Protease-Activated Receptor-1, PAR-1 Agonist TFA

Cat. No.: HY-P2518A

Molecular Formula: $C_{37}H_{59}F_3N_{10}O_{11}$

Molecular Weight: 876.92

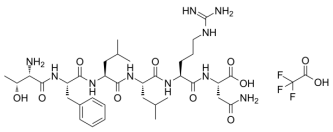
Sequence: Thr-Phe-Leu-Leu-Arg-Asn

Sequence Shortening: TFLLRN

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

H_2O : 6.67 mg/mL (7.61 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.1404 mL	5.7018 mL	11.4035 mL
	5 mM	0.2281 mL	1.1404 mL	2.2807 mL
	10 mM	---	---	---

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 5 mg/mL (5.70 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Protease-Activated Receptor-1, PAR-1 Agonist TFA is a selective proteinase-activated receptor1 (PAR-1) agonist peptide. Protease-Activated Receptor-1, PAR-1 Agonist TFA corresponds to PAR1 tethered ligand and which can selectively mimic theactions of thrombin via this receptor[1][2].

In Vitro

Protease-Activated Receptor-1, PAR-1 Agonist induces activation of protein kinase C isoenzymes alpha and epsilon in human HT-29 colon carcinoma cells expressing PAR1 endogeneously. On the cellular level, Protease-Activated Receptor-1, PAR-1 Agonist and thrombin prompted HT-29 cell migration and matrix adhesion by a PKCepsilon-dependent mechanism as concluded because of the inhibition of PAR1-mediated effects by the PKC inhibitor bisindolylmaleimide I and the PKCepsilon translocation inhibitory peptide EAVSLKPT but not by the PKC inhibitor Gö 6976[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Heider I, et al. PAR1-type thrombin receptor stimulates migration and matrix adhesion of human colon carcinoma cells by a PKCepsilon-dependent mechanism. Oncol Res. 2004;14(10):475-482.

[2]. Stefanie Gödecke, et al. Thrombin-induced ATP release from human umbilical vein endothelial cells. Am J Physiol Cell Physiol. 2012 Mar 15;302(6):C915-23.

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