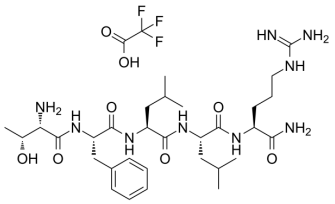
TFLLR-NH2(TFA)

Cat. No.:	HY-P0226A
CAS No.:	1313730-19-6
Molecular Formula:	$C_{33}H_{54}F_3N_9O_8$
Molecular Weight:	761.83
Sequence Shortening:	TFLLR-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 (131.26 mM; Need ultrasonic)

H_2O : 100 mg/mL (131.26 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.3126 mL	6.5631 mL	13.1263 mL
	5 mM	0.2625 mL	1.3126 mL	2.6253 mL
	10 mM	0.1313 mL	0.6563 mL	1.3126 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 33.33 mg/mL (43.75 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (3.28 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.28 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	TFLLR-NH2 (TFA) is a selective PAR1 agonist with an EC_{50} of 1.9 μM.
IC_{50} & Target	EC50: 1.9 μM (PAR1)[1]

In Vitro	PAR1 agonists stimulate concentration-dependent increases in $[Ca^{2+}]i$ and in the proportions of neurones. The maximal increase in $[Ca^{2+}]i$ above basal is detected in response to 10 μm TF-NH2 (peak 196.5±20.4 nM, n=25) when 50–80% of identified neurones responded[1]. SW620 cells cultured in the supernatant of TFLLR-NH2-activated platelets upregulate E-cadherin expression and downregulate the vimentin expression. In the in vitro platelet culture system, a TFLLR-NH2 dose-dependent increase of secreted TGF-β1 is detected in the supernatant[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Injection of TF-NH2 into the rat paw stimulates a marked and sustained oedema. An NK1R antagonist and ablation of sensory nerves with capsaicin inhibit oedema by 44% at 1 h and completely by 5 h. In wild-type but not $PAR1^{-/-}$ mice, TF-NH2 stimulates Evans blue extravasation in the bladder, oesophagus, stomach, intestine and pancreas by 2–8 fold. Extravasation in the bladder, oesophagus and stomach is abolished by an NK1R antagonist[1]. TFp-NH2 produces notable contraction at 3-50 μM and relaxation at 0.3-50 μM, in the absence of apamin. The concentration-response curve for TFp-NH2-induced contraction is remarkably shifted left, when the TFp-NH2-induced relaxation is blocked by apamin at 0.1 μM[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	Mice[1] Mice are anaesthetized with isofluorane, and saline or TF-NH2 (3 μmol/kg in 25 μL physiological saline) is injected into the lateral tail vein. Evans blue (33.3 mg/kg in 50 μL saline) is co-injected with the peptide. Mice are perfused transcardially at 10 min after administration of TF-NH2 with physiological saline containing 20 u/mL heparin at a pressure of 80-100 mmHg for 2-3 min. Excised tissues are incubated in 1 mL of formamide for 48 h, and Evans blue content is measured spectrophotometrically at 650 nm[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. de Garavilla L, et al. Agonists of proteinase-activated receptor 1 induce plasma extravasation by a neurogenic mechanism. Br J Pharmacol. 2001 Aug;133(7):975-87.

[2]. Kawabata A, et al. Characterization of the protease-activated receptor-1-mediated contraction and relaxation in the rat duodenal smooth muscle.

[3]. Jia Y, et al. Activation of platelet protease-activated receptor-1 induces epithelial-mesenchymal transition and chemotaxis of colon cancer cell line SW620. Oncol Rep. 2015 Jun;33(6):2681-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA