# α-Conotoxin Vc1.1 TFA

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| **Cat. No.:** | HY-125777A |
| **Molecular Formula:** | $C_{73}H_{104}F_3N_{23}O_{27}S_4$ |
| **Molecular Weight:** | 1921 |
| **Sequence:** | Gly-Cys-Cys-Ser-Asp-Pro-Arg-Cys-Asn-Tyr-Asp-His-Pro-Glu-Ile-Cys-NH2 (Disulfide bridge:Cys2-Cys8;Cys3-Cys16) |
| **Sequence Shortening:** | GCCSDPRCNYDHPEIC-NH2 (Disulfide bridge:Cys2-Cys8;Cys3-Cys16) |
| **Target:** | nAChR |
| **Pathway:** | Membrane Transporter/Ion Channel; Neuronal Signaling |
| **Storage:** | Please store the product under the recommended conditions in the Certificate of Analysis. |

## BIOLOGICAL ACTIVITY

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| **Description** | α-Conotoxin Vc1.1 TFA is a disulfide-bonded peptide isolated from Conus victoriae and is a selective nAChR antagonist. α-Conotoxin Vc1.1 TFA inhibits α3α5β2, α3β2 and α3β4 with $IC_{50}$s of 7.2 μM, 7.3 μM and 4.2 μM, respectively, and has less inhibitory effect on other nAChR subtypes. α-Conotoxin Vc1.1 TFA has the potential for neuropathic pain reserach[1][2]. |
| **$IC_{50}$ & Target** | IC50: 7.2 μM (α3α5β2), 7.3 μM (α3β2) and 4.2 μM (α3β4)[1] |
| **In Vitro** | The α-Conotoxin Vc1.1 is first discovered using a PCR screen of cDNAs from the venom ducts of Conus victoriae. α-Conotoxin Vc1.1 inhibits nicotine-evoked membrane currents in isolated bovine chromaffin cells in a concentration-dependent manner and preferentially targets peripheral nAChR subtypes over central subtypes. The three-dimensional structure of Vc1.1 comprises a small alpha-helix spanning residues Pro6 to Asp11 and is braced by the I-III, II-IV disulfide connectivity seen in other alpha-conotoxins. The cysteine spacing within the sequence of α-Conotoxin Vc1.1 suggests that it is a member of the 4/7 subclass of α-conotoxins, which includes the extensively studied conotoxins MII, EpI and PnIB[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | α-Conotoxin Vc1.1 (0.18-18 μg/μL; intramuscular injection; daily; for 7 days; male Sprague-Dawley rats) treatment suppresses pain behaviors and also accelerates functional recovery of injured neurons in CCI rats[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

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| Animal Model: | Outbred male Sprague-Dawley rats (3-4 months old; 250-350 g) bearing with chronic constriction injury (CCI)[1] |
| Dosage: | 0.18 μg/μL, 1.8 μg/μL or 18 μg/μL |
| Administration: | Intramuscular injection; daily; for 7 days |
| Result: | Suppressed pain behaviors and also accelerates functional recovery of injured neurones. |

## REFERENCES

[1]. Richard J Clark, et al. The Synthesis, Structural Characterization, and Receptor Specificity of the Alpha-Conotoxin Vc1.1. J Biol Chem. 2006 Aug 11;281(32):23254-63.

[2]. Narmatha Satkunanathan, et al. Alpha-conotoxin Vc1.1 Alleviates Neuropathic Pain and Accelerates Functional Recovery of Injured Neurones. Brain Res. 2005 Oct 19;1059(2):149-58.



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