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Review Article

Unravelling the role of the skin microbiome in immunodermatological diseases: implications for therapeutic interventions

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ABSTRACT

The skin microbiome, comprising diverse microbial communities, is pivotal in maintaining cutaneous homeostasis and modulating immune responses in immunodermatological diseases. This review provides an overview of recent research investigating the interplay between the skin microbiome and autoimmune, allergic, and inflammatory skin conditions, such as psoriasis, eczema, and acne vulgaris. Current evidence suggests that alterations in the skin microbiome composition, termed dysbiosis, may contribute to disease pathogenesis and exacerbate inflammation in immunodermatological disorders. Furthermore, microbial-derived metabolites and immune-modulating factors produced by commensal bacteria can influence local immune responses and skin barrier function. Future research directions include evaluating how the skin microbiome interacts with the host immune system, identifying microbial biomarkers for disease diagnosis and prognosis, and exploring microbiome-targeted therapeutic interventions, such as probiotics, microbial transplantation, and microbial metabolite supplementation. By leveraging insights from microbiome research, personalized approaches to managing immunodermatological diseases may offer novel therapeutic avenues for restoring skin immune homeostasis and improving patient outcomes.

Keywords: Skin microbiome, Cutaneous homeostasis, Immune responses, Immunodermatological diseases, Psoriasis, Eczema, Acne vulgaris, Dysbiosis

INTRODUCTION

The skin microbiome is essential for maintaining skin homeostasis and modulating immune responses in immunodermatological diseases. This diverse microbial ecosystem includes fungi, bacteria, viruses, and mites that interact dynamically with the host's immune system, epidermal cells, and environmental factors to preserve skin integrity.[1] It functions as a physical barrier and produces key metabolites for biochemical processes. The microbiome's adaptability to environmental factors and genetic variations is closely linked to its ability to maintain skin health. By producing antimicrobial peptides and other bioactive molecules, the skin microbiome helps regulate inflammation, respond to external stimuli, and prevent pathogenic microbes from causing infections. While the gut microbiome is widely recognized for its contribution to overall health, research into the skin microbiome's role in immune regulation and disease susceptibility has emerged more recently. The skin microbiome is now understood to be a key player in maintaining immune balance in the skin. Its intricate interaction with the immune system helps protect against autoimmune, allergic, and inflammatory skin conditions. The composition of the skin microbiome is highly variable, differing significantly between individuals and

across different regions of the same person's skin. Only 3% of microbial phylotypes are shared among all individuals, with a mere 0.1% present on all body surfaces.[1] This variability is essential to the microbiome's adaptability and its ability to maintain skin homeostasis despite changing environmental and biological factors. Antimicrobial peptides (AMPs) and protease enzymes, produced by both the host and the skin's microbial communities, are crucial to maintaining the integrity of the stratum corneum.[2] When this delicate balance of microbial colonization is disrupted, a state of dysbiosis can arise, leading to a variety of dermatological issues.

Dysbiosis, marked by an imbalance in microbial populations, often involves the overgrowth of specific bacterial taxa such as Staphylococcus and Cutibacterium. This microbial imbalance exacerbates inflammation and compromises the skin's barrier function, contributing to the pathogenesis of various skin disorders.[3] Research has shown that each immunodermatological disease tends to be associated with a distinct microbial profile compared to healthy skin.[4] Understanding how microbial populations interact with the host immune system is crucial for identifying new therapeutic targets and maintaining skin homeostasis.

This review provides an overview of recent advancements in understanding the role of the skin microbiome in immunodermatological diseases. It examines how shifts in microbial composition contribute to the pathogenesis and exacerbation of immunodermatological diseases such as eczema, psoriasis, and acne vulgaris. By analyzing recent research, the review highlights the importance of specific bacterial taxa in the development of these diseases and underscores the significance of maintaining microbial balance for skin health. Additionally, potential therapeutic interventions targeting the microbiome, including probiotics, microbial transplantation, and microbial metabolite supplementation, are discussed as promising strategies for restoring microbial homeostasis and treating these complex skin disorders.

INTERPLAY BETWEEN THE SKIN MICROBIOME AND IMMUNODERMATO-LOGICAL DISEASES

Bidirectional communication and homeostasis

Bidirectional communication between the skin microbiome and the immune system is essential for maintaining homeostasis and effectively responding to environmental stimuli. Commensal microbes, such as Staphylococcus epidermidis and Cutibacterium acnes, play a pivotal role in modulating immune responses and maintaining microbial balance.[5] Conversely, opportunistic pathogens like Staphylococcus aureus can disrupt this equilibrium, leading to disease by overwhelming the host's defenses. The immune system continuously interacts with microbial signals to preserve this balance, protect the skin from infections, and promote overall skin health. Probiotics and prebiotics have shown the potential to enhance these interactions by modulating immune responses, protecting against ultraviolet radiation-induced damage, and improving therapeutic outcomes.[6]

The mycobiome, composed of diverse fungal communities, also plays a critical role in immune regulation and skin health. Dominant fungal populations, such as Malassezia species, engage in complex interactions with both innate and adaptive immune systems, supporting skin barrier integrity and modulating inflammatory responses. Dysbiosis within the fungal community can disrupt these interactions, leading to immune dysregulation and conditions such as atopic dermatitis, where fungi interact with other microbes and the host to exacerbate inflammation.[7,8]

Additionally, the microbiome contributes to homeostasis through the production of metabolites that mediate host-microbe communication, promote tissue repair, and prevent wound infections.[9,10] These metabolites serve as signaling molecules that not only help maintain a balanced microbial community but also influence immune cell activity, regulate inflammatory pathways, and reinforce the skin barrier's resilience against environmental insults. This dynamic interaction underscores the microbiome's multifaceted role in preserving the skin's integrity as a physical, chemical, and immune barrier.

Dysbiosis in skin diseases

Microbial dysbiosis plays a pivotal role in the development and progression of inflammatory and autoimmune skin diseases by disrupting skin barrier function and immune regulation. This imbalance impairs the skin's ability to maintain homeostasis, triggering inappropriate immune responses that contribute to conditions such as psoriasis, atopic dermatitis (AD), and acne vulgaris. Olejniczak-Staruch et al, identified a significant decrease in microbiome alpha-diversity and beta-diversity in psoriasis, characterized by a reduction in beneficial bacteria like

Cutibacterium and Lactobacilli and an increase in potentially pathogenic species such as *Corynebacterium kroppenstedtii*, Corynebacterium simulans, and Finegoldia species.[11] Furthermore, psoriasis lesions exhibit an increased abundance of Firmicutes and a reduction in Actinobacteria, with Prevotella and Staphylococcus species associated with lesional skin, while Anaerococcus and Propionibacterium are more prevalent in non-lesional areas.[12] These findings emphasize the importance of targeting dysbiosis in developing new treatments for psoriasis. Atopic dermatitis is strongly associated with dysbiosis of the skin microbiome, particularly characterized by reduced bacterial diversity and a significant increase in

Staphylococcus aureus colonization. This microbial imbalance plays a pivotal role in disease pathogenesis, as S. aureus disrupts the skin barrier and drives inflammation through the production of virulence factors such as toxins, enzymes, and adhesion molecules.[13] These virulence factors not only degrade the structural integrity of the skin but also activate the immune system, leading to the release of proinflammatory cytokines and perpetuation of the inflammatory cycle characteristic of AD. The inherent properties of AD-affected skin further exacerbate this dysbiosis. Reduced skin acidity, a hallmark of AD, undermines the natural antimicrobial environment, creating conditions conducive to *S. aureus* overgrowth.

Additionally, decreased production of antimicrobial peptides, such as cathelicidins and defensins, leaves the skin vulnerable to colonization and invasion by pathogenic bacteria.[14] This combination of reduced microbial diversity and impaired innate defenses establishes a self-reinforcing loop, *S. aureus* colonization worsens barrier dysfunction and inflammation, which in turn promotes further bacterial proliferation and immune dysregulation.

Acne vulgaris is a multifactorial inflammatory condition strongly influenced by microbial dysbiosis, particularly the overgrowth of Cutibacterium acnes within the pilosebaceous unit. This overgrowth disrupts the delicate balance of the skin microbiome, triggering inflammatory responses and altering immune regulation, both of which are central to disease pathogenesis.[15] C. acnes interacts with sebaceous lipids to release proinflammatory mediators, including cytokines and proteases, that exacerbate inflammation and contribute to the formation of acne lesions. The ability of C. acnes to persist and dominate the microbiome is key to its pathogenic role, driving both localized inflammation and broader immune dysregulation. Epigenetic mechanisms further amplify these inflammatory responses.

Short-chain fatty acids produced by C. acnes can induce histone acetylation in keratinocytes, leading to increased expression of proinflammatory genes.[16] These epigenetic changes create a pro-inflammatory environment that sustains the chronic inflammation observed in acne vulgaris. Additionally, genetic polymorphisms in pattern recognition receptor (PRR) genes, which encode receptors responsible for identifying microbial components, can impair the immune system's ability to appropriately recognize and respond to C. acnes.[16] This impairment intensifies the inflammatory cascade, contributing to the severity and persistence of the condition.

The intricate relationship between microbial dysbiosis, immune responses, and skin barrier dysfunction offers critical insights into the pathogenesis of inflammatory and autoimmune skin diseases. Beyond the discussed conditions of atopic dermatitis, psoriasis, and acne vulgaris, epidermal dysbiosis also contributes to the development and progression of other skin disorders, underscoring its widespread impact on dermatological health. Understanding these interactions provides a foundation for developing innovative, integrative therapeutic strategies that aim to restore microbial balance, fortify skin barrier integrity, and modulate immune responses. By targeting these foundational processes, treatments can move beyond symptom management to address the root causes of skin diseases, enhancing both their efficacy and the potential for long-term management and improved patient outcomes.

THE SKIN MICROBIOME'S INTERACTIONS

Understanding the dynamic relationship between the skin microbiome and cytokine production is essential for advancing therapeutic strategies in immuno-dermatological diseases. Certain destructive microbes stimulate the production of proinflammatory cytokines, such as interleukin-1-beta (IL-1β) and TNF-α, exacerbating conditions like AD.[17] Conversely, some microbial species promote anti-inflammatory cytokines, such as IL-10, highlighting the microbiome's dual role in balancing immune responses.[17] In psoriasis, dysbiosis disrupts this balance, reducing microbial diversity and amplifying proinflammatory cytokines like IL-17 and TNF-α, which drive keratinocyte hyperproliferation and the formation of characteristic scaly plaques.[18]

As research progresses, new strategies targeting the microbiome's role in cytokine production are emerging. For example, the link between oxidative stress and cytokine dysregulation in AD and psoriasis suggests that antioxidants may serve as adjunctive therapies.[19] Other innovative treatments, such as monoclonal antibodies and RNA-interfering molecules targeting cytokines like thymic stromal lymphopoietin (TSLP), IL-25, and IL-33, hold promise for modulating microbial communities and improving cytokine profiles. These therapies can complement existing treatments, particularly pruritis, which is closely tied to microbial imbalances and inflammatory responses. Additionally, a diverse microbiome protects against pathogenic colonization by occupying ecological niches and producing antimicrobial substances. Beneficial microbes establish themselves in various skin environments, such as oily, moist, or dry areas, outcompeting pathogens for space and nutrients and preventing infections.[20] This competitive exclusion is fundamental to maintaining skin health and immunity.

The skin microbiome's interactions extend beyond host immune response, with external factors, including environmental changes and therapeutic interventions, also playing a critical role. For instance, ultraviolet radiation (UVR) can alter microbial communities, encouraging the growth of UV-resistant bacteria. Rai et al, demonstrated that narrow-band UVB phototherapy qualitatively and quantitatively modulates the skin microbiome, influencing skin pathology and immune responses.[21]

Similarly, research by Isler et al, highlights the impact of environmental factors such as temperature, humidity, air pollution, and UV exposure on the microbiome.[22] These changes can disrupt microbial balance, exacerbating inflammatory responses and worsening conditions such as AD, acne vulgaris, psoriasis, and even skin cancer.

Topical antibiotics and antiseptics also significantly affect the skin microbiome, often with lasting consequences. SanMiguel et al found that topical antibiotic treatments, including triple antibiotic ointment (TAO), caused profound shifts in microbial communities, reducing beneficial Staphylococcus species while increasing opportunistic bacteria like *Enterobacteriaceae*.[23] This imbalance persisted for days post-treatment, increasing susceptibility to Staphylococcus aureus colonization and highlighting the unintended consequences of disrupting commensal populations. In contrast, antiseptics like alcohol and povidone-iodine had more modest effects on microbial communities, although they also reduced beneficial *Staphylococcus spp*., which normally compete with pathogenic *S. aureus*. These findings emphasize the importance of preserving microbial diversity when developing dermatological treatments to maintain skin health and immune function. By recognizing and mitigating environmental and therapeutic factors that disrupt the microbiome, it is possible to maintain a healthy microbial balance. This balance can prevent or mitigate skin conditions, offering a pathway to refined, microbiome-conscious interventions that strengthen the skin's resilience and improve patient outcomes.

MICROBIAL SPECIES AND THEIR ROLE IN SKIN HOMEOSTASIS

The skin microbiome comprises a diverse array of microbial species, each contributing uniquely to maintaining skin homeostasis and health. Noteworthy species include bacteria, such as *Staphylococcus epidermidis* and *Corynebacterium kroppenstedtii*, and fungi like Malassezia species playing pivotal roles in maintaining skin health. These microorganisms contribute to barrier integrity, immune regulation, and protection against pathogens. *Staphylococcus epidermidis* supports antimicrobial defense and immune homeostasis, while *Corynebacterium kroppenstedtii* and Malassezia have more complex roles, sometimes acting as commensals and at other times contributing to inflammatory conditions. Examining the specific functions of these key microbial species provides deeper insight into their contributions to skin health and their potential as therapeutic targets.

Staphylococcus epidermidis

Staphylococcus epidermidis, a ubiquitous gram-positive commensal bacterium, is essential for maintaining skin health and immune homeostasis. This bacterium contributes to antimicrobial defense by producing antimicrobial peptides (AMPs) such as β-defensins and cathelicidins, which inhibit the growth of harmful bacteria and fungi while recruiting immune cells to infection sites.[24,25] These peptides not only enhance skin defenses but also regulate immune responses, preventing excessive inflammation. *S. epidermidis* also promotes the development of regulatory T cells (Tregs), which suppress immune overactivation and maintain equilibrium. Merena et al, demonstrated that *S. epidermidis* induced Tregs play a critical role in reducing inflammation, with colitis-linked reductions in these Tregs leading to increased tissue damage and neutrophil infiltration in the skin, further emphasizing the bacterium's importance in immune modulation.[26]

Beyond immune regulation, *S. epidermidis* plays a vital role in maintaining the structural integrity of the skin barrier. It stimulates the production of tight junction proteins, which seal gaps between epidermal cells and fortify the physical barrier.[27] Furthermore, short-chain fatty acids (SCFAs) such as butyrate, propionate, and acetate, produced through bacterial fermentation, enhance skin barrier resilience by reducing inflammation and promoting keratinocyte differentiation.[28,29] SCFAs also modulate the skin microenvironment, such as pH levels, creating conditions favorable for beneficial bacteria like *S. epidermidis* while suppressing pathogens. This symbiotic relationship underscores the bacterium's role in maintaining skin health, with disruptions increasing susceptibility to infections and inflammatory conditions.

Despite its commensal nature, *S. epidermidis* can transition to a pathogenic state under specific conditions, particularly in immunocompromised individuals or when medical devices are involved. It is a common cause of nosocomial infections, including periprosthetic joint infections, due to its ability to form biofilms that protect against immune responses and antibiotic treatment.[30] Additionally, S. epidermidis produces phenol-soluble modulins (PSMs), which can induce inflammation and exacerbate skin conditions such as atopic dermatitis.[31] PSMs not only disrupt host cell membranes but also promote neutrophil recruitment and inflammatory cytokine release, compounding tissue damage and exacerbating immune dysregulation. These effects underscore the duality of *S. epidermidis* as both a protector and potential aggressor, emphasizing the importance of context in determining its role within the skin microbiome. These pathogenic tendencies highlight the complexity of its dual roles and the fine balance required to maintain its beneficial contributions without tipping into disease states.

Corynebacterium kroppenstedtii

Corynebacterium kroppenstedtii is an integral member of the skin microbiome with dual roles in promoting skin health and contributing to disease under specific circumstances. This lipophilic, gram-positive rod is part of the diverse Corynebacterium genus, which plays a key

role in maintaining microbial diversity and supporting skin barrier integrity.[32] While generally considered a commensal organism, *C. kroppenstedtii* has been implicated in several inflammatory conditions, most notably granulomatous mastitis and its association as an endobacterium of Demodex folliculorum mites.

Granulomatous mastitis, a rare inflammatory breast condition, is characterized by the formation of granulomas-immune cell aggregates composed of macrophages and lymphocytes-that aim to isolate pathogens like C. kroppenstedtii.[33] Although granulomas initially serve as protective structures, persistent immune activation can lead to tissue damage and chronic inflammation.

Using histological and microbiological analyses, Johnstone et al, confirmed the presence of *C. kroppenstedtii* in granulomatous mastitis, underscoring its pathogenic potential. This bacterium's ability to trigger granuloma formation highlights its complex role in both defense against infection and contribution to disease. Targeting *C. kroppenstedtii* or modulating the immune response could offer therapeutic strategies to mitigate excessive granuloma formation and associated tissue damage, providing insights into managing other chronic inflammatory diseases.

Beyond its role in granulomatous mastitis, *C. kroppenstedtii* has been identified as the endobacterium of *Demodex folliculorum* mites, which are associated with inflammatory skin conditions such as rosacea.[34] This relationship underscores the intricate interplay between microbial and parasitic elements in the skin microbiome and their potential impact on human health. *C. kroppenstedtii* exhibits strain-specific biochemical traits, such as cobamide biosynthesis and antibiotic resistance profiles, which influence microbial community dynamics and therapeutic outcomes.[35] Understanding the dual nature of *C. kroppenstedtii* as a commensal organism and a potential pathogen highlights the broader complexities of the skin microbiome.

Its involvement in both maintaining skin health and driving inflammatory processes emphasizes the need for targeted therapeutic approaches. By unraveling the interactions between *C. kroppenstedtii*, the immune system, and other microbial and parasitic players, researchers can develop strategies to prevent and treat associated diseases while preserving overall skin homeostasis.

Malassezia species

Malassezia species are lipid-dependent fungi that play a complex dual role in the skin microbiome as both commensals and potential pathogens. Under normal conditions, Malassezia contributes to skin health by metabolizing lipids and producing free fatty acids, which reinforce the skin barrier and protect against microbial invasion. However, under certain circumstances, such as environmental changes or immune dysregulation, Malassezia can overgrow and transition to a pathogenic state. This shift contributes to inflammatory skin conditions like seborrheic dermatitis, psoriasis, and atopic dermatitis by producing inflammatory metabolites, such as oleic acid and arachidonic acid, that irritate the skin, promote keratinocyte proliferation, and trigger immune dysregulation.[36]

Furthermore, Malassezia interacts with immune cells, including dendritic cells and Langerhans cells, to amplify pro-inflammatory cytokine production, exacerbating inflammation in predisposed individuals.[37] Despite its potential for pathogenicity, Malassezia also plays a protective role in maintaining skin homeostasis. It can inhibit biofilm formation of pathogens like *Staphylococcus aureus* through the secretion of aspartyl protease 1 (MgSAP1), reducing the risk of bacterial infections.[38] This balance between its commensal and pathogenic states is influenced by host-microbe interactions and environmental factors, suggesting that targeting Malassezia's lipid metabolism or modulating the immune response could restore skin barrier function and reduce inflammation in affected individuals.

POTENTIAL THERAPEUTIC AVENUES IN IMMUNODERMATOLOGICAL DISEASES

Microbiome-based therapeutics

Harnessing the microbiome for therapeutic purposes offers promising strategies to address the underlying dysbiosis often implicated in immunodermatological diseases like psoriasis, atopic dermatitis, and acne. Probiotics, microbial transplantation, and microbial metabolite supplementation target the skin and gut microbiomes to restore microbial balance and modulate immune responses. For example, probiotics, live microorganisms administered in appropriate doses, have demonstrated the ability to reduce inflammatory markers such as IL-6, TNF-α, and CRP in psoriasis patients, contributing to symptom reduction and decreased recurrence rates.[39,40]

Additionally, probiotics improve skin barrier function and alleviate inflammation, benefiting conditions like eczema and acne. Clinical trials have shown that topical probiotics effectively reduce Staphylococcus aureus colonization in AD, increasing microbial diversity and reducing disease severity with minimal side effects.[41] These findings emphasize the importance of rebalancing the microbiota as a critical component of managing inflammatory disease severity.

Beyond probiotics, microbial metabolites such as short-chain fatty acids (SCFAs), tryptophan metabolites, and trimethylamine N-oxide (TMAO) play a systemic role in modulating immune responses.[42] These metabolites influence immune cell profiles and cytokine production,

creating a ripple effect on both local skin inflammation and systemic immune balance. By integrating microbial metabolite supplementation into treatment regimens, clinicians can address both the localized and systemic dimensions of dermatological diseases. These advances highlight the potential of microbiome-based therapies to complement existing treatments and provide a more holistic approach to managing immunodermatological conditions.

Personalized approaches to treatment

The advent of precision medicine has introduced a new dimension to managing immunodermatological diseases, emphasizing personalized approaches tailored to individual patient profiles. Genomic assessments of the human microbiome, combined with high-throughput immune profiling technologies such as transcriptomics, epigenomics, metabolomics, and proteomics, offer new diagnostic and therapeutic possibilities.[43,44] These approaches enable clinicians to identify biomarkers associated with disease risk and progression, facilitating early intervention and targeted treatment strategies. In psoriasis, for instance, precision medicine would allow for the identification of biomarkers to predict optimal biologic therapies, moving away from trial-and-error methods and improving both patient outcomes and satisfaction.

Machine learning and integrative multi-omics platforms further enhance the capacity for treatment personalization by analyzing patient-specific data to refine risk stratification and therapeutic decisions.[45] By incorporating these advanced tools, clinicians can better understand disease mechanisms, predict treatment responses, and optimize therapeutic regimens. This shift towards personalized medicine represents a paradigm change, offering more effective, targeted interventions that not only improve patient outcomes but also reduce the burden of unnecessary or ineffective treatments. Integrating microbiome-based therapies with precision medicine could revolutionize the management of immunodermatological diseases, setting the stage for a future where treatments are as unique as the patients they are designed for.

FUTURE RESEARCH DIRECTIONS

Future research in immunodermatological diseases should prioritize investigating specific mechanisms driving the disease using in vitro and in vivo models to advance our understanding of the molecular pathways involved in skin microbiome-host interactions. In vitro models, including organotypic and three-dimensional (3D) skin cultures, can uncover significant changes in epidermal thickness, differentiation, and barrier formation.[46] In vivo studies, particularly those using mouse models, are essential for observing changes in transcriptional profiles in response to microbial communities, focusing on genes involved in apoptosis, proliferation, and extracellular matrix regulation.[47] Genetic studies should identify host genes influencing skin microbiota through genome-wide association studies (GWAS), highlighting loci involved in innate immune signaling, environmental sensing, and cell differentiation.[48] Interspecies protein-protein interaction (PPI) data can further identify bacterial proteins interacting with human proteins, implicating various signaling pathways in diseases.

Emerging technologies like single-cell RNA sequencing (scRNA-seq) and spatial transcriptomics (ST) are furthering the understanding of cellular and molecular dynamics at the skin microbiome-immune interface. These technologies allow for a 3-dimensional spatial analysis of inflammatory skin conditions, including the location and distribution of various inflammatory cytokines.[49]

For example, recent studies combining single-cell and spatial RNA sequencing have provided in-depth views of psoriasis pathogenesis, revealing the role of specific fibroblast subsets in amplifying immune responses through ligand-receptor interactions with other cell types.[49] These technologies show great potential across many areas of dermatology, also assessing wound healing and skin cancer profiles. By understanding each patient's skin's unique cellular and molecular landscape, clinicians can tailor treatments to target specific pathways and cell types involved in their disease, improving treatment efficacy and patient outcomes. Future research should also focus on identifying and validating microbial biomarkers for predicting disease risk, severity, and treatment response in immunodermatological conditions.

Previous work like this has been done relating the gut microbiome and immunodermatological diseases.[50] The microbial risk score (MRS) framework can aggregate complex microbial profiles into a summarized risk score, predicting disease susceptibility and integrating multi-omics data to understand disease mechanisms comprehensively.[50] Although specific biomarkers in routine clinical practice for diseases like psoriasis are currently lacking, ongoing research and development of multi-omics approaches hold promise for improving patient management and outcomes by providing objective indications for disease prognosis and therapeutic response.

CONCLUSION

The skin microbiome maintains cutaneous homeostasis and modulates immune responses in immunodermatological diseases such as psoriasis, eczema, and acne vulgaris. The complex interplay between microbial communities and the host's immune system underscores the importance of maintaining a balanced microbiome to prevent and manage inflammatory skin conditions. Dysbiosis, characterized by microbial imbalances, contributes to disease

pathogenesis and exacerbates inflammation, highlighting the need for targeted microbiome therapies. Particular microbes, such as *Staphylococcus epidermidis*, *Corynebacterium kroppenstedtii*, and Malassezia species, play a significant role in the occupation of cutaneous niches, maintaining a healthy flora and preventing the overgrowth of pathogens.

Future research should focus on elucidating the specific mechanisms by which the skin microbiome influences immune responses and inflammation, using advanced in vitro and in vivo models. The potential of microbiome research to inspire new treatments and approaches is immense, potentially leading to more precise and effective treatments. Identifying and validating microbial biomarkers for disease diagnosis and prognosis is essential for advancing personalized medicine in dermatology.

Longitudinal studies and multi-omics approaches will be pivotal in capturing the dynamic nature of the skin microbiome and its impact on disease progression and treatment response. Overall, leveraging insights from microbiome research offers novel therapeutic avenues for restoring skin immune homeostasis and improving patient outcomes. Continued interdisciplinary research and clinical trials will be crucial in translating these findings into practical, personalized, and sustainable treatments for immunodermatological diseases.

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