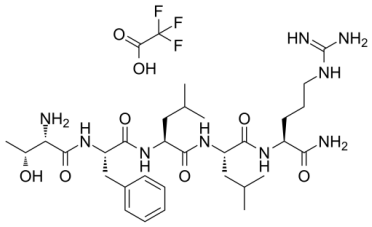
Data Sheet

Product Name:	TFLLR-NH2(TFA)
Cat. No.:	CS-0058948
CAS No.:	1313730-19-6
Molecular Formula:	C33H54F3N9O8
Molecular Weight:	761.83
Target:	Protease-Activated Receptor (PAR)
Pathway:	GPCR/G Protein
Solubility:	H2O : 100 mg/mL (131.26 mM; Need ultrasonic); DMSO : 100 mg/mL (131.26 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

TFLLR-NH2 (TFA) is a selective **PAR1** agonist with an **EC_{50}** of 1.9 μM. IC50 & 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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

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].

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.

CAIndexNames:

(S)-2-((S)-2-((2S,3R)-2-Amino-3-hydroxybutanamido)-3-phenylpropanamido)-N-((S)-1-(((S)-1-amino-5-guanidino-1-oxopentan-2-yl)amino)-4-methyl-1-

oxopentan-2-yl)-4-methylpentanamide 2,2,2-trifluoroacetate

SMILES:

N=C(N)NCCC[C@@H](C(N)=O)NC([C@H](CC(C)C)NC([C@H](CC(C)C)NC([C@H](CC1=CC=CC=C1)NC([C@H]([C@H](O)C)N)=O)=O)=O)=O.FC(C(O)=O)(F)F

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

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