PRODUCT INFORMATION

PAR1 (1-6) (mouse, rat) (trifluoroacetate salt)

Item No. 27111

Formal Name: L-seryl-L-phenylalanyl-L-phenylalanyl-L-leucyl-L-arginyl-L-asparagine, trifluoroacetate salt
Synonyms: H-Ser-Phe-Phe-Leu-Arg-Asn, PAR_1-AP, Proteinase-Activated Receptor 1, SFFLRN, TRAP, Thrombin Receptor Activating Peptide
MF: $C_{37}H_{54}N_{10}O_9 \bullet XCF_3COOH$
FW: 782.9
Purity: ≥95%
Supplied as: A solid
Storage: -20°C
Stability: ≥4 years

H— Ser— Phe — Phe—Leu —Arg— Asn—OH

• XCF_3COOH

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

PAR1 (1-6) (mouse, rat) (trifluoroacetate salt) is supplied as a solid. A stock solution may be made by dissolving the PAR1 (1-6) (mouse, rat) (trifluoroacetate salt) in water. The solubility of PAR1 (1-6) (mouse, rat) (trifluoroacetate salt) in water is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

PAR1 (1-6) is a hexapeptide that corresponds to amino acid residues 1-6 of the amino terminal tethered ligand sequence of mouse and rat proteinase-activated receptor 1 (PAR1), residues 42-47 of the full-length mouse sequence, and residues 46-51 of the full-length rat sequence.[1,2] PAR1 is a high-affinity thrombin receptor that induces platelet activation and deposition into thrombi as well as smooth muscle mitogenesis.[3] Unlike thrombin, PAR1 (1-6) acts as an agonist of PAR1 in smooth muscle cells but is inactive in rodent platelets.[4] It increases mitogenesis and the expression of the IGF-1 receptor (IGF-1R) by 47% in vascular smooth muscle cells (VSMCs), an effect that is inhibited by the thrombin inhibitor hirudin and by the protein tyrosine kinase inhibitor genistein (Item No. 10005167).[1] PAR1 (1-6) increases histamine and β-hexosaminidase release from rat peritoneal mast cells in a concentration-dependent manner, with the maximum release at a concentration of 50 µM.[2] It also mimics the effect of thrombin, increasing swelling-activated efflux of [^{3}H]glutamate induced by hypoosmotic medium in astrocytes *in vitro* when used at concentrations ranging from 5 to 10 µM.[5]

References

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3. Sidhu, T.S., French, S.L., and Hamilton, J.R. Differential signaling by protease-activated receptors: Implications for therapeutic targeting. *Int. J. Mol. Sci.* **15(4)**, 6169-6183 (2014).
4. Derian, C.K., Santulli, R.J., Tomko, K.A., *et al.* Species differences in platelet responses to thrombin and SFLLRN. Receptor-mediated calcium mobilization and aggregation, and regulation by protein kinases. *Thromb. Res.* **78(6)**, 505-519 (1995).
5. Ramos-Mandujano, G., Vázquez-Juárez, E., Hernández-Benítez, R., *et al.* Thrombin potently enhances swelling-sensitive glutamate efflux from cultured astrocytes. *Glia* **55(9)**, 917-925 (2007).

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