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Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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THE INFLUENCE OF SKIN MICROBIOME IMBALANCE ON SPECIFIC PATHOLOGICAL SKIN CONDITIONS

Michał Wycik *(Corresponding Author, Email: michalwycik98@gmail.com)*
University Clinical Hospital in Białystok, Białystok, Poland
ORCID ID: 0009-0007-3052-9924

Hubert Chmielewski
Medical University of Białystok, Białystok, Poland
ORCID ID: 0009-0000-5697-518X

Julia Komar
Medical University of Białystok, Białystok, Poland
ORCID ID: 0009-0008-9104-7934

Artur Wądołowski
University Clinical Hospital in Białystok, Białystok, Poland
ORCID ID: 0009-0006-0261-581X

Gabriela Kondratiuk
Medical University of Białystok, Białystok, Poland
ORCID ID: 0009-0009-4181-731X

Ada Kondrat
University Clinical Hospital in Białystok, Białystok, Poland
ORCID ID: 0009-0008-0823-0544

Michał Szczupak
University Clinical Hospital in Białystok, Białystok, Poland
ORCID ID: 0009-0005-5112-6978

Paweł Mierzejewski
University Clinical Hospital in Białystok, Białystok, Poland
ORCID ID: 0009-0009-2004-9972

Justyna Werno
University of Zielona Góra, Zielona Góra, Poland
ORCID ID: 0009-0002-9681-3323

Zuzanna Twarowska
University Clinical Hospital in Białystok, Białystok, Poland
ORCID ID: 0009-0006-8662-099X

ABSTRACT

The human skin microbiome, comprising bacteria, fungi, viruses, and other microorganisms, plays a crucial role in maintaining skin homeostasis, modulating immune responses, and protecting against pathogens. Emerging research highlights that disruptions in microbial balance - dysbiosis - contribute to the pathogenesis of various dermatological conditions, including atopic dermatitis, psoriasis, acne vulgaris, rosacea, skin cancer, and impaired wound healing. Dysbiosis arises from genetic, environmental, and therapeutic factors, leading to altered barrier function, chronic inflammation, and disease exacerbation.

In inflammatory disorders like atopic dermatitis and psoriasis, pathogenic overgrowth (e.g., Staphylococcus aureus) and reduced microbial diversity drive immune dysregulation. Similarly, acne and rosacea are influenced by shifts in Cutibacterium acnes phylotypes and Demodex-associated bacteria, which promote inflammation and biofilm formation. The skin microbiome also impacts skin cancer progression by modulating DNA damage responses and immunotherapy efficacy, while chronic wounds exhibit delayed healing due to pathogenic dominance and biofilm persistence.

Understanding the intricate host-microbiome interplay offers transformative potential for dermatology, paving the way for precision medicine interventions that harness microbial ecology to enhance skin health and treat disease.

KEYWORDS

Skin Microbiome, Dysbiosis, Atopic Dermatitis, Psoriasis, Acne, Rosacea, Skin Cancer, Wound Healing, Microbiome Therapeutics, Microbial Imbalance

CITATION

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Introduction

The human skin constitutes a complex ecosystem inhabited by a diverse array of microorganisms, including bacteria, fungi, and viruses, collectively referred to as the skin microbiota. Under physiological conditions, these microorganisms exist in a balanced symbiotic relationship with the host, playing essential roles in maintaining cutaneous homeostasis, protecting against pathogenic invasion, and modulating immune responses. In recent years, advancements in sequencing technologies have greatly enhanced our understanding of the composition and functional dynamics of the skin microbiome. Research has demonstrated that disturbances in this microbial equilibrium, known as dysbiosis, can contribute to the onset or exacerbation of various dermatological disorders. Dysbiosis may arise from intrinsic factors such as genetic predispositions or immune dysregulation, as well as extrinsic influences including antibiotic overuse, detergent exposure, and environmental pollutants. Alterations in the composition of the skin microbiome can impair barrier function, trigger aberrant immune responses, and promote chronic inflammation. The emerging concept of the "microbiome–skin axis" has gained prominence in dermatological research, emphasizing the bidirectional communication between microbial communities and the skin. A deeper understanding of these interactions opens new avenues for diagnosing and treating skin diseases. Despite notable progress, significant challenges remain, including the need for standardized research methodologies and improved insights into causal relationships within microbial ecology. This paper presents a comprehensive review of current knowledge on the role of the skin microbiota in health and disease, addressing both fundamental mechanisms and potential clinical applications. The analysis integrates molecular findings, clinical evidence, and therapeutic trials to provide a balanced overview of this rapidly evolving area in dermatology.

Material and Methods

A comprehensive review of the literature was conducted using the PubMed and Google Scholar databases, focusing on studies examining the relationship between skin microbiota dysbiosis and the development, progression, and management of dermatological diseases. The bibliographic search employed a combination of keywords, including: skin microbiota, skin microbiome, cutaneous dysbiosis, dermatological diseases, atopic dermatitis, psoriasis, acne vulgaris, rosacea, microbial-host interactions, skin barrier function, microbiome modulation, probiotics, prebiotics, phage therapy, skin cancer, wound, wound healing, and microbiome transplantation. The search was restricted to peer-reviewed, freely accessible articles published in English between 2005 and 2025. Both observational and interventional studies were considered, with particular attention given to clinical trials, mechanistic investigations, and systematic reviews. This methodological approach enabled a critical evaluation of the current understanding of skin microbiome alterations in dermatological conditions, as well as the emerging therapeutic strategies aimed at correcting microbial imbalances.

Structure and Function of the Epidermal Barrier

The epidermis consists of multiple specialized layers that work together to maintain the skin's protective barrier. At the base lies the basal layer, which contains keratinocyte stem cells attached to the basement membrane. These cells undergo asymmetric division, with some remaining as stem cells while others differentiate and migrate upward through the epidermal layers. [1]

As keratinocytes move up through the stratum spinosum, they change into a polyhedral shape and create strong intercellular connections with desmosomes, thus maintaining the structural integrity. When they get to the stratum granulosum, the cells become thinner and give off keratohyalin granules, which help to crosslink keratin filaments to create a waterproof barrier. The stratum corneum, the uppermost layer, consists of closely packed cells without nuclei that are surrounded by a lipid matrix. These corneocytes go through a process of constant shedding and replenishing in a period of four weeks. It is the main process to keep the skin barrier function. [1]

Skin Microbiome Composition and Diversity

The skin harbours a rich and diverse community of bacteria, viruses, fungi, and other microorganisms, collectively forming a sophisticated ecosystem that transcends conventional pathogen-centric views. Advanced DNA sequencing technologies have uncovered striking variations in the skin microbiome across different body sites and individuals, while also demonstrating its remarkable stability over time. These insights have redirected scientific focus toward elucidating the functional contributions of resident microbes to both skin homeostasis and disease pathogenesis. [2]

The human skin supports a sophisticated microbial ecosystem whose composition varies markedly across anatomical regions, reflecting local variations in humidity, temperature, pH, and antimicrobial peptide concentrations. 16S rRNA sequencing reveals four dominant bacterial phyla: Actinobacteria (51.8%), Firmicutes (24.4%), Proteobacteria (16.5%), and Bacteroidetes (6.3%). At the genus level, distinct ecological niches emerge: sebaceous areas (e.g., face) show predominance of Propionibacterium and Staphylococcus, moist regions (e.g., axilla) are characterized by Corynebacterium dominance, while dry sites harbour diverse communities of β-Proteobacteria and Flavobacteriales. [2]

Comparative methodological analyses indicate that while culture-dependent and sequencing approaches produce consistent results for resilient microbial species, molecular techniques uncover significantly greater diversity through detection of non-viable and unculturable organisms. Ecological characterization reveals that skin microbiota display moderate α-diversity but exceptionally high β-diversity relative to other epithelial surfaces. This pattern reflects pronounced inter-individual variation that demonstrates remarkable temporal stability. [2]

Beyond bacteria, the skin microbiome includes [2]:

1. Fungi: Predominantly lipophilic Malassezia species (*M. globosa, M. restricta, M. sympodialis*) in sebum-rich areas, with greater diversity on the feet (*Aspergillus, Rhodotorula, Cryptococcus*)
2. Arthropods: Demodex mites (*D. folliculorum* in hair follicles, *D. brevis* in sebaceous glands)
3. Viruses: Poorly characterized due to detection challenges, though emerging metagenomic studies reveal diverse DNA viruses, including potentially commensal human papillomaviruses

The Skin Microbiome as a Systemic Regulator

The skin microbiome, comprising diverse bacteria, fungi, and viruses, serves as a crucial interface between the body and its external environment. Beyond its well-established role in maintaining skin barrier function, emerging evidence highlights its systemic regulatory capacity, influencing both cutaneous and whole-body physiology. Functionally, the skin microbiome contributes to host defence through three primary mechanisms: physical barrier reinforcement by stimulating tight junction production, pathogen exclusion via microbial competition and antimicrobial peptide secretion, and immune system modulation through complex host-microbe interactions. These interactions primarily occur through pattern recognition receptor (PRR) activation, which maintain immune homeostasis by balancing pro- and anti-inflammatory responses. Dysbiosis of this delicate ecosystem has been linked to various dermatological conditions, including atopic dermatitis, psoriasis, and acne vulgaris. More significantly, such microbial imbalances may contribute to systemic disorders, particularly autoimmune diseases, through mechanisms involving aberrant immune activation and loss of tolerance. The microbiome's systemic influence extends through multiple pathways, including direct microbial metabolite circulation, immune cell priming, and cross-talk with other barrier sites. [3]

The skin microbiome in Atopic Dermatitis

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder characterized xerosis, pruritus, and eczematous lesions. The pathogenesis involves a triad of factors: genetic predisposition, particularly loss-of-function mutations in the filaggrin (FLG) gene that disrupt stratum corneum integrity, epidermal barrier dysfunction permitting enhanced allergen penetration and type 2 immune polarization with elevated Th2 cytokine production. This immunologic cascade results in chronic inflammation [4]. Environmental triggers, such as allergens and microbes, can worsen the condition by disrupting the skin microbiome and further impairing the barrier [5]. Clinically, AD presents as pruritic, erythematous patches, often leading to chronic scratching and skin thickening.

The cutaneous microbiome is essential for maintaining epidermal barrier integrity and immune regulation, with its disruption being a well-established contributor to atopic dermatitis (AD) pathogenesis. Robust clinical evidence demonstrates that AD flares are consistently associated with two key microbial alterations: significantly diminished bacterial diversity and selective overcolonization by Staphylococcus aureus. [6], [7] A healthy skin microbiota acts as a natural defence barrier, producing antimicrobial peptides and modulating inflammatory pathways [8]. In AD, colonization with *S. aureus* exacerbates barrier dysfunction and promotes a pro-inflammatory Th2 response [9]. Recent findings underline the importance of commensal species, such as *Staphylococcus epidermidis* and *Cutibacterium acnes*, in suppressing *S. aureus* overgrowth and supporting skin health. [4], [10], [11]

Dysbiosis is not only quantitative but also qualitative. For example, metagenomic analyses show functional impairments in the production of skin-protective molecules in AD patients [12]. Moreover, the infant skin microbiome is particularly susceptible to dysbiosis, suggesting early-life microbial imbalances could predispose individuals to AD [13].

Interestingly, host genetic factors, such as filaggrin mutations, interact with the microbiome, further impairing barrier integrity. [14]

Immune modulation by the microbiota involves intricate interactions between the innate and adaptive immune systems. Commensal microorganisms can promote the activation of regulatory T cells, thereby suppressing inflammation, while a dysbiotic microbiota enhances Th2, Th17, and ILC2-mediated immune responses. [15]

Recent studies explore innovative therapeutic avenues:

- Topical application of commensals such as *S. hominis* to outcompete *S. aureus*. [16]
- Microbiome transplantation and bacteriophage therapy targeting *S. aureus*. [8]
- Use of postbiotics and prebiotics to selectively modulate the skin microbiome. [10]

Moreover, an important observation is that treatment responses in AD (to corticosteroids or biologics) often correlate with restoration of a healthy microbiome. [17], [18]

Data also suggest that probiotic supplementation could offer systemic modulation of skin immunity, although evidence remains mixed. [7]

Newer metagenomic studies indicate that not just the presence, but the metabolic output of microbiota shapes disease outcomes. [9], [12]

Finally, longitudinal birth cohort studies are shedding light on how early-life environmental exposures and microbiome shifts predict AD risk. [11], [13]

Despite these advances, significant challenges remain, including high inter-individual variability, regional microbiome differences, and difficulties in distinguishing cause from effect. [14]

Future research must prioritize:

- Stratified clinical trials based on microbiome profiles
- Functional assays beyond taxonomic surveys
- Longitudinal studies tracking microbiome evolution and treatment responses [13], [17]

A deeper understanding of the skin microbiome's involvement in the pathogenesis of atopic dermatitis (AD) paves the way for personalized medicine and targeted therapies focused on sustained disease control. Reduced microbial diversity plays a key role in exacerbating AD-associated itch through non-histaminergic pathways, compromised skin barrier function, and central sensitization. Interventions like probiotics and microbiome modulation have shown promise in relieving itch, highlighting innovative therapeutic possibilities. Continued research into the interplay between microbiota and itch may uncover new targets for improving AD treatment. [16]

Psoriasis and skin microbiome

Psoriasis is a chronic, immune-mediated skin disorder marked by hyperproliferation and aberrant differentiation of keratinocytes. Its pathogenesis is driven by a dysregulated immune response, particularly involving the activation of dendritic cells and T lymphocytes, which leads to the overproduction of proinflammatory cytokines such as TNF-α, IL-17, and IL-23 [19], [20]. Genetic susceptibility and environmental factors such as infections and physical trauma contribute to both the onset and exacerbation of the disease [21]. Clinically, psoriasis manifests as well-demarcated, erythematous plaques with silvery scales, predominantly affecting the elbows, knees, scalp, and lower back. Nail involvement is common and includes pitting, onycholysis, and subungual hyperkeratosis, reflecting the systemic nature of the condition [22]. Recent research has emphasized the pivotal role of the skin microbiome in the development and progression of psoriasis. Dysbiosis, or disruption of the microbial equilibrium on the skin, is increasingly recognized as a contributing factor in disease pathogenesis, underscoring the intricate interplay among genetic, environmental, immune, and microbial components [19].

Multiple studies have demonstrated that psoriatic lesions exhibit reduced microbial diversity compared to healthy skin, suggesting a loss of microbial homeostasis [23]. Notably, the abundance of beneficial commensal bacteria such as *Cutibacterium acnes* (formerly *Propionibacterium acnes*) is diminished, while pathogenic genera such as *Streptococcus* and *Staphylococcus*, particularly *Staphylococcus aureus*, are elevated [24]. This microbial shift fosters a proinflammatory milieu through the heightened activation of both innate and adaptive immune responses [12].

Microbial dysbiosis is believed to initiate and sustain immune activation via key pathways, notably the IL-23/Th17 axis, which is central to psoriatic inflammation [25]. Pathogens like *S. aureus* can produce superantigens that non-specifically activate T cells, thereby amplifying the inflammatory cascade [26]. Moreover, bacterial components such as lipoteichoic acid and peptidoglycan from Gram-positive bacteria further stimulate toll-like receptor (TLR) signalling, promoting sustained keratinocyte activation [27].

The fungal microbiome, or mycobiome, also plays a significant role in psoriasis. While *Malassezia* species dominate healthy skin, their role in psoriatic disease remains complex. Some studies suggest that Malassezia may promote psoriatic inflammation through cytokine induction [17], others propose that different *Malassezia* strains might exert protective effects under specific conditions [28]. These findings imply that the relationship between fungal communities and the host is finely tuned and context-dependent.

In addition to the skin microbiota, the gut-skin axis has emerged as an important factor in psoriasis pathophysiology. Alterations in gut microbial composition, particularly a decrease in beneficial species like *Faecalibacterium prausnitzii*, have been linked to systemic inflammation and worsening of skin symptoms [26]. These findings suggest that both local (skin) and distant (gut) microbial communities are implicated in disease dynamics.

Therapeutic strategies targeting the microbiome are gaining attention as a novel approach to managing psoriasis. Biologic agents, such as anti-IL-17 and anti-TNF-α therapies, not only improve skin lesions but also appear to normalize the skin microbiome, indicating that microbial composition may serve as a biomarker of treatment response [29]. Furthermore, emerging therapies, including topical probiotics and microbiome transplants, are being explored for their potential to directly modulate microbial communities and attenuate inflammation [30].

Several studies have also found that reduced bacterial diversity, along with an overrepresentation of *Firmicutes* and underrepresentation of *Actinobacteria*, correlates with disease severity [17]. Restoring microbial diversity through targeted interventions could therefore represent a promising therapeutic strategy [31].

Lastly, recent evidence underscores the importance of host–microbiota interactions in maintaining skin immune homeostasis. Dysbiotic microbial communities can compromise skin barrier function, facilitating the entry of environmental antigens and aggravating inflammation [11]. This dual role of the microbiome, as both a protector and potential promoter of disease, highlights its importance in psoriatic pathogenesis, depending on microbial composition and barrier integrity.

In summary, psoriasis is increasingly understood not solely as a result of genetic predisposition and immune dysregulation, but also as a condition significantly influenced by both the skin and gut microbiomes. Deeper insights into these complex interactions offer promising avenues for future microbiome-targeted therapies aimed at restoring microbial balance and improving disease outcomes.

The influence of skin microbiota on the course of acne and rosacea

Acne vulgaris is a common multifactorial skin condition primarily affecting the pilosebaceous unit. Its complex pathogenesis involves excess sebum production, follicular hyperkeratinization, hormonal factors, immune dysregulation, and microbial colonization, most notably by *Cutibacterium acnes* (*C. acnes*) [25]. Formerly known as *Propionibacterium acnes*, *C. acnes* is a commensal bacterium that contributes to skin homeostasis under normal conditions by modulating immune responses and supporting barrier integrity [12]. However, dysbiosis, defined as an imbalance in the microbial ecosystem, can shift *C. acnes* from a symbiotic to a pathogenic role [32].

Acne-related dysbiosis is characterized by a decline in microbial diversity and an overabundance of virulent *C. acnes* phylotypes, particularly those of the IA1 lineage [33], [34]. These strains activate innate immune receptors such as Toll-like receptor 2 (TLR2), inducing proinflammatory cytokines including IL-1β, IL-8, and TNF-α[11], [35]. The ability of *C. acnes* to form biofilms exacerbates inflammation by enhancing bacterial resistance to antibiotics and shielding the microbes from host immune defences [36].

Concurrently, a reduction in beneficial commensals such as *Staphylococcus epidermidis* impairs protective mechanisms. *S. epidermidis* inhibits *C. acnes* pathogenicity through the production of antimicrobial peptides (AMPs) and competitive biofilm formation [37]. Loss of these protective species disrupts cutaneous immune balance and promotes an environment conducive to acne development [38].

Beyond its role in initiating disease, dysbiosis also affects acne severity and response to treatment. Persistent microbial imbalance contributes to chronic inflammation, while antibiotic therapies targeting *C. acnes* may inadvertently eliminate protective commensals, further exacerbating dysbiosis [39]. Increasing resistance of *C. acnes* to commonly used antibiotics such as clindamycin and erythromycin underscores the need for microbiome-sparing alternatives [40], [41].

Emerging therapies aim to modulate rather than eradicate the skin microbiota. Bacteriophage therapy, targeting specific pathogenic *C. acnes* strains, has shown promise in selectively rebalancing the microbiome without disrupting beneficial species [42]. Topical probiotics, including *Lactobacillus plantarum* and *Bifidobacterium* spp., demonstrate anti-inflammatory effects and promote skin barrier repair [17], [43].

Advances in next-generation sequencing (NGS) and metagenomics have provided deeper insight into the functional consequences of microbial shifts. Acne-associated microbiota exhibit increased activity in fatty acid metabolism, quorum sensing pathways, and oxidative stress responses, all of which contribute to comedogenesis and inflammation [34], [36].

The gut-skin axis also plays a crucial role in acne pathophysiology. Gut dysbiosis can drive systemic inflammation via microbial metabolites such as lipopolysaccharides (LPS) and secondary bile acids [32], [44]. Altered gut microbial profiles have been correlated with acne severity, suggesting that oral probiotics and dietary interventions may help regulate systemic immunity and support acne treatment [45], [46].

In addition to acne, microbial dysbiosis is implicated in the pathogenesis of rosacea. Elevated *Demodex* mite density and accompanying bacterial imbalance, particularly involving *Bacillus oleronius*, can trigger chronic inflammation through TLR2 activation and abnormal cathelicidin expression [47]. Treatments that reduce *Demodex* load, such as topical ivermectin, help restore microbial balance and reduce inflammation [40].

Therapeutic impacts on the microbiome are an important consideration. Isotretinoin, a gold-standard treatment for severe acne, not only decreases sebum production but also significantly alters the skin microbiota: reducing *C. acnes* density and enhancing microbial diversity [48]. However, due to its systemic effects, isotretinoin therapy requires careful monitoring of microbiome-related changes [49].

Additionally, microbial metabolites such as short-chain fatty acids (SCFAs), produced by commensal anaerobes, play an essential role in modulating skin inflammation and maintaining barrier integrity. SCFAs have been shown to strengthen the epidermal barrier and suppress inflammation, whereas dysbiosis-driven SCFA imbalances may impair these protective effects and sustain inflammatory lesions [50].

In conclusion, the skin microbiome plays a vital and multifaceted role in the development, progression, and treatment of acne vulgaris and rosacea. Dysbiosis, marked by reduced microbial diversity and pathogenic overgrowth, drives both local and systemic inflammation. Future acne therapies are likely to focus on restoring microbial homeostasis rather than broadly eliminating microbial populations. Ongoing research into microbiome-based interventions and personalized dermatological approaches will be essential for improving therapeutic outcomes and patient care [11], [35], [38], [51].

The Skin Microbiome and Skin Cancer

The skin microbiome plays a pivotal role in the pathogenesis of skin cancers, with growing evidence pointing to a complex interplay between microbial dysbiosis and oncogenesis. The composition of cutaneous microbiota can influence cancer development through several mechanisms, including modulation of immune responses, promotion of chronic inflammation, and direct impacts on genomic stability. Ultraviolet (UV) radiation, a well-established risk factor for skin cancer, has been shown to significantly disrupt the skin microbiome. It reduces protective commensals such as *Staphylococcus epidermidis* and facilitates the proliferation of pro-inflammatory species like *Staphylococcus aureus* and *Cutibacterium acnes* [52], [53]. These microbial alterations contribute to tumour development by increasing reactive oxygen species (ROS) production, which induces DNA damage and impairs repair processes, particularly relevant in squamous cell carcinoma (SCC) and basal cell carcinoma (BCC) [54], [55]. Specific pathogens, notably *S. aureus*, have been implicated in accelerating SCC progression by triggering the release of pro-inflammatory cytokines such as IL-6 and TNF-α, while simultaneously suppressing antitumor immune responses [56]. In melanoma, the gut-skin microbiome axis has emerged as an important modulator of therapeutic efficacy. Commensal bacteria such as *Bifidobacterium* and *Akkermansia muciniphila* enhance responses to immune checkpoint inhibitors (e.g., anti-PD-1 therapy) by stimulating dendritic cell activity and promoting CD8+ T-cell infiltration into tumours [57], [58]. Conversely, antibiotic-induced dysbiosis has been associated with diminished responsiveness to immunotherapy, further underscoring the microbiome's role in shaping treatment outcomes [37]. Additionally, organisms like *Demodex* mites and their associated bacteria (e.g., *Bacillus oleronius*) have been linked to enhanced tumour-associated inflammation via Toll-like receptor (TLR) activation and dysregulated cathelicidin expression, suggesting novel therapeutic targets for microbiome-based interventions [41], [59]. Beyond oncogenesis, microbial dysbiosis has been correlated with tumour aggressiveness, with distinct microbial signatures distinguishing between indolent and aggressive tumours. These findings point to the potential of the microbiome as a biomarker for disease stratification [60]. Current microbiome-modulating therapies, including probiotics and skin microbiota-targeted interventions, are primarily being explored for inflammatory skin conditions such as atopic dermatitis and psoriasis. These strategies aim to restore barrier integrity and suppress the overgrowth of pathogenic species. While they show promise in reducing cutaneous inflammation, their application as adjunctive treatments in oncology remains speculative and requires further clinical validation [56]. In summary, the skin microbiome plays a dual role in both promoting and inhibiting skin cancer. It represents a promising frontier for the development of diagnostic biomarkers and therapeutic targets in dermatologic oncology. Future research should aim to elucidate specific microbial mechanisms involved in tumour biology and to develop personalized microbiome-based interventions to enhance cancer prevention, treatment efficacy, and patient outcomes.

The relationship between intact skin microbiota and wound healing.

The skin microbiome plays a fundamental role in the process of wound healing, with its composition and equilibrium directly impacting both the speed and effectiveness of tissue repair. A healthy and diverse microbial community supports wound resolution through multiple mechanisms, including immune modulation, inhibition of pathogen colonization, and stimulation of tissue regeneration. Commensal bacteria such as *Staphylococcus epidermidis* and *Corynebacterium* species contribute positively to wound healing by producing antimicrobial peptides (AMPs) that suppress pathogenic microbes, while also promoting keratinocyte migration and proliferation—key processes in epidermal regeneration [1], [61]. These beneficial organisms help maintain a controlled inflammatory response by regulating cytokine production and neutrophil activity, which is essential during the early inflammatory phase of wound healing [62]. In contrast, microbial

dysbiosis—marked by an overgrowth of pathogenic species such as *Staphylococcus aureus* or *Pseudomonas aeruginosa*—disrupts the healing process by sustaining chronic inflammation, enhancing oxidative stress, and promoting biofilm formation. Biofilms act as protective barriers for pathogens, shielding them from immune clearance and contributing to wound chronicity [63], [64]. Chronic wounds, including diabetic foot ulcers and venous leg ulcers, frequently exhibit reduced microbial diversity and dominance of pathogenic bacteria, conditions that correlate with delayed wound healing and an increased risk of infection and complications [65], [66]. The interaction between the microbiota and the host immune system is especially critical; dysbiosis can impair macrophage polarization and fibroblast function, both of which are crucial for tissue remodelling and scar formation [67], [68]. Emerging therapeutic approaches aim to restore microbial balance to support effective healing. These include the use of probiotics, prebiotics, and bacteriophage therapy, which target harmful bacteria while preserving or enhancing beneficial microbial populations [69], [70]. Additionally, advancements in microbiome profiling technologies have enabled the identification of specific microbial signatures linked to impaired wound healing, offering potential biomarkers for prognosis and treatment monitoring. Future research should continue to investigate the precise mechanisms by which the skin microbiome influences each phase of wound repair. A deeper understanding will inform the development of targeted microbiome-based therapies that can optimize wound healing outcomes and reduce the burden of chronic wounds.

Future Directions in Skin Microbiome Research

The field of skin microbiome research holds significant transformative potential for dermatology, though it necessitates targeted advancements. Critical priorities include the standardization of sampling and analytical methodologies to facilitate cross-study comparisons, as well as a shift from correlative studies to investigations that establish causality, particularly through multi-omics approaches. Personalized modulation of the microbiome using targeted probiotics, phage therapy, or microbial transplants could revolutionize therapeutic strategies. Equally important is the development of microbiome-sparing alternatives to broad-spectrum antibiotics. Deeper exploration of the gut–skin axis and the microbiome's influence on immunotherapy responses is also warranted. Clinical translation will depend on robust trials validating the efficacy and safety of microbiome-based interventions. Collectively, these advances are expected to enable precision dermatology approaches that leverage microbial ecology to enhance skin health.

Conclusions

The skin microbiome plays a fundamental role in preserving cutaneous homeostasis by regulating barrier function, modulating immune responses, and protecting against pathogenic invasion. Emerging evidence has established that dysbiosis, defined as disruptions in microbial composition, contributes to the pathogenesis of numerous dermatological conditions, including atopic dermatitis, psoriasis, acne, rosacea, skin cancer, and impaired wound healing. These alterations can result from genetic predisposition, environmental exposures, or therapeutic interventions, underscoring the delicate balance between host and microbiota. In inflammatory skin diseases such as atopic dermatitis and psoriasis, dysbiosis fosters the overgrowth of pathogenic organisms like *Staphylococcus aureus* while suppressing beneficial commensals, thereby exacerbating inflammation and compromising barrier integrity. In acne and rosacea, microbial shifts involving virulent *Cutibacterium acnes* strains or *Demodex*-associated bacteria drive disease progression through immune activation and biofilm formation. The skin microbiome also impacts skin cancer development by modulating inflammatory pathways, DNA damage responses, and immunotherapy efficacy. In chronic wounds, microbial imbalances delay healing by perpetuating inflammation and promoting biofilm-associated infections. Therapeutic strategies targeting the microbiome including probiotics, prebiotics, phage therapy, and microbial transplantation hold promise for restoring microbial equilibrium and improving clinical outcomes. Nevertheless, challenges persist, including the lack of standardized methodologies, limited causal insights, and the need for personalized approaches. Future research should prioritize longitudinal studies, multi-omics integration, and rigorous clinical trials to validate these interventions. Ultimately, the skin microbiome represents a dynamic interface between the host and the environment, offering novel diagnostic and therapeutic avenues in dermatology. By advancing our understanding of host–microbiota interactions, precision medicine strategies can be developed to promote skin health and more effectively treat dermatological diseases.

Disclosure

Author's contribution

Conceptualization: Hubert Chmielewski

Methodology: Hubert Chmielewski, Julia Komar, Ada Kondrat, Gabriela Kondratiuk

Software: Michał Wycik, Artur Wądołowski, Justyna Werno, Zuzanna Twarowska

Formal analysis: Michał Szczupak, Julia Komar, Paweł Mierzejewski

Investigation: Hubert Chmielewski, Ada Kondrat, Gabriela Kondratiuk

Writing – rough preparation: Hubert Chmielewski, Julia Komar, Zuzanna Twarowska

Writing – review and editing: Hubert Chmielewski, Julia Komar, Artur Wądołowski, Gabriela Kondratiuk, Ada Kondrat, Michał Wycik, Paweł Mierzejewski, Michał Szczupak

Supervision: Justyna Werno, Zuzanna Twarowska

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REFERENCES

1. Smythe, P., & Wilkinson, H. N. (2023). The skin microbiome: Current landscape and future opportunities. *International Journal of Molecular Sciences, 24*(4), 3950. https://doi.org/10.3390/ijms24043950
2. Schommer, N. N., & Gallo, R. L. (2013). Structure and function of the human skin microbiome. *Trends in Microbiology, 21*(12), 660. https://doi.org/10.1016/j.tim.2013.10.001
3. Prescott, S. L., et al. (2017). The skin microbiome: Impact of modern environments on skin ecology, barrier integrity, and systemic immune programming. *World Allergy Organization Journal, 10*(1), 29. https://doi.org/10.1186/s40413-017-0160-5
4. Sroka-Tomaszewska, J., & Trzeciak, M. (2021). Molecular mechanisms of atopic dermatitis pathogenesis. *International Journal of Molecular Sciences, 22*(8), 4130. https://doi.org/10.3390/ijms22084130
5. Schuler, C. F., Billi, A. C., Maverakis, E., Tsoi, L. C., & Gudjonsson, J. E. (2022). Novel insights into atopic dermatitis. *Journal of Allergy and Clinical Immunology, 151*(5), 1145. https://doi.org/10.1016/j.jaci.2022.10.023
6. Kennedy, E. A., et al. (2017). Skin microbiome before development of atopic dermatitis: Early colonization with commensal staphylococci at 2 months is associated with a lower risk of atopic dermatitis at 1 year. *Journal of Allergy and Clinical Immunology, 139*(1), 166. https://doi.org/10.1016/j.jaci.2016.07.029
7. The skin microbiome: Is there a role in the pathogenesis of atopic dermatitis and psoriasis? (2025, April 26). *Journal of Drugs in Dermatology*. https://jddonline.com/articles/the-skin-microbiome-is-there-a-role-in-the-pathogenesis-of-atopic-dermatitis-and-psoriasis-S1545961615P0127X/
8. Kobayashi, T., et al. (2015). Dysbiosis and *Staphylococcus aureus* colonization drives inflammation in atopic dermatitis. *Immunity, 42*(4), 756. https://doi.org/10.1016/j.immuni.2015.03.014
9. Lee, S. Y., Lee, E., Park, Y. M., & Hong, S. J. (2018). Microbiome in the gut–skin axis in atopic dermatitis. *Allergy, Asthma & Immunology Research, 10*(4), 354. https://doi.org/10.4168/aair.2018.10.4.354
10. Park, D. H., Kim, J. W., Park, H. J., & Hahm, D. H. (2021). Comparative analysis of the microbiome across the gut–skin axis in atopic dermatitis. *International Journal of Molecular Sciences, 22*(8), 4228. https://doi.org/10.3390/ijms22084228
11. Carmona-Cruz, S., Orozco-Covarrubias, L., & Sáez-de-Ocariz, M. (2022). The human skin microbiome in selected cutaneous diseases. *Frontiers in Cellular and Infection Microbiology, 12*, 834135. https://doi.org/10.3389/fcimb.2022.834135
12. Lee, H. J., & Kim, M. (2022). Skin barrier function and the microbiome. *International Journal of Molecular Sciences, 23*(21), 13071. https://doi.org/10.3390/ijms232113071
13. Edslev, S. M., Agner, T., & Andersen, P. S. (2020). Skin microbiome in atopic dermatitis. *Acta Dermato-Venereologica, 100*(12), 5769. https://doi.org/10.2340/00015555-3514
14. Koh, L. F., Ong, R. Y., & Common, J. E. (2022). Skin microbiome of atopic dermatitis. *Allergology International, 71*(1), 31–39. https://doi.org/10.1016/j.alit.2021.11.001
15. Tham, E. H., Chia, M., Riggioni, C., Nagarajan, N., Common, J. E. A., & Kong, H. H. (2024). The skin microbiome in pediatric atopic dermatitis and food allergy. *Allergy, 79*(6), 1470–1484. https://doi.org/10.1111/all.16044

16. Moniaga, C. S., Tominaga, M., & Takamori, K. (2022). An altered skin and gut microbiota are involved in the modulation of itch in atopic dermatitis. *Cells, 11*(23), 3930. https://doi.org/10.3390/cells11233930
17. Zhang, X. E., et al. (2024). Microbiome: Role in inflammatory skin diseases. *Journal of Inflammation Research, 17*, 1057. https://doi.org/10.2147/jir.s441100
18. Paller, A. S., et al. (2018). The microbiome in patients with atopic dermatitis. *Journal of Allergy and Clinical Immunology, 143*(1), 26. https://doi.org/10.1016/j.jaci.2018.11.015
19. Orsmond, A., Bereza-Malcolm, L., Lynch, T., March, L., & Xue, M. (2021). Skin barrier dysregulation in psoriasis. *International Journal of Molecular Sciences, 22*(19), 10841. https://doi.org/10.3390/ijms221910841
20. Petit, R. G., et al. (2021). Psoriasis: From pathogenesis to pharmacological and nano-technological-based therapeutics. *International Journal of Molecular Sciences, 22*(9), 4983. https://doi.org/10.3390/ijms22094983
21. Raharja, A., Mahil, S. K., & Barker, J. N. (2021). Psoriasis: A brief overview. *Clinical Medicine, 21*(3), 170. https://doi.org/10.7861/clinmed.2021-0257
22. Canal-García, E., Bosch-Amate, X., Belinchón, I., & Puig, L. (2022). Nail psoriasis. *Actas Dermo-Sifiliográficas, 113*(5), T481–T490. https://doi.org/10.1016/j.ad.2022.01.032
23. Ito, Y., & Amagai, M. (2022). Controlling skin microbiome as a new bacteriotherapy for inflammatory skin diseases. *Inflammation and Regeneration, 42*(1). https://doi.org/10.1186/s41232-022-00212-y
24. Buhaș, M. C., et al. (2022). Gut microbiota in psoriasis. *Nutrients, 14*(14), 2970. https://doi.org/10.3390/nu14142970
25. Gallo, R. L., & Nakatsuji, T. (2011). Microbial symbiosis with the innate immune defense system of the skin. *Journal of Investigative Dermatology, 131*(10), 1974. https://doi.org/10.1038/jid.2011.182
26. Celoria, V., et al. (2023). The skin microbiome and its role in psoriasis: A review. *Psoriasis: Targets and Therapy, 13*, 71. https://doi.org/10.2147/ptt.s328439
27. Cogen, A. L., Nizet, V., & Gallo, R. L. (2008). Skin microbiota: A source of disease or defence? *British Journal of Dermatology, 158*(3), 442. https://doi.org/10.1111/j.1365-2133.2008.08437.x
28. Thye, A. Y. K., et al. (2022). Gut–skin axis: Unravelling the connection between the gut microbiome and psoriasis. *Biomedicines, 10*(5), 1037. https://doi.org/10.3390/biomedicines10051037
29. Olejniczak-Staruch, I., et al. (2021). Alterations of the skin and gut microbiome in psoriasis and psoriatic arthritis. *International Journal of Molecular Sciences, 22*(8), 3998. https://doi.org/10.3390/ijms22083998
30. Arya, P., Kaur, M., Chosyang, S., Kushwaha, N., & Singh, B. (2023). Decrypting skin microbiome in psoriasis: Current status. *Journal of Psoriasis and Psoriatic Arthritis, 8*(4), 166. https://doi.org/10.1177/24755303231194293
31. Mazur, M., Tomczak, H., Lodyga, M., Czajkowski, R., Zaba, R., & Adamski, Z. (2021). The microbiome of the human skin and its variability in psoriasis and atopic dermatitis. *Advances in Dermatology and Allergology, 38*(2), 205. https://doi.org/10.5114/ada.2021.106197
32. Dréno, B., Dagnelie, M. A., Khammari, A., & Corvec, S. (2020). The skin microbiome: A new actor in inflammatory acne. *American Journal of Clinical Dermatology, 21*(Suppl 1), 18. https://doi.org/10.1007/s40257-020-00531-1
33. Natarelli, N., Gahoonia, N., & Sivamani, R. K. (2023). Bacteriophages and the microbiome in dermatology: The role of the phageome and a potential therapeutic strategy. *International Journal of Molecular Sciences, 24*(3), 2695. https://doi.org/10.3390/ijms24032695
34. Sánchez-Pellicer, P., et al. (2022). Acne, microbiome, and probiotics: The gut–skin axis. *Microorganisms, 10*(7), 1303. https://doi.org/10.3390/microorganisms10071303
35. Zhu, W., Hamblin, M. R., & Wen, X. (2023). Role of the skin microbiota and intestinal microbiome in rosacea. *Frontiers in Microbiology, 14*, 1108661. https://doi.org/10.3389/fmicb.2023.1108661
36. Severn, M. M., & Horswill, A. R. (2022). *Staphylococcus epidermidis* and its dual lifestyle in skin health and infection. *Nature Reviews Microbiology, 21*(2), 97. https://doi.org/10.1038/s41579-022-00780-3
37. El-Sayed, A., Aleya, L., & Kamel, M. (2021). Microbiota's role in health and diseases. *Environmental Science and Pollution Research, 28*(28), 36967. https://doi.org/10.1007/s11356-021-14593-z
38. Xu, H., & Li, H. (2019). Acne, the skin microbiome, and antibiotic treatment. *American Journal of Clinical Dermatology, 20*(3), 335. https://doi.org/10.1007/s40257-018-00417-3
39. Ryguła, I., Pikiewicz, W., & Kaminiów, K. (2024). Impact of diet and nutrition in patients with acne vulgaris. *Nutrients, 16*(10), 1476. https://doi.org/10.3390/nu16101476
40. Mahmud, M. R., et al. (2022). Impact of gut microbiome on skin health: Gut–skin axis observed through the lenses of therapeutics and skin diseases. *Gut Microbes, 14*(1), 2096995. https://doi.org/10.1080/19490976.2022.2096995
41. Manos, J. (2022). The human microbiome in disease and pathology. *APMIS, 130*(12), 690. https://doi.org/10.1111/apm.13225
42. Jin, Z., Song, Y., & He, L. (2023). A review of skin immune processes in acne. *Frontiers in Immunology, 14*, 1324930. https://doi.org/10.3389/fimmu.2023.1324930
43. Kang, D., Shi, B., Erfe, M. C., Craft, N., & Li, H. (2015). Vitamin B12 modulates the transcriptome of the skin microbiota in acne pathogenesis. *Science Translational Medicine, 7*(293), 293ra103. https://doi.org/10.1126/scitranslmed.aab2009
44. Huang, C., et al. (2023). The updates and implications of cutaneous microbiota in acne. *Cell & Bioscience, 13*(1), 113. https://doi.org/10.1186/s13578-023-01072-w

45. Lam, M., Hu, A., Fleming, P., & Lynde, C. W. (2022). The impact of acne treatment on skin bacterial microbiota: A systematic review. *Journal of Cutaneous Medicine and Surgery, 26*(1), 93–97. https://doi.org/10.1177/12034754211037994
46. Chilicka, K., Dzieńdziora-Urbińska, I., Szyguła, R., Asanova, B., & Nowicka, D. (2022). Microbiome and probiotics in acne vulgaris: A narrative review. *Life, 12*(3), 422. https://doi.org/10.3390/life12030422
47. Yang, Y., Qu, L., Mijakovic, I., & Wei, Y. (2022). Advances in the human skin microbiota and its roles in cutaneous diseases. *Microbial Cell Factories, 21*(1), 176. https://doi.org/10.1186/s12934-022-01901-6
48. Dreno, B., et al. (2024). Acne microbiome: From phyla to phylotypes. *Journal of the European Academy of Dermatology and Venereology, 38*(4), 657–664. https://doi.org/10.1111/jdv.19540
49. Sánchez-Pellicer, P., et al. (2024). Rosacea, microbiome and probiotics: The gut–skin axis. *Frontiers in Microbiology, 14*, 1323644. https://doi.org/10.3389/fmicb.2023.1323644
50. Xiao, X., et al. (2023). The role of short-chain fatty acids in inflammatory skin diseases. *Frontiers in Microbiology, 13*, 1083432. https://doi.org/10.3389/fmicb.2022.1083432
51. Dreno, B., Martin, R., Moyal, D., Henley, J. B., Khammari, A., & Seité, S. (2017). Skin microbiome and acne vulgaris: *Staphylococcus*, a new actor in acne. *Experimental Dermatology, 26*(9), 798–803. https://doi.org/10.1111/exd.13296
52. Morgenroth, S., Roggo, A., Pawlik, L., Dummer, R., & Ramelyte, E. (2023). What is new in cutaneous T-cell lymphoma? *Current Oncology Reports, 25*(11), 1397. https://doi.org/10.1007/s11912-023-01464-8
53. Woo, Y. R., Cho, S. H., Lee, J. D., & Kim, H. S. (2022). The human microbiota and skin cancer. *International Journal of Molecular Sciences, 23*(3), 1813. https://doi.org/10.3390/ijms23031813
54. Azzimonti, B., et al. (2023). Microbiota, oxidative stress, and skin cancer: An unexpected triangle. *Antioxidants, 12*(3), 546. https://doi.org/10.3390/antiox12030546
55. Hou, K., et al. (2022). Microbiota in health and diseases. *Signal Transduction and Targeted Therapy, 7*(1), 135. https://doi.org/10.1038/s41392-022-00974-4
56. Chambers, E. S., & Vukmanovic-Stejic, M. (2019). Skin barrier immunity and ageing. *Immunology, 160*(2), 116. https://doi.org/10.1111/imm.13152
57. De Pessemier, B., Grine, L., Debaere, M., Maes, A., Paetzold, B., & Callewaert, C. (2021). Gut–skin axis: Current knowledge of the interrelationship between microbial dysbiosis and skin conditions. *Microorganisms, 9*(2), 353. https://doi.org/10.3390/microorganisms9020353
58. Flowers, L., & Grice, E. A. (2020). The skin microbiota: Balancing risk and reward. *Cell Host & Microbe, 28*(2), 190. https://doi.org/10.1016/j.chom.2020.06.017
59. Zhu, Y., Yu, X., & Cheng, G. (2023). Human skin bacterial microbiota homeostasis: A delicate balance between health and disease. *mLife, 2*(2), 107. https://doi.org/10.1002/mlf2.12064
60. Pereira, M. S., Redanz, S., & Kriegel, M. A. (2022). Skin deep: The role of the microbiota in cutaneous autoimmunity. *Journal of Investigative Dermatology, 142*(3), 834–840. https://doi.org/10.1016/j.jid.2021.12.005
61. Yang, Y., Huang, J., Zeng, A., Long, X., Yu, N., & Wang, X. (2024). The role of the skin microbiome in wound healing. *Burns & Trauma, 12*, tkad059. https://doi.org/10.1093/burnst/tkad059
62. Fernandes, A., Rodrigues, P. M., Pintado, M., & Tavaria, F. K. (2023). A systematic review of natural products for skin applications: Targeting inflammation, wound healing, and photo-aging. *Phytomedicine, 115*, 154824. https://doi.org/10.1016/j.phymed.2023.154824
63. Tomic-Canic, M., Burgess, J. L., O'Neill, K. E., Strbo, N., & Pastar, I. (2020). Skin microbiota and its interplay with wound healing. *American Journal of Clinical Dermatology, 21*(Suppl 1), 36. https://doi.org/10.1007/s40257-020-00536-w
64. Canchy, L., Kerob, D., Demessant, A., & Amici, J. M. (2023). Wound healing and microbiome: An unexpected relationship. *Journal of the European Academy of Dermatology and Venereology, 37*(S3), 7–15. https://doi.org/10.1111/jdv.18854
65. Wang, G., et al. (2021). Bacteria induce skin regeneration via IL-1β signaling. *Cell Host & Microbe, 29*(5), 777. https://doi.org/10.1016/j.chom.2021.03.003
66. Swaney, M. H., & Kalan, L. R. (2021). Living in your skin: Microbes, molecules, and mechanisms. *Infection and Immunity, 89*(4), e00695-20. https://doi.org/10.1128/iai.00695-20
67. Piazzesi, A., Scanu, M., Ciprandi, G., & Putignani, L. (2024). Modulations of the skin microbiome in skin disorders: A narrative review from a wound care perspective. *International Wound Journal, 21*(10), e70087. https://doi.org/10.1111/iwj.70087
68. Li, C., et al. (2022). Insights on gut and skin wound microbiome in stranded Indo-Pacific finless porpoise (*Neophocaena phocaenoides*). *Microorganisms, 10*(7), 1295. https://doi.org/10.3390/microorganisms10071295
69. Wang, G., et al. (2023). Commensal microbiome promotes hair follicle regeneration by inducing keratinocyte HIF-1α signaling and glutamine metabolism. *Science Advances, 9*(1), eabo7555. https://doi.org/10.1126/sciadv.abo7555
70. White, E. K., & Grice, E. A. (2023). The wound microbiome. *Cold Spring Harbor Perspectives in Biology, 15*(6), a041218. https://doi.org/10.1101/cshperspect.a041218