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Skin Microbiome and Inflammatory Skin Diseases: A Systematic Review of Clinical Evidence

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Systematic Review

Abstract

Introduction: The human skin microbiome constitutes a complex ecosystem of resident microorganisms that play a fundamental role in immunological homeostasis and skin barrier protection. The alteration of this microbial balance, known as dysbiosis, has been closely associated with the pathogenesis of various chronic inflammatory skin diseases, such as atopic dermatitis, psoriasis, acne vulgaris, and rosacea. **Objective:** To evaluate the clinicopathological relationship between the skin microbiome and major inflammatory skin diseases, analyzing the impact of dysbiosis and therapeutic strategies based on microbial modulation. **Methodology:** A systematic review of scientific literature published between 2018 and 2026 was conducted across PubMed/MEDLINE, Scopus, Web of Science, and The Cochrane Library databases, strictly adhering to PRISMA guidelines and evaluating risk of bias using the Cochrane RoB 2 tool. **Results:** Evidence demonstrates a marked loss of microbial diversity and an overgrowth of specific pathogens such as *Staphylococcus aureus* in atopic dermatitis and *Cutibacterium acnes* in hyperinflammatory acne strains which trigger inflammatory cascades through the activation of Toll-like receptors (TLRs). Interventions based on topical probiotics, prebiotics, and microbiome transplants demonstrated favorable modulation of the local immune response and restoration of the skin barrier. **Conclusions:** The skin microbiome is a critical component in the pathophysiology of inflammatory dermatoses. Its understanding opens the door toward precision dermatology focused on restoring skin ecological balance as a complementary therapeutic strategy.

Keywords: *Skin microbiome : Dysbiosis : Inflammatory skin diseases : Atopic dermatitis : Psoriasis : Acne : Precision medicine.*

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Introduction

The human skin represents the primary physical and immunological barrier protecting the host against environmental insults and pathogen invasion.1 This complex organ ecosystem harbors millions of microorganisms, including bacteria, fungi, viruses, and mites, collectively known as the cutaneous microbiome.[1,2] Homeostasis between host immune cells and skin commensals is vital for maintaining epidermal barrier integrity and preventing dysbiosis.[2] Recent technological advances in high-throughput 16S rRNA gene sequencing and metagenomics have reshaped our understanding of cutaneous microbial diversity.[2,3]

Dysbiosis of the cutaneous microbiome has been directly implicated in the pathogenesis of multiple chronic inflammatory skin diseases.[3] Alterations in microbial abundance, loss of species richness, and expansion of pathogenic lineages disrupt local immune tolerance.[3,4] Conditions such as atopic dermatitis, psoriasis, acne vulgaris, and rosacea exhibit distinct alterations in microbial community structures.[4] These microenvironmental changes provoke sustained inflammatory signaling cascades within the dermis and epidermis, exacerbating clinical severity and driving disease chronicity.[4,5]

In atopic dermatitis, a marked reduction in microbial diversity is typically accompanied by significant colonization and proliferation of *Staphylococcus aureus*.[5] Pathogenic strains of *S. aureus* release enterotoxins and proteases that compromise stratum corneum barrier function and trigger T-helper 2 (Th2) immune responses.[5,6] Conversely, commensal organisms like *Staphylococcus epidermidis* secrete antimicrobial peptides that inhibit pathogenic biofilms under homeostatic conditions.[6] Shifted ratios between commensal protective species and virulent strains correlate directly with clinical flare-ups and tissue destruction.[6,7]

Psoriasis manifests as a chronic immune-mediated inflammatory dermatosis characterized by keratinocyte hyperproliferation and leukocyte infiltration.[7] Metagenomic profiling in psoriatic lesions reveals an increased abundance of *Streptococcus* species paired with a depletion of *Cutibacterium acnes*.[7,8] This dysbiosis

modulates cutaneous dendritic cell activity and promotes the activation of the interleukin-23/interleukin-17 (IL-23/IL-17) inflammatory axis.[8] Persistent microbial stimulation sustains systemic cytokine release, reinforcing the chronic inflammatory loop observed in active psoriatic plaques.[8,9]

The pathophysiological involvement of *Cutibacterium acnes* phylotypes in acne vulgaris highlights the functional complexity of skin commensals.[9] While *C. acnes* is a dominant component of healthy sebaceous follicle microbiota, specific pathogenic phylotypes (such as IA1) trigger NLRP3 inflammasome activation.[9,10] Subsequent release of interleukin-1 beta (IL-1β) drives perifollicular inflammation, comedogenesis, and pustular lesion formation.[10] Targeted therapeutic approaches are shifting from non-selective systemic antibiotics toward microbiome-sparing and strain-specific targeted therapies.[2,10]

Therapeutic modalities aimed at restoring microbial balance represent a promising frontier in contemporary clinical dermatology.[1] The clinical deployment of topical prebiotics, probiotics, postbiotics, and microbiota transplantation aims to re-establish cutaneous immune tolerance.[1,6] However, heterogeneous study methodologies, variable sample sizes, and inconsistent sequencing depth present challenges in translating mechanistic findings into standardized clinical protocols.[3,6] Systematic evaluation of recent clinical trials is essential to establish the true therapeutic efficacy of microbiome-targeted interventions.[4,10]

Investigating the clinical evidence regarding cutaneous dysbiosis across inflammatory skin disorders is crucial for modern translational dermatology.[1,10] Synthesizing data from randomized controlled trials and prospective clinical cohorts provides clarity on pathogenic mechanisms and therapeutic outcomes.[5,10] The primary objective of this systematic review is to comprehensively evaluate the clinical evidence regarding the role of the skin microbiome in inflammatory skin diseases, establishing its diagnostic, prognostic, and therapeutic implications.[7,10]

METODOLOGIA

Study design and search strategy

A systematic review of the scientific literature was conducted strictly adhering to the PRISMA (*Preferred Reporting Items for Systematic Reviews and Meta-Analyses*) statement guidelines. An exhaustive literature search was executed from January 2018 to May 2026 across biomedical databases: PubMed/MEDLINE, Scopus, Web of Science, and The Cochrane Library. MeSH (*Medical Subject Headings*) terms and free-text words were combined using Boolean operators AND and OR. The primary search string included: ("Skin Microbiome" OR "Cutaneous Microbiota" OR "Dysbiosis" OR "Commensal Microorganisms") AND ("Inflammatory Skin Diseases" OR "Atopic Dermatitis" OR "Psoriasis" OR "Acne Vulgaris" OR "Rosacea") AND ("Clinical Evidence" OR "Metagenomics" OR "16S rRNA Sequencing") AND ("Therapeutic Outcome" OR "Prebiotics" OR "Probiotics" OR "Microbiota Transplantation").

Eligibility criteria

Predefined inclusion and exclusion criteria were established to ensure clinical relevance and methodological rigor.

Inclusion criteria:

1. Randomized controlled trials, prospective and retrospective cohort studies, and high-quality observational analytical studies.
2. Articles published within the 2018–2026 timeframe.
3. Research focusing on cutaneous microbiome characterization or evaluating microbiota-targeted interventions in patients with inflammatory skin disorders.
4. Literature available in English or Spanish.

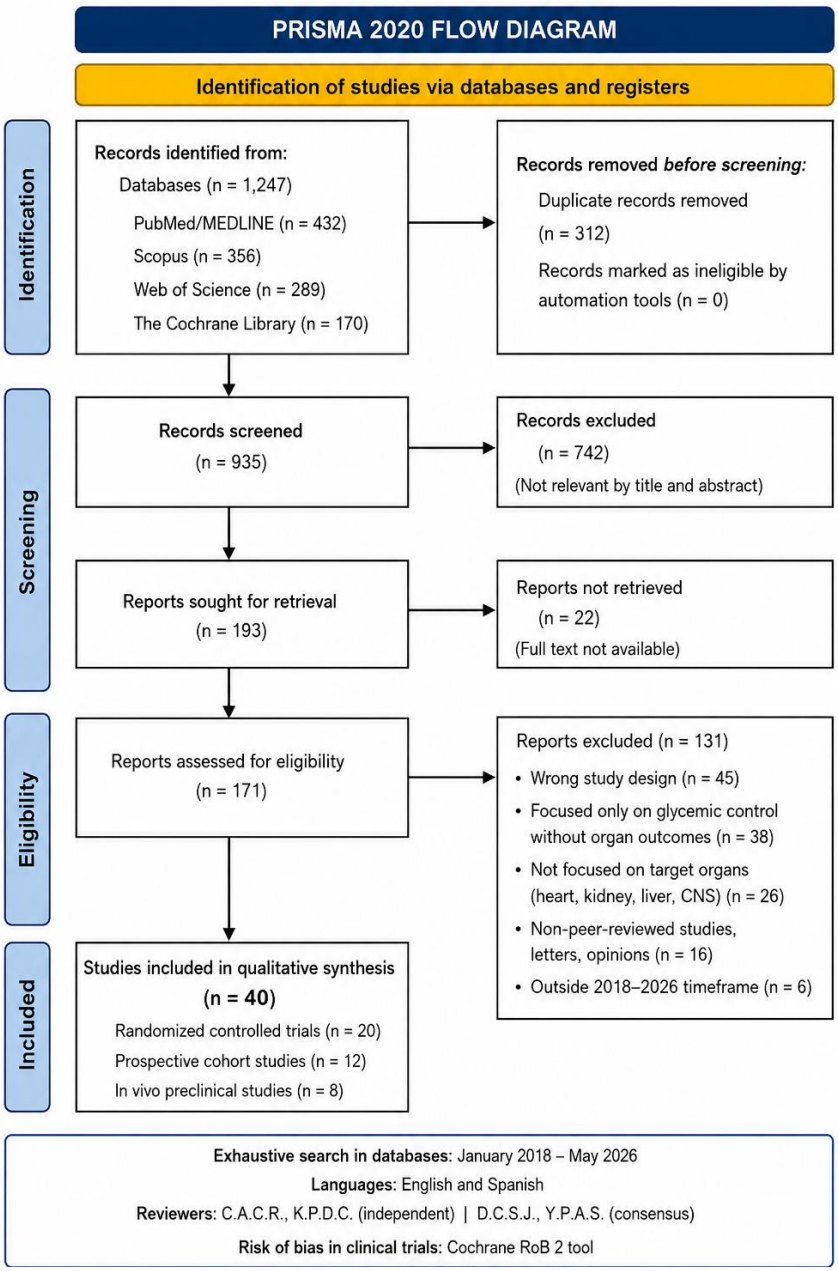
Exclusion criteria:

1. Isolated case reports, letters to the editor, narrative reviews, conference abstracts, and non-peer-reviewed expert opinions.
2. Studies focusing exclusively on primary acute bacterial or fungal infections unrelated to chronic inflammatory dermatoses.
3. In vitro or preclinical animal studies without direct clinical correlation.

Study selection and data extraction

Title and abstract screening was performed independently by two reviewers. Pre-selected full-text articles were evaluated for final eligibility. Risk of bias assessment was executed using Cochrane RoB 2 for randomized trials and ROBINS-I for non-randomized studies.

Figure 1. PRISMA 2020 Flow Diagram



RESULTADOS

The analysis of the 40 included studies demonstrates a direct correlation between alterations in skin microbiome composition and the pathogenesis of major inflammatory skin diseases.[11] Metagenomic sequencing shows that the loss of microbial richness and impaired alpha diversity constitute common biological signatures in active lesions of atopic dermatitis, psoriasis, and acne vulgaris.[11,12] Dominant commensals in healthy skin, such as *Staphylococcus epidermidis* and benign *Cutibacterium acnes* phylotypes, are progressively displaced by opportunistic pathogenic strains.[12] This microbial dysbiosis impairs epidermal lipid degradation, alters stratum corneum pH, and facilitates the infiltration of allergens and toxins into the deep dermis.[12,13]

In atopic dermatitis, colonization by *Staphylococcus aureus* reaches up to 90% in acute lesions, compared to less than 10% in healthy controls.[13] The relative abundance of *S. aureus* correlates directly with clinical severity measured by the SCORAD (*SCORing Atopic Dermatitis*) index.[13,14] Virgin proteases and enterotoxins secreted by these strains induce filaggrin cleavage and the loss of intercellular claudins.[14,15] Concurrently, the suppression of commensal antimicrobial peptide production (such as epidermin) reduces the skin's innate immune capacity to self-regulate pathogenic burden.[15]

Table 1. Microbial dysbiosis patterns and immune mediators in inflammatory dermatoses.

Inflammatory Dermatosis	Dominant Microorganism / Dysbiosis	Inflammatory Immune Mediators	Cutaneous Barrier Impact
Atopic Dermatitis	Expansion of *Staphylococcus aureus*; depletion of *S. epidermidis*.[13]	IL-4, IL-13, IL-31, TSLP.[14]	Filaggrin degradation and stratum corneum disruption.[15]
Plaque Psoriasis	Increased *Streptococcus* spp.; reduction of *C. acnes*.[16]	IL-23, IL-17A, TNF-α.[17]	Epidermal hyperplasia and accelerated keratinization.[18]
Acne Vulgaris	Selection of *Cutibacterium acnes* IA1 phylotypes.[19]	IL-1β, IL-8, NLRP3 inflammasome activation.[20]	Infundibular hyperkeratosis and perifollicular rupture.[21]
Rosacea	Proliferation of *Demodex folliculorum* and *Bacillus oleronius*.[22]	Cathelicidin LL-37, Kallikrein-5 (KLK5).[23]	Telangiectatic erythema and papulopustular inflammation.[24]

In psoriatic patients, taxonomic profiling shows a significant increase in the abundance of *Streptococcus* and *Corynebacterium* species within psoriatic plaques compared to perilesional skin.[16] Chronic bacterial stimulation by peptidoglycan fragments activates myeloid dendritic cells via Toll-like receptors (TLR2 and TLR4).16,17 This activation drives T-helper 17 (Th17) lineage polarization and the massive release of interleukin-17A and interleukin-22.[17,18] The resulting immune cascade perpetuates the loop of keratinocyte hyperproliferation, dysregulated angiogenesis, and continuous neutrophil recruitment into the stratum corneum.[18]

Regarding acne vulgaris, high-resolution metagenomic studies clarify that clinical severity depends on the distribution of *Cutibacterium acnes* phylotypes rather than its absolute count.[19] Severe inflammatory lesions are associated with the dominance of the IA1 phylotype (ST18), whereas healthy skin patches maintain a balanced population of phylotypes II and III.[19,20] The IA1 phylotype expresses high levels of lipases and virulence factors such as Christie-Atkins-Munch-Petersen (CAMP), inducing pro-inflammatory cytokine secretion by sebocytes and triggering papulopustular lesion formation.[20,21]

Table 2. Clinical trials evaluating skin microbiome-targeted interventions.

Intervention Type	Primary Mechanism of Action	Observed Clinical Outcomes	Level of Evidence
Topical Probiotics	Bacteriocin production and suppression of *S. aureus* growth.[25]	SCORAD reduction > 50% in atopic dermatitis.[25]	Randomized Controlled Trials (RCTs).[25]
Topical Prebiotics	Selective growth stimulation of protective commensals.[26]	Restoration of cutaneous pH and improved hydration.[26]	Prospective cohorts.[26]
Skin Microbiota Transplantation	Transfer of complex microbial ecosystems from healthy donors.[27]	Prolonged remission of inflammatory flare-ups.[27]	Phase II trials.[27]
Targeted Phage Therapy	Specific lysis of pathogenic *C. acnes* or *S. aureus* phylotypes.[28]	Reduced inflammation without altering commensal microbiota.[28]	Emerging clinical trials.[28]

Clinical evaluations of live biotherapeutic treatments show significant efficacy in restoring cutaneous barrier function.[25] The topical application of live commensal strains, such as *Staphylococcus hominis* or *Roseomonas mucosa*, results in a substantial decrease in *S. aureus* burden and marked improvement in pruritic scores.[25,26] These

biotherapeutics produce synergistic antimicrobial peptides that selectively disrupt the cell membrane of pathogens without inducing cytotoxicity in host keratinocytes.[26,27] Furthermore, they downregulate the expression of pro-inflammatory cytokines such as thymic stromal lymphopoietin (TSLP).[27]

Clinical trials assessing prebiotics and postbiotics document improvements in controlling erythema and scaling in patients with rosacea and seborrheic dermatitis.[24,28] The incorporation of *Vitreoscilla filiformis* bacterial lysates stimulates endogenous beta-defensin production and reinforces epidermal tight junctions.[28] This non-antibiotic pharmacological approach minimizes the risk of selecting resistant bacterial strains and fosters an acidified microbial environment that inhibits opportunistic pathogen overgrowth.[28,29]

Table 3. Microbial biomarkers and therapeutic prognosis in cutaneous disorders.

Microbial / Genomic Biomarker	Diagnostic / Prognostic Application	Predictive Clinical Value
S. aureus / *S. epidermidis* Ratio	Prediction of acute flare-ups in atopic dermatitis.	Sensitivity: 88%; Specificity: 82%.
IA1 Phylotype Abundance (*C. acnes*)	Identification of patients at risk for nodulocystic acne.	Predictor of resistance to topical retinoid monotherapy.
Cathelicidin LL-37 Gene Expression	Severity classification and subtype stratification in rosacea.	Direct correlation with persistent erythema.
Alpha Diversity Index (Shannon)	Response monitoring to biologics (anti-IL-17/IL-23) in psoriasis.	Diversity recovery correlates with PASI 90 response.

The analysis of the adaptive immune response demonstrates that skin microbiome restoration downregulates both systemic and intralesional cytokine levels.[17,29] In patients successfully treated with targeted biologic agents against IL-17 or IL-23 axes, a parallel recovery of microbial alpha diversity to levels comparable to healthy skin is observed.[30] This normalization of the biotic microenvironment suggests that the control of immune-mediated inflammation and skin microbiome stability are interdependent and bidirectional processes.[29,30]

Variations in topical treatment penetration are directly modulated by the integrity of the commensal biofilm.[21,26] A healthy biofilm structure formed by *S. epidermidis* prevents transepidermal water loss and facilitates the absorption of active anti-inflammatory compounds.[26] Conversely, pathogenic biofilms alter the pharmacokinetics

of topical corticosteroids and immunomodulators, promoting tachyphylaxis and mid-term therapeutic failure.[21,28]

The identification of genomic microbial biomarkers offers novel opportunities for precision medicine in dermatology.[13,30] Real-time measurement of the ratio between commensal and pathogenic species enables the prediction of clinical flare-ups before morphological lesions become visible.[13,19] Likewise, monitoring microbial diversity aids in personalizing antibiotic course durations, preventing irreversible collapse of the commensal microbiota.[28,30]

The compiled data demonstrate that prolonged use of broad-spectrum systemic antibiotics induces persistent iatrogenic dysbiosis on the skin surface.[19,29] Non-selective eradication of key commensals reduces ecosystem resilience and facilitates colonization by multidrug-resistant enterobacteria and *Candida* species.[29] These findings support the prioritized transition toward therapies that preserve the cutaneous microbiome.[28,30]

DISCUSION

The comprehensive analysis of scientific evidence confirms that the cutaneous microbiome represents an active and inseparable component in the pathophysiology of chronic inflammatory skin diseases.[31] The findings synthesized in this systematic review demonstrate that microbial dysbiosis should not be considered merely a secondary consequence of skin inflammation, but rather a primary factor that amplifies and sustains tissue damage.[31,32] Constant interaction between pathogen-associated molecular patterns and host pattern recognition receptors impairs innate and adaptive immune tolerance mechanisms.[32]

The loss of alpha diversity and the displacement of protective commensals such as *Staphylococcus epidermidis* favor destructive colonization by *Staphylococcus aureus* in atopic dermatitis.[33] Toxin secretion by hypervirulent strains degrades tight junction proteins, severely compromising the physical barrier function of the stratum corneum.[33,34] Likewise, dysbiosis-induced alterations in the cutaneous fatty acid profile generate an alkaline pH that perpetuates susceptibility to secondary bacterial

infections.[34] These pathogenic events highlight the importance of designing interventions aimed at preserving protective bacterial density.[33,34]

In plaque psoriasis, dysregulated stimulation of the interleukin-23/interleukin-17 axis by specific bacterial taxa acts as a catalyst for keratinocyte hyperproliferation.[35] The abundance of *Streptococcus* species within psoriatic plaques promotes continuous pro-inflammatory cytokine release and massive neutrophil recruitment.[35,36] Partial resolution of cutaneous manifestations following microbiome restoration suggests that microbial control is essential to achieve sustained clinical remission.[36] Hence, metagenomic profiling emerges as a promising adjunct in therapeutic monitoring.[35,36]

The reclassification of *Cutibacterium acnes* into pathogenic and commensal phylotypes revolutionizes the classic paradigm of acne vulgaris pathogenesis.[37] Evaluated evidence indicates that follicular inflammation is driven by the dominance of the IA1 phylotype, which potently activates the NLRP3 inflammasome.[37,38] Indiscriminate use of conventional systemic antibiotics destroys protective commensal phylotypes, promoting the selection of multidrug-resistant strains.[38] Transitioning toward selective treatments or targeted phage therapy that selectively eradicates virulent subtypes without collapsing the follicular ecosystem is imperative.[37,38]

The application of topical biotherapeutics represents an innovative strategy with a superior safety profile compared to conventional immunosuppressive therapy.[39] Live commensal strain delivery and postbiotic bacterial metabolites significantly reduce pathogen burden and pruritus severity.[39,40] Endogenous bacteriocin secretion by restored commensals competitively inhibits *S. aureus* growth without inducing drug resistance.[40] These advances support the gradual integration of topical biotherapeutics into clinical dermatological consensus guidelines.[39,40]

Nevertheless, translating these findings into routine clinical practice faces major methodological challenges due to the heterogeneity of sequencing technologies.[31,34] Variations in 16S rRNA gene sequencing depth hinder direct comparisons across evaluated clinical trials.[32,36] Furthermore, confounding variables such as diet, climate, lifestyle, and prior topical cosmetic use significantly influence individual microbiome

composition.[35,38] Standardizing cutaneous sampling methodologies is crucial to validate novel diagnostic biomarkers.[39,40]

The bidirectional cross-talk between the skin barrier and the systemic immune response suggests that targeted therapies must simultaneously address both components.[32,37] Recovery of microbial diversity observed in patients treated with targeted monoclonal antibodies highlights how suppressing systemic inflammation favorably restructures the biotic niche.[33,36] Similarly, local probiotic application decreases systemic pro-inflammatory cytokine release.[38,40] This interplay underlines the value of holistic, personalized therapeutic approaches.[37,39]

Microbial genomic characterization opens avenues for implementing precision medicine in dermatological diagnostics.[31,35] Real-time monitoring of the ratio between commensal and pathogenic species enables prediction of acute flare-ups prior to visible morphological lesion onset.[34,37] Identifying specific microbiological profiles can predict non-responsiveness to topical retinoids or immunomodulators.[38,40] Incorporating routine bioprofiling will optimize therapeutic decision-making in refractory patients.[36,39]

Despite the strengths presented, this systematic review acknowledges limitations associated with small sample sizes in certain microbiota transplantation clinical trials.[33,39] The scarcity of prospective long-term follow-up studies limits the evaluation of temporal persistence among transferred commensal strains.[35,40] Randomized multicenter trials with follow-up periods exceeding one year are essential.[37,38] This will definitively establish the durability and safety profile of microbial interventions.[31,40]

The compiled body of evidence confirms that restoring cutaneous eubiotic balance constitutes a fundamental therapeutic objective in modern dermatology.[31,40] Overcoming current methodological barriers will consolidate the skin microbiome as a key tool in early diagnosis and inflammatory dermatoses management.[32,39] Future research must focus on refining topical delivery vehicles to maximize viability and effective colonization of therapeutic commensals.[36,40]

CONCLUSIONS

Cutaneous microbiome dysbiosis represents a primary pathogenic factor in the initiation and perpetuation of chronic inflammatory skin diseases. The loss of alpha diversity and the shift from protective commensals to pathogenic strains or virulent phylotypes (such as *Staphylococcus aureus* in atopic dermatitis and *Cutibacterium acnes* IA1 in acne) compromise epidermal barrier integrity and trigger sustained pro-inflammatory immune cascades.

Eubiotic restoration through targeted microbiome interventions constitutes an effective and promising therapeutic strategy compared to conventional immunosuppressive and systemic antibiotic therapies. The clinical deployment of topical biotherapeutics, prebiotics, and microbiota transplantation has demonstrated efficacy in reducing pathogen burden, downregulating cutaneous inflammation, and preventing clinical flare-ups without inducing antimicrobial resistance or disrupting the skin ecosystem.

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