrix metalloproteinases 2 and 9 in aneurysmal aortae, with no apparent effect on PPARγ-regulated gene expression[2]. Telmisartan (1 mg/kg/day) significantly ameliorates neuronal loss and the spatial acquisition impairment in 5XFAD mice, but without any changes of NeuN expression in the hippocampus layer. Telmisartan (1 mg/kg/day) treatment reduces amyloid burden and microglial accumulation in 5XFAD mice brain, induces microglial polarization towards neuroprotective phenotype, but does not alter the expression levels of NEP and IDE in 5XFAD mice specific brain areas[3]. Telmisartan (0.05, 0.1, 1 mg/kg, p.o.) shows significant reduction in immobility time, antagonizes depression and anxiety, and also significantly attenuates serum cortisol, NO, IL-6 and IL-1β in rats[4]. Telmisartan (50 μg, i.p.) leads to a 73.2% reduction in tumor growth in mice bearing xenografts derived from OE19 cells. Furthermore, miRNA expression is significantly altered by telmisartan in vivo[5].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## PROTOCOL

| | |
|---|---|
| **Cell Assay** [5] | Cell proliferation is assayed using the CCK-8 cell counting kit. Briefly, $5\times10^3$ cells are seeded into each well of a 96-well plate and cultured in 100 μL of RPMI-1640 supplemented with 10% FBS. After 24 h, ARBs (telmisartan, irbesartan, losartan, and valsartan at 0, 1, 10, or 100 μM) or vehicle is added to each well, and cells are cultured for an additional 48 h. CCK-8 reagent (10 μL) is added to each well, and the plates are incubated at 37°C for 3 h. The absorbance is measured at 450 nm using a microplate reader.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration** [5] | Male athymic mice (BALB/c-nu/nu; 6 weeks old; 20-25 g) are maintained under specific pathogen-free conditions using a laminar airflow rack. The mice have continuous free access to sterilized (γ-irradiated) food and autoclaved water. Each mouse is subcutaneously inoculated with OE19 cells ($5\times10^6$ cells per animal) in the flank. One week later, the xenografts are identifiable as masses with a maximal diameter > 4 mm. The animals are randomly assigned to treatment with telmisartan (50 μg per day) or diluent only (control). The telmisartan group is intraperitoneally (i.p.) injected five times per week with 2 mg/kg telmisartan for four weeks; the control group is administered 5% DMSO alone for four weeks. Tumor growth is monitored daily by the same investigators, and tumor size is measured weekly. The tumor volume ($mm^3$) is calculated as the tumor length (mm) × tumor width $(mm)^2/2$. All animals are sacrificed on day 22 after treatment, and all animals survive during this period. Between-group differences in tumor growth are analyzed by two-way ANOVA.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Int Immunopharmacol. 2023 Aug 4;123:110761.
- J Cell Mol Med. 2022 Feb 27.
- Int J Obes. 2020 Mar;44(3):697-706.
- Saudi Pharm J. 13 January 2022.
- Biosci Rep. 2018 Dec 14;38(6):BSR20181501.

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## REFERENCES


[1]. Maillard MP, et al. In vitro and in vivo characterization of the activity of telmisartan: an insurmountable angiotensin II receptor antagonist. J Pharmacol Exp Ther. 2002 Sep;302(3):1089-95.

[2]. Xuan H, et al. Inhibition or deletion of angiotensin II type 1 receptor suppresses elastase-induced experimental abdominal aortic aneurysms. J Vasc Surg. 2017 Apr 20. pii: S0741-5214(17)30100-3.

[3]. Torika N, et al. Intranasal telmisartan ameliorates brain pathology in five familial Alzheimer's disease mice. Brain Behav Immun. 2017 Apr 3.

[4]. Aswar U, et al. Telmisartan attenuates diabetes induced depression in rats. Pharmacol Rep. 2017 Apr;69(2):358-364.

[5]. Fujihara S, et al. The angiotensin II type 1 receptor antagonist telmisartan inhibits cell proliferation and tumor growth of esophageal adenocarcinoma via the AMPKα/mTOR pathway in vitro and in vivo. Oncotarget. 2017 Jan 31;8(5):8536-8549.



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