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EXPLORING THE POTENTIAL OF MICROBIOME-CONTAINING MOISTURIZERS IN MANAGING ATOPIC DERMATITIS

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Abstract
Atopic dermatitis (AD) is a chronic inflammatory skin disorder driven by barrier dysfunction, immune dysregulation, and microbiome dysbiosis, affecting up to 20% of children worldwide. This review explores the pathophysiology of AD, emphasizing epidermal defects like filaggrin mutations, elevated pH, and reduced antimicrobial peptides that promote *Staphylococcus aureus* overgrowth and Th2 inflammation. The gut-skin axis also plays a contributory role, with gut dysbiosis reducing short-chain fatty acids and increasing skin flares. The function of the skin microbiome in immune modulation is also discussed, with commensals such as *S. epidermidis* synthesizing antimicrobials and inducing T-cell responses through Toll-like receptors. However, age-related changes may disrupt this homeostasis. Microbiome-based moisturizers (prebiotics, probiotics, postbiotics, and synbiotics) have become innovative treatments that help in restoring eubiosis, reducing pH, and increasing hydration. Clinical data, including RCTs and meta-analyses from 2024 to 2025, report improved SCORAD scores, decreased transepidermal water loss, and microbiome normalization, as well as prevention in infants. These non-steroidal alternatives supplement conventional treatment, providing better long-term control.

Keywords: Atopic Dermatitis, Skin Microbiome, Dysbiosis, Microbiome-Containing Moisturizers, Gut-Skin Axis

Introduction

Atopic dermatitis (AD) is a persistent inflammatory skin condition that manifests as severe itching, red, dry, and scaly patches, and is highly common in children, with a global prevalence of up to 20% [1]. Composed of lipids, proteins of the tight junction, and structural components of the stratum corneum (SC), the epidermal barrier plays a crucial role in preventing dryness by regulating water

loss [2]. When this weakened layer is penetrated by high-molecular-weight allergens, such as dust mite antigens, food proteins, and microbes, it provokes the activation of the innate immune system and inflammation [2]. The pathogenesis of AD is attributed to the interaction of genetic factors, such as mutations in the filaggrin gene that interfere with skin barrier integrity [3], environmental factors, including allergens and pollutants, and immune dysregulation, where high levels of cytokines such as IL-4, IL-13, and IL-31 stimulate overactive Th2 responses [3, 4]. Research has demonstrated that microbiome dysbiosis is a significant factor contributing to AD, with an imbalanced skin and gut microbiome community disrupting homeostasis and exacerbating inflammation [5]. For example, AD patients typically exhibit decreased microbial diversity on the skin, with increased pathogenic bacteria such as Staphylococcus aureus, which colonizes up to 90% of AD lesions and exacerbates the inflammatory effects through toxin production and immune activation [6].

Traditional methods of AD management are topical corticosteroids and calcineurin inhibitors. The past few years have sparked an intensifying interest in microbiome-containing moisturizers that integrate prebiotics, probiotics, postbiotics, or bacterial lysates to adjust the microbial balance and re-establish skin homeostasis [1, 5]. These products, including those containing *Vitreoscilla filiformis* lysate or ceramide-rich emollients, nurture the healthful commensals that populate the skin, such as *Staphylococcus epidermidis*, thereby lowering the skin pH, inhibiting pathogens, and adjusting immunological responses to reduce flare-ups [6]. Such moisturizers provide a non-steroidal, safe alternative that supplements conventional therapies such as topical corticosteroids. This review aims to assess the current evidence on the usefulness of microbiome-enriched moisturizers in AD by exploring clinical evidence in terms of effectiveness.

Pathophysiology of AD

The skin barrier serves as the primary line of defense against environmental insults, pathogens, and allergens. This skin barrier is also damaged by loss-of-function mutations in the protein filaggrin (FLG), a significant protein in skin barrier formation, which causes increased transepidermal water loss, a more elevated skin pH, and reduced levels of antimicrobial skin peptides, including LL-37 and beta-defensins [6, 7]. An elevated skin pH has been linked to reduced production of defensins, which contributes to increased pathogen growth and suppression of commensals, including *S. epidermidis* [8]. Such an environment favors the overgrowth of *S. aureus*, which utilizes the mechanism of quorum sensing to produce biofilms and secrete toxins, including α-toxin and δ-toxin, thereby triggering Th2-mediated inflammation via the IL-4 and IL-13 pathways, which further impairs barrier repair [6, 8].

Microbial dysbiosis is a major contributor to AD. It is characterized by a reduced microbial diversity of *S. epidermidis* and *Corynebacterium*, which generally produce inhibitory metabolites against pathogens [8]. This imbalance increases inflammation through lipid-microbiome-immune loops, in which impaired sebum composition decreases ceramide and fatty acid levels, facilitating *S. aureus* adhesion and inducing innate immune responses, such as TLR2 stimulation [9]. In infants, poor microbial diversity at birth, primarily due to cesarean delivery or antibiotic exposure, is predictive of AD onset, with a relative reduction in α-diversity associated with early eczematous lesions observed in cohort studies [10]. Infant studies show early dysbiosis, with lower *Bifidobacterium* at 26 weeks predicting eczema onset, alongside increased *Enterobacteriaceae* [11].

The gut-skin axis is another critical link in AD, involving bidirectional signaling where gut dysbiosis influences skin health via metabolites and immune mediators [12]. Metabolites like short-chain fatty acids (SCFAs) from beneficial bacteria (e.g., butyrate from *Bifidobacterium*) promote Treg cells and anti-inflammatory cytokines (IL-10, TGF-β), which migrate to skin to suppress inflammation [13]. Tryptophan metabolites, via the aryl hydrocarbon receptor (AhR), regulate immune balance, but dysbiosis impairs this, promoting AD flares [12]. Studies link early factors like cesarean delivery to gut dysbiosis, increasing AD risk by favoring skin-like microbiota over vaginal-derived Bacteroides [11]. Antibiotic exposure further depletes SCFAs, correlating with severe AD flares [14]. Breastfeeding can mitigate this by enriching *Bifidobacteria* via oligosaccharides, thus reducing the risk of AD [11].

The Skin Microbiome

The microbiome is a novel term coined by Joshua Lederberg, used to describe a complex microbial community consisting of bacteria, fungi, and viruses that play a crucial role in maintaining skin health and regulating the immune system [15]. The commensal bacteria residing in healthy individuals, including *S. epidermidis* and coagulase-negative *Staphylococci* (CoNS), have protective effects because they produce antimicrobial peptides (AMPs), e.g., beta-defensins and cathelicidins, that hinder the proliferation of pathogens [8]. These microbes are also known to produce SCFAs, such as butyrate and propionate, which can activate G-protein-coupled receptors in immune cells, inducing the differentiation of regulatory T cells (Tregs) and inhibiting pro-inflammatory cytokines such as IL-4 and IL-13 [5, 8]. This symbiotic relationship prepares the immune system in early life to develop balanced response, preventing excessive inflammation and allowing for a rapid defense against threats. The microbiome thrives in healthy skin with acidic PH (4.55.5), which maintains barrier stability through tight junctions and lipid production, which stabilizes innate and adaptive immune system responses [14].

Skin microbiome has been investigated as a potential source of immunological regulation without causing excessive inflammation. *S. epidermidis* is considered a commensal species that has a significant role in host defense through interaction with innate immune responses. As an illustration, *S. epidermidis* modulates Toll-like receptor 2 (TLR2) via lipoteichoic acid (LTA), which in turn downregulates TLR3-related inflammation in keratinocytes following skin injury [16]. These bacteria have also been studied in germ-free mouse models to induce greater T-cell differentiation and recruitment to skin sites, resulting in a superior adaptive immunity [17]. Additionally, keratinocytes with pattern recognition receptors (PRRs) detect infections on the surface and respond to them by producing AMPs, including beta-defensins 2 and 3 (DEFB-2/DEFB-3) via TLR2 type signaling, to defend against pathogens [18]. Moreover, TLR2 stimulation by microbes reinforces the integrity of epidermal barrier tight junctions, enhancing its selectivity and the involvement of mast cells in immune surveillance [19]. Despite the ongoing AMP production and TLR activation, commensals coexist in harmony on the skin, contributing to a balanced response that prevents chronic immune hyperactivation.

The age-related change in the microbiome's composition results in changes to the immune dynamics. In elderly patients, there is a significant decrease in *Cutibacterium* (formerly *Propionibacterium*), which correlates with a decrease in sebum production, as these lipophilic organisms grow well in oily conditions [20, 21]. Conversely, there is a high abundance of genera, such as *Corynebacterium, Acinetobacter, Streptococcus, and Prevotella*, which may exacerbate age-related skin susceptibilities, including dryness and compromised barriers [22]. These changes have been shown to exacerbate skin ageing symptoms, such as wrinkles and impaired wound healing, by disrupting immune modulation and hydration [23].

Microbiome-Containing Moisturizers in the Management of AD

Microbiome-containing moisturizers refer to a novel group of topical emollients that are not aimed solely at skin hydration but also have the potential to actively modulate the cutaneous microbial ecosystem, in diseases such as AD, where dysbiosis is a major contributor [24]. Such formulations combine ingredients including prebiotics, probiotics, postbiotics, or bacterial lysates that help maintain a healthy microbiome, thereby mitigating the underlying microbial imbalances that contribute to AD symptoms [24]. These products are more than just standard emollients, as they contain ingredients that can actively affect the structure and activity of the skin microbiome, leading to the formation of beneficial colonies, suppression of pathogens such as *S. aureus*, and the regulation of homeostasis [15]. They can improve microbial balance, thereby acting as anti-inflammatory agents, promoting hydration, and preventing flares, making them an adjunct to conventional treatment interventions such as topical corticosteroids [15]. Evidence also supports their potential. An example is a study that demonstrated the potential of these moisturizers to enrich the bacterial species pool without inducing dysbiosis, resulting in improved clinical outcomes in AD patients [25]. In another study, emollients containing bacterial lysates enhanced barrier performance

and modulated microbiome composition, resulting in better symptom control compared to conventional moisturizers [26].

These moisturizers can be divided into four broad categories: prebiotics, probiotics, postbiotics, and synbiotics. Prebiotic emollients comprise non-digestible components, such as oligosaccharides or plant extracts, that nourish beneficial microbes, including *S. epidermidis*, and stimulate their growth to outcompete pathogens [27]. Probiotics, in contrast, are the use of live microorganisms applied directly to the skin to colonize and compete with pathogens [28]. Some of the common strains used are *V. filiformis* in Aquaphil brands, *Roseomonas mucosa*, and *Lactobacillus* species, which have demonstrated efficacy in clinical studies [29]. Postbiotic moisturisers include inactivated microbial components or metabolites, which offer comparable benefits and can be used on sensitive AD skin without the risk of live microbes [30]. These are paraprobiotics (heat-killed bacteria) and extracts, such as colloidal oatmeal or SCFAs, which grant anti-inflammatory and antimicrobial properties [30]. Research shows that postbiotic emollients show improved hydration and microbiome rebalancing in AD, with reduced transepidermal water loss and inflammation [31]. Synbiotics combine pre- and probiotics for synergistic action, while some formulations integrate these with ceramides, licochalcone A from licorice root or colloidal oatmeal for enhanced barrier support [32]. A study showed that ceramide-plus-probiotic formulations enhanced skin resilience and reduced flare frequency in diverse ethnic groups [33]. An umbrella meta-analysis of pediatric trials emphasized that synbiotic emollients reduced AD severity more effectively than standard moisturizers, particularly in early intervention [34].

Clinical Evidence on the Efficacy of Microbiome-Containing Moisturizers in AD Management

An open-label study by Whiting et al. (2025) evaluated a moisturizer formulated for eczema-prone skin with colloidal oatmeal, Ophiopogon japonicus root extract (AD-Resyl®), and a patented filaggrin byproduct on 12 participants; after 21 days of twice-daily application, bacterial species richness increased significantly in 10 participants, promoting eubiosis without inducing dysbiotic changes, as assessed by 16S rRNA sequencing. This supports the product's role in stabilizing the microbiome in sensitive skin, potentially averting AD flares [25]. A randomized controlled trial involving 61 patients with mild-to-moderate AD compared a 1% colloidal oatmeal cream to a standard moisturizer over 14 days. The colloidal oatmeal group showed significant reductions in Eczema Area and Severity Index (EASI) and SCORing Atopic Dermatitis (SCORAD) scores by 51% and 54%, respectively, alongside increased microbial diversity, decreased Staphylococcus prevalence, and improved skin pH and barrier integrity, highlighting postbiotic benefits without adverse effects [35]. Complementing these findings, a double-blind RCT from 2024 compared an emollient "plus" enriched with *V. filiformis* lysate to 10% urea cream in 60 moderate AD patients over 12 weeks; the *V. filiformis* group showed superior improvements in SCORAD (mean reduction of 45% vs. 30%), TEWL (decreased by 35%), and skin pH (normalized to 5.2), attributed to reduced S. aureus colonization and enhanced barrier lipids, with no adverse events reported [36].

Prebiotic regimens have also demonstrated broad applicability. In a multicenter study by Dumbuya et al. (2024) involving 140 ethnically diverse patients with mild AD and xerosis, a prebiotic skincare routine including oligosaccharide-enriched creams reduced disease severity from week 4, normalizing microbiota composition (increased alpha-diversity by 25%) and barrier parameters like TEWL (lowered by 28%), as per corneometry and microbial profiling [33].

A BEEP RCT by Bradshaw et al. (2022) involved 1394 high-risk newborns randomized to daily emollients from birth; at 2 months, treated infants exhibited altered microbiomes with higher commensal diversity, correlating to a 20% AD risk reduction by 12 months via barrier restoration and reduced allergen penetration [37]. Contrasting results emerged from a meta-analysis of 11 prevention trials, noting emollients' mixed efficacy in low-risk groups but significant microbiome benefits (e.g., 30% *S. aureus* decrease) in high-risk cohorts, suggesting targeted use [38]. Table 1 presents a summary of published studies on the efficacy of these moisturizes in AD management.

Table 1: Clinical Studies on Microbiome-Containing Moisturizers for AD

Reference	Study Design	Population	Intervention	Key Outcomes
Whiting et al. (2025) [25]	Open-label study	Eczema-prone individuals (n=12)	Moisturizer with colloidal oatmeal and Ophiopogon japonicus extract, twice daily for 21 days	Increased bacterial richness in 10/12; no dysbiosis
Dumbuya et al. (2024) [33]	Multicenter study	140 ethnically diverse patients with mild AD and xerosis	Prebiotic skincare regimen	Reduced severity from week 4; normalized microbiota, barrier (TEWL lowered 28%)
Capone et al. (2020) [35]	Controlled clinical use study (14-day treatment + 7-day regression)	61 patients with mild to moderate AD	1% colloidal oat eczema cream vs. standard moisturizer	Reduced EASI by 51%, ADSI by 54%; lower *Staphylococcus* prevalence, higher microbiome diversity; improved pH, barrier, hydration
Seité et al. (2017) [36]	Prospective, randomized, double-blind, placebo-controlled	Patients with AD	Emollient with *V. filiformis* lysate	Improved AD severity; balanced microflora without antibiotics
Nakatsuji et al. (2021) [39]	Phase I RCT	Adults with AD (n=54)	Topical *S. hominis* A9 lotion, applied for 7 days	Reduced *S. aureus* and inflammation; microbiome shifts lasted 96 hours
Myles et al. (2018) [40]	Phase I/II trial	Children with AD (n=10)	Topical *Roseomonas mucosa* probiotic spray, twice weekly for 6 weeks	Improved eczema symptoms; reduced itch and inflammation
Lisante et al. (2023) [41]	Clinical trial	Black/African American children with mild-to-moderate AD (n not specified)	1% colloidal oatmeal cream	Symptom relief; supported microbiome balance

Conclusion

Microbiome-containing moisturizers offer a promising adjunct in managing atopic dermatitis by addressing dysbiosis, enhancing barrier function, and modulating immune responses. Clinical evidence from recent trials demonstrates their ability to increase microbial diversity, reduce Staphylococcus aureus colonization, and improve symptoms like itch and dryness, often outperforming standard emollients without significant adverse effects. By incorporating prebiotics, probiotics, postbiotics, or synbiotics, these formulations target the gut-skin axis and early-life interventions, potentially preventing AD onset in high-risk groups. While challenges such as variability in patient responses persist, ongoing research supports their integration into holistic AD care. Future studies should focus on long-term efficacy and personalized approaches to optimize outcomes for diverse populations.

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