

Product Data Sheet

Product Name: ω -Hexatoxin-Hv1a TFA

Cat. No.: GC80469

Chemical Properties

Cas. No.

Formula $C_{162}H_{247}N_{49}O_{61}S_6 \cdot xC_2HF_3O_2$ M.Wt 4049.38

Solubility H_2O : ≥ 50 mg/mL Storage Sealed storage, away from moisture

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure

Ser-Pro-Thr-Cys-Ile-Pro-Ser-Gly-Gln-
Pro-Cys-Pro-Tyr-Asn-Glu-Asn-Cys-Cys-
Ser-Gln-Ser-Cys-Thr-Phe-Lys-Glu-Asn-
Glu-Asn-Gly-Asn-Thr-Val-Lys-Arg-Cys-
Asp (Disulfide bridge: Cys4-Cys18,
Cys11-Cys22, Cys17-Cys36) (TFA salt)

Background

ω -Hexatoxin-Hv1a (ω -ACTX-Hv1; ω -Atracotoxin-HV1) TFA is an orally active insecticidal neurotoxin containing an inhibitor cystine knot motif and a selective calcium channel inhibitor. ω -Hexatoxin-Hv1a TFA blocks L-type voltage-dependent Ca^{2+} channels and reduces intracellular calcium ion concentration, thereby decreasing apoptosis, necroptosis and oxidative stress, and promoting cell recovery and energy level elevation. ω -Hexatoxin-Hv1a TFA causes larval paralysis and death by impairing neurotransmission in the central nervous system of insects. It shows high injectable toxicity

Caution: Product has not been fully validated for medical applications. For research use only.

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against insects of multiple orders, but exhibits weak oral toxicity. ω -Hexatoxin-Hv1a TFA is widely applicable to studies related to ischemia-reperfusion injury, atopic dermatitis, and ischemic injury of cardiomyocytes and neurons [1] [2] [3].

In Vivo, ω -Hexatoxin-Hv1a (46-184 $\mu\text{g/g}$ insect; injection) TFA causes dose-dependent paralysis and mortality in fifth stadium *Mamestra brassicae* larvae, with 90% mortality achieved at 184 $\mu\text{g/g}$ insect^[2]. ω -Hexatoxin-Hv1a (9.6 μg per larva; p.o.; daily; 4 days) TFA alone via daily sucrose droplets has no insecticidal activity against fifth stadium *Mamestra brassicae* larvae^[2].

In Vitro, ω -Hexatoxin-Hv1a TFA (10-50 nM; 3 h of reperfusion) inhibits ischemia/reperfusion-induced apoptosis, suppresses ischemia/reperfusion-induced necrosis, alleviates intracellular calcium overload, and promotes the recovery of cell index in CHO-K1 epithelial cells^[1]. Recombinant ω -Hexatoxin-Hv1a TFA (100-200 ng) retains immunoreactivity after fusion with GNA, which is confirmed by Western blotting analysis of 200 ng and 100 ng loaded samples of the intact Hv1a/GNA fusion protein^[2]. ω -Hexatoxin-Hv1a TFA (10-50 nM; 18 h) blocks AC-1001 H3-induced apoptosis in CHO-K1 cells in a dose-dependent manner in vitro. At 50 nM (18 h incubation), it reduces the apoptosis level to that of the control group, while 10 nM used alone does not alter the baseline apoptosis level^[3].

References:

- [1]. Iurova EV, et al. Effect of the Peptide Calcium Channel Blocker ω -hexatoxin-Hv1a on Cell Death during Ischemia/Reperfusion in vitro. *Sovrem Tekhnologii Med.* 2023;15(1):21-27.
- [2]. Fitches EC, et al. Fusion to snowdrop lectin magnifies the oral activity of insecticidal ω -Hexatoxin-Hv1a peptide by enabling its delivery to the central nervous system. *PLoS One.* 2012;7(6):e39389.
- [3]. Iurova E, et al. Arthropod toxins inhibiting Ca^{2+} and Na^{+} channels prevent AC-1001 H3 peptide-induced apoptosis. *J Pept Sci.* 2021;27(1):e3288.

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