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CURRENT PERSPECTIVES ON THE ROLE OF THE CUTANEOUS MICROBIOME IN CHRONIC INFLAMMATORY SKIN DISEASES

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ABSTRACT

Objective: The skin microbiome is a complex ecosystem of bacteria, viruses, fungi, and archaea that interacts with host cells to regulate defense mechanisms, immune responses, colonization resistance, and tissue repair. This review aims to summarize current knowledge on the role of the skin microbiome in the course and treatment of chronic inflammatory dermatoses, including atopic dermatitis, psoriasis, and acne vulgaris.
Materials and methods: A literature review was performed using PubMed and Google Scholar databases. Keywords included: skin microbiota, skin microbiome, chronic inflammatory dermatoses, atopic dermatitis, psoriasis, acne vulgaris, microbiome transplantation, probiotics, prebiotics, and postbiotics. A total of 36 articles published between 2008 and 2024 were analyzed for relevance.
Description: Patients with atopic dermatitis show reduced microbial diversity, characterized by Staphylococcus aureus overgrowth and decreased commensal bacteria such as Streptococcus, Corynebacterium, Cutibacterium, and Proteobacteria. In psoriasis, decreased levels of Corynebacterium spp., Lactobacillus spp., and Cutibacterium acnes are common. Acne vulgaris is linked to excessive sebum production, abnormal follicular keratinization, Cutibacterium acnes colonization, and inflammation.
Strategies to modulate the skin microbiome include microbiome transplantation and bacteriotherapy, which applies selected beneficial strains to disinfected skin. These methods may offer more practical alternatives than full transplantation. Additionally, prebiotic compounds can promote the growth of protective microbes, while probiotics and postbiotics are increasingly studied for their therapeutic effects.
Conclusions: Chronic inflammatory dermatoses significantly impair quality of life. Evidence suggests that targeting the skin microbiome holds therapeutic potential. Further studies are needed to clarify microbiota–host interactions, consequences of dysbiosis, and the clinical utility of microbiome-based interventions.

KEYWORDS

Skin Microbiota, Chronic Inflammatory Dermatoses, Atopic Dermatitis, Psoriasis, Acne Vulgaris, Microbiome Transplantation

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Introduction

The skin microbiome is an ecosystem consisting of many species of microorganisms, such as bacteria, viruses, fungi, and archaea, which interact with their environment, including other microorganisms as well as the host's epithelial and immune cells. The interactions between skin microbes and the host can have both mutualistic and pathogenic characteristics [1, 2, 3].

The skin microbiota consists of 18 phyla of bacteria, among which the dominant ones are Actinobacteria, Firmicutes, Proteobacteria, and Bacteroidetes. The fungal skin microbiome is mainly composed of Malassezia, Aspergillus, Cryptococcus, Rhodotorula, Debaryomyces, Epicoccum, and Candida [4].

One of the most numerous species of bacteria constituting the skin microbiome of adults is Cutibacterium acnes. This bacterium produces propionic acid, which helps maintain the acidic pH of healthy skin, thereby inhibiting colonization by more pathogenic microorganisms. At the same time, C. acnes has the potential to directly and indirectly cause inflammation and tissue damage, contributing to the development of common acne. Bacteria of the genus Corynebacterium are also commonly part of the skin microbiota. Corynebacterium species are frequent etiological agents of skin, soft tissue, and organ infections in immunocompromised, hospitalized, and chronically ill individuals. Interestingly, Corynebacterium species play an important role in producing volatile odor compounds characteristic of axillary sweat. A very numerous groups of bacteria in the skin microbiome consists of bacteria from the genus Staphylococcus, including the coagulase-positive S. aureus and coagulase-negative species such as S. epidermidis, S. capitis, S. caprae, S.

hominis, S. lugdunensis, and S. haemolyticus. Malassezia fungi are the most abundant fungi in the skin microbiome. Malassezia species, including M. globosa, M. furfur, and M. restricta, can cause skin diseases such as dandruff and seborrheic dermatitis [2].

The skin is a highly dynamic organ composed of a variety of cell types and structures that constitute the main barrier between the organism and the external environment. Similar to other barriers of the body, the skin barrier is formed by microbiological, immunological, chemical, and physical components [2, 3, 4]. The surface of the skin consists of a highly organized structure of proteins and lipids. Microorganisms covering the skin, by metabolizing host's proteins and lipids, produce bioactive molecules such as free fatty acids, antimicrobial peptides (AMP), phenol-soluble modulins (PSM), cell wall components, and antibiotics. These molecules act on other microorganisms, the host's epithelium, and the host's immune cells in the epidermis and dermis, contributing to the maintenance of barrier homeostasis [1, 4].

The skin microbiota, interacting with each other and with host's cells, promotes defense and immune responses, inhibits colonization and infection by opportunistic or pathogenic organisms, and promotes tissue repair and barrier functions [2]. The skin microbiome plays a fundamental role in the induction and functioning of the skin immune barrier through a range of antimicrobial mechanisms, such as the release of antimicrobial peptides, short-chain fatty acids, and polyamines. Uberoi A et al., in studies on mice, demonstrated that commensal microbes are essential for proper epidermal differentiation, functioning of the epidermal permeability barrier (EPB), and its repair. Furthermore, they concluded that the aryl hydrocarbon receptor (AHR) is a potential mechanism by which skin microbes modulate epithelial barrier integrity [3].

Tomic-Canic M et al. demonstrated that microorganisms forming the skin microbiome play a significant role in the wound healing process. The interaction of commensal microorganisms with skin cells during the normal process of skin wound healing is beneficial in modulating the innate immune response, whereas pathogenic microorganisms influence delayed wound healing. The microbiota of chronic wounds differs from the surrounding healthy skin microbiota. The majority of bacteria colonizing the tissue of chronic wounds are bacteria from the genera Staphylococcus and Pseudomonas [5, 6].

The skin microbiota begins to form at the moment of birth. The colonization of newborns by microbiota has a long-lasting impact on the immune barrier of adults. The initial colonization by microorganisms depends on the mode of delivery – newborns born by cesarean section exhibit microbiome profiles more resembling the mother's skin (dominated by Staphylococcus, Corynebacterium, and Cutibacterium) than the vagina (dominated by Lactobacillus and Prevotella). By the sixth week of life, the microbiological composition of the skin can no longer be determined based on the mode of delivery. The composition of the skin microbiome changes significantly during puberty, with an increased predominance of Corynebacterium and a decreased abundance of Firmicutes. Moreover, both Cutibacterium acnes and Staphylococcus epidermidis increase in a site- specific manner upon reaching sexual maturity in men and women. In adulthood, despite the disturbances to which the skin microbiota is exposed daily, its composition remains relatively stable [1, 4]. It is estimated that there are about 10^{12} bacteria on the skin of an adult human [7]. Changes in the microbiological composition of the skin coincide with physical changes, as infant skin is thinner, more alkaline, and has a higher cell turnover rate compared to adult skin [4].

The skin microbiome is topographically diverse, temporally complex, and distinct from other organs. The skin offers protective niches and nutrients for the survival, competition, and cooperation of microorganisms. Chen YE et al. observed that the chemistry of the skin niche drives the composition of its microbiome. Staphylococcus species are common throughout the body. In sebaceous areas, such as the trunk, back, and face, bacteria capable of metabolically utilizing sebum and tolerating low pH, such as Cutibacterium, dominate. In moist areas, such as the groin crease, the antecubital fossa, and the popliteal fossa, bacteria from the genus Corynebacterium dominate. Dry sites, including the hypothenar palm and the volar forearm, exhibit high microbial diversity and low temporal stability. Moreover, the dominant member of the fungal microbiota across the entire body surface, except for the feet, is Malassezia. The foot mycobiome is significantly more diverse and includes Malassezia, Aspergillus, Cryptococcus, Rhodotorula, and Epicoccum [1, 2, 3, 4].

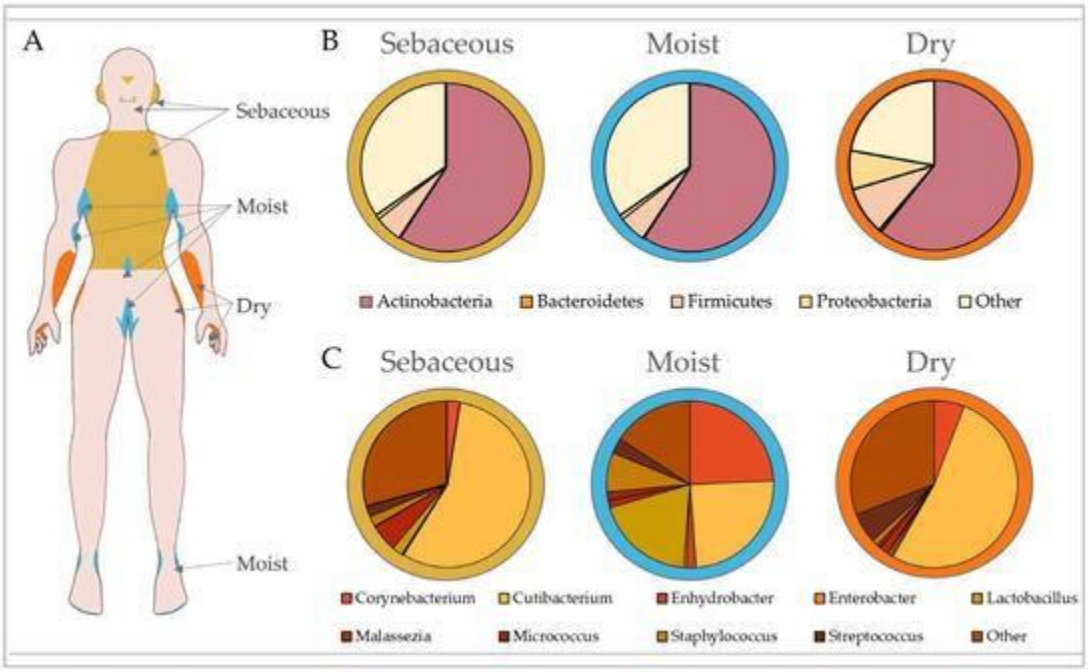

Fig. 1. *Microbial composition of the skin is dictated by topography.*

Skin exhibits biogeographically distinct regions that are generally categorised according to their unique physiological characteristics into sebaceous, moist and dry sites (A). These regions exhibit different microbial community structures influenced by skin pH, temperature, sebum content and moisture levels. Microbiota are separated by phylum according to site-specific variation in (B), with top contributing genera shown in (C). In healthy adults, sebaceous (e.g., torso, glabella and retroauricular crease) and dry (e.g., volar forearm and hypothenar palm) sites are predominantly colonised by Cutibacterium, while moist sites (e.g., antecubital fossa, inguinal crease and axillary crease) show equally high abundances of Corynebacterium, Cutibacterium and Staphylococcus. Data analysed from Oh et al [8].

The composition of the skin microbiome depends on many factors, such as the external environment, the host's age, ethnic origin, genetic predispositions, skin care, the presence of skin diseases or systemic diseases. The microbiological composition of the skin may change during inflammatory conditions. Disturbances in skin homeostasis, such as aging, diabetes, and skin diseases, can cause microbial dysbiosis and increase the risk of infection [1, 2, 4, 5].

The nature of the interaction between skin microbes and the host can change depending on the context. Pathogens such as Staphylococcus aureus can colonize the skin asymptomatically.

Staphylococcus epidermidis can both stimulate and suppress inflammation. Other microbial species, such as Roseomonas mucosa, Malassezia spp., or Corynebacterium accolens, can also influence the host's immune response in a context-dependent manner. Even within the same species, different strains can vary significantly in their impact on the host. Such microbe-host interactions contribute to the stability of the microbial ecosystem and the integrity of the skin [1, 2].

The aim of this study is to present the current state of knowledge regarding the skin microbiome and its influence on the course and treatment of chronic inflammatory dermatoses, such as atopic dermatitis, psoriasis and acne vulgaris. This is an important topic that offers prospects for new therapeutic solutions.

Microbiome in Atopic Dermatitis

Atopic dermatitis (AD) is an inflammatory, chronic, non-infectious dermatosis that occurs primarily in children. The peak incidence is observed during infancy: approximately 45% of cases begin within the first six months of life, and 80–90% occur before the age of five. AD is also seen in adults, with a prevalence in this age group estimated at 1% to 10% [9, 10, 11].

Itching is a hallmark and the most bothersome symptom of AD, initiating a vicious cycle of irritation→ scratching → pruritus → skin damage. It is typically associated with erythematous, scaly lesions of varying morphology. The presentation and distribution of skin lesions change with age. In the first year of life, AD

usually manifests as erythematous papules, patches, or plaques on the face (particularly the cheeks), scalp, trunk, and extremities. In older children, lesions are typically localized to flexural areas. In adults, dry, scaly patches commonly appear on the head and limbs [12, 13, 14].

The pathogenesis of this condition is complex and not fully understood. It is believed to involve a combination of genetic predisposition, epidermal barrier dysfunction, aberrant immune responses, dysbiosis of the skin and gut microbiota, as well as environmental factors such as exposure to airborne or food allergens, pollutants, infections, and diet [9, 10, 12, 13].

The composition of the skin microbiome differs significantly between healthy individuals and patients with AD. Studies have shown that in individuals with AD, microbial diversity on the skin is reduced, although the total bacterial load does not significantly differ from that of healthy individuals. Skin microbiota dysbiosis, which becomes more pronounced during disease flares, is characterized by increased colonization by *Staphylococcus aureus* and a concomitant reduction in commensal bacteria, including *Streptococcus*, *Corynebacterium*, *Cutibacterium*, and members of the *Proteobacteria* phylum. Importantly, the degree of *S. aureus* colonization correlates with disease severity. Following the resolution of skin lesions, microbial diversity is partially restored, which may play a role in remission and the reestablishment of skin barrier function [9, 10, 12, 13, 15].

When describing the influence of the skin microbiota on the development of AD, it is essential to highlight the role of filaggrin—a key structural protein of the epidermis. Filaggrin plays a critical role in keratinocyte differentiation as well as in the formation and maintenance of epidermal barrier integrity. Its degradation products, such as urocanic acid and pyrrolidone carboxylic acid, are major components of the natural moisturizing factor (NMF) and contribute to proper skin hydration and the acidic pH of the skin surface, which helps to limit pathogenic colonization. In AD, filaggrin deficiency may occur due to loss-of-function mutations in the *FLG* gene or as a result of the action of Th2 and Th22 cytokines, which suppress *FLG* gene expression independently of genetic mutations. This deficiency disrupts the structure of the stratum corneum, increases transepidermal water loss (TEWL), elevates skin pH, and reduces the production of antimicrobial peptides—factors that collectively impair the innate immune defense of the skin. Such conditions promote colonization by *Staphylococcus aureus* and facilitate the penetration of allergens and toxins through the compromised barrier, thereby contributing to chronic inflammation and disease exacerbation [9, 12, 13, 15].

Staphylococcus aureus is a Gram-positive opportunistic bacterium capable of causing both superficial and invasive skin infections. It possesses numerous colonization and virulence factors, including superantigens (such as enterotoxins and toxic shock syndrome toxin-1 [TSST-1]), exotoxins, phenol-soluble modulins, and proteases. The presence of S. aureus is associated with more severe AD, owing to its adaptive capacity in inflammatory environments and its ability to adhere to the epidermis through surface-associated adhesins.

This pathogen incorporates specific fatty acids into its cell membrane, enhancing its resistance to host immune responses. By secreting toxins—including alpha-toxin, enterotoxins, and TSST-1—as well as proteases, S. aureus disrupts intercellular junctions within the epidermis, compromising skin barrier integrity and facilitating infection. Enterotoxins, in particular, are considered key inflammatory mediators in S. aureus-driven pathology. For example, topical application of staphylococcal enterotoxin B (SEB) has been shown to induce erythema and epidermal thickening in both healthy individuals and AD patients, likely due to heightened T cell activation. Additionally, S. aureus promotes mast cell degranulation, leading to the release of inflammatory cytokines and histamine. This contributes to elevated IgE levels and is closely linked to the occurrence of pruritus, one of the hallmark symptoms of AD [9, 10, 12, 13, 15].

Scientific studies indicate that the skin microbiome of healthy children displays greater microbial diversity compared to adults. However, in individuals with AD, both children and adults exhibit a marked reduction in microbial diversity and a concurrent increase in the abundance of *Staphylococcus aureus* within lesional skin. In infants who develop AD, an increase in *S. aureus* colonization may be detectable several weeks before the clinical onset of symptoms. Excessive colonization by clonal strains of *S. aureus* has been associated with more severe AD flares, whereas the presence of more diverse strains of *Staphylococcus epidermidis* may exert a protective effect and help mitigate disease severity. Interestingly, some evidence suggests that *S. aureus* colonization is not consistently observed during the first year of life, and that a key factor may be the reduction of commensal staphylococci populations, indicating their potential protective role against disease development. These discrepancies in findings may reflect the dynamic and maturing nature of the infant skin microbiome, which undergoes substantial compositional changes during the first year of life [15, 16].

In patients with AD, the composition of the skin mycobiome shows greater richness and diversity compared to healthy individuals, although the dominant species—*Malassezia globosa* and *Malassezia restricta*—are commonly found in both groups. In individuals with a history of AD, there is an increased presence of species such as *M. sympodialis* and *M. dermatitis*, although their proportions in active lesions do not differ significantly from those found in healthy skin. *Malassezia* spp., particularly under conditions of epidermal barrier disruption, may contribute to the initiation and persistence of cutaneous inflammation. Despite growing interest in the role of the eukaryotic skin microbiota in AD, further studies involving larger patient cohorts are necessary to fully elucidate its contribution to disease pathogenesis [10, 13, 15].

The treatment of AD primarily focuses on restoring the epidermal barrier and alleviating inflammation; however, increasing attention is being paid to its impact on the skin microbiota. Topical glucocorticosteroids, as standard anti-inflammatory therapies, not only reduce clinical symptoms but, after several weeks of use, also contribute to increased microbial diversity while reducing the dominance of *Staphylococcus aureus*, which may support disease remission. In the short term, despite clinical improvement, no significant changes in the microbiome structure are typically observed, suggesting that modifications to the microbial composition may require a longer duration of treatment [9, 13].

Microbiome in Psoriasis

Psoriasis is a chronic inflammatory skin disease with genetic, autoimmune, and autoinflammatory components. It affects approximately 2% of the global population, though its prevalence varies by geographic region and skin type—being most common among individuals of Caucasian descent and in Scandinavian populations, where it may affect up to 11% of people. The disease can develop at any age, and its onset is influenced by both genetic predisposition and environmental factors such as stress, infections, sun exposure, skin injuries (e.g., the Koebner phenomenon), and the use of certain medications, such as β-blockers [17, 18, 19].

The most common clinical form of psoriasis, accounting for about 90% of cases, is plaque psoriasis. It is characterized by well-demarcated, erythematous plaques covered with silvery scales, often accompanied by itching. Beyond the skin, psoriasis can also affect the joints, leading to psoriatic arthritis, and is frequently associated with comorbidities such as hypertension, type 2 diabetes, obesity, and cardiovascular diseases. The disease significantly impacts patients' quality of life, often contributing to depression, anxiety, and suicidal ideation [17, 18, 19].

The pathogenesis of psoriasis involves dysregulation of both the innate and adaptive immune systems. Key roles are played by dendritic cells, macrophages, T lymphocytes, and keratinocytes, which interact through the secretion of various cytokines, including IL-17, IL-23, and TNF-α. A hallmark of psoriasis is a persistent inflammatory state that leads to uncontrolled keratinocyte proliferation and impaired epidermal differentiation. Histologically, this is characterized by acanthosis, inflammatory infiltrates composed of immune cells, and neovascularization. In plaque psoriasis, innate immune responses predominate, whereas other forms may involve more pronounced autoimmune reactions, explaining phenotypic differences and varied treatment responses [17, 18, 19].

Based on current data, the skin microbiota of patients with psoriasis shows significant differences compared to that of healthy individuals, although study results remain somewhat inconsistent. In healthy skin, the dominant bacterial phyla are Actinobacteria, Bacteroidetes, Firmicutes, and Proteobacteria, with the most common genera being Cutibacterium, Corynebacterium, and Staphylococcus [20]. In psoriatic lesions, skin barrier disruption—partly due to scratching caused by pruritus—can facilitate bacterial penetration into the deeper dermis and even the bloodstream. This promotes interactions between bacteria and the immune system, triggering inflammatory responses and exacerbating skin microbiota dysbiosis. Consequently, a reduction in the abundance of Corynebacterium spp., Lactobacillus spp., and Cutibacterium acnes is often observed in psoriatic skin lesions [20, 21].

Despite a general consensus on the presence of dysbiosis, detailed data regarding changes in the composition of the skin microbiota in psoriasis are inconsistent. Some studies, such as those conducted by Gao et al., indicate a decrease in the abundance of Actinobacteria and Propionibacterium spp., accompanied by an increase in Firmicutes, whereas Alekseyenko et al. reported an increase in both Firmicutes and Actinobacteria alongside a reduction in taxonomic diversity [22, 23]. Regarding the genus *Staphylococcus*, some studies report an increased abundance in psoriatic lesions (particularly *Staphylococcus aureus*) alongside a simultaneous decrease in *Staphylococcus epidermidis*, whereas other studies, such as those by Fahlén et al., report an overall decline in staphylococci [17, 24, 25]. Data concerning *Corynebacterium* spp. are also inconsistent — some

studies suggest its reduction, while others indicate increased presence in more inflamed lesions and a positive correlation with disease severity (PASI) [20, 24].

One of the most commonly occurring fungi on human skin, both healthy and diseased, is *Malassezia* spp. Studies have shown that although *Malassezia* is present in both types of skin, its abundance tends to be lower in patients with psoriasis compared to healthy individuals [17, 24, 26]. At the same time, greater fungal diversity is observed in psoriatic lesions than in healthy skin. This may indicate a disturbed microbial balance of the skin in psoriasis patients, potentially contributing to the maintenance or exacerbation of the inflammatory process [20, 24, 26]. Some studies indicate that increased *Malassezia* spp. levels on the skin may primarily occur during disease flare-ups, suggesting a possible association with increased inflammation [26]. Additionally, the presence of fungi, especially *Malassezia restricta*, was most frequently identified in patients with psoriasis [26].

Conversely, other analyses indicate no significant differences in the composition of the fungal microbiome between healthy and psoriatic skin, highlighting the complexity and variability of this research area [24].

Mechanisms through which fungi may influence psoriasis pathogenesis include the potential ability of certain species, such as *M. sympodialis*, to induce the expression of pro-inflammatory cytokines including TNF-α, IL-1, IL-6, and IL-8. These cytokines are important mediators of inflammation in psoriasis, leading to activation of immune cells and keratinocyte hyperproliferation. Furthermore, these fungi may promote dendritic cell maturation and mast cell activation, further amplifying the inflammatory process [20].

This phenomenon may also affect treatment responses — it has been shown that high *Malassezia* spp. concentrations within psoriatic lesions may be associated with an increased tendency for irritation after using topical vitamin D analogues, such as calcipotriol. Interestingly, the use of antifungal agents may lead to improvement, especially in nail psoriasis, which may indirectly indicate a role of fungi in the pathogenesis of nail psoriasis [26, 27].

Ultimately, although all analyzed studies confirm the presence of alterations in the skin microbiome composition in psoriasis, their results are ambiguous and sometimes contradictory. This may be due to differences in analytical methods used, sampling locations on the skin, disease stage, or individual characteristics of the patients studied. Currently, there is no clear answer as to whether skin microbiome dysbiosis is a cause or a consequence of psoriasis, making the causal relationship difficult to determine [24].

Microbiome in Acne Vulgaris

Acne vulgaris is a chronic inflammatory skin disease that most commonly affects teenagers and young adults, although it can persist into the 30s and 40s. In the United States alone, this condition affects approximately 50 million people annually, making it the most common dermatological disease in the country. Although acne does not pose a direct threat to life, its consequences—such as scarring, hyperpigmentation, low self-esteem, depression, and anxiety—can significantly impair patients' quality of life. Acne lesions primarily occur on the face, chest, and back, and are classified by severity into mild, moderate, and severe forms. In clinical practice, due to the lack of a uniform and widely accepted grading system, physicians most often base their assessment on the number, type, and location of acne lesions [28].

The foundation of acne development lies in changes occurring within the pilosebaceous unit—a structure composed of the hair follicle and its associated sebaceous gland. The key pathophysiological mechanisms include excessive sebum production, abnormal shedding of follicular epithelial cells, colonization by the bacterium *Cutibacterium acnes*, and the accompanying inflammatory response. Overproduction of sebum most often results from increased androgen activity or heightened sensitivity of sebaceous glands to these hormones. The inflammatory process can occur at any stage of the disease—when the integrity of the hair follicle is disrupted, its contents are released into the surrounding tissues, triggering an inflammatory response that leads to the formation of papules, pustules, nodules, and even cysts. In addition to inflammatory lesions, non- inflammatory forms also occur, such as open (blackheads) and closed (whiteheads) comedones, which develop due to the accumulation of sebum and keratin at the follicular opening [28, 29, 30].

Cutibacterium acnes (formerly *Propionibacterium acnes*) is a Gram-positive, anaerobic bacterium that constitutes a dominant component of the human skin microbiome, especially in areas rich in sebaceous glands such as the face, scalp, back, and chest. It was first described by Unna in 1896, and a year later isolated by Sabouraud from acne lesions, laying the groundwork for the hypothesis regarding its role in acne pathogenesis. Initially named *Bacillus acnes*, then *Corynebacterium acnes*, it was eventually renamed *Propionibacterium acnes* due to its ability to produce propionic acid. In 2015, phylogenetic analyses and genome sequencing led to the proposal of dividing it into three subspecies: *P. acnes* subsp. *acnes*, *defendens*, and *elongatum*. A year

later, a new name was introduced—*Cutibacterium acnes*—which better reflects the taxonomic classification of this bacterium [29, 31].

Cutibacterium acnes is not only a commensal bacterium but also an opportunistic pathogen. Although its presence does not necessarily indicate disease, significant changes in the microbiota composition and the proportions between C. acnes phylotypes are observed in individuals with acne. Importantly, it is not the overall bacterial abundance that differs—since it is similar in both acne patients and healthy individuals—but rather an imbalance between different C. acnes types, known as dysbiosis. Based on analysis of the recA gene sequence and multilocus sequence typing (MLST), four main phylotypes have been distinguished: IA, IB, II, and III, with phylotype IA further divided into subtypes IA1 and IA2. Among these, phylotype IA1 has the greatest pathogenic significance and is significantly overrepresented in acne lesions. The IA1 phylotype shows increased ability to colonize the pilosebaceous units and induce inflammatory responses. Strains belonging to this group exhibit elevated expression of genes encoding virulence factors such as proteases, lipases, phosphatases, and porphyrins. In contrast, phylotypes IB and II are mainly found on healthy skin and are considered less pro-inflammatory. They are characterized by limited porphyrin production and lower expression of virulence genes [27, 29, 30, 31, 32].

In the pilosebaceous unit, this bacterium utilizes sebum lipids as a metabolic substrate, and its presence influences the local skin environment. *C. acnes* increases sebum secretion, likely through the activation of enzymes such as diacylglycerol acyltransferase. It breaks down triglycerides in sebum, releasing free fatty acids that can induce comedone formation and create a pro- inflammatory environment. Additionally, porphyrins secreted by *C. acnes* catalyze the oxidation of squalene, which increases oxidative stress and promotes comedogenesis [27, 29, 31, 32].

The inflammatory response triggered by *Cutibacterium acnes* involves both the innate and adaptive immune systems. The bacterium produces toxins such as CAMP factors, whose expression and activity vary depending on the phylotype. In particular, CAMP1 can activate Toll-like receptors, initiating a signaling cascade that leads to the production of numerous proinflammatory cytokines, including interleukins IL-1α, IL-1β, IL-6, IL-8, IL-12, and tumor necrosis factor alpha (TNFα). These cytokines indirectly induce the production of extracellular matrix metalloproteinases, which participate in tissue remodeling and the development of inflammation, often resulting in scarring. Furthermore, *C. acnes* influences the activation of T helper 17 (Th17) cells, which are a primary source of cytokines that recruit neutrophils and sustain chronic inflammation in the skin tissue. In this way, the bacterium contributes to the exacerbation and persistence of inflammation characteristic of acne vulgaris [27, 29, 32].

The ability of *C. acnes* to form biofilms plays a key role in its resistance to treatment, as the biofilm protects the bacteria from the effects of antibiotics and aids adherence to the walls of the pilosebaceous unit. The presence of biofilm is significantly more common in acne lesions than on healthy skin and contributes to the maintenance of chronic inflammation [32].

Staphylococcus epidermidis and other coagulase-negative staphylococci, such as *Staphylococcus hominis*, are found on the skin of both healthy individuals and those with acne. In acne-affected skin, the number of *S. epidermidis* increases, which inhibits the growth of other microorganisms through the production of succinic acid. Additionally, studies report that *S. epidermidis* secretes polymorphic toxins and staphylococcal lipoteichoic acid, which reduces inflammation by inhibiting the TLR-2 receptor, leading to decreased production of proinflammatory cytokines such as IL-6 and TNF-α [29, 30].

Fungi of the genus *Malassezia*, which dominate the skin mycobiome, also participate in the pathogenesis of acne. In particular, *Malassezia restricta* and *Malassezia globosa* have been identified in acne lesions. Their lipases, with activity up to 100 times higher than other skin enzymes, hydrolyze triglycerides into free fatty acids, which induce hyperkeratinization of hair follicles and the formation of comedones. Free fatty acids also stimulate neutrophil migration and the production of proinflammatory cytokines by keratinocytes and monocytes. Antifungal treatment in some patients reduces skin lesions, suggesting the involvement of *Malassezia* in therapy-resistant acne. The role of these fungi requires further research [29, 30, 31].

Topical and systemic use of probiotics, prebiotics and postbiotics

Probiotics are defined as "live microorganisms which, when consumed in adequate amounts, confer health benefits to the host organism." As early as 1900, Louis Pasteur was the first to identify the microorganisms responsible for the fermentation process. His research led to the discovery that consuming fermented products, such as yogurt—containing beneficial bacteria—improved health and influenced the longevity of the inhabitants of a Bulgarian village. Pasteur proposed the hypothesis that "lactic acid bacilli

(lactobacilli) may help counteract the harmful effects of metabolic changes in the digestive tract, which contribute to diseases and aging." [33].

Over the past ten years, interest in the use of probiotics—both oral and topical—in skin care and the treatment of dermatological conditions has significantly increased. With the emergence of new products on the market, research teams have been intensively working on evaluating their efficacy, mechanisms of action, safety, and appropriate applications. According to Lee et al., a limited number of clinical studies have demonstrated the potential benefits of using probiotics in the therapy of acne, atopic dermatitis, and rosacea [33].

The microbiota of individuals with rosacea differs from that of people without this condition— patients with rosacea exhibit a distinct microbiological profile. Systemic antibiotics used in treatment can modify the composition of this microbiota, particularly affecting the diversity and presence of Cutibacterium acnes (formerly Propionibacterium acnes), a bacterium linked to the severity of acne symptoms. One of the main challenges in contemporary acne therapy is patient adherence—topical treatments often damage the skin's protective barrier, causing dryness and irritation. Park et al. observed that probiotics influence various aspects of acne pathophysiology and may also improve patient engagement in treatment. Increasing attention is also being paid to the connections between gut microbiota and acne. New research on the gut–brain–skin axis and acne immunobiology suggests that stress can disrupt the balance of gut microbiota, contributing to skin inflammation and disease flare-ups. These findings indicate that dietary interventions and probiotic supplementation may help restore microbial balance in the gut and thereby alleviate acne symptoms [33].

Di Marzio et al. demonstrated that Streptococcus thermophilus, found among others in yogurt, increases ceramide production in the skin after just 7 days of topical application. Bowe et al. emphasize the significance of this in the context of acne, as certain ceramides—such as phytosphingosine—exhibit antibacterial and anti-inflammatory properties against Cutibacterium acnes (formerly Propionibacterium acnes). It has been proven that topical application of phytosphingosine reduces the number of acne lesions. In vitro studies also showed that probiotic strains combined with konjac glucomannan hydrolysates (GMH) effectively inhibit the growth of C. acnes. Moreover, Enterococcus faecalis SL-5, a lactic acid bacterium, demonstrated strong antibacterial activity, and an emulsion containing this strain significantly reduced pustule counts, suggesting its potential use as an alternative to topical antibiotics [33].

In a study on atopic dermatitis (AD), Di Marzio et al. showed that applying a cream containing Streptococcus thermophilus for two weeks significantly increased ceramide levels in the stratum corneum of 11 patients. This effect likely results from the bacterial sphingomyelinase activity. Patients also showed improvements in erythema, scaling, and itching, indicating beneficial effects of probiotics in other dermatological conditions as well [33].

One method of modifying the skin microbiome is skin bacteriotherapy, which involves the application of one or several beneficial microbial strains onto previously cleansed skin. Depending on the form used, these can include:

1. Live probiotics – active, live bacteria that, in adequate amounts, exert a beneficial effect on skin health.
2. Tyndallized or heat-inactivated bacteria – inactive microorganisms that, despite being deactivated, retain their structures and secreted factors.
3. Cell lysates – physically destroyed bacteria whose enzymes and cellular contents still exhibit biological activity.
4. Purified enzymes – isolated bacterial enzymes applied without the presence of the microorganisms themselves.
5. Fermentation products – bacterial supernatants rich in antioxidants, amino acids, lipids, and vitamins.

The use of these various forms of bacteriotherapy represents a modern approach to supporting skin health by modulating its microbiome, without the need for traditional antibiotics or anti- inflammatory drugs.

The approaches described above are more scalable and industry-friendly than full skin microbiome transplants. Live probiotics, in particular, can be used at higher concentrations, potentially increasing their effectiveness. Both probiotics and postbiotics are most commonly delivered in the form of creams, emollients, or other suitable carriers. However, certain challenges remain. Bacteria cultured in sugar-rich environments may struggle to adapt to the skin environment, which is rich in sebum. Furthermore, their stable colonization (engraftment) on the skin is hindered by competition with the host's natural microbiota. Additionally, high doses of bacteria can provoke immune reactions, leading to irritation or adverse effects. [34]

Another method of altering the skin microbiome is prebiotic stimulation, which involves applying compounds that promote the growth of beneficial skin microbes. Prebiotics are non-living ingredients with selective activity that support host health.

Advantages include:

- No use of live bacteria, reducing the risk of immune reactions.
- Prebiotics are well-characterized, with established safety profiles and standardized INCI listings.
- Their indirect action makes them generally safe and stable.
- However, there are drawbacks:
- The effects are less direct and may take longer to manifest.
- Prebiotics might also stimulate unintended or non-beneficial microbes.
- Outcomes can vary due to individual differences in skin microbiome composition, physiology, and immune response [34].

A study on 31 individuals with atopic dermatitis (AD) examined the effects of a lotion containing heat-treated Lactobacillus johnsonii NCC 533. After 3 weeks of use, the lotion reduced Staphylococcus aureus colonization and was linked to local clinical improvement, as measured by the SCORAD index [33].

Skin microbiota transplantation – emerging directions in research

Skin microbiome transplantation is an emerging method inspired by fecal microbiota transplantation (FMT), which is used to treat *Clostridium difficile* infections by transferring gut microbes from a healthy donor to a patient. Similarly, skin microbiome transplantation involves transferring microorganisms from the surface of healthy skin onto a disinfected area of another person's skin to improve its condition. This approach delivers microbes in their natural form, potentially enhancing the effectiveness of the intervention. However, this method faces significant limitations. Only small amounts of bacteria can be collected, and their multiplication often requires laboratory culturing. Additionally, scaling up this procedure for widespread commercial use is challenging. There is also uncertainty about which specific microorganisms—beneficial or potentially harmful—are actually transferred with the microbiome, raising safety concerns. In summary, while skin microbiome transplantation holds promise as a novel therapeutic strategy, practical and safety issues currently restrict its broader application and commercialization. [34]

Transplanting microbes from one body site to another within the same subject

Costello et al. conducted a study aimed at determining whether changes in microbial communities depend on environmental factors or result from prior exposures. To investigate this, they transplanted bacteria from the tongue to the forehead or forearm, as well as from the forearm to the forehead or tongue. Samples were collected 2, 4, and 8 hours after transplantation. It was found that bacteria originating from the tongue successfully colonized the forearm but did not establish themselves on the forehead, suggesting that local environmental conditions of the skin play a decisive role in the ability of microorganisms to inhabit a given area. [34]

Transplanting skin microbes from one skin site to another within the same subject

Many studies highlight the importance of syntrophy—that is, metabolic cooperation between different microbial species—in maintaining the balance of the skin microbiome. Although most experiments focus on transferring single bacterial strains, often overlooking complex nutritional interdependencies (known as cross-feeding), some research examines the effects of transplanting the entire native microbiome from one skin area to another. This approach allows the preservation of natural interactions between different microbial species. [34]

Leyden et al. (1981) investigated whether bacteria responsible for unpleasant odors in the underarm area could induce similar effects when transferred to another part of the body. Two types of diphtheroid bacteria were applied to the forearms of volunteers, and the emergence of a strong odor confirmed that these microorganisms can successfully transplant and retain their properties even outside their original environment. [34]

S.epidermidis application on facial skin

In a randomized, double-blind clinical trial, strains of *Staphylococcus epidermidis* were isolated from participants, cultured, and then reapplied to the facial skin twice a week for four weeks. Compared to the control group, individuals undergoing the therapy showed an increase in skin lipid and water content, a reduction in transepidermal water loss, and a decrease in skin pH from 5.5 to 5.0. The pH reduction may have resulted from increased production of lactic and propionic acids. The study results confirmed the beneficial effect of *S. epidermidis* on facial skin, highlighting its potential as a cosmetic ingredient. [34]

The impact of treatment eg. Antibiotics on the skin microbiome

In a study of the skin microbiome across nine uninfected body sites in individuals with acute purulent skin and soft tissue infections (SSTIs) and those without, no significant differences in microbiome diversity were found between the groups—except in the gluteal fold. Similarly, Cranendonk and colleagues did not observe significant differences in the microbiota of patients with non-purulent SSTIs (e.g., cellulitis) compared to a control group, instead noting a correlation between the microbiota in infected lesions and that on the contralateral healthy limb. It is worth noting, however, that the discussed study focused on purulent SSTIs, which may have a different pathogenesis. [35]

Interestingly, these results differ from the observations of Horton et al., who in a cross-sectional study demonstrated a similar microbiota composition—including a high level of *Staphylococcus aureus*—both on infected skin and on the healthy contralateral skin, distinguishing them from individuals without infection. However, unlike Horton's study, the discussed research included diverse body sites characterized by different skin environments (dry, moist, sebaceous) as well as mucous membranes such as the nares and throat, which are known colonization sites for *S. Aureus.* [35]

The gluteal fold was the only site where clear differences in microbiome composition were observed—it showed increased species diversity and a higher relative abundance of Corynebacterium species compared to the control group. In individuals with purulent skin and soft tissue infections (SSTIs), the infection site after treatment also exhibited a similar increase in diversity and abundance of Corynebacterium. Consistent with previous studies, no significant differences were found in the microbiota of the nares and groin areas. Additionally, at the SSTI site, an inverse relationship was observed between the presence of Corynebacterium and Staphylococcus aureus, both before and after antibiotic therapy. [35]

A review of four studies examining the impact of systemic or oral antibiotics—including minocycline, doxycycline, and lymecycline—on the skin microbiome revealed varied effects on microbial diversity. Two of these studies included healthy control groups, and one compared the effects of oral lymecycline with isotretinoin. [36]

All four studies reported measurements of alpha diversity. Three observed an increase in alpha diversity following treatment, while one noted a decrease. Notably, two studies reported statistically significant increases in alpha diversity among acne patients, assessed using the Shannon and Inverse Simpson indices. For instance, Park et al. demonstrated a 1.27-fold increase in the Shannon index ($P= 0.03$) and a 1.11-fold increase in the Inverse Simpson index ($P = 0.03$) after six weeks of doxycycline therapy. Similarly, Kelhälä and colleagues observed significant increases in alpha diversity on the back ($P \leq 0.05$) and cheeks ($P \leq 0.01$) following treatment with lymecycline or isotretinoin, although they did not differentiate the effects of each therapy. However, the same study also noted a significant decrease in armpit microbiome diversity post-treatment ($P \leq 0.01$). [36]

Thompson et al. observed an increase in alpha diversity following treatment; however, these changes were not statistically significant when compared to baseline values or to healthy control subjects. Conversely, Chien and colleagues reported an overall decrease in alpha diversity after antibiotic therapy. While this reduction was not statistically significant at the group level, significant decreases were noted in two of the four individual participants. [36]

Three out of four studies reported a significant decrease in the abundance of Cutibacterium species, particularly C. acnes, following antibiotic treatment. The fourth study, conducted by Thompson et al., also demonstrated a notable reduction in C. acnes levels when compared to healthy control subjects. Additionally, Dreno and colleagues, investigating the topical application of 4% erythromycin, did not observe changes in alpha diversity; however, they did report a significant decrease in Cutibacterium within comedones after therapy. [36]

Conclusions

Chronic inflammatory dermatoses, such as atopic dermatitis, psoriasis and acne vulgaris, are serious skin conditions characterized by inflammation that negatively impact the quality of life of patients. The skin microbiota plays a significant role in the pathophysiology of skin inflammatory dermatoses. At the same time, an increasing number of studies point to the therapeutic potential of the microbiome of the skin. Further understanding of the skin microbiota, its role in forming the skin's protective barrier, the consequences of dysbiosis, as well as the therapeutic potential of the microbiota of the skin is essential. Continued research is needed to determinate the significance of the influence of the skin microbiome on the course and treatment of chronic inflammatory dermatoses.

Disclosures

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