Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(1), 2488-2494 | ISSN:2251-6727

Cathelicidin LL-37 Level in Leprosy Patients and Household Contacts - A Systematic Review

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(Received: 05 December 2025 Revised: 15 January 2026 Accepted: 10 February 2026)

KEYWORDS

Antimicrobial Peptide, Cathelicidin, Enzyme-Linked Immunoabsorbent Assay, Leprosy

ABSTRACT:

Cathelicidin LL-37 is a multifunctional antimicrobial peptide central to innate immunity. Its dysregulated expression in leprosy patients and household contacts suggests a significant role in pathogenesis and potential as a prognostic biomarker. This systematic review followed PRISMA guidelines, searching PubMed, Cochrane Library, and Google Scholar for studies published between 2010–2025. Inclusion criteria focused on human observational research assessing LL-37 at protein or gene levels in serum, skin, or blood. From 55,212 records, four studies were analyzed. Matzner et al. (2010) identified significantly lower serum LL-37 in untreated leprosy patients. Conversely, Argentina et al. (2023a, 2023b) reported elevated LL-37 protein and *CAMP* gene expression in skin lesions, with intermediate levels found in household contacts. Oliveira et al. (2022) observed reduced *CAMP* and vitamin D receptor expression in peripheral blood across both patients and contacts, correlating with inflammatory cytokine profiles that persisted post-therapy. The evidence reveals a compartmentalized paradox: systemic suppression of LL-37 alongside localized overexpression in lesional skin. Systemic downregulation may be linked to impaired vitamin D–*CAMP* pathways, while local upregulation reflects innate activation against bacillary load. Intermediate levels in contacts suggest immunological priming from exposure. The vitamin D–cathelicidin axis emerges as a potential therapeutic target and a valuable biomarker for disease activity and risk stratification in endemic regions.

1. Introduction

Cathelicidins are a class of multifunctional antimicrobial peptides that play a central role in the innate immune defense system. This peptide is widely recognized for its broad-spectrum antimicrobial activity, demonstrating the ability to combat a wide array of pathogens, including those resistant to conventional antibiotics. Its action involves disrupting microbial membranes, immunomodulating, and enhancing host defense pathways, underscoring its importance in maintaining immune surveillance and resilience against infectious agents[1]. Beyond its antimicrobial action, cathelicidin has also been implicated in the pathophysiology of several inflammatory and infectious diseases. Elevated levels of this peptide have been reported in conditions such as pneumonitis and Inflammatory Bowel Disease (IBD), where it is thought to contribute to disease progression as well as modulation of inflammatory responses[1,2].

In the context of leprosy, a chronic infectious disease caused by *Mycobacterium leprae*, cathelicidin expression has demonstrated distinct alterations. Gene expression analyses have revealed that cathelicidin is significantly elevated in skin lesions of leprosy patients compared to their normal skin, and also shows higher levels in both household contacts and patients when contrasted with healthy, non-exposed individuals[3,4]. This pattern of increased expression suggests that cathelicidin may serve not only as a marker of host immune activation but also as a potential biomarker for prognosis and treatment stratification. The ability to differentiate expression profiles between patients, contacts, and healthy controls has strong implications, especially in endemic areas where close household exposure is a major risk factor. Epidemiological data indicate that the incidence of leprosy among household contacts is approximately 1.4 cases per 1,000 person-years, with a cumulative finding of 192 out of 9,024 contacts

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JCHR (2026) 16(1), 2488-2494 | ISSN:2251-6727

developing leprosy over a 33-year follow-up period[5]. Such data highlight the necessity of identifying predictive biomarkers that can distinguish between individuals at risk of disease progression and those who remain resistant despite exposure.

Given the persistent global burden of leprosy, particularly in endemic regions such as Brazil and parts of Asia, the development of reliable biomarkers like cathelicidin is of increasing significance. Traditional diagnostic methods, including slit-skin smears and histopathological examinations, often have limitations in sensitivity and cannot always predict disease evolution or exposure-related risk. Therefore, the prospect of cathelicidin serving as a prognostic tool could facilitate earlier diagnosis, more targeted treatment, and improved monitoring of household contacts. Its potential incorporation into clinical practice may allow for stratified therapeutic approaches, ultimately supporting the goal of reducing transmission and disability associated with the disease[6],[7].

2. Materials and Method

This systematic review aims to quantify and synthesize the differences in Cathelicidin LL-37 level among leprosy patients and their household contacts, while adhering to PRISMA guideline. The PICO for the following study are:

- **Population:** Humans with leprosy (any Ridley-Jopling class) and/or **household/close contacts**; healthy controls eligible. (Terminology around "contacts" is heterogeneous; we'll accept study-defined contacts and capture definitions.)
- **Index/Exposure:** Cathelicidin/LL-37 measured in any matrix (serum/plasma, skin biopsy, PBMCs; mRNA, protein).
- **Comparator:** Healthy or non-exposed controls; between-group contrasts (patients vs contacts, contacts vs healthy).
- **Outcomes:** Primary—level/expression of LL-37 (or CAMP/hCAP18) as reported (pg/mL, AU, fold-change, ΔCt). Secondary—associations with clinical form (PB/MB), reactions (ENL/Type 1), treatment status, vitamin D/VDR.
- **Design:** Observational (cross-sectional, case-control, cohort); interventional if LL-37 measured at baseline or outcome. No date/language limits.

3. Search Strategy

Article search will be conducted on three databases (Pubmed, Cochrane Library and Google Scholar) using search queries tailored for those individual databases. Search process will include Randomized Clinical Trial, Cohort Study and any research articles while excluding any case reports and review articles.

Pubmed: ("Cathelicidins"[MeSH] OR cathelicidin*[tiab] OR "LL-37"[tiab] OR LL37[tiab] OR hCAP18[tiab]) AND ("Leprosy"[MeSH] OR leprosy[tiab] OR "Hansen's disease"[tiab] OR "Hansen disease"[tiab] OR "Mycobacterium leprae"[tiab])

Cochrane Library: (leprosy OR Hansen OR "Mycobacterium leprae"):ti,ab,kw AND (cathelicidin OR "LL-37" OR LL37 OR hCAP18):ti,ab,kw

Google Scholar: "leprosy" OR "Hansen's disease" OR "Mycobacterium leprae" (LL 37 OR "cathelicidin" OR hCAP18)

4. Selection and data extraction

Authors independently reviewed titles, abstracts, and full texts. Four studies ; Matzner et al. (2011, Yemen), two studies by Argentina et al. (2023a,b, Indonesia), and Oliveira et al. (2022, Brazil), were included. Data extracted included author, year, country, study design, sample size, biospecimens, assay methods, comparator groups, and main outcomes. Household contacts were defined as individuals living in the same residence as leprosy patients.

5. Data synthesis and analysis

Study characteristics and results were narratively summarized and presented in tabular form. Quantitative data were extracted as means ± SD, medians with interquartile ranges, or fold-change values where reported. Because of heterogeneity in study designs, populations, and measurement matrices (serum vs skin), no pooled meta-analysis was attempted. Instead, results were synthesized descriptively, highlighting systemic versus local expression patterns and the position of household contacts relative to patients and healthy groups.

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6. Result

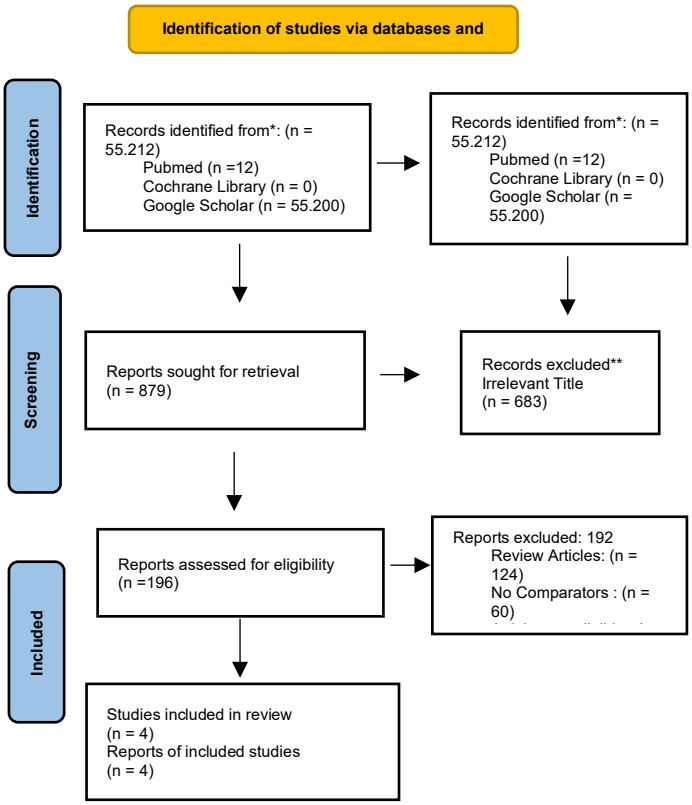

Figure 1 PRISMA Guideline for Article selection process

Table 1 Study Synthesis

Author, Year	Population	Sample / Method	Comparator	Key Findings
Matzner et al., 2010 (Acta Trop)	29 leprosy patients (14 untreated, 15 treated), 19 healthy controls (Yemen)	Serum, ELISA (LL-37), Vitamin D by immunoassay	Patients vs controls; untreated vs treated	Serum LL-37 significantly **lower in leprosy patients** vs healthy (p<0.001). **Untreated** patients had lowest levels (median 48 ng/mL), treated higher (median 98 ng/mL) but still < controls (median 160 ng/mL). Vitamin D3 levels not different. Suggests LL-37 suppressed in active disease.
Argentina et al., 2023 (Acta Med Acad)	18 leprosy patients, 18 household contacts, 18 healthy	Skin scrapings, ELISA for cathelicidin	Patients vs contacts vs healthy	LL-37 protein: **Leprosy > contacts > healthy**. Mean ±SD: patients 256.8±22.9 pg/mL; contacts 25.9±2.7; healthy 1.4±0.1 (p<0.0001). Demonstrates intermediate elevation in HHCs.
Argentina et al., 2023 (Clin Cosmet Invest Derm)	18 leprosy patients, 18 household contacts, 18 healthy	Skin swabs, qPCR for **CAMP (cathelicidin gene)** and HBD-3	Patients vs contacts vs healthy	**CAMP expression**: markedly ↑ in leprosy lesion skin (median 38.72 fold), intermediate in contacts (9.8), baseline in healthy (1.0). Normal skin of patients lower (0.48). Strongly significant (p<0.0001). HBD-3 also upregulated.
Oliveira et al., 2022 (Microbes Infect)	34 leprosy patients (PB & MB, untreated + 6mo MDT), 25 household contacts, 18 healthy (Brazil)	Peripheral blood, RT-qPCR for **VDR** & **CAMP**; serum cathelicidin & cytokines (ELISA & bead array)	Patients vs contacts vs healthy; pre vs post MDT	**Reduced CAMP & VDR expression** in patients and HHCs vs healthy (log10FC < –1 to –2). Expression correlated with IL-2, IFN-γ, IL-17F. Path analysis: VDR/CAMP regulate inflammatory cytokine cascades. Persistent inflammatory profile even after 6mo MDT. Suggests impaired vitamin D–cathelicidin axis in disease.

7. Study Description

Matzner et al. (2010)

This study, conducted in Yemen, assessed serum LL-37 and vitamin D3 concentrations in 29 leprosy patients (14 untreated, 15 treated) and 19 healthy controls. Using ELISA, the authors found that serum LL-37 levels were markedly lower in both untreated and treated leprosy patients compared to controls. The lowest levels were detected in newly diagnosed, untreated patients, while treated patients showed partial restoration of LL-37 but remained below healthy levels. No significant differences in serum vitamin D3 were observed across groups. The study concluded that cathelicidin suppression may be linked to active *M. leprae* infection, independent of vitamin D status.

Argentina et al. (2023a) – Acta Medica Academica

This cross-sectional study in Indonesia compared cathelicidin protein levels in skin scrapings from 18 leprosy patients, 18 household contacts, and 18 healthy controls. Cathelicidin levels were quantified using ELISA, showing a gradient pattern: highest in patients (mean 256.8 ± 22.9 pg/mL), intermediate in household contacts (25.9 ± 2.7 pg/mL), and lowest in healthy individuals (1.4 ± 0.1 pg/mL). Statistical analysis confirmed significant differences between groups ($p < 0.0001$). The findings suggest that household contacts exhibit partial innate immune activation, reflecting their close exposure to *M. leprae*.

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Argentina et al. (2023b) – Clinical, Cosmetic and Investigational Dermatology

In this related study, also based in Indonesia, 18 leprosy patients, 18 household contacts, and 18 healthy controls were examined for gene expression of cathelicidin (CAMP) and human beta-defensin-3 (HBD-3). Skin swabs from lesional and non-lesional sites were analyzed using quantitative PCR. Results demonstrated a marked increase in CAMP expression in leprosy lesions (median 38.72 fold), intermediate expression in household contacts (9.8 fold), and baseline in healthy controls (1.0). Normal skin from patients had much lower expression (0.48 fold). HBD-3 followed a similar pattern. These results highlight local upregulation of antimicrobial peptide genes in response to bacillary exposure, in both patients and contacts.

Oliveira et al. (2022)

This Brazilian study analyzed 34 leprosy patients (14 paucibacillary, 20 multibacillary), 25 household contacts, and 18 healthy controls. Peripheral blood samples were assessed for gene expression of vitamin D receptor (VDR) and CAMP using RT-qPCR, alongside serum vitamin D, cathelicidin, and cytokine levels. Both CAMP and VDR expression were significantly reduced in patients and contacts compared to healthy controls (log10FC < -1 to -2), and strong correlations were observed with inflammatory cytokines (IL-2, IFN-γ, IL-17F). Importantly, these alterations persisted even after six months of multidrug therapy. Path analysis suggested that VDR–CAMP interactions modulate the inflammatory response to *M. leprae*. The authors concluded that impaired vitamin D–cathelicidin signaling contributes to chronic inflammation in leprosy.

8. Discussion

Leprosy, otherwise known as Hansen's Disease, is a chronic infectious disease that is caused by *Mycobacterium leprae* that primarily affects the skin and peripheral nerves. This bacteria is a slow-growing bacterium of which the infection will eventually leads to nerve damage and skin lesions[8], This disease causes a range of symptoms which includes skin injuries, muscle weakness, numbness and in more advance cases may lead to disfiguring mutilations and disabilities such as blindness and paralysis[6].

Diagnosis of this condition often involves clinical examination for cardinal signs that is then supported by laboratory tests such as slit-skin smear and histopathology[9]. Due to its inflammatory nature, biomarkers can be used to accurately diagnose the condition. Bioinformatics analysis showed 10 gene markers which includes ITK, CD48, IL2RG, CCR5, FGR, JAK3, STAT1, LCK, PTPRC, and CXCR4 which are considered to be an active immune system pathway for Chemokine signalling and T-Cell receptor pathway[10].

Another form of biomarker that can and should be considered in the treatment and diagnostic of leprosy are peptides. These molecules play a significant role in the pathogenesis and treatment strategy for leprosy. For example, a specific peptide has been designed to target phenolic glycolipid-1 (PGL-1) of Mycobacterium leprae, which is crucial for the bacterium's entry into Schwann cells. This peptide, derived from the laminin subunit alpha-2 (LAMA2), was shown to potentially block the interaction between PGL-1 and Schwann cells, thereby inhibiting the initial stages of infection. The peptide's stability and preferential binding site were confirmed through molecular dynamics simulations, suggesting its potential as a therapeutic agent to prevent neural damage in leprosy[11].

Peptide that is specifically studied in this review is Cathelicidin LL-37 which exhibits antimicrobial activity against a wide range of pathogens, including bacteria, viruses and fungi by targeting bacterial membrane lipids and other structures, disrupting their integrity and leading to cell death[12]. The earliest specific study that was found in this study is done by Matzner et al (2010), which showed that circulating LL-37 concentrations in serum are significantly reduced in leprosy patients compared to healthy individuals, with untreated cases demonstrating the lowest levels. This finding suggests a systemic suppression of cathelicidin in active infection. Importantly, vitamin D levels were not significantly different between patients and controls, raising the possibility of vitamin D–independent mechanisms contributing to LL-37 downregulation in leprosy[13].

In contrast, more recent studies evaluating skin-derived cathelicidin revealed increased expression in lesional tissue. Argentina et al. (2023) demonstrated that LL-37 protein levels were markedly higher in the skin of leprosy patients than in household contacts and healthy

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individuals, with contacts showing intermediate concentrations. This supports the concept that continuous exposure to *M. leprae* can prime the innate immune system of household contacts, leading to partial upregulation of antimicrobial peptides. A complementary gene expression study by the same group confirmed that CAMP mRNA was elevated in leprosy lesions and, to a lesser extent, in household contacts, relative to healthy skin[3,4]. Together, these findings highlight a divergence between systemic and local expression, where blood levels appear reduced while tissue-specific expression is enhanced in response to bacillary burden.

The mechanistic insights from Oliveira et al. (2022) further elucidate this paradox. Their study showed that both CAMP and vitamin D receptor (VDR) gene expression were significantly reduced in the peripheral blood of patients and household contacts compared to healthy controls. This downregulation correlated with inflammatory cytokine profiles, including IL-2, IFN-γ, and IL-17F, and persisted despite six months of multidrug therapy. These results suggest that the vitamin D–CAMP axis is impaired in leprosy and may underpin the systemic reduction in LL-37 observed in serum. At the same time, persistent inflammatory signaling could explain the increased cathelicidin detected locally in skin tissue[14].

In terms of its function, it has a dual function in pro-inflammation and immuno-response, which are important in fighting bacterial infections, but, under certain condition it has been observed to be able to inhibit inflammatory process[15]. Beyond leprosy, LL-37 has been studied in other conditions such as sepsis where it shows a release of antimicrobial ectosome in neutrophils, reducing bacterial load and improving survival of patients with sepsis[16].

Furthermore, it has also been studied in its role in other inflammatory diseases, such as Type 2 Diabetes and Chronic Lung illness, highlighting its multifunctional nature in immune regulation and disease outcome[17-20].

Having established a potential prognostic modality for Leprosy, it is worth noting that there are other biomarkers that can be used in identifying leprosy, such as ITK, CD48, IL2RG, CCR5, FGR, JAK3, STAT1, LCK, PTPRC, and CXCR4, which are active in immune system pathways such as Chemokine signaling and T cell receptor signaling[21]. Tumour Necrosis Factor (TNF) and Interleukin 10 (IL-10) is also considered to an accurate biomarker for classifying leprosy patients and household contacts[22]. PCR-based detection can also be used in detecting the main cause of Leprosy, which is the *Mycobacterium Leprae*, in which the PCR can be used to detect the bacterium's DNA[23].

Elevated levels of IgA antibodies, which occurred due to reaction of *Mycobacterium Leprae*-specific antigens such as NDO-HSA, LID-1, and NDO-LID, can be used as biomarkers for leprosy detection, aiding in the diagnosis and classification of the disease, especially in multibacillary patients and household contacts[24-27].

Treating leprosy requires a multi-drug treatment, which is the primary treatment. The standard multi-drug treatment involved is a regiment of rifampicin, dapsone and clofazimine, which is effective for both paucibacillary and multibacillary type of leprosy[28,29]. The main challenge in treating leprosy is one of the more drug therapy problem which is drug resistance, relapses and the need for new therapeutic agents, thus it is important, going forward, to improve and overcome this persisting problem[30].

9. Conclusion

Cathelicidin LL-37 is dysregulated in leprosy, showing reduced systemic levels but enhanced local expression in lesions and contacts. These consistent differences across compartments underline its dual role in immune defense and inflammation. LL-37 and its regulatory pathways may serve as valuable biomarkers for exposure and disease activity, and future studies should further explore their diagnostic and therapeutic potential.

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