



Review Article

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Bacterial contributions to superficial skin diseases: Ecology, host interactions, and therapeutic insights

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ABSTRACT

Superficial dermatoses are traditionally attributed to fungal overgrowth, particularly *Malassezia* spp. However, emerging evidence highlights the significant role of the skin bacterial microbiome in modulating disease onset, progression, and severity. This narrative review aims to synthesize current knowledge on bacterial contributions to superficial dermatoses, with emphasis on microbial interactions, host responses, and therapeutic implications. A comprehensive literature search was conducted using PubMed, Scopus, and Web of Science databases. Selected studies published from 2010 to 2025 were selected based on predefined inclusion and exclusion criteria. Commensal bacteria, including *Staphylococcus*, *Cutibacterium*, and *Corynebacterium*, play active roles in regulating microbial balance, lipid metabolism, and inflammatory pathways. Mechanisms such as quorum sensing, biofilm formation, and enzymatic activity facilitate complex bacterial–fungal crosstalk that can either suppress or exacerbate disease. Host factors, including immune responses, skin barrier integrity, and circadian rhythms, further influence these interactions. Advances in diagnostic approaches, such as microbial profiling, enzymatic biomarkers, and volatile organic compounds, provide new insights into disease monitoring. Emerging therapeutic strategies, including probiotics, plant-derived antibacterials, metabolic modulation, and chronotherapy, show promise in restoring microbial balance and improving clinical outcomes. Bacteria are key modulators of superficial skin disease ecology and should be considered central components in disease pathogenesis and management. Integrating microbiome-based diagnostics and targeted therapeutic strategies may enhance precision dermatology and improve patient outcomes.

KEYWORDS: Skin microbiome; Bacterial–fungal interactions;

Superficial dermatoses; Host–microbe interface; Probiotics and therapeutic strategies

1. Introduction

The human skin represents a complex and dynamic ecosystem that harbors a diverse community of microorganisms, collectively known as the skin microbiome. While fungi and viruses are integral components of this ecosystem, bacteria constitute the most abundant and metabolically active members, playing essential roles in maintaining skin homeostasis and modulating disease processes[1]. Traditionally, superficial skin diseases—including acne vulgaris, seborrheic dermatitis, intertrigo, atopic dermatitis, and pityriasis versicolor—have been primarily attributed to fungal overgrowth or host-related factors. However, emerging evidence suggests that bacterial communities are not merely passive inhabitants but active contributors that influence disease onset, severity, and progression[2,3].

Bacteria interact with fungi and the host through multiple mechanisms, including quorum sensing, nutrient competition, enzymatic activity, and antimicrobial peptide production. For instance, *Staphylococcus* (*S.*) *epidermidis* can produce antimicrobial

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factors that suppress fungal proliferation, whereas *Cutibacterium (C.) acnes* hydrolyzes sebum into free fatty acids that may indirectly support fungal growth[2]. These interactions underscore a complex microbial crosstalk in which bacterial communities can either mitigate or exacerbate dermatological conditions, depending on host immunity, skin physiology, and environmental influences. Factors such as humidity, temperature, sebum production, and skin pH further shape microbial composition, creating a dynamic ecosystem that governs disease expression[3,4].

Understanding the role of bacteria in superficial dermatoses has important diagnostic and therapeutic implications. Bacterial-derived markers, enzymatic profiles, and volatile metabolites have emerged as promising tools for early detection and disease monitoring. In

parallel, therapeutic strategies targeting bacterial communities—including probiotics, plant-derived antibacterials, metabolic modulation, and chronotherapy—offer innovative approaches to restore microbial balance and improve clinical outcomes[2,5].

Despite these advances, the role of bacterial communities in superficial skin diseases remains incompletely understood, particularly in the context of their interactions with fungi and the host. Therefore, the aim of this narrative review is to synthesize current evidence on bacterial contributions to superficial skin diseases, with a particular focus on bacterial–fungal interactions, host–microbe dynamics, and environmental influences. Pityriasis versicolor is included as a representative model to illustrate how bacterial activity can modulate fungal behavior within the broader

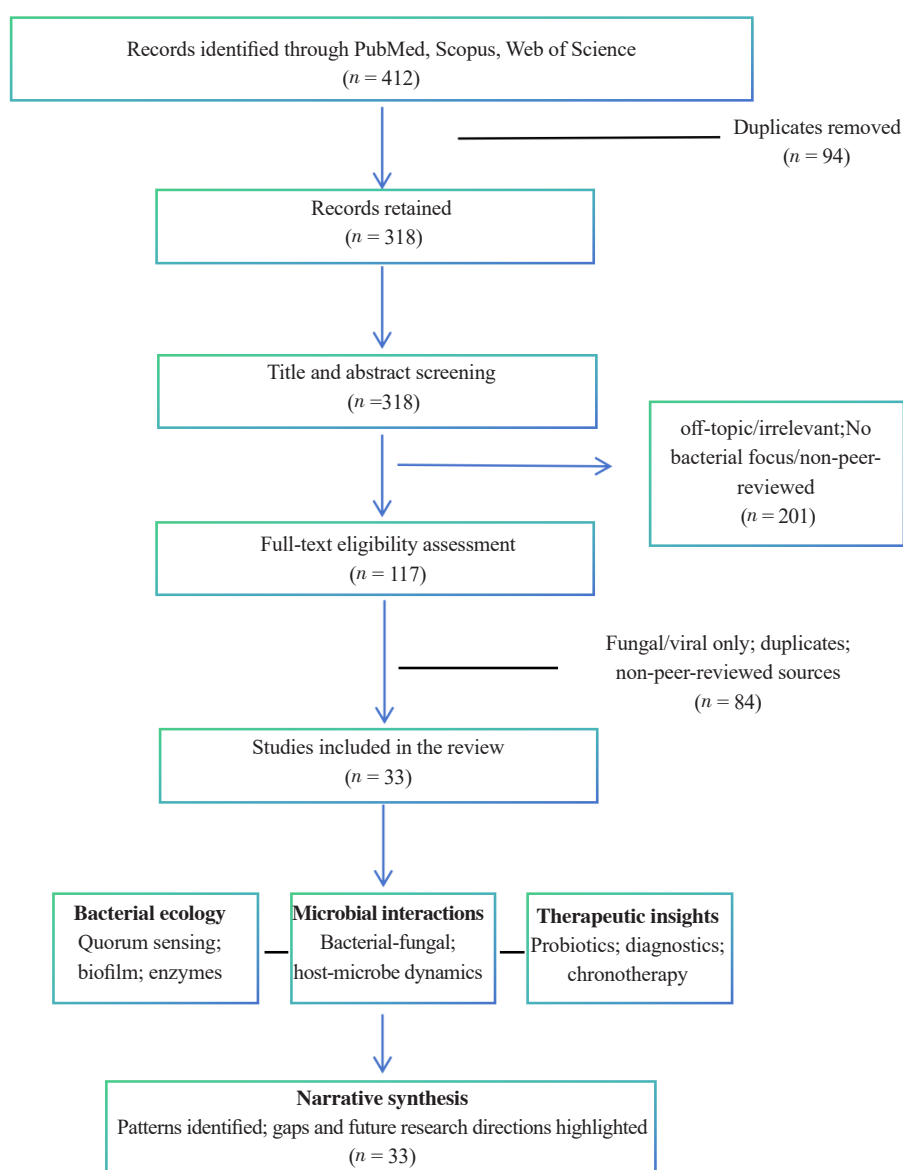


Figure 1. Flowchart of the literature screening.

framework of skin health. This review also seeks to provide a foundation for future research and to highlight potential microbiome-informed precision therapeutic strategies in dermatology.

2. Methods

2.1. Search strategy

This study was conducted as a narrative review with a structured literature selection approach inspired by PRISMA guidelines to enhance transparency and reproducibility. The process included systematic searching, screening, eligibility assessment, and final inclusion of relevant studies. A flowchart illustrating the study selection process is provided to improve methodological clarity (Figure 1).

A comprehensive literature search was conducted to identify relevant studies addressing bacterial contributions to superficial skin diseases. Electronic databases including PubMed, Scopus, and Web of Science were systematically searched. The search strategy incorporated a combination of keywords such as “skin microbiome,” “bacterial–fungal interactions,” “superficial dermatoses,” “*Malassezia*,” “*Staphylococcus*,” and “*Cutibacterium*.” The search was restricted to articles published within the last 10–15 years to capture recent advances in microbial ecology and dermatology.

2.2. Eligibility criteria

Studies were included if they addressed the role of bacterial communities in superficial skin diseases, with particular emphasis on bacterial–fungal interactions and their influence on host immunity, disease progression, or severity. Both original research articles and review studies with clear clinical or experimental relevance were considered to ensure a comprehensive understanding of microbial mechanisms and their implications.

Studies were excluded if they focused exclusively on fungal or viral pathogens without addressing bacterial involvement, or if they lacked sufficient clinical, experimental, or mechanistic relevance. Additionally, duplicate publications and non-peer-reviewed sources were excluded to maintain the quality and reliability of the included literature.

2.3. Study selection process

Titles and abstracts of identified studies were screened for relevance. Full-text articles were then assessed based on the predefined eligibility criteria. Studies meeting the inclusion criteria were selected for qualitative synthesis.

2.4. Data extraction and synthesis

Relevant data were extracted and analyzed to identify key themes in bacterial ecology, microbial interactions, and therapeutic implications. Particular emphasis was placed on mechanisms such as quorum sensing, biofilm formation, enzymatic activity, and host–microbe interactions. Findings were synthesized narratively to highlight patterns across studies and to identify gaps requiring further investigation.

3. Results

3.1. Skin microbiome and bacterial ecology

The skin microbiome is a highly diverse ecosystem, comprising bacteria, fungi, viruses, and other microorganisms, which collectively contribute to the maintenance of skin health and barrier function. Among these, bacteria represent the most abundant and metabolically active members, with key genera including *Staphylococcus* spp., *Cutibacterium*, and *Corynebacterium*[6]. *Staphylococcus* spp., particularly *S. epidermidis*, act as commensals that produce antimicrobial peptides and modulate local immune responses, thereby limiting pathogen overgrowth. *C. acnes* resides primarily in sebaceous regions, where it metabolizes sebum into free fatty acids, influencing both local pH and microbial composition. *Corynebacterium* spp. are commonly found in moist areas such as skin folds and contribute to nutrient competition and microbial balance[7].

Bacterial communities on the skin exhibit greater diversity and abundance compared to fungi, with densities varying across anatomical sites. While fungi such as *Malassezia* spp. are restricted to sebaceous regions, bacteria colonize a wider range of microenvironments, providing resilience to perturbations and interacting dynamically with other microbes[8]. This bacterial dominance highlights their potential to shape disease susceptibility, influence inflammatory responses, and affect the success of therapeutic interventions.

Environmental and host-related factors strongly influence bacterial ecology. Humidity and temperature affect bacterial growth and community composition, with moist areas favoring certain *Corynebacterium* and *Staphylococcus* spp. Sebum-rich regions promote *Cutibacterium* proliferation, while skin pH modulates enzyme activity, microbial colonization, and interspecies interactions. Climatic conditions, personal hygiene practices, and host genetics further contribute to the spatial and temporal heterogeneity of bacterial populations, which in turn affects disease

manifestation and severity[9,10].

Understanding the ecology of skin bacteria and their interactions with environmental factors provides a foundation for exploring how bacterial communities contribute to superficial skin diseases. It also informs the development of diagnostic markers and therapeutic strategies aimed at modulating bacterial populations to restore microbial balance and improve clinical outcomes.

3.2. Bacterial–fungal interactions in superficial dermatoses

Bacteria and fungi cohabit the skin and engage in complex interactions that influence microbial balance and disease outcomes. One key mechanism is quorum sensing, through which bacteria release signaling molecules that can modulate fungal behavior, including the switch between yeast and hyphal forms. This bacterial communication can either suppress fungal virulence or, in some contexts, trigger pathogenic transitions[11].

Competition for nutrients, particularly lipids, constitutes another important mode of bacterial–fungal interaction. *C. acnes*, for instance, hydrolyzes sebum triglycerides into free fatty acids, which may inadvertently provide substrates that support fungal growth. Conversely, bacterial species such as *S. epidermidis* can consume shared nutrients or secrete metabolic byproducts that inhibit fungal proliferation, highlighting a dynamic balance shaped by microbial metabolism[12].

Bacteria also influence fungal populations through the production of antimicrobial peptides, enzymes, and other secondary metabolites. These molecules can degrade fungal cell walls, inhibit enzymatic activity, or disrupt biofilm formation, thereby modulating fungal colonization and disease severity[13]. The net effect of these interactions is context-dependent: bacterial communities can

either exacerbate dermatological conditions by supporting fungal expansion or mitigate symptoms by restraining fungal overgrowth.

As a representative example, pityriasis versicolor illustrates the interplay between bacteria and fungi. While *Malassezia* spp. are the primary etiological agents, the presence of certain bacterial communities may influence lesion severity and distribution. *Staphylococcus* spp., through antimicrobial peptide secretion, may limit fungal overgrowth in some individuals, whereas imbalances in bacterial populations, such as overabundance of *Cutibacterium*, may enhance fungal activity, demonstrating how bacteria act as modulators rather than passive bystanders in disease progression[14]. Understanding these bacterial–fungal interactions provides insight into the mechanisms underlying superficial dermatoses and informs potential therapeutic strategies that target both bacterial and fungal components to restore microbial balance (Figure 2).

3.3. Bacterial contributions to major superficial skin diseases

Acne vulgaris is a common inflammatory skin condition primarily associated with *C. acnes*, a lipophilic bacterium residing in sebaceous follicles. *C. acnes* contributes to acne pathogenesis by interacting with the host immune system, triggering the production of pro-inflammatory cytokines, and promoting neutrophil chemotaxis. Its metabolic activities, including sebum hydrolysis and biofilm formation, can exacerbate follicular obstruction and lesion development. These bacterial activities also modulate the local microenvironment, potentially influencing the growth of co-existing fungi and other microorganisms, highlighting the complex interplay between microbial communities in acne pathophysiology[15].

Seborrheic dermatitis is characterized by erythematous, scaly lesions commonly found in sebaceous-rich areas. While *Malassezia*

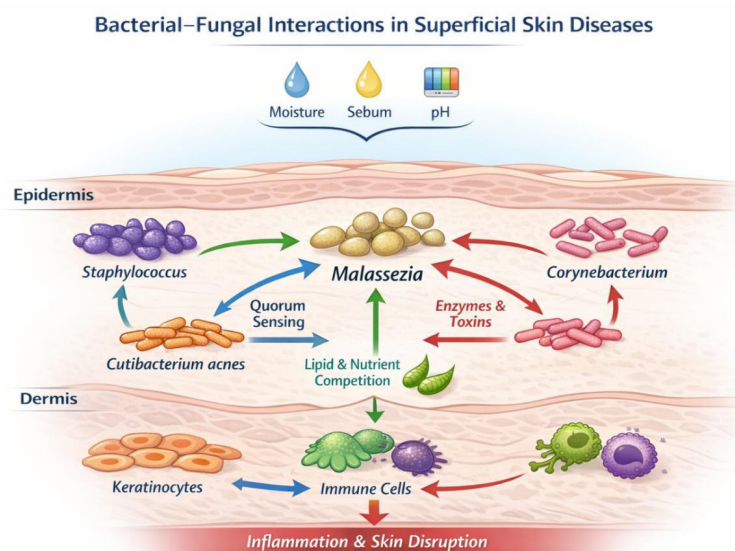


Figure 2. Bacterial–fungal interactions in superficial dermatoses.

Table 1. Major bacteria and their roles in superficial skin diseases.

Diseases	Key bacteria	Effect on fungi	Clinical impact	Evidence
Acne vulgaris	<i>Corynebacterium acnes</i>	Provides lipids, biofilm formation	Promotes follicular inflammation and lesion formation	[15]
Seborrheic dermatitis	<i>Staphylococcus</i> spp.; <i>Corynebacterium</i> spp.	Compete for nutrients, produce inhibitory metabolites	Modulate fungal overgrowth and lesion severity	[16]
Atopic dermatitis	<i>Staphylococcus aureus</i>	Toxins and enzymes disrupt barrier	Exacerbates inflammation, increases infection risk	[17]
Intertrigo/Skin fold infections	<i>Corynebacterium</i> spp.; <i>Staphylococcus</i> spp.	Supports fungal growth in moist environments	Inflammation, maceration	[18]

spp. have been traditionally considered the primary agents, bacterial communities significantly influence disease expression. Commensal bacteria such as *Staphylococcus* spp. and *Corynebacterium* spp. can compete with fungi for nutrients, secrete inhibitory metabolites, or modulate local immune responses, thereby affecting the distribution and severity of lesions. These bacterial–fungal interactions suggest that seborrheic dermatitis is a multifactorial disease where microbial balance plays a critical role in maintaining skin homeostasis or promoting pathology[16].

Atopic dermatitis is a chronic inflammatory disorder frequently associated with dysbiosis, particularly the overgrowth of *S. aureus*. This bacterium exacerbates disease through the production of toxins, superantigens, and proteolytic enzymes, which disrupt the skin barrier and amplify Th2-mediated immune responses. In contrast, commensal bacterial populations, including *S. epidermidis*, can counteract pathogenic effects by producing antimicrobial peptides and promoting microbial diversity. The dynamic balance between pathogenic and commensal bacteria thus critically influences disease severity and susceptibility to secondary infections[17].

Intertrigo and skin fold infections occur in moist, friction-prone areas of the body, creating ideal conditions for bacterial overgrowth. *Corynebacterium* and *Staphylococcus* spp. thrive in these niches, often in conjunction with fungal colonizers, leading to inflammation, maceration, and discomfort. Environmental factors such as humidity, temperature, and occlusion modulate microbial composition, emphasizing the importance of local conditions in shaping bacterial–fungal interactions and clinical outcomes. Understanding these dynamics is essential for developing targeted interventions that restore microbial equilibrium and mitigate disease progression[18] (Table 1).

3.4. Host–microbe interface

The interaction between host immunity and skin microbiota plays a central role in maintaining skin homeostasis and modulating disease susceptibility. The immune system operates at both local and systemic levels to regulate microbial communities. Locally, keratinocytes, Langerhans cells, and resident immune cells produce

antimicrobial peptides, cytokines, and chemokines that shape bacterial colonization and prevent overgrowth of pathogenic species. Systemically, adaptive immune responses, including T-cell and B-cell activity, help maintain a balanced microbial ecosystem, ensuring that commensal bacteria provide protective functions without triggering excessive inflammation[19,20].

Environmental factors further influence the host–microbe interface by altering bacterial composition and activity. Skin moisture, temperature, sebum production, and pH are major determinants of microbial colonization patterns. High humidity and occlusion promote the proliferation of moisture-loving species such as *Corynebacterium*, while sebaceous, lipid-rich regions favor *Cutibacterium* spp. growth. Skin pH modulates enzymatic activity and microbial competition, creating niches that can either suppress or support pathogenic organisms. Variations in these environmental parameters contribute to the heterogeneity of skin diseases and influence the severity and distribution of lesions[21,22].

Pharmacological interventions, including antibiotics, antifungals, and topical therapies, can significantly impact the skin microbiome. Broad-spectrum antibiotics may disrupt commensal bacterial populations, reducing microbial diversity and allowing opportunistic pathogens or fungi to expand. Topical treatments, depending on their formulation and frequency, can similarly shift microbial equilibrium, sometimes improving clinical outcomes but also potentially creating conditions for dysbiosis. Understanding how medications interact with the host–microbe interface is essential for designing interventions that target pathogenic species while preserving beneficial bacteria[23].

Collectively, the host–microbe interface represents a dynamic and context-dependent system where immunity, environmental conditions, and pharmacological exposures interact to shape microbial composition and influence disease manifestation. Insights into this interface provide critical guidance for microbiome-informed therapeutic strategies, aiming to restore microbial balance and improve patient outcomes in superficial skin disorders.

3.5. Diagnostics and biomarkers

The increasing understanding of the skin microbiome has opened new avenues for diagnostics and biomarker development in superficial skin diseases. Bacteria produce a variety of enzymes, metabolites, and signaling molecules that can serve as indicators of disease activity and microbial imbalance. Enzymatic profiling, including the measurement of bacterial lipases, proteases, and other hydrolytic enzymes, allows researchers and clinicians to detect bacterial activity associated with inflammation or fungal overgrowth. Elevated enzyme levels can indicate shifts in microbial balance, providing an early warning of disease onset or flare-ups[24].

Volatile organic compounds (VOCs) represent another promising diagnostic tool. Skin-associated bacteria metabolize lipids and amino acids, generating unique VOC signatures that can be detected non-invasively using techniques such as gas chromatography-mass spectrometry. These bacterial VOCs reflect both microbial composition and metabolic activity, offering potential for early disease detection, monitoring treatment response, and distinguishing between bacterial-driven versus fungal-driven pathologies[25].

Advances in sequencing-based microbiome analysis, including 16S rRNA gene sequencing and metagenomics, allow comprehensive profiling of bacterial communities. These approaches enable the identification of dysbiotic patterns, strain-level variation, and functional potential of bacterial populations. Integrating microbiome data with clinical parameters provides a powerful framework for precision diagnostics, identifying patients at risk for disease exacerbation, and guiding personalized interventions[26].

Together, enzymatic profiling, VOC analysis, and sequencing-based microbiome assessment offer complementary insights into bacterial contributions to superficial skin diseases. By leveraging these diagnostic tools, clinicians can implement earlier interventions, tailor treatments based on microbial signatures, and potentially prevent disease progression through targeted modulation of bacterial communities. These strategies mark a shift from symptom-based diagnosis toward microbiome-informed precision dermatology.

3.6. Therapeutic perspectives

Advances in understanding the skin microbiome have enabled a shift from conventional, pathogen-focused therapies to microbiome-informed, ecology-conscious strategies. Targeting bacterial communities directly or indirectly offers a new avenue for managing superficial skin diseases.

One of the most promising strategies is the use of probiotics and bacterial modulation. Beneficial bacteria, such as *Lactobacillus* strains or commensal *S. epidermidis*, can be applied topically or systemically to restore microbial balance. These probiotics not

only compete with pathogenic bacteria but also enhance the skin's innate defenses by stimulating antimicrobial peptide production. This approach allows the skin microbiome to actively suppress overgrowth of harmful bacteria and fungi, providing a natural and sustainable method for disease control. Moreover, the introduction of probiotics can help maintain long-term stability of microbial communities, reducing the frequency of disease flare-ups[27].

Topical antibacterials provide another critical therapeutic avenue. Plant-derived compounds, including tea tree oil and other botanical extracts, possess antimicrobial properties that selectively inhibit pathogenic species while preserving commensals. Unlike broad-spectrum antibiotics, these agents minimize disruption of the microbial ecosystem and reduce the risk of antimicrobial resistance. Regular or targeted application of these natural compounds can reduce inflammation, microbial load, and fungal synergy in diseases such as seborrheic dermatitis and intertrigo[28].

Targeting bacterial metabolism and communication represents a more precise intervention. Techniques such as lipid sequestration limit nutrient availability for bacteria like *Cutibacterium* spp., indirectly reducing the proliferation of associated fungi. Similarly, quorum-quenching peptides interfere with bacterial signaling pathways, disrupting coordination of virulence factors and biofilm formation. This approach enables selective modulation of microbial activity, preserving beneficial species while suppressing pathogenic behavior[29].

Chronotherapy, or timing interventions according to microbial and host circadian rhythms, is an emerging strategy that maximizes therapeutic efficacy. Skin microbial populations and host immune activity fluctuate throughout the day, influencing bacterial metabolism, sebum production, and inflammatory responses. Administering treatments at peak microbial activity or during periods of heightened immune responsiveness can enhance outcomes, reduce required dosages, and limit side effects[30].

Beyond these targeted strategies, combination therapies that integrate probiotics, topical antibacterials, metabolic modulation, and chronotherapy could offer synergistic benefits. By addressing multiple aspects of microbial ecology simultaneously, clinicians may achieve more robust disease control, reduce recurrence, and promote the resilience of beneficial skin microbiota.

Finally, the development of personalized microbiome-based therapies holds significant potential. Advances in sequencing, enzymatic profiling, and VOC analysis allow clinicians to identify individual microbial imbalances and tailor interventions accordingly. This precision approach moves dermatology toward a future where treatments are guided not only by visible symptoms but also by the underlying microbial ecosystem, optimizing patient outcomes and advancing the concept of microbiome-informed medicine.

4. Discussion

The accumulated evidence underscores the intricate interplay between bacteria and fungi in superficial skin diseases. Studies reveal that bacterial communities are not passive inhabitants but active modulators of fungal growth and disease expression[31]. Such interactions demonstrate that disease outcomes are often the result of dynamic microbial balances rather than the presence of a single pathogen.

Importantly, this review integrates ecological, immunological, and therapeutic perspectives, demonstrating that bacterial contributions extend beyond simple microbial coexistence to actively shaping the skin microenvironment and influencing disease trajectories.

Integrating findings across multiple studies highlights consistent patterns in bacterial–fungal crosstalk. Quorum sensing, nutrient competition, and enzymatic activity emerge as recurring mechanisms by which bacteria influence fungal virulence. This crosstalk can either attenuate or exacerbate disease severity depending on the composition and activity of the microbiome, host immune status, and environmental conditions. For instance, the overgrowth of *S. aureus* in atopic dermatitis disrupts the microbial equilibrium, intensifying inflammation and increasing susceptibility to secondary infections[32].

Beyond these mechanisms, bacterial ecology particularly spatial distribution across skin niches such as sebaceous, moist, and dry environments plays a decisive role in determining microbial dominance and interaction outcomes. This ecological perspective aligns with findings presented in earlier sections of this review and reinforces the concept that disease manifestation is context-dependent rather than universally pathogen-driven.

Despite these advances, significant knowledge gaps remain. Many studies are observational, providing correlations without definitive mechanistic insight. The strain-specific effects of bacteria, their temporal dynamics, and the influence of host genetics on microbial interactions are still poorly understood. Additionally, most research has focused on a limited set of bacteria and fungi, leaving the role of less abundant species largely unexplored. Understanding these gaps is critical for designing targeted and effective interventions.

Another critical limitation is the insufficient integration of host-related factors, including immune signaling pathways, barrier integrity, and circadian rhythms, into current microbiome models. These host–microbe interactions are essential for understanding why similar microbial profiles may result in different clinical outcomes among individuals.

The evidence also supports the generation of novel hypotheses for future experimental studies. One potential avenue is to investigate how modulation of bacterial quorum sensing can control fungal virulence *in vivo*, using co-culture or animal models. Another hypothesis involves exploring the circadian rhythms of bacterial

activity to optimize timing of topical therapies. Additionally, manipulating bacterial metabolic pathways to indirectly suppress fungal growth could provide innovative therapeutic strategies. Integrating microbiome profiling with clinical outcomes may allow for the development of personalized microbiome-based treatments in superficial skin diseases[33].

Furthermore, emerging diagnostic approaches, such as VOC profiling and sequencing-based microbiome analysis, should be incorporated into future studies to establish functional links between microbial activity and disease phenotypes. These tools may enable early detection of dysbiosis and facilitate real-time monitoring of therapeutic responses.

From a therapeutic perspective, the findings of this review strongly support a paradigm shift toward microbiome-informed interventions. Strategies such as targeted probiotics, plant-derived antibacterials, quorum-sensing inhibitors, and chronotherapy are not merely adjunctive but may represent central components of future dermatological treatment frameworks. Importantly, combination approaches that integrate microbial, metabolic, and temporal targeting are likely to yield synergistic effects and improve long-term disease control.

Bacterial–fungal interactions are central to the pathogenesis and management of superficial dermatoses. Addressing the current knowledge gaps through mechanistic studies and innovative experimental designs will pave the way for more precise, effective, and microbiome-informed interventions.

This discussion reinforces the central thesis of the review: Bacteria are dynamic regulators of superficial skin disease ecology, interacting with fungi, the host, and the environment in a multidimensional network. Recognizing this complexity is essential for advancing both scientific understanding and clinical practice in dermatology.

5. Conclusions

The evidence presented in this review highlights the active and multifaceted role of bacteria in the development and progression of superficial skin diseases. Far from being passive inhabitants, bacterial communities interact dynamically with fungi, the host immune system, and environmental factors to influence disease onset, severity, and chronicity. Mechanisms such as quorum sensing, nutrient competition, enzymatic activity, and biofilm formation demonstrate that bacteria are central players in shaping the skin microenvironment and modulating fungal virulence.

In the context of clinical practice for health professionals, these findings support shifting from pathogen-focused algorithms toward microbiome-informed assessments that consider bacterial–fungal balance, skin barrier function, and environmental factors such as

pH, humidity, and hygiene practices. Regarding policy development and implementation, regulatory frameworks should incentivize the development of narrow-spectrum, microbiome-sparing topical agents, while clinical practice guidelines should be updated by dermatological societies to include probiotics, metabolic modulators, and chronotherapy as standard considerations rather than adjunctive options. For future research, priorities must include longitudinal mechanistic studies, strain-level analyses, and interventional trials that establish causality and translate microbial insights into precision dermatology across diverse populations.

Understanding these bacterial–fungal interactions provides critical insights that can enhance diagnostic and therapeutic strategies. Novel approaches, including enzymatic profiling, VOC detection, microbiome sequencing, probiotics, metabolic modulation, and chronotherapy, have the potential to transform patient care by targeting microbial imbalances rather than focusing solely on individual pathogens. By integrating microbiome knowledge into clinical practice, it is possible to develop personalized interventions that restore microbial equilibrium, reduce disease recurrence, and improve overall outcomes in superficial dermatoses.

Despite growing awareness, substantial knowledge gaps remain, including the strain-specific effects of bacteria, temporal dynamics of microbial interactions, and the influence of host genetics and environmental factors. Addressing these gaps requires multidisciplinary research, combining expertise in microbiology, dermatology, immunology, and therapeutic development. Collaborative efforts in these areas will not only elucidate the complex mechanisms of microbial crosstalk but also guide the design of innovative, targeted, and sustainable interventions.

Bacteria play an integral and dynamic role in superficial skin diseases. Future research that bridges microbiome science with clinical dermatology and therapeutic innovation holds the promise of redefining diagnosis and treatment paradigms, ultimately improving patient care and advancing our understanding of skin microbial ecology.

Conflict of interest statement

The authors declare that there is no conflict of interest.

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Data availability statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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