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THE ROLE OF THERAPY IN MODULATING THE SKIN MICROBIOME IN CHILDREN WITH ATOPIC DERMATITIS

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ABSTRACT

Objectives. Atopic dermatitis is a chronic dermatological condition characterized by epidermal barrier dysfunction, intensified pruritus, and the coexistence of other atopic diseases, most often beginning in early childhood. The disease has a multifactorial etiology involving genetic, immunological, and environmental factors, and is associated with reduced quality of life, particularly in children.

Methods. A literature review was performed using PubMed, Google Scholar, and ResearchGate for studies in Polish and English using the keywords “atopic dermatitis,” “dysbiosis,” “microbiota,” “skin microbiota,” and “S. aureus.” Incomplete, off-topic, methodologically insufficient, unreliable, or outdated studies were excluded.

Key findings. In children with atopic dermatitis, skin microbiota dysbiosis has been observed, manifested as reduced microbial diversity and a predominance of bacteria such as *Staphylococcus aureus*. In contrast, healthy skin exhibits a more diverse microbiome, with commensal bacteria like *Staphylococcus epidermidis* playing a crucial role in maintaining homeostasis. Dysbiosis in atopic dermatitis disrupts epidermal barrier function, exacerbates inflammatory symptoms, and facilitates colonization by pathogens. Skin microbiome fluctuations can vary depending on the therapy employed, including glucocorticosteroids, emollients, or biological treatments, which restore microbial balance.

Conclusions. Microbiome-modulating therapies show potential in alleviating clinical symptoms and supporting beneficial bacteria, yet research on the impact of microbiota on the progression of atopic dermatitis, especially in children, remains limited. Further studies are necessary to better understand the disease mechanisms, develop more targeted therapeutic approaches, and evaluate the long-term efficacy of microbiome-focused treatments.

KEYWORDS

Atopic Dermatitis, Microbiota, Dysbiosis, S.aureus

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Introduction.

Atopic dermatitis (AD) is defined as a chronic or relapsing inflammatory skin disease characterized by impaired epidermal barrier function and abnormalities in immune regulation. In AD, a characteristic morphology and distribution of lesions are observed, accompanied by pronounced pruritus and the coexistence of other atopic conditions in the patient or their family members (Bylund et al, 2020). It typically manifests in early childhood and is more prevalent in pediatric populations, affecting up to 25% of children. Approximately 80% of patients develop AD within the first years of life, while remission is observed in about 60% of patients during adolescence (Bylund et al, 2020). AD is a condition that may predispose patients, in later stages of life, to comorbid diseases such as asthma, food allergies, and allergic rhinitis (Maintz et al, 2022). Studies indicate that children with AD experience a reduced quality of life and are more likely to require hospitalization compared to their healthy peers (Djurović et al, 2020)(Hua & Silverberg, 2019). Behavioral issues among pediatric patients with AD include increased dependency on caregivers, elevated anxiety levels, and sleep disturbances—typically as a consequence of the chronic discomfort associated with the disease (Langan, Irvine & Weidinger, 2020). The etiology of AD is multifactorial, involving genetic predisposition, dysregulation of the immune system, defects in the epidermal barrier, and environmental influences. Moreover, the role of the microbiome in the pathogenesis of this condition is garnering increasing attention in the scientific literature (Kong et al, 2012).

Objective.

The aim of this study is to elucidate the role of the skin microbiome in the pathogenesis, progression, and treatment of AD. A review of the literature and scientific research will assess how disruptions in the microbiome affect skin barrier function and the development of the inflammatory process in AD. Additionally, the study intends to outline potential therapeutic strategies that may support the restoration of a balanced skin microbiome in patients with AD.

Materials and Methods.

A literature review was conducted using the PubMed, Google Scholar, and ResearchGate databases. The review included studies published in both Polish and English. Articles were identified using the keywords: “atopic dermatitis,” “dysbiosis,” “microbiota,” “skin microbiota,” and “S. aureus.” Studies that were incomplete, outside the scope of the discussed topics, lacked an adequate description of the methodology, were unreliable, or contained outdated medical data were excluded.

How Does the Skin Microbiome Change in Children with Atopic Dermatitis?

In patients with AD, there are marked differences in microbial diversity and the structure of microorganisms between areas of the skin affected by lesions and unaffected regions of the epidermis. During exacerbations, children with AD exhibit a reduction in the diversity of microorganisms in the skin microbiome, which correlates with an increase in the severity of clinical symptoms (Bjerre et al, 2017). In individuals with AD, the skin microbiome is characterized by a predominance of bacteria such as *Staphylococcus aureus* and *Staphylococcus epidermidis*, alongside a reduced abundance of bacteria belonging to the genera *Streptococcus*, *Propionibacterium*, *Corynebacterium*, *Acinetobacter*, and *Prevotella* (Kong et al, 2012). Research findings indicate that the dominance of *S. aureus* in the skin’s bacterial composition may initiate damage to the epidermal barrier, thereby facilitating further penetration of the bacterium into the dermis. Once in the dermis, the presence of *S. aureus* induces the production of proinflammatory cytokines through internal dysregulation

of the host's immune system and the expression of virulence genes. Virulence factors—such as alpha-toxin, protein A, lipoteichoic acid, phenol-soluble modulins, and proteases—can lead to keratinocyte damage (Nakatsuji et al, 2016) (Cheung, Bae & Otto, 2021). Strains of *S. aureus* residing in patients with AD exhibited an enrichment in virulence factors, whereas in individuals with milder forms of the disease, *S. epidermidis* strains predominated (Deng et al, 2023). Colonization of the skin by *S. epidermidis*, a commensal staphylococcal species, has been associated with a decreased incidence of AD in infants, suggesting that these microorganisms may potentially participate in the processes underlying the disease's pathogenesis (Kennedy et al, 2017). Nakatsuji and colleagues demonstrated that bacterial species such as *S. epidermidis* and *S. hominis* stimulate the synthesis of antimicrobial peptides by host cells—including cathelicidins and human beta-defensins—and produce antimicrobial factors that limit the proliferation of pathogenic bacteria (Nakatsuji et al, 2017). Dysbiosis of the skin microbiota significantly impacts the composition and integrity of the skin's hydro-lipid barrier. In a study conducted by Kim et al., lipid profiles of the stratum corneum as well as the microbiological composition of the skin in children with AD were analyzed. The results indicated a predominance of short-chain fatty acids in these children, with particularly elevated concentrations in areas of skin exhibiting pathological changes. Elevated levels of these short-chain fatty acids correlated with impaired epidermal barrier function and dysbiosis of the skin microbiota (Kim et al, 2023).

Factors Shaping the Skin Microbiome in Children

Dysbiosis of the skin microbiome may result from various environmental and behavioral factors, including cesarean section (CS) deliveries, diet, frequent exposure to antibiotics, the use of cosmetics and chemical detergents, and urban living (Renz & Skevaki, 2021). Studies have shown that neonates delivered vaginally exhibit a skin microbiota resembling the maternal vaginal microflora—comprising bacteria such as *Lactobacillus* and *Prevotella*. In contrast, neonates born via CS predominantly harbor bacteria from the maternal skin microbiota, including *Staphylococcus*, *Corynebacterium*, and *Cutibacterium* (Dominguez-Bello et al, 2010). Furthermore, the mode of cesarean delivery itself influences the formation of the neonatal skin microbiota; infants born by emergency CS demonstrate a lower abundance of *Lactobacillus* compared to those born via planned CS (Rapin et al, 2023). Environmental conditions also significantly impact the risk of developing AD. Research indicates that children residing in rural areas have a lower risk of AD compared to their urban counterparts. The heightened risk among urban children is attributed to increased exposure to air pollutants, which play a pivotal role in disrupting skin homeostasis and initiating inflammatory processes (Levin et al, 2020). Exposure to air pollutants, including particulate matter, can markedly affect the skin microbiota and its metabolome by promoting increased colonization by *S. aureus* (Leung et al, 2023).

How Does the Skin Microbiome Change as a Result of Atopic Dermatitis (AD) Therapy?

Therapy targeting AD exacerbations plays a crucial role in alleviating clinical symptoms and influences the taxonomic composition of the skin microbiome by reducing the relative abundance of *S. aureus* (Bjerre et al, 2017). One therapeutic modality involves the topical application of glucocorticosteroids (GCS). Gonzalez et al. reported that treatment with GCS-containing creams facilitates the restoration of microbial balance in areas affected by AD. Upon completion of treatment, the bacterial composition of pathologically altered skin became more similar to that of healthy skin, although differences persisted when compared to the microbiota of control subjects with no AD symptoms (Gonzalez et al, 2016). Both topical steroids alone and combination therapy with steroids and antimicrobial agents resulted in a reduction in AD severity, as well as a decrease in the abundance of *Cutibacterium* in diseased skin areas. Notably, combined topical treatment with an antimicrobial agent and a steroid demonstrated superior efficacy in restoring the bacterial skin microbiome, whereas monotherapy with a topical steroid did not ameliorate the dysbiosis (Tingting et al, 2025). Patients with AD undergoing topical GCS therapy exhibited increased bacterial diversity and a lower relative abundance of *S. aureus* compared to untreated individuals (Edslev, Agner & Andersen, 2020).

The use of emollient therapy plays a significant role in modulating the skin microbiota. Xu et al. conducted a study involving children aged 4 to 18 years with mild to moderate AD, during which emollients were incorporated into their skin care regimen. The study results revealed a significant reduction in the abundance of *S. aureus* and an increase in microbial diversity in areas of damaged skin (Xu et al, 2020). Glatz et al. demonstrated that emollient use promotes the restoration of the skin microbiota by lowering skin pH and enhancing bacterial diversity. A notable increase in the relative abundance of *S. salivarius* was observed in the groups using emollients compared to the control group. As AD progresses and exacerbations occur, the proportion of *S. salivarius* diminishes while the abundance of *S. aureus* increases. Given that *S. salivarius*

exhibits immunomodulatory properties and the ability to regulate skin pH, these findings suggest a beneficial effect of emollient therapy in the treatment of AD (Glatz et al, 2018).

Biologic therapy demonstrates significant advances in the treatment of AD. Dupilumab is a monoclonal antibody used in AD management that targets components of the Th2 inflammatory axis by blocking the IL-4R α receptor, thereby effectively inhibiting IL-4 and IL-13 cytokine signaling (Drucker et al, 2020). The pathogenesis of AD is driven by a complex immuno-inflammatory response in which Th2 lymphocytes and B cells play a crucial role, leading to the production of specific IgE antibodies. Disruptions in the skin's lipid structure, resulting from decreased expression of filaggrin and loricrin, facilitate the penetration of antigens, allergens, and pollutants through the stratum corneum, ultimately compromising the epidermal barrier. In this process, alarmins such as IL-25, IL-33, and thymic stromal lymphopoietin (TSLP) are released, triggering the activation of type 2 innate lymphoid cells and dendritic cells, which in turn stimulate the production of IL-5 and IL-13. These cytokines further initiate the activation of Th2 lymphocytes and eosinophils, and promote class switching in B cells, leading to the generation of specific IgE (Facheris et al, 2023)(Nowowiejska, Baran & Flisiak, 2023).

Studies have demonstrated that dupilumab therapy results in significant clinical improvement, a reduction in inflammatory infiltrates, and decreased expression of proinflammatory cytokines and chemokines in affected areas (Möbus et al, 2022). Simpson et al. conducted a study involving AD patients, all of whom exhibited skin colonization by *S. aureus*. Patients receiving dupilumab, compared with those receiving placebo, showed a reduction in *S. aureus* abundance on the skin as early as three days into the study. This reduction in *S. aureus* colonization was significantly associated with more pronounced clinical improvement. Moreover, dupilumab therapy led to a significantly greater decrease in the abundance of *S. aureus* in lesional skin compared to the less marked reduction observed in non-lesional areas (Simpson et al, 2023). In one study, dupilumab treatment was associated with a reduction in *S. aureus* abundance along with a simultaneous increase in *S. hominis* abundance, both in lesional skin and, to a lesser extent, in non-lesional skin (Callewaert et al, 2020). Hartmann et al. described that dupilumab therapy altered the composition of the skin microbiota, aligning it more closely with the pattern characteristic of healthy individuals in the control group; notably, these changes were largely independent of the degree of clinical improvement achieved (Hartmann et al, 2023). Further studies are required to precisely evaluate the effectiveness of IL-4R α receptor blockade in AD treatment and its impact on restoring skin microbiota homeostasis.

Conclusions

The skin microbiome plays a pivotal role in the pathogenesis of AD by regulating immune processes and maintaining the integrity of the epidermal barrier. In patients, including children with AD, a reduced diversity of the skin microbiota has been observed, which contributes to disrupted microbial homeostasis and increases susceptibility to colonization by pathogens such as *S. aureus*. Studies also indicate that the composition of the skin microbiota dynamically shifts in response to various therapeutic interventions, including biologic treatments. Therapies that target microbiome modulation can not only enhance epidermal barrier function but also alleviate clinical symptoms by reducing the predominance of pathogenic bacteria and fostering the growth of commensal flora. The limited number of studies focusing on the role of the skin microbiome in the pathogenesis of AD in pediatric populations underscores the need for further research. Such studies are essential to deepen our understanding of disease mechanisms and to develop more targeted and effective therapeutic strategies.

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Ethical consideration

This review is based exclusively on previously published data, all of which are publicly accessible through academic databases and journal publications. As no new patient data was collected or analyzed, ethical approval was not required.

Conflict of interest statement

The authors declare no conflict of interest.

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