tcY-NH2 TFA

Cat. No.: HY-P1263A

CAS No.: 1262750-73-1

Molecular Formula: $C_{42}H_{50}F_3N_7O_9$

Molecular Weight: 853.88

Sequence Shortening: {trans-Cinnamoyl}-YPGKF-NH2

Target: Protease Activated Receptor (PAR)

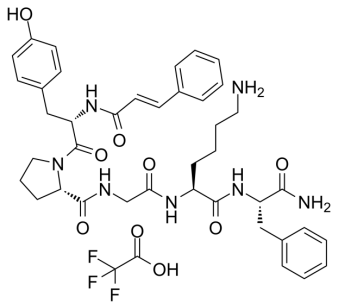
Pathway: GPCR/G Protein

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

DMSO : 100 mg/mL (117.11 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.1711 mL	5.8556 mL	11.7112 mL
	5 mM	0.2342 mL	1.1711 mL	2.3422 mL
	10 mM	0.1171 mL	0.5856 mL	1.1711 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: ≥ 2.08 mg/mL (2.44 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (2.44 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (2.44 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

tcY-NH2 ((trans-Cinnamoyl)-YPGKF-NH2) TFA is a potent selective PAR4 antagonist peptide. tcY-NH2 TFA inhibits thrombin- and AY-NH_2-induced platelet aggregation and endostatin release, and can be used in the research of inflammation, immunology[1][2][6].

IC_{50} & Target

PAR4

In Vitro

tcY-NH2 TFA (0-500 μM) inhibits AYPGKF-NH_2 (10 μM)-induced platelet (obtained from male albino Sprague–Dawley rats) aggregation, with an IC_{50} value of 95 μM[1].

tcY-NH2 TFA potently activates aorta relaxation (RA) and gastric (LM) contraction, with IC_{50} values of 64 μM (RA) and 1 μM (LM)[1].

tcY-NH2 TFA (Tc-YPGKF-NH_2, 400 μM, 5 min) prevents endostatin release and platelet aggregation induced by thrombin or by AY-NH_2[2].

tcY-NH2 TFA (5 μM, 15 min) decreases infarct size (IS) by 51%, and increases recovery of ventricular function by 26% in an isolated heart model[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

tcY-NH2 TFA (tail vein injection, 0.6 mg/kg for a single dose) alleviates liver injury in Brain death (BD) rat model, indicated by lower serum ALT/AST levels and better histomorphology[3].

tcY-NH2 TFA (intraperitoneal injection, 0.6 mg/kg for a single dose) increases posttraumatic activation of $CD4^+$ Tregs within the draining lymph nodes in burn injury mice model [4].

tcY-NH2 TFA (intrapleural injection, 40 ng/kg for a single dose) inhibits neutrophil recruitment in experimental inflammation in mice[6].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Brain death (BD) rat model[3]
Dosage:	0.6 mg/kg for a single dose
Administration:	Tail vein injection for a single dose
Result:	Reduced blood platelet activation and hepatic platelet accumulation. Attenuated the inflammatory response and apoptosis in the livers. Inhibited the activation of NF-κB and MAPK pathways induced by Brain death (BD).

Animal Model:	Burn injury model of C57BL/6 N mice[4]
Dosage:	0.6 mg/kg for a single dose
Administration:	Intraperitoneal injection
Result:	Increased expression and phosphorylation of PKC-θ in the presence of platelets, without affecting early posttraumatic hemostasis.

Animal Model:	BALB/c mice[6]
Dosage:	40 ng/kg for a single dose
Administration:	Intrapleural injection
Result:	Abolished the number of rolling and adhering neutrophils on the vessel wall. Inhibited CXCL8- and Cg-induced neutrophil migration into the pleural cavity of mice.

CUSTOMER VALIDATION

- Mol Nutr Food Res. 2022 May 1;e2200166.

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REFERENCES

[1]. Hongbo Fang, et al. Blocking protease-activated receptor 4 alleviates liver injury induced by brain death. Biochem Biophys Res Commun. 2022 Mar 5;595:47-53.

[2]. Matthias Bock, et al. Platelets differentially modulate CD4 + Treg activation via GPIIa/IIIb-, fibrinogen-, and PAR4-dependent pathways. Immunol Res. 2022 Apr;70(2):185-196.

[3]. Jennifer L Strande, et al. Inhibiting protease-activated receptor 4 limits myocardial ischemia/reperfusion injury in rat hearts by unmasking adenosine signaling. J Pharmacol Exp Ther. 2008 Mar;324(3):1045-54.

[4]. Lindisley F Gomides, et al. Blockade of proteinase-activated receptor 4 inhibits neutrophil recruitment in experimental inflammation in mice. Inflamm Res. 2014 Nov;63(11):935-41.

[5]. Morley D Hollenberg, et al. Proteinase-activated receptor-4: evaluation of tethered ligand-derived peptides as probes for receptor function and as inflammatory agonists in vivo. Br J Pharmacol. 2004 Oct;143(4):443-54.

[6]. L Ma, et al. Thrombin-induced platelet endostatin release is blocked by a proteinase activated receptor-4 (PAR4) antagonist. Br J Pharmacol. 2001 Oct;134(4):701-4.

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