# Talfirastide

**Cat. No.:** HY-12403

**CAS No.:** 51833-78-4

**Molecular Formula:** $C_{41}H_{62}N_{12}O_{11}$

**Molecular Weight:** 899

**Sequence Shortening:** DRVYIHP

**Target:** Angiotensin Receptor; Angiotensin-converting Enzyme (ACE); Endogenous Metabolite

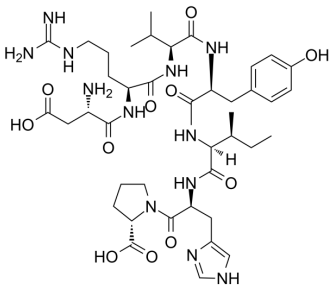
**Pathway:** GPCR/G Protein; Metabolic Enzyme/Protease

**Storage:** Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

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



## SOLVENT & SOLUBILITY

**In Vitro**

$H_2O$ : ≥ 30.2 mg/mL (33.59 mM)

* "≥" means soluble, but saturation unknown.

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 1.1123 mL | 5.5617 mL | 11.1235 mL |
| | 5 mM | 0.2225 mL | 1.1123 mL | 2.2247 mL |
| | 10 mM | 0.1112 mL | 0.5562 mL | 1.1123 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 (111.23 mM); Clear solution; Need ultrasonic

## BIOLOGICAL ACTIVITY

**Description**

Angiotensin 1-7 (Ang-(1-7)) is an endogenous heptapeptide from the renin-angiotensin system (RAS) with a cardioprotective role due to its anti-inflammatory and anti-fibrotic activities in cardiac cells. Angiotensin 1-7 inhibits purified canine ACE activity ($IC_{50}$=0.65 μM). Angiotensin 1-7 acts as a local synergistic modulator of kinin-induced vasodilation by inhibiting ACE and releasing nitric oxide. Angiotensin 1-7 blocks Ang II-induced smooth muscle cell proliferation and hypertrophy and shows antiangiogenic and growth-inhibitory effects on the endothelium. Angiotensin 1-7 shows anti-inflammatory activity [1][2][3].

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

AT1 Receptor

**In Vitro**

Angiotensin 1-7 (Ang-(1-7)) inhibits cultured vascular smooth muscle cell growth, whereas equal molar concentration of Ang

| | |
|---|---|
| | II stimulates cell growth[2].<br>?Angiotensin 1-7 (Ang 1-7) abrogates the methylglyoxal-modified albumin (MGA)-stimulated myofibroblast phenotype by inhibiting the chronic stimulation of the TGF-β-ERK pathway in NRK-52E cells[4].<br>?Angiotensin 1-7 signals through the Mas receptor ( MasR) in opposition to Ang II/angiotensin II type 1 receptor (AT1R), promoting anti-inflammatory,vasodilatory, and neuroprotective effects[5].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | Daily Angiotensin 1-7 (Ang-(1-7)) treatment (0.01-0.06 mg/kg) results in significant amelioration of DSS-induced colitis. Colitis-associated phosphorylation of p38, ERK1/2 and Akt is reduced by Ang 1-7 treatment[3].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## PROTOCOL

| | |
|---|---|
| **Kinase Assay** [1] | Competition assays using purified canine ACE are determined using a fixed concentration of the substrate Hip-His-Leu (1 mM) and varying the concentrations of the competing agents [Lisinopril (0.1 to 100 nM), Angiotensin (1-7) (10 nM to 10 μM), or $Sar^1$, $Thr^8$-Ang II (10 nM to 10 μM)]. Inhibitory constants ($IC_{50}$) are determined from the respective competition curves. To study the effect of Angiotensin (1-7) on BK metabolism in intact coronary rings, $^{125}$I-[$Tyr^0$]-BK (final concentration of 1 nM) is added to the tubes containing three rings preincubated with 1 mL Krebs' buffer and aerated with 95% $O_2$ and 5% $CO_2$ at 37°C. Lisinopril (2 μM), Angiotensin (1-7) (2 μM), or Krebs' buffer as control are added to the rings 10 minutes before addition of the radiolabeled BK. Aliquots of the incubation medium are removed at 5, 10, and 20 minutes and diluted with 1% HFBA to inhibit peptidase activity[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Cell Assay** [2] | 500 μM Methylglyoxal is incubated with 100 μM BSA dissolved in phosphate buffered saline (PBS) for 24 hours, then washed on 10 kDa filters to remove excess methyl glyoxal, reconstituted with DMEM/F12 serum free media and passed through a 0.2 μmicron filter. TGF-β (5 ng/mL) is prepared to treat cells in a subset of experiments. Cells are co-treated with one or combinations of the following: Angiotensin (1-7) (100 nM), D-Ala7-Ang-(1-7) (10 μM), ERK1/2 kinase inhibitor, PD 98059 (1 μM), TGF-β receptor kinase inhibitor; SB525334 (1 μM), the $AT_1$ receptor antagonist Losartan (1 μM), the renin inhibitor Aliskerin (1 μM) and the ACE inhibitor Lisinopril (1 μM)[2].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration** [3][4] | Mice[3]<br>Male and female BALB/c mice (1:1 ratio, 6-10 weeks old, mean weight 20 g.) are used. Angiotensin fragment 1-7 acetate salt hydrate (Ang 1-7) is dissolved in 0.9% saline (vehicle) at 1 mg/mL and stored at -80°C. Various doses (0.01, 0.06, 0.1, 0.3 and 1 mg/kg) are freshly prepared from the stock each day of the experiment, and administered to mice by daily intra-peritoneal (i.p) injections in a volume of 500 μL per injection, either before (prophylactic approach) or after (treatment approach) DSS treatment. A779 (MAS-1 R antagonist) is similarly dissolved in distilled water at 1 mg/mL and stored at -80°C. A freshly prepared dose of 1 mg/kg is administered to a second group of mice by daily i.p injections in a volume of 500 μL daily (for 4 days) along with colitis induction (prophylactic approach). A third group of mice receive DSS containing water and daily i.p injections of 0.9% saline (vehicle). The fourth group receive DSS containing water along with daily i.p injections with Dexamethasone (DEX) at doses of 0.01-1.0 mg/kg or its vehicle (0.9% saline) (prophylactic approach).<br>Rats[4]<br>Twenty six ovariectomized female Wistar rats weighing 200±20 g are used. Angiotensin (1-7) is administered intravenously by a microsyringe pump at two different continuous doses of 100 and 300 ng/kg/min after antagonist/saline infusion. Each dose is infused for 15 min; and MAP, RPP, and RBF are recorded during Angiotensin (1-7) infusion and the last 3-5 min of each dose measured as “response to Angiotensin (1-7) infusion”. During Angiotensin (1-7) infusion, RPP is sustained at pre-Ang1-7 infusion levels via an adjustable aortic clamp.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Chin Chem Lett. 2022 May 16.
- Cell Biosci. 2023 Feb 4;13(1):23.
- Biol Proced Online. 2022 Oct 25;24(1):15.
- Front Cell Dev Biol. 2021 Jun 11;9:659809.
- J Inflamm Res. 2024 Jan 23.

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## REFERENCES


[1]. Gómez-Mendoza DP, et al. Angiotensin-(1-7) oral treatment after experimental myocardial infarction leads to downregulation of CXCR4. J Proteomics. 2019;208:103486.

[2]. Li P, et al. Angiotensin-(1-7) augments bradykinin-induced vasodilation by competing with ACE and releasing nitric oxide. Hypertension. 1997 Jan;29(1 Pt 2):394-400.

[3]. Khajah MA, et al. Anti-Inflammatory Action of Angiotensin 1-7 in Experimental Colitis. PLoS One. 2016 Mar 10;11(3):e0150861.

[4]. Alzayadneh EM, et al. Angiotensin-(1-7) abolishes AGE-induced cellular hypertrophy and myofibroblast transformation via inhibition of ERK1/2. Cell Signal. 2014 Sep 19. pii: S0898-6568(14)00314-3.

[5]. Janatpour ZC, et al. Subcutaneous Administration of Angiotensin-(1-7) Improves Recovery after Traumatic Brain Injury in Mice. J Neurotrauma. 2019;36(22):3115-3131.





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