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- Expressversand

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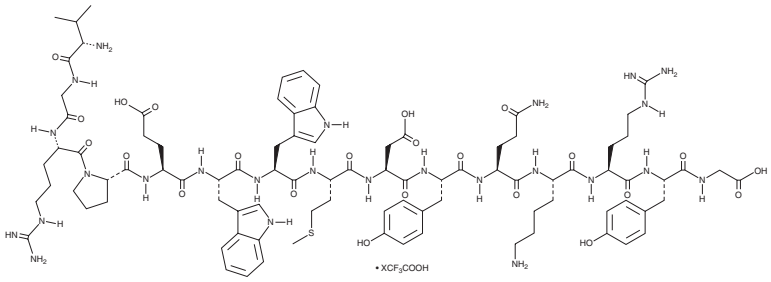
PRODUCT INFORMATION

BAM-22P (8-22) (human, mouse, rat, bovine) (trifluoroacetate salt)

Item No. 35263

Formal Name: L-valylglycyl-L-arginyl-L-prolyl-L-α-glutamyl-L-tryptophyl-L-tryptophyl-L-methionyl-L-α-aspartyl-L-tyrosyl-L-glutaminyl-L-lysyl-L-arginyl-L-tyrosyl-glycine, trifluoroacetate salt
Synonyms: BAM 8-22, Bovine Adrenal Medulla 8-22
MF: $C_{91}H_{127}N_{25}O_{23}S \bullet XCF_3COOH$
FW: 1,971.2
Purity: ≥95%
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

BAM-22P (8-22) (human, mouse, rat, bovine) (trifluoroacetate salt) is supplied as a solid. A stock solution may be made by dissolving the BAM-22P (8-22) (human, mouse, rat, bovine) (trifluoroacetate salt) in water. The solubility of BAM-22P (8-22) (human, mouse, rat, bovine) (trifluoroacetate salt) in water is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

BAM-22P (8-22) is an endogenous neuropeptide derived from proenkephalin A and an agonist of MAS-related G protein-coupled receptor family member X1 (MRGPRX1), previously known as sensory neuron-specific G protein-coupled receptor 4 (SNSR4).[1] It is involved in prurition and nociception. BAM-22P (8-22) induces calcium mobilization in HEK293S cells expressing human MRGPRX1 or SNSR3 (EC_{50}s = 14 and 28 nM, respectively) and inhibits voltage-induced calcium currents in isolated rat neurons expressing human MRGPRX1 (EC_{50} = 0.6 µM).[2] Intrathecal administration of BAM-22P (8-22) (30 nmol/animal) decreases the tail-flick latency in rats stimulated with the $G\alpha_i$ inhibitor pertussin toxin and increases the tail-flick latency in rats stimulated with the phospholipase C (PLC) inhibitor U-73122, indicating changes in pain sensitivity.[3] BAM-22P (8-22) (100 µg/animal) increases scratching behavior in wild-type mice and enhances bile-duct ligation-induced scratching behavior in a mouse model of cholestasis.[4]

References

1. Lembo, P.M.C., Grazzini, E., Groblewski, T., *et al.* Proenkephalin A gene products activate a new family of sensory neuron-specific GPCRs. *Nat. Neurosci.* **5(3),** 201-209 (2002).
2. Chen, H. and Ikeda, S.R. Modulation of ion channels and synaptic transmission by a human sensory neuron-specific G-protein-coupled receptor, SNSR4/*mrg*X1, heterologously expressed in cultured rat neurons. *J. Neurosci.* **24(21),** 5044-5053 (2004).
3. Chen, T., Wang, D., and Hong, Y. Dual modulation effects of Mas-related gene (Mrg) receptors on pain sensitivity in rats. *Neurosci. Lett.* **514(1),** 82-85 (2012).
4. Sanjel, B., Maeng, H.-J., and Shim, W.-S. BAM8-22 and its receptor MRGPRX1 may attribute to cholestatic pruritus. *Sci. Rep.* **9(1),** 10888 (2019).

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