

Product Data Sheet

Product Name: Chlorotoxin

Cat. No.: GC33024

Chemical Properties

Cas. No. 163515-35-3

SMILES Met-Cys-Met-Pro-Cys-Phe-Thr-Thr-Asp-His-Gln-Met-Ala-Arg-Lys-Cys-Asp-Asp-Cys-Cys-Gly-Gly-Lys-Gly-Arg-Gly-Lys-Cys-Tyr-Gly-Pro-Gln-Cys-Leu-Cys-Arg-NH₂ (Disulfide bridge: Cys2-Cys19,Cys5-Cys28,Cys16-Cys33,Cys20-Cys35)

Formula C₁₅₈H₂₄₉N₅₃O₄₇S₁₁ M.Wt 3995.71

Solubility Soluble in DMSO Storage Store at -20°C

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

MCMPCFTTDHQMARCDDCCGGKGR
GKCYGPQCLCR-NH₂(Disulfide bridge:
Cys2-Cys19,Cys5-Cys28,,Cys16-
Cys33,Cys20-Cys35)

Protocol

Cell experiment [1]:

Cell lines MCF-7, MDA-MB-231, T47D cells (human breast cancer cell lines)

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Preparation Method Cells were maintained in DMEM (MCF-7, MDA-MB-231) or RPMI-1640 medium (T47D) supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO₂. Cells were treated with Chlorotoxin at concentrations of 0, 0.05, 0.5, and 5µM for 12, 24, 48, or 72 hours.

Reaction Conditions 0.05–5µM; 12–72h.

Applications Chlorotoxin significantly inhibited the proliferation, migration, and invasion of breast cancer cells in a concentration- and time-dependent manner. Chlorotoxin also downregulated the mRNA and protein expression levels of ERα, VASP, and MMP2, and inhibited the activity of the ERα promoter reporter gene. Immunofluorescence showed that Chlorotoxin treatment reduced nuclear ERα expression, decreased VASP and actin protein levels, disrupted actin morphology, and decreased MMP2 expression.

Animal experiment [2]:

Animal models Adult and juvenile Sprague-Dawley rats

Preparation Method Chlorotoxin (2µM; 10µL) was administered intrathecally into the subarachnoid space of the L4-L6 spinal cord once every 2 days. The treatment was applied in the context of established pain models (e.g., spared nerve injury).

Dosage form 2µM; 10µL; Intrathecal injection; Once every 2 days.

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Applications Chlorotoxin significantly increased the pain vulnerability of juvenile rats in neuropathic, chemotherapeutic, and inflammatory pain models, abolishing the inherent lower pain vulnerability typically observed in juvenile compared to adult rats.

References:

- [1] Wang Y, Li K, Han S, et al. Chlorotoxin targets ER α /VASP signaling pathway to combat breast cancer. *Cancer Med.* 2019 Apr;8(4):1679-1693.
- [2] Zhang XL, Zhang JJ, Chen ZH, et al. Difference of pain vulnerability in adult and juvenile rodents: the role of SIRT1-mediated CIC-3 trafficking in sensory neurons. *Pain.* 2021 Jun 1;162(6):1882-1896.

Background

Chlorotoxin is a polypeptide toxin isolated from the venom of the Israeli deathstalker scorpion [1-2]. Chlorotoxin selectively binds to the MMP-2 protein and chloride ion channels on the surface of glioma cells to inhibit tumor cell migration and invasion, while simultaneously blocking chloride ion currents to affect cellular volume changes. Chlorotoxin is applicable in the diagnosis, imaging, and therapeutic research of glioblastoma[3-4].

In vitro, Chlorotoxin (10nM-100 μ M) bound to superparamagnetic nanoparticles (NPC) and was applied to rat C6 glioma cells for 2 hours. Chlorotoxin significantly inhibited cell invasion and induced the internalization of lipid rafts containing MMP-2 and ion channels[5]. Chlorotoxin (0.05–5 μ M) was applied to human ER-positive breast cancer cells MCF-7, T47D, and ER-negative cells MDA-MB-231 for 12–72 hours. Chlorotoxin significantly inhibited cell proliferation, migration, and invasion in a concentration- and time-dependent manner, and downregulated the mRNA and protein expression levels of ER α , VASP, and MMP2 in the cells[6].

In vivo, Chlorotoxin (60 μ g/kg) was administered via intraperitoneal injection to tumor-bearing (SiHa cells) BALB/c nude mice, beginning when the tumor volume reached 50-100mm³ and repeated every 4 days. Chlorotoxin significantly enhanced the tumor-suppressive effect of cisplatin and downregulated the expression level of P-glycoprotein in tumor tissue[7]. Chlorotoxin (2 μ M; 10 μ L;

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single injection) was administered intrathecally to adult rats. Chlorotoxin significantly increased sciatic nerve injury and pain vulnerability in the rats^[8].

References:

- [1] Dardevet L, Rani D, Aziz TA, et al. Chlorotoxin: a helpful natural scorpion peptide to diagnose glioma and fight tumor invasion. *Toxins (Basel)*. 2015 Mar 27;7(4):1079-101.
- [2] Fu Y, An N, Li K, et al. Chlorotoxin-conjugated nanoparticles as potential glioma-targeted drugs. *J Neurooncol*. 2012 May;107(3):457-62.
- [3] Cohen G, Burks SR, Frank JA. Chlorotoxin-A Multimodal Imaging Platform for Targeting Glioma Tumors. *Toxins (Basel)*. 2018 Nov 26;10(12):496.
- [4] Veisheh O, Gunn JW, Kievit FM, et al. Inhibition of tumor-cell invasion with chlorotoxin-bound superparamagnetic nanoparticles. *Small*. 2009 Feb;5(2):256-64.
- [5] Tang J, Chen S, Deng Y, et al. MA104 cell line is permissive for human bocavirus 1 infection. *J Virol*. 2025 Feb 25;99(2):e0153924.
- [6] Wang Y, Li K, Han S, et al. Chlorotoxin targets ER α /VASP signaling pathway to combat breast cancer. *Cancer Med*. 2019 Apr;8(4):1679-1693.
- [7] Shen J, Zhang D, Zheng Q, et al. CIC-3 inhibition induces autophagy to reverse cisplatin resistance in cervical cancer via the Akt/mTOR pathway. *Biochim Biophys Acta Mol Basis Dis*. 2026 Jan;1872(1):168030.
- [8] Zhang XL, Zhang JJ, Chen ZH, et al. Difference of pain vulnerability in adult and juvenile rodents: the role of SIRT1-mediated CIC-3 trafficking in sensory neurons. *Pain*. 2021 Jun 1;162(6):1882-1896.

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