Data Sheet (Cat.No.TMPH-00055)

TargetMol

Alpha-toxin Amm8 Protein, Androctonus mauritanicus, Recombinant (His & Myc)

General Information

Synonyms: Alpha-toxin Amm8;Neurotoxin 8;Alpha-anatoxin Amm VIII (Amm VIII;AmmVIII);P4

Protein Construction: 20-84 aa

Species: Androctonus mauritanicus

Expression Host: Baculovirus Insect Cells

Accession: Q7YXD3

Molecular Weight: 11.3 kDa (predicted)

AA Sequence: LKDGYIVNDINCTYFCGRNAYCNELCIKLKGESGYCQWASPYGNSCYCYKLPDHVRTKGPGRCND

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: > 85% as determined by SDS-PAGE.

Endotoxin: < 1.0 EU/μg of the protein as determined by the LAL method.

Formulation: If the delivery form is liquid, the default storage buffer is Tris/PBS-based buffer, 5%-50% glycerol. If the delivery form is lyophilized powder, the buffer before lyophilization is Tris/PBS-based buffer, 6% Trehalose, pH 8.0.

Preparation and Storage

Reconstitution:

Reconstitute the lyophilized protein in sterile deionized water. The product concentration should not be less than 100 μg/mL. Before opening, centrifuge the tube to collect powder at the bottom. After adding the reconstitution buffer, avoid vortexing or pipetting for mixing.

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

Alpha toxins bind voltage-independently at site-3 of sodium channels (Nav) and inhibit the inactivation of the activated channels, thereby blocking neuronal transmission. The toxin principally slows the inactivation process of TTX-sensitive sodium channels. It discriminates neuronal versus muscular sodium channel, as it is more potent on rat brain Nav1.2/SCN2A (EC(50)=29 nM) than on rat skeletal muscle Nav1.4/SCN4A (EC(50)=416 nM). It also shows

a weak activity on Nav1.7/SCN9A (EC(50)=1.76 uM). In vivo, the toxin produces pain hypersensibility to mechanical and thermal stimuli.(PubMed:23685008). It also exhibits potent analgesic activity (when injected intraperitoneally), increasing hot plate and tail flick withdrawal latencies in a dose-dependent fashion. This paradoxical analgesic action, is significantly suppressed by opioid receptor antagonists, suggesting a pain-induced analgesia mechanism that involves an endogenous opioid system. This led to hypothesis that pain relief induced by peripheral administration of Amm VIII may result from sensitization of primary afferent neurons and subsequent activation of an opioid-dependent noxious inhibitory control.

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