Talfirastide acetate

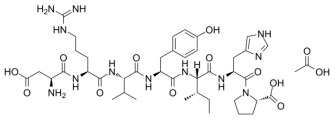
Cat. No.: HY-12403A

CAS No.: 2855063-75-9

Molecular Formula: $C_{43}H_{66}N_{12}O_{13}$

Molecular Weight: 959.06

Sequence: Asp-Arg-Val-Tyr-Ile-His-Pro



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 : 62.5 mg/mL (65.17 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.0427 mL	5.2134 mL	10.4269 mL
	5 mM	0.2085 mL	1.0427 mL	2.0854 mL
	10 mM	0.1043 mL	0.5213 mL	1.0427 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 (104.27 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Angiotensin 1-7 (Ang-(1-7)) acetate 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 acetate inhibits purified canine ACE activity (IC_{50}=0.65 μM). Angiotensin 1-7 acetate acts as a local synergistic modulator of kinin-induced vasodilation by inhibiting ACE and releasing nitric oxide. Angiotensin 1-7 acetate blocks Ang II-induced smooth muscle cell proliferation and hypertrophy and shows antiangiogenic and growth-inhibitory effects on the endothelium[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]. ?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]. ?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]. 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]. 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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