PRODUCT INFORMATION

PKI (14-22) amide (myristoylated) (trifluoroacetate salt)

Item No. 34431

Formal Name: N-(1-oxotetradecyl)glycyl-L-arginyl-L-threonylglycyl-L-arginyl-L-arginyl-L-asparaginyl-L-alanyl-L-isoleucinamide, trifluoroacetate salt

Synonyms: Myr-Gly-Arg-Thr-Gly-Arg-Arg-Asn-Ala-Ile-NH_2, Myr-GRTGRRNAI-NH_2, Myristoylated PKI-(14-22)-amide, PKI-(Myr-14-22)-amide

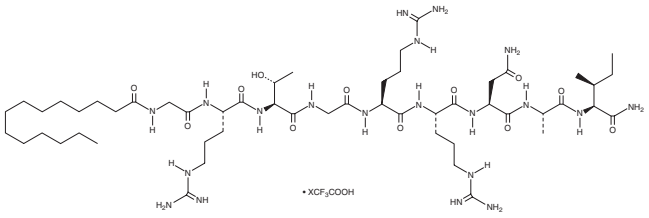
MF: $C_{53}H_{100}N_{20}O_{12} \bullet XCF_3COOH$

FW: 1,209.5

Supplied as: A solid

Storage: -20°C

Stability: ≥4 years



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

Laboratory Procedures

PKI (14-22) amide (myristoylated) (trifluoroacetate salt) is supplied as a solid. A stock solution may be made by dissolving the PKI (14-22) amide (myristoylated) (trifluoroacetate salt) in the solvent of choice, which should be purged with an inert gas. PKI (14-22) amide (myristoylated) (trifluoroacetate salt) is soluble in the organic solvent DMSO at a concentration of approximately 1 mg/ml.

PKI (14-22) amide (myristoylated) (trifluoroacetate salt) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, PKI (14-22) amide (myristoylated) (trifluoroacetate salt) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. PKI (14-22) amide (myristoylated) (trifluoroacetate salt) has a solubility of approximately 0.25 mg/ml in a 1:3 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

PKI (14-22) amide (myristoylated) is a cell-permeable peptide inhibitor of protein kinase A (PKA).[1-3] It inhibits PKA activity in mouse brain or spinal cord lysates when used at a concentration of 75 µM.[1] PKI (14-22) amide (myristoylated) reduces IgG-dependent phagocytosis of heat-killed *S. cerevisiae* by isolated human neutrophils in a concentration-dependent manner.[2] It inhibits the replication of several strains of Zika virus, including IbH 30656, MR766, H/FP/2013, and PRVABC59, in human umbilical vein endothelial cells (HUVECs; IC_{50}s = 17.75, 22.29, 34.09, and 19.19 µM, respectively).[3] PKI (14-22) amide (myristoylated) (2.5 nmol, i.c.v.) increases latency to tail withdrawal in the tail-flick test in morphine-tolerant mice.[1]

References

1. Dalton, G.D., Smith, F.L., Smith, P.A., *et al.* Alterations in brain Protein Kinase A activity and reversal of morphine tolerance by two fragments of native Protein Kinase A inhibitor peptide (PKI). *Neuropharmacology* **48(5)**, 648-657 (2005).
2. Ydrenius, L., Majeed, M., Rasmusson, B.J., *et al.* Activation of cAMP-dependent protein kinase is necessary for actin rearrangements in human neutrophils during phagocytosis. *J. Leukoc. Biol.* **67(4)**, 520-528 (2000).
3. Cheng, F., da Silva, S.R., Huang, I.-C., *et al.* Suppression of Zika virus infection and replication in endothelial cells and astrocytes by PKA inhibitor PKI 14-22. *J. Virol.* **92(4)**, e02019-17 (2018).

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