# α-Conotoxin EI

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| **Cat. No.:** | HY-P1266 |
| **CAS No.:** | 170663-33-9 |
| **Molecular Formula:** | $C_{83}H_{125}N_{27}O_{27}S_5$ |
| **Molecular Weight:** | 2093.37 |
| **Sequence:** | Arg-Asp-{Hyp}-Cys-Cys-Tyr-His-Pro-Thr-Cys-Asn-Met-Ser-Asn-Pro-Gln-Ile-Cys-NH2 (Disulfide bridge:Cys4-Cys10;Cys5-Cys18) |
| **Sequence Shortening:** | RD-{Hyp}-CCYHPTCNMSNPQIC-NH2 (Disulfide bridge: Cys4-Cys10;Cys5-Cys18) |
| **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 EI is a selective nicotinic acetylcholine α1β1γδ receptor (nAChR) antagonist ($IC_{50}$=187 nM) and an α3β4 receptor inhibitor. α-Conotoxin EI can block muscle and ganglionic receptors[1][2][3]. |
| **$IC_{50}$ & Target** | $IC_{50}$: 187 nM (nicotinic acetylcholine α1β1γδ receptor)[3] |
| **In Vitro** | α-Conotoxin EI (10 μM) blocks muscle and ganglionic receptors, but less potent for the central nevous system receptor[3].<br>α-Conotoxin EI (10 μM) blocks the α1β1γδ receptors with a τon of 4.9 s, and blocks the α3β4 receptor with a τon of 11 s[3].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | α-Conotoxin EI (intraperitoneal injection) into mice induces muscular weakness, followed by flaccid paralysis and ultimately death[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## REFERENCES

[1]. Martinez JS, et al. alpha-Conotoxin EI, a new nicotinic acetylcholine receptor antagonist with novel selectivity. Biochemistry. 1995 Nov 7;34(44):14519-26.

[2]. Ning J, et al. Identification of Crucial Residues in α-Conotoxin EI Inhibiting Muscle Nicotinic Acetylcholine Receptor. Toxins (Basel). 2019 Oct 16;11(10):603.

[3]. López-Vera E, et al. Novel alpha-conotoxins from Conus spurius and the alpha-conotoxin EI share high-affinity potentiation and low-affinity inhibition of nicotinic acetylcholine receptors. FEBS J. 2007 Aug;274(15):3972-85.



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