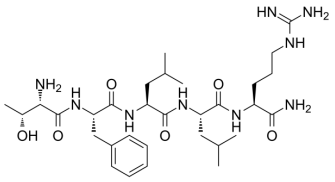
TFLLR-NH2

Cat. No.:	HY-P0226
CAS No.:	197794-83-5
Molecular Formula:	$C_{31}H_{53}N_9O_6$
Molecular Weight:	647.81
Sequence:	Thr-Phe-Leu-Leu-Arg-NH2
Sequence Shortening:	TFLLR-NH2
Target:	Protease Activated Receptor (PAR)
Pathway:	GPCR/G Protein
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	TFLLR-NH2 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: 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.

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