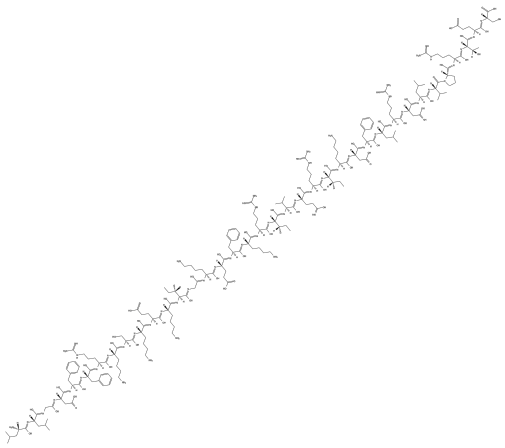
Product Data Sheet

Product Name: LL-37 (trifluoroacetate salt)
Cat. No.: GC14377

Chemical Properties

Cas. No.	154947-66-7		
SMILES	CC[C@]([C@@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/C/N=C(O)/[C@](/N=C(O)/[C@](N)([H])CC(C)C)([H])CC(C)C)([H])CC(O)=O)([H])CC1=CC=CC=C1)([H])CC2=CC=CC=C2)([H])CCCNC(N)=N		
Formula	$C_{205}H_{340}N_{60}O_{53} \cdot XCF_3COOH$	M.Wt	4493.3
Solubility	Water: 1 mg/ml	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			

Protocol

Cell experiment [1]:

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Cell lines	HNEpCs (human nasal epithelial cells) and J774.2 cells (mouse monocyte macrophage cell line)
Preparation Method	HNEpCs and J774.2 cells were maintained in complete medium at 37°C, 5% CO_2. HNEpCs and J774.2 cells were treated with LL-37 (10μM) for 15 minutes, followed by detection of ATP release, Annexin V/7-AAD flow cytometry for cell death, light microscopy for morphology, ELISA for IL-6 and IL-8 (60min treatment for HNEpCs), and FAM-FLICA caspase-1/-3/7/-8 activation assays.
Reaction Conditions	10μM; 15-60min
Applications	LL-37 increased ATP release from HNEpCs and J774.2 cells. LL-37 increased IL-6 and IL-8 secretion from HNEpCs. LL-37 caused cell swelling and rupture in HNEpCs and J774.2 cells. LL-37 increased Annexin V and 7-AAD positive cell populations in HNEpCs and J774.2 cells. LL-37 activated caspase-1 and caspase-8 in HNEpCs and J774.2 cells. LL-37 did not activate caspase-3/7 in HNEpCs and J774.2 cells.
Animal experiment [2]:	
Animal models	BALB/c male mice (4-week-old)
Preparation Method	Mice were intradermally injected with 40μL LL-37 (320μM) once daily for 20 days, with simultaneous intraperitoneal injection of 100μL normal saline once daily from day 10 to day 20. Mice were photographed before each injection and sacrificed 24 hours after the last injection. Back skin was collected for HE staining, VG staining, immunofluorescence staining (TNF-α, α-SMA, vimentin) and Western blot (TNF-α, IL-1β, COL1, vimentin, N-Cadherin).

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Dosage form	320μM; 40μL; i.d.; once daily for 20 days
Applications	LL-37 induced erythema, capillary angiogenesis, papules, pustules and crusts on mouse back skin. LL-37 increased skin lesion area. LL-37 thickened epidermis and dermis. LL-37 increased dermal inflammatory cell infiltration. LL-37 destroyed fat vacuoles and muscle structure. LL-37 increased TNF-α and IL-1β protein expression in skin. LL-37 increased α-SMA, vimentin and TNF-α immunofluorescence intensity in skin. LL-37 increased dermal collagen deposition (VG staining). LL-37 upregulated COL1, vimentin and N-Cadherin protein expression in skin.

References:

[1] Thomas AJ, Pulsipher A, Davis BM, et al. LL-37 causes cell death of human nasal epithelial cells, which is inhibited with a synthetic glycosaminoglycan. PLoS ONE. 2017 Aug 24;12(8):e0183542.

[2] Kang Y, Zhang C, He Y, et al. Thalidomide attenuates skin lesions and inflammation in rosacea-like mice induced long-term exposure of LL-37. Drug Des Devel Ther. 2022 Dec 2;16:4127-4138.

Background

LL-37 (trifluoroacetate salt) is a novel cationic amphiphilic cathelicidin antimicrobial peptide that disrupts the membrane integrity of bacteria, fungi and enveloped viruses. LL-37 regulates inflammatory responses by binding to LPS and modulating TLR4 and other signaling pathways, and promotes keratinocyte migration, proliferation and angiogenesis. LL-37 can be used in studies related to infectious diseases, chronic inflammation, wound healing and tumor immunity[1-4].

In vitro, treatment of HNEpCs cells and J774.2 cells with 10μM LL-37 for 15 minutes increased ATP release, increased IL-6 and IL-8 secretion, caused morphological changes such as cell swelling and rupture, and led to cell death[5]. Treatment of A549 cells with 50μg/mL LL-37 for 24 hours increased cell viability, decreased lactate dehydrogenase release, reactive oxygen species levels and

malondialdehyde content, and reduced apoptosis[6]. Treatment of NCI-H292 cells with 1-20μg/mL LL-37 for 24-48 hours dose-dependently promoted wound closure in mechanically injured monolayers and increased lamellipodia area at the wound edge[7].

In vivo, intradermal injection of LL-37 (320μM; 40μL) into male BALB/c mice once daily for 20 consecutive days induced erythema, capillary hyperplasia, papules, pustules and crusts, increased skin lesion area, thickened epidermis and dermis, increased dermal inflammatory cell infiltration, and destroyed fat vacuoles and muscle structure[8]. Twice-daily intradermal injection of 50μL 320μM LL-37 into the back of C57BL/6N mice for 2 consecutive days induced erythema and small skin lesions, increased skin redness and swelling scores, elevated serum MCP-1 levels, increased epidermal thickness, and increased the proportion of degranulated mast cells in the skin[9]. Intravenous injection of 2μg/mouse LL-37 immediately after cecal ligation and puncture (CLP) surgery into male BALB/c mice improved 7-day survival rate, inhibited peritoneal macrophage caspase-1 activation and pyroptosis, reduced caspase-1 p10 subunit levels in peritoneal lavage fluid, and inhibited caspase-1 activation in splenic white pulp macrophages[10].

References:

[1] Leite ML, Duque HM, Rodrigues GR, et al. The LL-37 domain: A clue to cathelicidin immunomodulatory response? Peptides. 2023 Jul;165:171011.

[2] Chinipardaz Z, Zhong JM, Yang S. Regulation of LL-37 in Bone and Periodontium Regeneration. Life (Basel). 2022 Sep 30;12(10):1533.

[3] Memariani H, Memariani M. Antibiofilm properties of cathelicidin LL-37: an in-depth review. World J Microbiol Biotechnol. 2023 Feb 14;39(4):99.

[4] Radic M, Muller S. LL-37, a Multi-Faceted Amphipathic Peptide Involved in NETosis. Cells. 2022 Aug 8;11(15):2463.

[5] Thomas AJ, Pulsipher A, Davis BM, et al. LL-37 causes cell death of human nasal epithelial cells, which is inhibited with a synthetic glycosaminoglycan. PLoS ONE. 2017 Aug 24;12(8):e0183542.

[6] Gao H, Tang F, Chen B, et al. LL-37 Attenuates Sepsis-Induced Lung Injury by Alleviating Inflammatory Response and Epithelial Cell Oxidative Injury via ZBP1-Mediated Autophagy. Toxins.

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[7] Shaykhiev R, BeiBwenger C, Kandler K, et al. Human endogenous antibiotic LL-37 stimulates airway epithelial cell proliferation and wound closure. Am J Physiol Lung Cell Mol Physiol. 2005 Nov;289(5):L842-8.

[8] Kang Y, Zhang C, He Y, et al. Thalidomide attenuates skin lesions and inflammation in rosacea-like mice induced long-term exposure of LL-37. Drug Des Devel Ther. 2022 Dec 2;16:4127-4138.

[9] Chow BK, Choi YG, Wong TK, et al. Therapeutic effect of an MRGPRX2/MRGPRB2 antagonist on LL-37-induced rosacea-like inflammation in mice. Inflamm Res. 2025;74:174.

[10] Hu Z, Murakami T, Suzuki K, et al. Antimicrobial cathelicidin peptide LL-37 inhibits the pyroptosis of macrophages and improves the survival of polybacterial septic mice. Int Immunol. 2016 May;28(5):245-253.

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