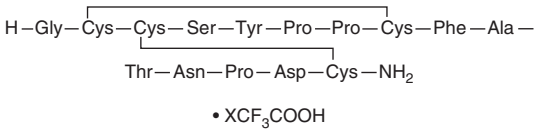
PRODUCT INFORMATION

α-Conotoxin AuIB (trifluoroacetate salt)

Item No. 33795

Formal Name: cyclic (2→8), (3→15)-*bis*(disulfide), glycyl-L-cysteinyl-L-cysteinyl-L-seryl-L-tyrosyl-L-prolyl-L-prolyl-L-cysteinyl-L-phenylalanyl-L-alanyl-L-threonyl-L-asparaginyl-L-prolyl-L-α-aspartyl-L-cysteinamide, trifluoroacetate salt



Synonym: GCCSYPPCFATNPDC

MF: $C_{65}H_{89}N_{17}O_{21}S_4 \bullet XCF_3COOH$

FW: 1572.8

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

α-Conotoxin AuIB (trifluoroacetate salt) is supplied as a solid. A stock solution may be made by dissolving the α-conotoxin AuIB (trifluoroacetate salt) in water. The solubility of α-conotoxin AuIB (trifluoroacetate salt) in water is approximately 5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

α-Conotoxin AuIB is a conotoxin that has been found in *C. aulicus* and has receptor antagonist and analgesic activity.[1] It is a peptide antagonist of α3β4 subunit-containing nicotinic acetylcholine receptors (nAChRs; IC_{50} = 0.75 µM). It is greater than 100-fold selective for α3β4 subunit-containing nAChRs over those containing α2β2, α2β4, α3β2, α4β2, α4β4, or α1β1γδ subunits but does inhibit homomeric α7 nAChRs by 34% at 3 µM. Intrathecal administration of α-conotoxin AuIB (0.2 and 2 nmol/animal) reduces mechanical allodynia in a rat model of neuropathic pain induced by partial sciatic nerve ligation.[2] It also reverses somatic signs of withdrawal in a mouse model of morphine withdrawal when administered intracerebroventricularly at doses of 1.75 and 3.5 pmol/animal.[3]

References

1. Luo, S., Kulak, J.M., Cartier, G.E., *et al.* α-Conotoxin AuIB selectively blocks α3 β4 nicotinic acetylcholine receptors and nicotine-evoked norepinephrine release. *J. Neurosci.* **18(21)**, 8571-8579 (1998).
2. Napier, I.A., Klimis, H., Rycroft, B.K., *et al.* Intrathecal α-conotoxins Vc1.1, AuIB and MII acting on distinct nicotinic receptor subtypes reverse signs of neuropathic pain. *Neuropharmacology* **62(7)**, 2202-2207 (2012).
3. Muldoon, P.P., Jackson, K.J., Perez, E., *et al.* The α3β4* nicotinic ACh receptor subtype mediates physical dependence to morphine: mouse and human studies. *Br. J. Pharmacol.* **171(16)**, 3845-3857 (2014).

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