RonanRx
PERSONALIZED MEDICINE

CLINICAL MONOGRAPH · TISSUE REPAIR (UNDER FDA REVIEW)

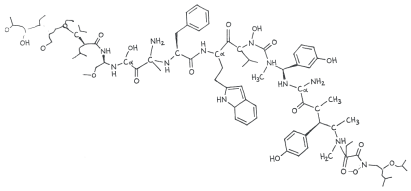
LL-37

Cathelicidin host-defense peptide with case-by-case review

LL-37 is a small protein your own body makes as part of its first line of immune defense. It kills bacteria, fungi, and some viruses, and it helps wounds heal. It is the only member of the cathelicidin family that humans produce.

LL-37 is interesting to researchers because vitamin D turns it on, low levels are linked to skin infections, and high levels are linked to inflammatory skin diseases like rosacea and psoriasis [liu2006]. Despite that biology, LL-37 is not an FDA-approved drug and there is very little human clinical data outside of a few small wound-healing trials [gronberg2014].

LL-37 has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.



EVIDENCE POSTURE PRECLINICAL

REVIEWED **2026-05-11**

State-licensed 503A | Pharmacist reviewed | Doctor led | Cold-chain ready | Patient choice preserved

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FOR CLINICIANS

LL-37 is the C-terminal 37-amino-acid antimicrobial peptide cleaved from hCAP-18, the protein product of the single human cathelicidin gene CAMP [fda503a] [turner1998; durr2006]. It was first identified by Agerberth and colleagues as FALL-39 [agerberth1995] and characterized in granulocytes by Gudmundsson [gudmundsson1996]. Extracellular processing by proteinase 3 releases mature LL-37 from hCAP-18 stored in neutrophil secondary granules [sorensen2001]. CAMP transcription is induced by 1,25-dihydroxyvitamin D_3 acting through the vitamin D receptor; this pathway underlies the Toll-like receptor 2/1 induction of cathelicidin in human macrophages described by Liu et al. (2006, Science) as a mechanism for vitamin D-dependent host defense against Mycobacterium tuberculosis [liu2006].

Mature LL-37 is amphipathic, cationic, and predominantly α-helical, with direct broad-spectrum antimicrobial activity against Gram-positive and Gram-negative bacteria, mycobacteria, fungi, and several enveloped viruses [fda503a] [lopezgarcia2005; tripathi2015]. Beyond direct microbicidal activity, LL-37 chemoattracts neutrophils, monocytes, and T cells through formyl peptide receptor-like 1 (FPRL1/FPR2) [deyang2000], neutralizes LPS, transactivates the epidermal growth factor receptor in airway epithelium [tjabringa2003], stimulates keratinocyte migration and proliferation [niyonsaba2007], and is angiogenic [koczulla2003]. In dysregulated states, LL-37 contributes to disease: deficiency tracks with Staphylococcus aureus colonization in atopic dermatitis [ong2002] and with severe bacterial infections in morbus Kostmann [putsep2002], whereas excess and aberrant processing are implicated in rosacea [yamasaki2007], psoriasis through self-DNA-LL-37 complexes that activate plasmacytoid dendritic cells [lande2007], and lupus through LL-37-modified NETs that drive NLRP3 inflammasome activation [kahlenberg2013].

LL-37 has no FDA approval in the United States. This ingredient is part of an evolving FDA review process. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case, and availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance.

Why Personalized LL-37

The evidence base for LL-37 is largely mechanistic and preclinical. It is a human antimicrobial and immune-signaling peptide with complex inflammatory biology, not an FDA-approved anti-infective or wound-care drug product.

Physicians may submit patient-specific prescription requests for LL-37 for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, supported by patient-specific documentation, and approved by the dispensing pharmacy. Availability is determined case by case. This is not a consumer access promise; it is a clinical, sourcing, formulation, and regulatory review process. This ingredient is part of an evolving FDA review process for peptide-related bulk substances used in compounding.

A patient-specific pharmacy review matters for LL-37 because immune and antimicrobial claims can outrun the evidence quickly. Requests must stay tied to clinician rationale, source verification, formulation controls, and pharmacist release.

Quick Facts About LL-37

Category: Cathelicidin antimicrobial host defense peptide (the only human cathelicidin)

Active ingredient: LL-37, a 37-amino-acid amphipathic α-helical peptide cleaved from the C-terminus of hCAP-18, the product of the human CAMP gene

FDA-approved branded forms: None. LL-37 is not the active ingredient in any FDA-approved drug product.

Route (investigational): Topical and subcutaneous in published research; no FDA-cleared dosage form

Evidence posture: Preclinical and early-phase clinical. Mechanistic in vitro and animal data are extensive; human translation is limited to small wound-healing trials (Grönberg 2014, Mahlapuu 2021) and a research-grade derivative (OP-145) in chronic ear infection.

FDA compounding status: Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

RonanRx compounding status: Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY

Physicians may submit patient-specific prescription requests for LL-37 for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case.

- **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
- **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
- **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
- **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
- **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is LL-37?

LL-37 is the mature 37-amino-acid antimicrobial peptide cleaved from the C-terminus of human cationic antimicrobial protein 18 (hCAP-18), the only cathelicidin encoded in the human genome. The CAMP gene (chromosome 3p21.3) encodes a preproprotein consisting of a signal peptide, a conserved cathelin-like N-terminal domain, and the variable C-terminal antimicrobial peptide. In humans, that C-terminal peptide is LL-37, named for the two leucines at its N-terminus and its length of 37 residues.

LL-37 was first reported as FALL-39 by Agerberth and colleagues in 1995, isolated from human bone marrow and testis as a putative cysteine-free peptide antibiotic [agerberth1995]. Gudmundsson et al. (1996) characterized the human gene FALL39 and cathelin precursor processing in granulocytes, defining the inactive proform stored in neutrophil secondary granules [gudmundsson1996]. Sørensen et al. (2001) demonstrated that extracellular cleavage by proteinase 3, also from neutrophils, releases the mature antimicrobial peptide LL-37 from hCAP-18 [sorensen2001]. Mature LL-37 adopts an amphipathic α-helical conformation in lipid environments [wang2008] and is cationic at physiological pH, properties that underlie its direct antimicrobial activity.

LL-37 is expressed by neutrophils, mast cells, keratinocytes, airway and intestinal epithelium, salivary glands, and reproductive tract epithelium. Tissue expression is upregulated by injury, infection, and 1,25-dihydroxyvitamin D3 acting through the vitamin D receptor at a defined response element in the CAMP promoter [liu2006]. It is the only cathelicidin in humans; other mammals express multiple cathelicidins (e.g., the mouse homolog CRAMP) with distinct sequence and activity profiles [nizet2001, durr2006].

How LL-37 Works

LL-37 has two functional modes. The first is direct microbicidal activity: the cationic amphipathic α-helix binds anionic microbial membranes and permeabilizes them, producing rapid concentration-dependent killing of Gram-positive bacteria, Gram-negative bacteria, mycobacteria, fungi, and several enveloped viruses [turner1998, durr2006, lopezgarcia2005]. The second is immunomodulation: at concentrations below the direct microbicidal threshold, LL-37 reshapes innate and adaptive immune responses without directly killing pathogens [bowdish2005, mookherjee2020].

Immunomodulatory effects include chemoattraction of neutrophils, monocytes, mast cells, and T cells via formyl peptide receptor-like 1 (FPRL1/FPR2) [deyang2000]; neutralization of bacterial lipopolysaccharide and dampening of LPS-induced cytokine production in macrophages [bowdish2005]; induction of keratinocyte migration, proliferation, and chemokine secretion in wound contexts [heilborn2003, niyonsaba2007]; transactivation of the epidermal growth factor receptor in airway epithelium [tjabringa2003]; and stimulation of endothelial proliferation and angiogenesis [koczulla2003].

These effects are protective in normal physiology but contribute to disease in dysregulated states. In atopic dermatitis, LL-37 is deficient and S. aureus colonization increases [ong2002]. In morbus Kostmann (severe congenital neutropenia), LL-37 and α-defensins are absent and life-threatening bacterial infections result [putsep2002]. Conversely, in rosacea, aberrant proteolytic processing of hCAP-18 by elevated kallikrein 5 generates cathelicidin fragments that drive cutaneous inflammation [yamasaki2007]. In psoriasis, LL-37 binds host self-DNA, and the complexes activate plasmacytoid dendritic cells through TLR9 to produce type I interferons that perpetuate the psoriatic phenotype [lande2007]. In systemic lupus erythematosus, LL-37 associated with neutrophil extracellular traps activates the NLRP3 inflammasome in macrophages [kahlenberg2013].

Biological Role of LL-37

Cathelicidins are an evolutionarily conserved family of antimicrobial peptides defined by a shared N-terminal cathelin domain and a variable C-terminal antimicrobial peptide [ong2002; lande2007]. Most mammals encode multiple cathelicidins; humans encode only one, CAMP, whose product hCAP-18 is processed to mature LL-37. Mouse CRAMP and pig PR-39 are the most commonly studied non-human

cathelicidins; the mouse Cnlp/Camp knockout demonstrated that cathelicidin deficiency increases susceptibility to invasive bacterial skin infection [nizet2001] [yamasaki2007].

LL-37 sits at the interface of innate antimicrobial defense and immunomodulation. It contributes to mucosal and cutaneous protection (skin, airway, gut, oral cavity, reproductive tract), to neutrophil-mediated host defense following degranulation, and to wound repair through keratinocyte and endothelial effects [heilborn2003, koczulla2003]. Its expression integrates several stress and immune inputs, injury, infection, vitamin D signaling, and inflammatory cytokines, making it a node where deficiency drives infection susceptibility and overexpression or aberrant processing drives sterile inflammation [alford2020].

Detailed Mechanism of LL-37

Mature LL-37 (LLGDFFRKSKEKIGKEFKRIVQRIKDFLRNLVPRTES) is 37 residues long, carries a net charge of approximately +6 at physiological pH, and adopts a primarily α-helical conformation when bound to lipid surfaces. Solution and lipid-micelle NMR structures by Wang (2008) defined the helix-break-helix architecture of the membrane-associated form and identified the central helical core (residues ~13, 31) as the principal antimicrobial determinant; the smaller fragment KR-12 was shown to retain antibacterial activity [wang2008]. Structure-function dissection by Braff et al. (2005) demonstrated that antimicrobial and host-immunostimulatory activities of human cathelicidin peptides can be partially dissociated by sequence variation, supporting the view that LL-37's two functional modes are separable [braff2005].

Direct microbicidal activity proceeds by binding to anionic outer leaflets of microbial membranes through electrostatic and hydrophobic interactions, followed by membrane disruption, likely via carpet-, toroidal-pore-, or detergent-like mechanisms depending on lipid composition and peptide concentration [durr2006, bandurska2015]. LL-37 is active against Gram-positive cocci (including Staphylococcus aureus and Group A Streptococcus), Gram-negative rods (E. coli, Pseudomonas aeruginosa, Klebsiella), mycobacteria, Candida albicans and other fungi, and a range of enveloped viruses [turner1998, lopezgarcia2005].

Vitamin D dependence is a defining feature of human CAMP regulation. Liu et al. (2006, Science) showed that human macrophages exposed to Mycobacterium tuberculosis antigens upregulate the vitamin D receptor and CYP27B1, generating active 1,25-dihydroxyvitamin D3 intracellularly and inducing cathelicidin expression at a vitamin D response element in the CAMP promoter [liu2006]. This pathway depends on adequate systemic 25-hydroxyvitamin D and is one mechanism linking vitamin D status to mycobacterial host defense in humans. Topical calcipotriol upregulates LL-37 in wounded human skin in vivo [heilborn2010], reinforcing the vitamin D-cathelicidin axis as a clinically tractable lever.

Antiviral activity has been characterized in vitro against influenza A virus [tripathi2015, white2017] and SARS-CoV-2, where Roth et al. (2025) demonstrated direct binding of LL-37 to the SARS-CoV-2 Spike protein and to accessory proteins ORF7a and ORF8 [roth2025]. Antiviral mechanism is multimodal, direct envelope disruption, modulation of innate antiviral signaling, and (for some viruses) interference with cell

entry receptor engagement. Whether these in vitro and animal-model effects translate to clinical antiviral benefit remains unestablished.

Pro-tumorigenic and anti-tumorigenic effects of LL-37 have both been reported, depending on tumor type and microenvironmental context. Sainz et al. (2015) showed that microenvironmental hCAP-18/LL-37 promotes pancreatic ductal adenocarcinoma by activating the cancer stem cell compartment via FPR2 and P2X7 signaling [sainz2015], underscoring that cathelicidin expression cannot be treated as uniformly beneficial in oncology contexts.

LL-37 Research History

LL-37 was first reported in 1995 by Agerberth and colleagues as FALL-39, a putative cysteine-free human peptide antibiotic isolated from bone marrow and testis cDNA libraries [agerberth1995]. Gudmundsson et al. (1996) cloned the human FALL39 (later CAMP) gene, defined the cathelin precursor architecture, and described processing of the proform in granulocytes [gudmundsson1996]. Sørensen et al. (2001) identified extracellular proteinase 3, also a neutrophil granule protein, as the protease that cleaves stored hCAP-18 to mature LL-37, anchoring the regulated-release model of cathelicidin function [sorensen2001]. Turner et al. (1998) reported the first systematic antimicrobial profiling of LL-37 against Gram-positive and Gram-negative bacteria [turner1998].

Through the late 1990s and 2000s, immunomodulatory functions of LL-37 were progressively defined. De Yang et al. (2000) identified FPRL1 as the chemotactic receptor for LL-37 on neutrophils, monocytes, and T cells [deyang2000]. Tjabringa et al. (2003) showed transactivation of the EGFR by LL-37 in airway epithelium, linking the peptide to wound-context epithelial signaling [tjabringa2003]. Heilborn et al. (2003) and Koczulla et al. (2003) defined wound-healing and angiogenic roles of LL-37 [heilborn2003, koczulla2003], and Niyonsaba et al. (2007) characterized keratinocyte responses to cathelicidins and defensins [niyonsaba2007]. Bowdish et al. (2005) consolidated the immunomodulatory framework that distinguishes LL-37's direct antimicrobial and signaling functions [bowdish2005].

Disease associations were established in parallel. Ong et al. (2002, NEJM) reported that atopic dermatitis lesional skin is deficient in LL-37 and β-defensin compared with psoriatic skin, providing a mechanistic explanation for the elevated S. aureus colonization characteristic of atopic dermatitis [ong2002]. Pütsep et al. (2002, Lancet) demonstrated near-complete absence of cathelicidin and α-defensins in patients with morbus Kostmann (severe congenital neutropenia of the ELANE/HAX1 type), correlating with the severe bacterial-infection phenotype [putsep2002]. Yamasaki et al. (2007, Nat Med) showed that rosacea skin has elevated kallikrein 5 protease activity and aberrant cathelicidin processing producing pro-inflammatory cathelicidin peptides [yamasaki2007]. Lande et al. (2007, Nature) demonstrated that LL-37 forms complexes with self-DNA that activate plasmacytoid dendritic cells through TLR9 to produce type I interferon, a mechanism that drives the psoriatic interferon signature [lande2007]. Kahlenberg et al. (2013)

extended the autoimmune-disease framework to systemic lupus erythematosus by showing that NET-associated LL-37 enhances NLRP3 inflammasome activation in macrophages [kahlenberg2013].

Liu et al. (2006, Science) connected vitamin D status to cathelicidin-dependent host defense, demonstrating Toll-like receptor 2/1 triggering of vitamin D receptor-mediated cathelicidin induction in human macrophages exposed to Mycobacterium tuberculosis [liu2006]. The vitamin D response element in the CAMP promoter is a human and primate innovation absent from mouse CAMP, complicating direct translation of mouse cathelicidin data to human biology [durr2006, mookherjee2020].

Antiviral investigation has produced consistent in vitro signal but limited clinical translation. Tripathi et al. (2015) characterized direct activity of LL-37 and derived peptides against seasonal and pandemic influenza A viruses [tripathi2015]; White et al. (2017) extended this to collectin and ficolin-LL-37 combinations in human monocyte infection [white2017]. Roth et al. (2025) demonstrated direct binding of LL-37 to SARS-CoV-2 Spike and to ORF7a/ORF8 accessory proteins, supporting an antiviral or immunomodulatory rationale in COVID-19 that has not been clinically established [roth2025].

Clinical translation has been narrow. Grönberg et al. (2014) reported a phase 2a randomized placebo-controlled trial of topical LL-37 in hard-to-heal venous leg ulcers, demonstrating safety and a dose-response signal at intermediate doses [gronberg2014]. The follow-on multicenter phase 2 trial by Mahlapuu et al. (2021) did not establish a clear efficacy benefit and did not lead to regulatory approval [mahlapuu2021]. Research-grade LL-37-derived peptides (e.g., OP-145, SAAP-148) have advanced in early-phase work for chronic ear infection and topical antimicrobial use but are not FDA-approved.

LL-37 Timeline

1995 • Agerberth et al [agerberth1995]. (PNAS) isolate FALL-39 from human bone marrow and testis as a putative cysteine-free peptide antibiotic

1996 • Gudmundsson et al [gudmundsson1996]. clone the human FALL39 gene and describe processing of the cathelin precursor to LL-37 in granulocytes

1998 • Turner et al [turner1998]. characterize the broad-spectrum antimicrobial activity of LL-37 against Gram-positive and Gram-negative bacteria

2000 • De Yang et al [deyang2000]. (J Exp Med) identify FPRL1 as the chemotactic receptor for LL-37 on neutrophils, monocytes, and T cells

2001 • Sørensen et al [sorensen2001]. (Blood) demonstrate proteinase 3-mediated extracellular cleavage of hCAP-18 to mature LL-37 in neutrophils

- **2001** • Nizet et al [nizet2001]. (Nature) show that cathelicidin knockout mice are susceptible to invasive Group A Streptococcus skin infection, establishing cathelicidin as a non-redundant innate skin defense factor
- **2001** • Dorschner et al [dorschner2001]. report that cutaneous injury induces cathelicidin release with activity against group A Streptococcus
- **2002** • Ong et al. (NEJM) report LL-37 and β-defensin deficiency in atopic dermatitis lesional skin, providing a mechanism for S [ong2002]. aureus colonization
- **2002** • Pütsep et al [putsep2002]. (Lancet) demonstrate cathelicidin and α-defensin deficiency in morbus Kostmann (severe congenital neutropenia)
- **2003** • Heilborn et al [heilborn2003]. show LL-37 involvement in re-epithelialization of human skin wounds and its absence in chronic ulcer epithelium
- **2003** • Koczulla et al [koczulla2003]. (JCI) demonstrate angiogenic activity of LL-37/hCAP-18 in vivo
- **2003** • Tjabringa et al [tjabringa2003]. (J Immunol) show LL-37 activates innate immunity at the airway epithelial surface through EGFR transactivation
- **2005** • Bowdish et al [bowdish2005]. consolidate the immunomodulatory framework for small host defense peptides including LL-37
- **2005** • Braff et al [braff2005]. (J Immunol) dissect structure-function relationships of cathelicidin peptides, separating antimicrobial and host-immunostimulatory activities
- **2005** • López-García et al [lopezgarcia2005]. (JID) characterize antifungal activity of cathelicidins including LL-37 against Candida albicans
- **2006** • Liu et al [liu2006]. (Science) demonstrate Toll-like receptor 2/1 triggering of vitamin D-mediated cathelicidin induction in human macrophages exposed to Mycobacterium tuberculosis
- **2006** • Dürr et al [durr2006]. publish the landmark review framing LL-37 as the only human cathelicidin
- **2007** • Yamasaki et al [yamasaki2007]. (Nat Med) identify aberrant kallikrein 5-mediated cathelicidin processing as the molecular basis for rosacea cutaneous inflammation
- **2007** • Lande et al [lande2007]. (Nature) demonstrate that LL-37 forms complexes with self-DNA that activate plasmacytoid dendritic cells through TLR9 to drive type I interferon production in psoriasis
- **2007** • Niyonsaba et al [niyonsaba2007]. (JID) characterize keratinocyte migration, proliferation, and cytokine responses to human β-defensins and cathelicidin

- **2008** • Wang (J Biol Chem) reports the lipid-micelle NMR structure of LL-37 and the minimal antimicrobial peptide KR-12 [wang2008]
- **2008** • Kaus et al [steinstraesser2008]. (Burns) survey host defence peptide expression including LL-37 in human burn wounds
- **2010** • Heilborn et al [heilborn2010]. report that topical calcipotriol upregulates hCAP-18/LL-37 in wounded human skin in vivo
- **2013** • Kahlenberg et al [kahlenberg2013]. (J Immunol) show that NET-associated LL-37 enhances NLRP3 inflammasome activation in lupus macrophages
- **2014** • Grönberg et al [gronberg2014]. report a phase 2a placebo-controlled randomized trial of topical LL-37 in hard-to-heal venous leg ulcers, demonstrating safety and a dose-response signal at intermediate doses
- **2015** • Tripathi et al [tripathi2015]. characterize antiviral activity of LL-37 and derived peptides against seasonal and pandemic influenza A viruses
- **2015** • Sainz et al [sainz2015]. (Gut) demonstrate that microenvironmental hCAP-18/LL-37 promotes pancreatic ductal adenocarcinoma by activating cancer stem cells via FPR2
- **2015** • Bandurska et al [bandurska2015]. publish a comprehensive review of unique features of human cathelicidin LL-37
- **2017** • White et al [white2017]. (Innate Immun) extend antiviral findings to collectin/H-ficolin-LL-37 combinations in human monocyte influenza infection
- **2020** • Mookherjee et al [mookherjee2020]. (Nat Rev Drug Discov) review antimicrobial host defence peptide functions and clinical translation prospects
- **2020** • Alford et al [alford2020]. (Front Microbiol) review the cathelicidin balance between protective and pathological inflammatory signaling
- **2021** • Mahlapuu et al [mahlapuu2021]. report a multicentre randomized placebo-controlled clinical trial of LL-37 for hard-to-heal venous leg ulcers; the follow-on did not establish a clear efficacy benefit
- **2025** • Roth et al [roth2025]. (Front Cell Infect Microbiol) demonstrate that LL-37 binds the SARS-CoV-2 Spike protein and accessory proteins ORF7a and ORF8
- **2026** • Target window for FDA decision on a broader peptide review that may reclassify Category 2 peptides; LL-37's status is unresolved at the time of writing [fda503a]

Clinical Contexts for LL-37

Chronic, hard-to-heal venous leg ulcers PRECLINICAL

Evidence should be interpreted in context for LL-37. Any patient-specific request requires prescriber rationale and pharmacy review; availability is determined case by case.

Grönberg et al. (2014) randomized adults with hard-to-heal venous leg ulcers to topical LL-37 (0.5, 1.6, or 3.2 mg/mL) or placebo twice weekly for 4 weeks. Safety was favorable and intermediate doses produced greater ulcer-area reduction than placebo, with a non-monotonic dose-response [gronberg2014]. The follow-on multicenter randomized placebo-controlled trial by Mahlapuu et al. (2021) did not establish a clear efficacy benefit at the primary endpoint, and LL-37 has not progressed to approval for venous leg ulcer healing [mahlapuu2021]. This remains the only formal randomized clinical trial program for native LL-37 in any indication.

Atopic dermatitis (mechanistic context) PRECLINICAL

Disease association, not a therapeutic use. LL-37 deficiency is mechanistically implicated in S. aureus colonization in atopic dermatitis; LL-37 has not been studied as a therapeutic in atopic dermatitis.

Ong et al. (2002, NEJM) showed that lesional atopic dermatitis skin has substantially lower LL-37 and β-defensin expression than psoriatic skin, providing a mechanistic explanation for elevated S. aureus colonization characteristic of atopic dermatitis [ong2002]. The pathophysiologic insight has not translated into a therapeutic indication for exogenous LL-37 administration.

Rosacea (mechanistic context) PRECLINICAL

Disease association, pathological, not therapeutic. Aberrant cathelicidin processing drives rosacea inflammation; the implication is that LL-37 should not be administered in this context.

Yamasaki et al. (2007, Nat Med) identified elevated kallikrein 5 protease activity in rosacea skin generating abnormal cathelicidin peptide fragments that induce cutaneous inflammation [yamasaki2007]. Cathelicidin in rosacea is a driver of pathology, increasing LL-37 in patients with rosacea would be expected to worsen rather than improve disease.

Psoriasis (mechanistic context) PRECLINICAL

Disease association, pathological, not therapeutic. LL-37-self-DNA complexes drive the interferon signature of psoriasis; LL-37 administration would be contraindicated in this disease biology.

Lande et al. (2007, Nature) demonstrated that LL-37 binds self-DNA to form complexes that activate plasmacytoid dendritic cells via TLR9, producing the type I interferon signature that perpetuates psoriasis [lande2007]. As with rosacea, the cathelicidin biology of psoriasis argues against exogenous LL-37 as a therapeutic strategy.

Antiviral / SARS-CoV-2 and influenza (mechanistic context) PRECLINICAL

In vitro and animal-model signal; no clinical evidence. Not a clinical indication.

LL-37 has direct in vitro activity against influenza A viruses [tripathi2015, white2017] and binds the SARS-CoV-2 Spike protein and accessory proteins ORF7a/ORF8 in biochemical assays [roth2025]. No clinical trials have demonstrated antiviral benefit of exogenous LL-37 in humans for influenza, SARS-CoV-2, or any other viral infection.

Oncology (mechanistic context, both pro- and anti-tumorigenic signals reported) PRECLINICAL

Preclinical; tumor-type dependent and bidirectional. Not a clinical indication; reported pro-tumorigenic effects in some cancers are a cautionary consideration.

Sainz et al. (2015, Gut) showed that microenvironmental hCAP-18/LL-37 promotes pancreatic ductal adenocarcinoma by activating the cancer stem cell compartment through FPR2 and P2X7 signaling [sainz2015]. Other tumor types have shown growth-suppressive effects of LL-37 in vitro. The bidirectional preclinical literature does not support a clinical oncology indication and signals that systemic LL-37 administration could be harmful in patients with cathelicidin-responsive tumors.

Compounded LL-37 (503A)

Physicians may submit patient-specific prescription requests for pharmacy review. For LL-37, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for LL-37 is largely mechanistic and preclinical. It is a human antimicrobial and immune-signaling peptide with complex inflammatory biology, not an FDA-approved anti-infective or wound-care drug product.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. This ingredient is part of an evolving FDA review process for peptide-related bulk substances used in compounding. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For LL-37, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for LL-37 are reviewed before any preparation is made or released. A patient-specific pharmacy review matters for LL-37 because immune and antimicrobial claims can outrun the evidence quickly. Requests must stay tied to clinician rationale, source verification, formulation controls, and pharmacist release.

LL-37 Formulations and Routes

Form	Concentration	Description
Investigational topical solution (research only)	—	Used in the Grönberg 2014 and Mahlapuu 2021 venous leg ulcer trials as an aqueous solution at 0.5, 1.6, or 3.2 mg/mL applied twice weekly. Not commercially available; not FDA-approved.
Research-grade synthetic peptide	—	Sold to research laboratories by peptide synthesis vendors as a synthetic 37-amino-acid peptide. Not a finished pharmaceutical product, not GMP-manufactured for human administration, and not legal for use in 503A compounding while LL-37 remains in FDA Category 2.

Routes used in published literature: topical.

LL-37 Dosing

Route	Population	Range	Duration	Study type
Topical	Adults with hard-to-heal venous leg ulcers (investigational only)	0.5, 1.6, or 3.2 mg/mL aqueous solution applied twice weekly for 4 weeks in the phase 2a trial; the intermediate (1.6 mg/mL) dose produced the largest ulcer-area reduction signal	4 weeks in the phase 2a trial; up to 12 weeks of treatment in the follow-on multicenter trial	Phase 2a and phase 2 randomized placebo-controlled trials (investigational)

RonanRx does not publish a consumer dosing schedule for LL-37. Any request requires a valid patient-specific prescription, supporting clinical rationale, and pharmacist review. Route, strength, dosing interval, monitoring expectations, and dispensing quantity would be determined case by case from the prescriber's documentation and pharmacy feasibility review.

LL-37 Safety

Published human safety data for native LL-37 are limited to the two venous leg ulcer trials. In Grönberg et al. (2014), topical LL-37 at 0.5, 1.6, and 3.2 mg/mL applied twice weekly for 4 weeks was reported as well-tolerated with adverse event rates comparable to placebo across the dose range [25]. The multicenter follow-on by Mahlapuu et al. (2021) similarly reported a favorable local safety profile at topical doses tested in

venous leg ulcers [32]. Systemic, parenteral, or sustained administration of LL-37 has not been characterized in human safety studies [34].

Preclinical and mechanistic concerns argue against treating LL-37 as a benign supplement [34]. LL-37 has clear pro-inflammatory and pro-pathological roles in rosacea [18], psoriasis [19], and systemic lupus erythematosus [24], administering exogenous LL-37 to patients with these conditions or with a predisposition to type I interferon-driven autoimmunity would be expected to exacerbate disease. Pro-tumorigenic effects of cathelicidin in pancreatic ductal adenocarcinoma [28] are a further consideration; the bidirectional oncology literature does not support an assumption of safety in cancer patients.

LL-37 is currently FDA Category 2 [34]. The classification reflects outstanding safety, characterization, or stability questions FDA has not resolved for the substance, and is independent of any specific clinical trial result. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case.

Contraindications

Honest gap. No formal contraindications are defined because LL-37 has no FDA-approved indication or labeled use. Preclinical mechanistic data argue against exogenous LL-37 in patients with rosacea, psoriasis, systemic lupus erythematosus, or cathelicidin-responsive malignancy (e.g., pancreatic ductal adenocarcinoma), but these are mechanistic cautions rather than labeled contraindications.

Searched: PubMed, FDA Drugs@FDA, ClinicalTrials.gov on 2026-05-11 · terms *LL-37 contraindications, cathelicidin contraindications, hCAP-18 safety*.

Drug interactions

Honest gap. No formal drug-drug interaction studies have been performed in humans. LL-37 is a peptide cleared by proteolytic catabolism and is not expected to participate in CYP450-mediated interactions. Mechanistic interactions with vitamin D status (which upregulates endogenous CAMP transcription) and with neutrophil proteases (which process hCAP-18) are biology-of-the-system effects rather than conventional drug-drug interactions.

Searched: PubMed, FDA Drugs@FDA, DailyMed on 2026-05-11 · terms *LL-37 drug interaction, cathelicidin interaction*.

Adverse events

Adverse-event data are confined to the two venous leg ulcer trials. Local skin reactions, transient irritation, and burning at the application site were the most common topical events; rates were comparable between active and placebo in both trials [25][32]. No systemic safety signals were reported, but systemic exposure with topical wound application is expected to be low and these trials were not designed to characterize systemic safety. There is no human safety database for subcutaneous, intravenous, intranasal, or inhaled LL-37.

Monitoring LL-37 Therapy

Not applicable in routine practice, LL-37 is not FDA-approved and is not compounded at RonanRx. In the venous leg ulcer trials, monitoring consisted of wound-area measurement, local skin tolerability assessment at each application, and standard adverse-event reporting [gronberg2014, mahlapuu2021].

LL-37 in Special Populations

LL-37 Evidence Quality

The mechanistic and preclinical evidence base for LL-37 is large, mature, and reproducible across laboratories, direct antimicrobial activity [turner1998, lopezgarcia2005, durr2006], immunomodulatory activity, wound-healing and angiogenic activity [heilborn2003, koczulla2003, niyonsaba2007], vitamin D-driven expression [liu2006, heilborn2010], and disease associations across atopic dermatitis, morbus Kostmann, rosacea, psoriasis, and lupus [fda503a]. The antiviral literature, including SARS-CoV-2 Spike binding [roth2025] and influenza neutralization [tripathi2015, white2017], extends the mechanistic story but has not produced clinical translation.

Human clinical evidence is narrow [fda503a] [mookherjee2020]. Two randomized placebo-controlled trials in venous leg ulcers [gronberg2014, mahlapuu2021] form the published human therapeutic evidence base for native LL-37. The phase 2a trial showed a favorable safety profile and a dose-response signal at intermediate doses; the follow-on multicenter trial did not establish a clear efficacy benefit, and LL-37 has not advanced to FDA approval for any indication [lande2007; kahlenberg2013; bowdish2005].

From a 503A regulatory perspective, LL-37 sits in FDA Category 2. The classification reflects unresolved FDA-level questions about the substance and excludes its use in 503A compounding regardless of the strength of the preclinical literature. Physicians may submit patient-specific prescription requests for pharmacy review. Availability is determined case by case. This monograph documents the science and the regulatory state for partner clinics and patients who may encounter LL-37 in gray-market peptide channels [ong2002; putsep2002; yamasaki2007].

Major LL-37 Clinical Studies

Study	Design	Participants	Duration	Finding
Agerberth et al. (1995, PNAS),	Bone marrow and testis cDNA library screen plus	—	—	Identified FALL-39 (later renamed LL-37) as a putative

Study	Design	Participants	Duration	Finding
Discovery of FALL-39	chemical synthesis and antibacterial testing of the predicted peptide			cysteine-free human peptide antibiotic, establishing the existence of a single human cathelicidin [agerberth1995]
Gudmundsson et al. (1996, Eur J Biochem), Human FALL39 gene and granulocyte processing	Gene cloning, expression analysis, and proform processing in human granulocytes	—	—	Defined the human CAMP gene architecture (cathelin domain plus variable C-terminal antimicrobial peptide) and demonstrated cleavage of the proform to mature LL-37 in granulocytes [gudmundsson1996]
Turner et al. (1998, AAC), Antibacterial activities of LL-37	In vitro antimicrobial susceptibility testing of synthetic LL-37 against Gram-positive and Gram-negative bacteria	—	—	Established broad-spectrum direct antibacterial activity of LL-37; the foundational pharmacology paper for the molecule [turner1998]
De Yang et al. (2000, J Exp Med), FPRL1 chemotaxis	Receptor pharmacology and chemotaxis assays on primary human leukocytes	—	—	Identified formyl peptide receptor-like 1 (FPRL1/FPR2) as the chemotactic receptor for LL-37 on neutrophils, monocytes, and T cells, defining the immunomodulatory receptor axis [deyang2000]
Sørensen et al. (2001, Blood), Proteinase 3 processing of hCAP-18	Biochemical characterization of hCAP-18 processing in neutrophil exocytosis	—	—	Showed that extracellular proteinase 3 cleaves stored hCAP-18 to release mature LL-37, the regulated activation step of the cathelicidin system [sorensen2001]
Nizet et al. (2001, Nature), Cathelicidin knockout skin susceptibility	Cathelicidin (Cnlp/Camp) gene-knockout mouse model with Group A Streptococcus skin infection	—	—	Demonstrated that cathelicidin is a non-redundant innate skin defense factor; CRAMP-knockout mice show invasive bacterial infection from skin inoculum [nizet2001]
		—	—	

Study	Design	Participants	Duration	Finding
Ong et al. (2002, NEJM), Atopic dermatitis cathelicidin deficiency	Comparative analysis of LL-37 and β-defensin expression in atopic dermatitis vs psoriasis lesional skin			Lesional atopic dermatitis skin had substantially reduced LL-37 and β-defensin expression vs psoriatic skin, providing a mechanism for elevated S [ong2002]. aureus colonization in atopic dermatitis
Pütsep et al. (2002, Lancet), Morbus Kostmann cathelicidin deficiency	Observational study of patients with severe congenital neutropenia	—	—	Demonstrated near-complete absence of cathelicidin and α-defensins in patients with morbus Kostmann, correlating with the severe bacterial-infection phenotype [putsep2002]
Heilborn et al. (2003, JID), LL-37 in re-epithelialization	Immunohistochemistry and functional studies of LL-37 in human acute and chronic wounds	—	—	LL-37 is upregulated in re-epithelializing acute wounds and absent or reduced in chronic ulcer epithelium; antibody-mediated neutralization of LL-37 impaired re-epithelialization in vitro [heilborn2003]
Koczulla et al. (2003, JCI), Angiogenic role of LL-37/hCAP-18	In vitro and in vivo angiogenesis assays including a rabbit hindlimb ischemia model	—	—	LL-37 induced endothelial proliferation, sprouting, and in vivo angiogenesis, establishing a vascular function for cathelicidin beyond antimicrobial activity [koczulla2003]
Tjabringa et al. (2003, J Immunol), Airway EGFR transactivation	In vitro studies in human airway epithelial cells	—	—	LL-37 activated innate airway epithelial responses by transactivating the EGFR, linking cathelicidin to mucosal epithelial signaling pathways [tjabringa2003]
Liu et al. (2006, Science), Vitamin	Human macrophage cell culture with Mycobacterium	—	—	TLR2/1 triggering upregulates vitamin D receptor and CYP27B1 in macrophages,

Study	Design	Participants	Duration	Finding
D induction of cathelicidin	tuberculosis ligands and vitamin D pathway interrogation			generating intracellular 1,25-dihydroxyvitamin D3 that induces CAMP through a vitamin D response element; defined the mechanism linking vitamin D status to mycobacterial host defense [liu2006]
Yamasaki et al. (2007, Nature Medicine), Rosacea cathelicidin processing	Comparative analysis of cathelicidin processing and protease activity in rosacea vs control skin, plus murine cathelicidin injection	—	—	Identified elevated kallikrein 5 protease activity in rosacea skin producing abnormal cathelicidin peptide fragments that induce inflammation; defined the molecular basis of cathelicidin's pathological role in rosacea [yamasaki2007]
Lande et al. (2007, Nature), LL-37-self-DNA in psoriasis	In vitro plasmacytoid dendritic cell stimulation assays plus psoriatic skin analysis	—	—	LL-37 forms complexes with self-DNA that activate pDCs through TLR9 to produce type I interferon, identifying a mechanism for the IFN signature of psoriasis [lande2007]
Wang (2008, J Biol Chem), NMR structure of LL-37 and KR-12	Solution and lipid-micelle NMR structural determination	—	—	Established the membrane-associated helical structure of LL-37 and identified KR-12 (residues 18, 29) as a minimal antimicrobial peptide retaining bacterial-killing activity [wang2008]
Kahlenberg et al. (2013, J Immunol), NET-LL-37 and lupus inflammasome	In vitro macrophage and neutrophil studies plus SLE patient sample analysis	—	—	Showed that LL-37 associated with neutrophil extracellular traps enhances NLRP3 inflammasome activation in macrophages, linking cathelicidin biology to SLE inflammation [kahlenberg2013]
		34		

Study	Design	Participants	Duration	Finding
Grönberg et al. (2014, Wound Repair Regen), Phase 2a LL-37 in venous leg ulcers	Phase 2a randomized double-blind placebo-controlled trial in adults with hard-to-heal venous leg ulcers		4 weeks treatment	Topical LL-37 (0.5, 1.6, 3.2 mg/mL) twice weekly was safe and well-tolerated; the 1.6 mg/mL intermediate dose produced significantly greater ulcer-area reduction than placebo, with a non-monotonic dose-response, the foundational human safety and efficacy signal for native LL-37 [gronberg2014]
Tripathi et al. (2015, PLoS One), Antiviral activity against influenza A	In vitro antiviral assays with seasonal and pandemic influenza A viruses	—	—	Demonstrated direct antiviral activity of LL-37 and derived peptides against multiple influenza A strains, characterizing structural determinants of antiviral activity [tripathi2015]
Sainz et al. (2015, Gut), Pancreatic cancer pro-tumorigenic LL-37	Human PDAC tissue analysis plus tumor-stroma co-culture and xenograft studies	—	—	Microenvironmental hCAP-18/LL-37 from tumor-associated macrophages activated the PDAC cancer stem cell compartment through FPR2 and P2X7 signaling, defining a pro-tumorigenic role of cathelicidin in pancreatic cancer [sainz2015]
Mahlapuu et al. (2021, Wound Repair Regen), Phase 2 LL-37 in venous leg ulcers	Multicentre prospective randomized placebo-controlled clinical trial of topical LL-37 in hard-to-heal venous leg ulcers	—	Up to 12 weeks treatment	Did not establish a clear efficacy benefit on the primary wound-healing endpoint vs placebo; safety remained favorable [mahlapuu2021]. LL-37 has not progressed to regulatory approval for venous leg ulcer healing on the basis of this program
Mookherjee et al. (2020, Nat Rev Drug Discov), Host	Comprehensive narrative review of antimicrobial host defence peptide	—	—	Frames the broader cathelicidin and defensin drug-development landscape, including the gap between

Study	Design	Participants	Duration	Finding
defence peptide review	functions and clinical translation prospects			preclinical promise and human regulatory approval, directly relevant to interpreting LL-37's current status [mookherjee2020]
Roth et al. (2025, Front Cell Infect Microbiol), LL-37 binds SARS-CoV-2 Spike	Biochemical binding assays and structural studies of LL-37 interactions with SARS-CoV-2 proteins	—	—	Demonstrated direct binding of LL-37 to the SARS-CoV-2 Spike protein and to accessory proteins ORF7a and ORF8, providing a molecular rationale for cathelicidin-based antiviral strategies that has not yet been clinically translated [roth2025]

LL-37 Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Native LL-37 is a 37-amino-acid linear peptide cleared by proteolytic catabolism in plasma and tissues. No formal pharmacokinetic studies have been published in humans. Topical application to wounds (as in the venous leg ulcer trials) produces local tissue exposure with minimal expected systemic absorption [gronberg2014, mahlapuu2021]. The peptide is susceptible to proteolysis by host enzymes and by microbial proteases, limiting half-life in biological fluids, a recognized challenge in cathelicidin therapeutic development [mookherjee2020].

Pharmacodynamics

Pharmacodynamic effects characterized in vitro and in animal models include direct concentration-dependent killing of bacteria, fungi, and enveloped viruses; chemoattraction of neutrophils, monocytes, mast cells, and T cells via FPRL1; transactivation of EGFR in airway epithelium; keratinocyte migration and proliferation; endothelial proliferation and angiogenesis; LPS neutralization; and modulation of inflammatory cytokine production [turner1998; deyang2000; tjabringa2003; niyonsaba2007]. Clinically measurable pharmacodynamic endpoints in the venous leg ulcer trials were wound area and re-epithelialization rate [gronberg2014, mahlapuu2021] [koczulla2003; bowdish2005].

LL-37 Storage and Handling

Storage guidance is not applicable, RonanRx does not stock LL-37 as a bulk drug substance or as a finished compounded preparation while it remains in FDA Category 2.

LL-37 Compounding & Operations

503A compounding

Physicians may submit patient-specific prescription requests for pharmacy review. For LL-37, certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case and may depend on patient-specific documentation, ingredient status, source qualification, formulation feasibility, state requirements, and pharmacist judgment. The review starts with the evidence constraint: The evidence base for LL-37 is largely mechanistic and preclinical. It is a human antimicrobial and immune-signaling peptide with complex inflammatory biology, not an FDA-approved anti-infective or wound-care drug product.

This ingredient is part of an evolving FDA review process. RonanRx is monitoring FDA's PCAC process and any subsequent agency action. This ingredient is part of an evolving FDA review process for peptide-related bulk substances used in compounding. Availability may change after FDA review, PCAC discussion, final agency action, or state-board guidance. For LL-37, RonanRx ties that monitoring to the evidence limits described above and to any patient-specific documentation submitted by the prescriber.

Valid patient-specific prescription required. Supporting clinical rationale may be requested. Compounded medications are not FDA-approved. No consumer self-ordering, no office stock, no bulk dispensing. Requests for LL-37 are reviewed before any preparation is made or released. A patient-specific pharmacy review matters for LL-37 because immune and antimicrobial claims can outrun the evidence quickly. Requests must stay tied to clinician rationale, source verification, formulation controls, and pharmacist release.

Pharmacist review

For LL-37, the pharmacist review starts before any preparation is made. Valid patient-specific prescription required. Supporting clinical rationale may be requested. The pharmacist reviews ingredient status, sourcing, formulation feasibility, state requirements, patient-specific documentation, and whether dispensing is appropriate case by case.

Frequently Asked Questions About LL-37

Can physicians request LL-37 through RonanRx?

Physicians may submit patient-specific prescription requests for pharmacy review. Certain preparations may be available now when clinically appropriate, lawfully prescribed, and approved by the dispensing pharmacy. Availability is determined case by case. Compounded medications are not FDA-approved, and no consumer self-ordering, office stock, or bulk dispensing is offered.

What is LL-37?

LL-37 is a 37-amino-acid antimicrobial peptide that humans naturally produce as part of innate immune defense [agerberth1995; gudmundsson1996; durr2006]. It is the C-terminal active fragment of hCAP-18, the protein product of the single human cathelicidin gene (CAMP). It kills bacteria, fungi, and some viruses, helps recruit immune cells to sites of infection or injury, and contributes to wound healing.

Is there clinical evidence for LL-37 as a treatment?

Limited. Two randomized placebo-controlled trials have studied topical LL-37 in hard-to-heal venous leg ulcers [gronberg2014; mahlapuu2021]. The phase 2a trial (Grönberg 2014) showed safety and a dose-response signal at an intermediate dose; the follow-on multicenter phase 2 trial (Mahlapuu 2021) did not establish a clear efficacy benefit. LL-37 has not been approved by FDA for any indication.

Why are people interested in LL-37?

LL-37 has extensive preclinical biology, broad-spectrum antimicrobial activity, immunomodulation, wound-healing effects, and antiviral activity in vitro against influenza A and SARS-CoV-2, and it is induced by vitamin D [liu2006; tripathi2015; roth2025]. The combination of mechanisms made it a popular research-peptide candidate. Clinical translation has been slow, and FDA still has unresolved questions about the substance [mookherjee2020].

Why might LL-37 be harmful in some conditions?

LL-37 drives the inflammation of rosacea (through aberrant proteolytic processing), psoriasis (through self-DNA complexes that activate plasmacytoid dendritic cells and produce type I interferon), and contributes to autoimmune signaling in systemic lupus erythematosus [yamasaki2007; lande2007; kahlenberg2013]. Microenvironmental LL-37 also promotes pancreatic ductal adenocarcinoma in preclinical models [sainz2015]. Administering exogenous LL-37 to patients with these conditions would be expected to worsen disease rather than help it.

When might LL-37 become available through 503A compounding?

Unknown. A broader FDA review of previously-Category-2 peptides has been publicly discussed, with a decision target window of July 2026. Whether LL-37 will be among the substances moved to Category 1 or receive a positive determination is unresolved. RonanRx will update this monograph and publish compounding guidance only if and when FDA's status for LL-37 changes [fda503a].

Where else is LL-37 sold?

Research-grade LL-37 is sold by peptide synthesis vendors to laboratories; LL-37 also appears in the gray-market peptide channel sold direct-to-consumer without prescription review, USP-aligned compounding, or lot-traceable quality testing. Those products are not 503A-compounded medications and carry the safety and identity risks documented across the unregulated peptide supply chain [mookherjee2020].

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How to Access LL-37

Compounded LL-37 is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed LL-37, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)