PRODUCT INFORMATION

Neuromedin U-25 (human) (trifluoroacetate salt)

Item No. 17617

Formal Name: L-phenylalanyl-L-arginyl-L-valyl-L-α-aspartyl-L-α-glutamyl-L-α-glutamyl-L-phenylalanyl-L-glutaminyl-L-seryl-L-prolyl-L-phenylalanyl-L-alanyl-L-seryl-L-glutaminyl-L-seryl-L-arginylglycyl-L-tyrosyl-L-phenylalanyl-L-leucyl-L-phenylalanyl-L-arginyl-L-prolyl-L-arginyl-L-aspartamide, trifluoroacetate salt

Synonym: NMU-25

MF: $C_{141}H_{203}N_{41}O_{38} \bullet XCF_3COOH$

FW: 3,080.4

Purity: ≥95%

Supplied as: A crystalline solid

Storage: -20°C

Stability: ≥4 years

Phe—Arg—Val—Asp—Glu—Glu—Phe—Gln—Ser—Pro—Phe—Ala—Ser—Gln—Ser—Arg—Gly—Tyr—Phe—Leu—Phe—Arg—Pro—Arg—Asn—NH_2

$\bullet XCF_3COOH$

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

Laboratory Procedures

Neuromedin U-25 (human) (trifluoroacetate salt) is supplied as a crystalline solid. A stock solution may be made by dissolving the neuromedin U-25 (human) (trifluoroacetate salt) in the solvent of choice. Neuromedin U-25 (human) (trifluoroacetate salt) is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of neuromedin U-25 (human) (trifluoroacetate salt) in these solvents is approximately 1 and 5 mg/ml, respectively.

Description

Neuromedin U (NMU) is a neuropeptide first demonstrated to drive smooth muscle contraction.[1] Translated as a 174 amino acid propeptide, NMU is cleaved to different lengths in different animals. It has diverse receptor-mediated roles *in vivo*, as it regulates feeding, vasoconstriction, nociception, and bone remodeling and contributes to obesity, cancer and septic shock.[1,2] NMU-25 is the active form of NMU in humans. It binds with high affinity to receptors on human left ventricle and coronary artery (K_Ds = 0.26 and 0.11 nM, respectively), eliciting endothelium-independent vasoconstriction.[3] NMU-25 also suppresses glucose-stimulated insulin secretion in human islets, and this effect is lost in NMU R165W mutants, resulting in early-onset obesity.[4]

References

1. Mitchell, J.D., Maguire, J.J., and Davenport, A.P. Emerging pharmacology and physiology of neuromedin U and the structurally related peptide neuromedin S. *Br. J. Pharmacol.* **158(1)**, 87-103 (2009).
2. Greenwood, H.C., Bloom, S.R., and Murphy, K.G. Peptides and their potential role in the treatment of diabetes and obesity. *Rev. Diabet. Stud.* **8(3)**, 355-368 (2011).
3. Mitchell, J.D., Maguire, J.J., Kuc, R.E., *et al*. Expression and vasoconstrictor function of anorexigenic peptides neuromedin U-25 and S in the human cardiovascular system. *Cardiovasc. Res.* **81(2)**, 353-361 (2009).
4. Alfa, R.W., Park, S., Skelly, K.R., *et al*. Suppression of insulin production and secretion by a decretin hormone. *Cell Metab.* **21(2)**, 323-333 (2015).

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