Data Sheet (Cat.No.TMPH-00429)

TargetMol

Con-ikot-ikot Protein, Conus striatus, Recombinant (His)

General Information

Synonyms:	CII;Con-ikot-ikot
Protein Construction:	38-123 aa
Species:	Conus striatus
Expression Host:	E. coli
Accession:	P0CB20
Molecular Weight:	13.4 kDa (predicted)
AA Sequence:	SGPADCCRMKECCTDRVNECLQRYSGREDKFVSFCYQEATVTCGSFNEIVGCCYGYQMCMIRVVKPNSLSGA HEACKTVSCGNPCA

QC Testing

Biological Activity:	Activity has not been tested. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 90% as determined by SDS-PAGE.
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Tris-based buffer, 50% glycerol

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Potently and selectively blocks the desensitization of ionotropic glutamate AMPA receptor (GRIA1, GRIA2, GRIA3 and GRIA4). Can also open already desensitized GRIA1 receptors. Binds to a different site than does the drug cyclothiazide. The toxin acts like a straightjacket on the ligand-binding domain (LBD) 'gating ring' of the receptor, restraining the domains via both intra- and interdimer cross-links such that agonist-induced closure of the LBD 'clamshells' is transduced into an irislike expansion of the gating ring. Application of the toxin to hippocampal slices causes a large and rapid increase in resting AMPAR-mediated current leading to neuronal death.

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