


Skin Barrier Repair and Nursing Care in Patients with Atopic Dermatitis: A Narrative Review

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Abstract: Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disorder characterized by impaired skin barrier function and immune dysregulation. Despite advancements in understanding its pathogenesis, clinical management remains challenging due to high recurrence rates, complex symptom control, and diminished quality of life, particularly highlighting the lack of standardized protocols for skin barrier repair. Compromised skin barrier integrity in AD leads to increased transepidermal water loss (TEWL) and heightened susceptibility to irritants and allergens, exacerbating inflammation. Thus, restoring skin barrier function is pivotal in AD management. Current therapeutic strategies predominantly prioritize anti-inflammatory treatments while undervaluing barrier repair. Traditional care models relying on qualitative symptom assessments inadequately guide personalized interventions, necessitating an integrated approach combining barrier restoration and immunomodulation. Although diverse barrier repair methods and moisturizers are available, systematic evaluation and evidence-based selection criteria remain limited. This review aims to comprehensively summarize the structure and function of the skin barrier in AD, the role of skin barrier dysfunction in AD pathogenesis, current barrier repair strategies, and evidence-based emollient selection criteria. By analyzing existing research, we provide clinical recommendations for skin barrier restoration and long-term management in AD patients, while proposing future research directions. Emphasis should be placed on developing multi-omics-driven personalized barrier interventions and constructing a “barrier-immune-environment” interaction model to advance precision medicine in AD care.

Keywords: transepidermal water loss, moisturizers, immune dysregulation, skin management, therapeutic strategies, emollients

Introduction

Atopic dermatitis (AD), a chronic immune-mediated dermatosis characterized by xerosis, pruritus, and recurrent inflammation, clinically manifests with polymorphic lesions including erythema, exudation, and lichenification.^{1–4} Beyond its dermatological manifestations, AD imposes significant psychosocial burdens, contributing to sleep disturbances, anxiety, depression, and frequent comorbidities such as allergic rhinitis and asthma, thereby escalating healthcare costs and socioeconomic challenges globally.^{5–7}

Epidemiological data reveal marked age- and region-dependent variations in AD prevalence, with pediatric rates reaching 15–30% and adult incidence approximating 2.1%–8.1%.⁸ Notably, rapid increases in AD prevalence have been documented across 108 countries, including China and Russia, with the highest disease burdens observed in Central Asia, high-income Asia-Pacific regions, and Western Europe, implicating gene-environment interactions in disease etiology.⁹ The pathogenesis of AD involves multifaceted interplay among skin barrier dysfunction, immune-inflammatory dysregulation, and microbial dysbiosis.¹⁰ The cutaneous barrier, a dynamic system sustained by physical, chemical, immunological, and microbial components, serves as the frontline defense against environmental insults. Dysregulation of any barrier component amplifies inflammatory responses, increases cutaneous permeability, and facilitates pathogen invasion, perpetuating disease activity and complications.¹¹ Comprehensive impairment of barrier function thus constitutes a pivotal node in the AD pathogenic cascade.

Despite substantial advances in elucidating the fundamental pathomechanisms of AD, systematic approaches to achieve durable barrier restoration and long-term disease management remain clinically elusive. Current therapeutic paradigms



prioritize anti-inflammatory interventions and pruritus control; however, evidence supporting sustained barrier integrity maintenance remains limited. Innovations in targeted repair strategies—such as bioengineered keratinocytes and selective microbial colonization—hold promise for transcending the limitations of conventional anti-inflammatory therapies. While further investigation is required to clarify specific risk determinants, maintaining epidermal barrier integrity is universally recognized as critical for preventing AD relapse and mitigating symptom severity. This can be achieved through the use of moisturizers, avoidance of excessive cleansing, and implementation of other skin care measures. Emerging evidence underscores the prophylactic potential of early interventions, such as routine emollient application during infancy [11]. Nevertheless, the evidence base for AD-specific barrier repair protocols remains fragmented, necessitating further research to refine clinical guidelines. Particular emphasis should be placed on developing age-stratified, severity-adjusted, and comorbidity-tailored therapeutic regimens. Elucidating the dynamic regulatory networks governing the “immune-barrier-microbiome axis” may provide critical insights into the chronicity and recurrence mechanisms of AD.

This narrative review synthesizes contemporary evidence on AD-related skin barrier repair and care, with the following objectives: (1) delineate the structural and functional architecture of the cutaneous barrier; (2) summarize mechanistic insights into barrier dysfunction in AD pathogenesis; (3) evaluate therapeutic strategies for barrier restoration and their clinical implementation; (4) appraise the composition, selection criteria, and practical efficacy of emollients in AD management; and (5) identify knowledge gaps and propose future research priorities. By integrating mechanistic research with translational perspectives, this review aims to inform clinical decision-making for dermatologists, nursing professionals, researchers, and healthcare policymakers.

Structure and Function of the Skin Barrier

The skin barrier encompasses a multi-layered defense system conventionally categorized into four interdependent components: physical, chemical, immune, and microbial barriers. Skin Barrier Structure (Figure 1).

Physical Barrier

Stratum Corneum

The stratum corneum, as the outermost defensive structure of the epidermis, primarily consists of corneocytes and intercellular lipids. These components collectively form a unique “brick-and-mortar” composite system. Within this

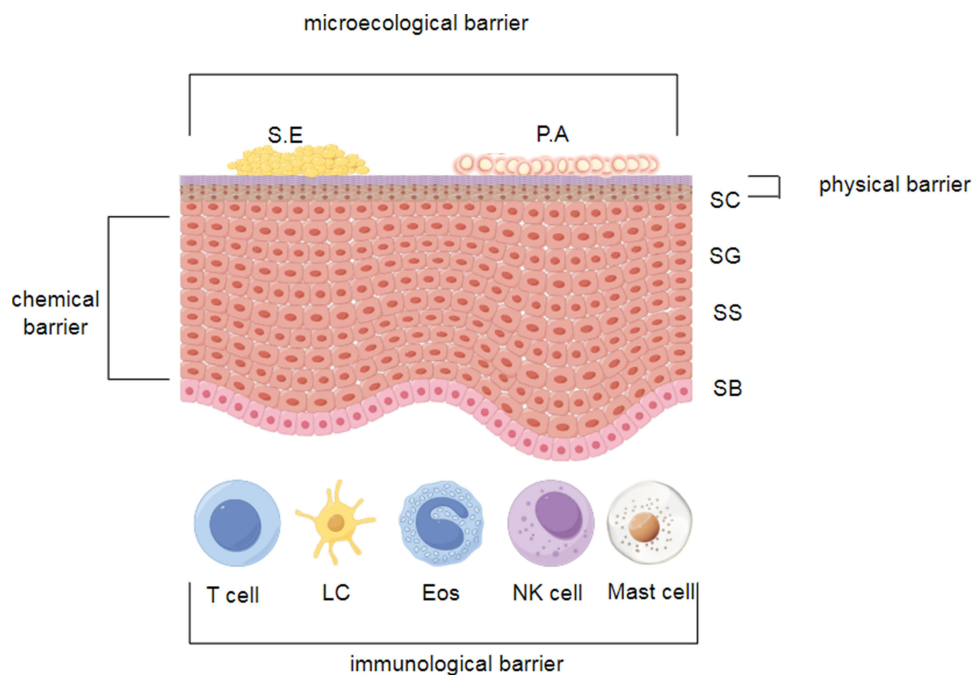


Figure 1 Skin barrier structure.

three-dimensional architecture, corneocytes exhibit tightly arranged lamellar distributions, constructing a scaffold analogous to architectural bricks. Meanwhile, intercellular lipids exist as a continuous phase in the cellular interstices, functioning as mortar-like cementing agents. The synergistic interaction between these two components is critical for maintaining the stratum corneum's physiological functions.

Intercellular lipids are principally composed of three classes of substances: ceramides, cholesterol, and free fatty acids.¹² Studies have demonstrated^{13,14} that ceramides, as core lipid components, exert their functions through two primary mechanisms: (1) establishing a hydrophobic barrier to reduce excessive water evaporation, maintaining the dermal-epidermal moisture gradient essential for normal skin metabolism; and (2) constructing a selective permeability barrier via the physicochemical properties of lipid bilayers, effectively blocking the penetration of foreign particles (>500 Da), microorganisms, and chemical irritants. Covalent binding between ceramides and corneocyte cornified envelope proteins enhances intercorneocyte adhesion. Furthermore, ceramides interact with cholesterol at specific molar ratios to form liquid crystal structures that collaboratively regulate barrier homeostasis.

Notably, disproportional ratios of these three lipids (ceramide levels are reduced by 60% in AD patients) can induce abnormal lamellar body secretion, subsequently triggering structural disorganization of the stratum corneum.¹⁵

Sebum Film

The sebum film, a critical biological barrier system on the skin surface, is formed through the emulsification of lipids secreted by sebaceous glands (triglycerides, wax esters, and squalene), electrolytes from sweat glands, and terminal differentiation products of keratinocytes (eg, ceramides).¹⁶ This composite structure exerts multiple physiological regulatory functions:^{17–20}

(1) Hydration regulation mechanism: By forming a hydrophobic lipid film layer, it suppresses transepidermal water loss (TEWL). Simultaneously, polar lipids such as ceramides capture water molecules from the external environment through hygroscopic properties, replenishing epidermal moisture and maintaining cutaneous homeostasis and stratum corneum hydration balance. A study demonstrated²¹ that applying a multilamellar emulsion cream containing lipids structurally analogous to those in the stratum corneum for 14 days significantly increased the density and length of lipid layers within the stratum corneum, elevated total lipid, ceramide, and free fatty acid levels, thereby improving skin barrier integrity and enhancing water retention capacity. (2) Microbial homeostasis modulation: The sebum film's weakly acidic environment (pH 4.5–6.0) and lipid components like squalene exhibit broad-spectrum antimicrobial properties, inhibiting colonization by pathogenic microorganisms such as *Staphylococcus aureus*. This synergizes with epidermal antimicrobial peptides (AMPs) to form a cutaneous immune defense system. (3) Oxidative stress antagonism: Vitamin E (α -tocopherol), enriched in sebum, acts as a lipid-soluble antioxidant by scavenging reactive oxygen species (ROS), thereby mitigating ultraviolet-induced lipid peroxidation and delaying photoaging processes. (4) Circadian rhythm regulation: Recent studies have revealed dynamic variations in sebum film functionality. Both the quantity of surface lipids and physiological parameters (eg, sebum secretion, TEWL) exhibit significant circadian rhythmic fluctuations. Notably, lipid species critical for barrier function—such as fatty acids and ceramides with specific chain lengths—display rhythmic changes strongly correlated with skin hydration status. This suggests that circadian disruption may impair skin barrier integrity by interfering with lipid metabolism.

Through its multifunctional mechanisms—encompassing physical, chemical, immune, and microecological barriers—this biological film plays a central role in maintaining epidermal permeability barrier function. Dysregulation of its composition is closely associated with the pathogenesis of various dermatological disorders.

Immunological Barrier

The cutaneous immunological barrier, a critical component of the host defense system, comprises a coordinated network of T lymphocytes, mast cells, Langerhans cells, natural killer (NK) cells, and immunologically active molecules. This system achieves multilayered pathogen defense through the integrated regulation of innate and adaptive immunity.

In innate immunity, Langerhans cells, as professional antigen-presenting cells in the epidermis, capture pathogen-associated molecular patterns via pattern recognition receptors (PRRs). These cells then present antigenic information to draining lymph nodes through major histocompatibility complex (MHC) molecules, activating naïve T cells to initiate

adaptive immune responses. Mast cells, upon recognizing pathogens via high-affinity IgE receptors (FcεRI), trigger degranulation to release histamine, prostaglandins, and other inflammatory mediators. This process induces local vasodilation and immune cell chemotaxis, establishing an acute inflammatory microenvironment to accelerate pathogen clearance.^{22–24} In adaptive immunity, CD4⁺ T lymphocytes are activated through T-cell receptor (TCR) recognition of antigenic peptide-MHC II complexes, subsequently differentiating into Th1, Th2, or Th17 effector subsets. Th1 cells secrete IFN-γ to activate macrophage bactericidal functions, Th2 cells produce IL-4/IL-13 to promote B-cell antibody class switching, and Th17 cells enhance epithelial barrier function by inducing antimicrobial peptide secretion from keratinocytes via IL-17A. Recent studies reveal that skin-resident memory T cells establish long-term immunological memory, enabling rapid responses upon secondary antigen exposure.^{25–28}

The immune molecular network achieves precise defense through three-phase regulation:^{29–34} (1) Antimicrobial phase: Antimicrobial peptides (AMPs) such as β-defensins and cathelicidin exert broad-spectrum antibacterial effects by disrupting pathogen membrane potentials and inhibiting DNA replication. Their expression is regulated by the vitamin D3/VDR signaling pathway. (2) Chemotactic phase: Adhesion molecules (eg, E-selectin, ICAM-1) mediate neutrophil rolling-adhesion-migration processes, while chemokine gradients (CXCL8/CCL20) direct immune cell migration to infected sites. (3) Immunomodulatory phase: Proinflammatory cytokines (IL-1β/TNF-α) amplify immune responses via NF-κB signaling, whereas anti-inflammatory cytokines (IL-10/TGF-β) maintain response homeostasis through STAT3/Smad pathways, preventing excessive inflammatory damage.

Notably, skin commensal microbiota enhance AMP expression in keratinocytes via TLR2-mediated PI3K-Akt signaling and induce regulatory T cells to secrete IL-10, thereby suppressing hyperactive immune responses. This symbiotic microbe-host immune crosstalk mechanism provides novel therapeutic targets for immune-related dermatoses such as AD.

Chemical Barrier

Normal skin exhibits a weakly acidic pH of approximately 4.1–5.8. This acidic milieu plays a critical role in maintaining cutaneous microbiome equilibrium by inhibiting pathogenic microbial proliferation while promoting the survival and colonization of beneficial skin commensals. Consequently, preserving this weakly acidic environment aids in preventing cutaneous infections and facilitating barrier repair and homeostasis.^{35,36}

As the largest organ in direct contact with the external environment, the skin is chronically exposed to oxidative stress. Oxidative stress arises from the generation of reactive free radicals, which readily induce structural and functional cellular damage, accelerating skin aging. The skin harbors multiple endogenous antioxidant systems capable of neutralizing free radicals and mitigating oxidative injury. Core antioxidant components include vitamin C, vitamin E, and glutathione. Vitamin C, a water-soluble antioxidant, is abundantly present in the skin. It effectively scavenges free radicals to prevent oxidative damage while stimulating collagen synthesis, thereby enhancing skin elasticity and radiance.³⁷ Vitamin E, a lipid-soluble antioxidant, neutralizes free radicals and protects cellular membrane lipids from oxidative degradation, preserving membrane integrity and functionality. Glutathione, one of the primary endogenous antioxidants in the skin, participates in diverse antioxidant reactions. It directly eliminates free radicals and modulates the activity of other antioxidant molecules, collectively safeguarding cutaneous cells from oxidative insults.³⁸

Microecological Barrier

The human skin surface hosts at least 100 microbial species. *Staphylococcus epidermidis* (S.E) predominantly colonizes the epidermis, while *propionibacterium acnes* (P.A) typically resides within pilosebaceous units. These microorganisms collectively establish the skin's microecological barrier through mutualistic and interdependent survival strategies. This barrier—composed of residential microorganisms and skin secretions adhering to the cutaneous surface—serves as the outermost primary defense layer of the skin barrier system.³⁹

Multiple studies^{40–43} confirm that the skin microbiota directly participates in and regulates the formation, maintenance, and repair of the skin barrier. Notably, microbial communities not only provide protection against pathogens but also modulate host immune responses and enhance epithelial integrity.

Impact of Skin Barrier Dysfunction on AD Pathogenesis

The human skin barrier system constitutes an integrated defense network comprising physical, chemical, immune, and microbial barriers. Their synergistic interactions confer dual physiological significance:^{17,44} (1) Establishing a defensive interface against exogenous pathogenic factors; (2) Maintaining internal homeostasis through precise regulation of transepidermal water loss and dynamic equilibrium of bioactive substances. The multi-layered immune barrier constructed by keratinocytes and immune cells exhibits a dual defense mechanism: it effectively resists pathogenic microbial invasion while interrupting the vicious “itch-scratch-itch” cycle. Notably, epidermal-resident antimicrobial peptides (AMPs)—core effector molecules of the chemical-immune barrier—demonstrate broad-spectrum antimicrobial activity despite significant heterogeneity in their encoding gene structures, post-translational modifications, and immune regulatory pathways. Commensal microbiota in the skin microecosystem participate in maintaining local immune homeostasis through intricate biological mechanisms. Specifically, *S.E* is the predominant skin commensal, secretes lipoteichoic acid that modulates inflammatory responses and plays pivotal regulatory roles in cutaneous wound repair. Concurrently, short-chain fatty acids produced by *P.A* in hair follicles exhibit potent antimicrobial activity against pathogens. AMPs, as key molecular components of this defense system, exert immunomodulatory functions through two distinct modes (1) Direct pathogen suppression: Rapid inhibition of microbial proliferation; (2) Immune cascade orchestration: Precision activation of cytokine cascades to recruit neutrophils, mast cells, and monocytes to lesion sites, thereby dynamically regulating innate immune responses.

Skin barrier dysfunction has been conclusively identified as a central driver in AD pathophysiology, with the degree of barrier integrity disruption demonstrating a significant positive correlation with clinical disease staging.⁴⁵ Molecular mechanistic studies reveal that filaggrin (FLG), a core structural protein encoded by the epidermal differentiation complex (EDC), maintains barrier homeostasis through dual pathways: (1) Regulating the formation of keratin intermediate filament cross-linking networks to ensure orderly stratum corneum cell differentiation; (2) Generating natural moisturizing factors (NMFs, eg, histidine derivatives) via proteolytic cascade reactions, thereby maintaining epidermal osmotic equilibrium. Aberrant FLG gene expression disrupts NMF synthesis, leading to characteristic pathological alterations such as stratum corneum desquamation and increased transepidermal water loss (TEWL). Extensive genetic studies and clinical observations^{46–50} have demonstrated that even minor reductions in epidermal FLG content significantly elevate AD susceptibility. Loss-of-function mutations in the FLG gene represent the strongest genetic risk factor for AD, where haploinsufficiency markedly enhances disease predisposition, demonstrating a clear dose-dependent relationship between allelic burden and disease risk severity. Histopathological investigations further confirm⁵¹ that AD patients exhibit diminished FLG immunofluorescence intensity in both lesional and non-lesional skin compared to healthy controls, with expression levels inversely correlating with SCORAD index scores. Beyond established physical barrier defects, dysbiosis of commensal microbiota plays a critical role in disease progression. Natural moisturizing factors (NMFs), while primarily maintaining epidermal hydration, additionally regulate microbial homeostasis by modulating the cutaneous acidic microenvironment. The pronounced NMF deficiency observed in AD skin leads to xerosis and elevated surface pH, disrupting local microecological balance and inducing characteristic microbial dysbiosis.⁵² Notably, a bidirectional regulatory relationship exists between microbial dysbiosis and inflammatory responses. Kim et al demonstrated that *Staphylococcus aureus* (*S. aureus*, SA) colonization density positively correlates with the severity of skin barrier damage.⁵³ SA-derived metabolites (eg, δ -toxin, serine proteases, and superantigens) directly activate Toll-like receptor 2 (TLR2) signaling in keratinocytes, inducing the release of Th2-associated cytokines such as IL-4 and IL-13. This microbe-host interaction establishes a self-perpetuating vicious cycle: compromised barrier integrity promotes SA overcolonization, while SA-derived cytotoxic products further impair stratum corneum cohesion, exacerbating epidermal permeability barrier dysfunction. 16S rRNA sequencing analyses reveal^{54–56} a 5–10-fold increase in SA relative abundance within AD lesions compared to healthy skin, concomitant with significant depletion of antimicrobial commensals (eg, *Staphylococcus epidermidis*). This structural microbial shift drives chronic AD inflammation through multiple mechanisms, including sustained immune activation and impaired barrier repair capacity.

Previous studies have established that a subset of AD cases correlate with epidermal barrier defects caused by filaggrin (FLG) gene mutations. However, two critical observations warrant attention:^{57,58} (1) most AD patients exhibit no detectable FLG mutations, and (2) mutation carriers demonstrate spontaneous clinical symptom resolution in

adulthood. This paradox suggests that AD pathogenesis involves regulatory mechanisms extending beyond genetic mutations. Emerging evidence highlights the central role of immune dysregulation in AD development. Histopathological analyses reveal persistent immune activation in lesional skin:⁵⁹ increased perivascular Th2 lymphocyte infiltration is observed in non-lesional skin of patients with elevated IgE levels, even prior to overt lesion formation. Furthermore, specific AD subtypes exhibit immune-mediated suppression of keratinocyte differentiation, where IL-4/IL-13 impair epidermal barrier function via dual mechanisms—downregulating FLG expression and upregulating kallikrein activity—while IL-22 exacerbates barrier dysfunction by inducing keratinocyte hyperproliferation and inhibiting FLG synthesis, collectively driving epidermal barrier failure. Notably, interventional studies in moderate-to-severe AD patients demonstrate that immunomodulatory therapies such as cyclosporine A and narrow-band UVB significantly improve disease activity, providing clinical validation of immune mechanisms' central role.^{60,61} Immunological micro-environment analyses of chronic AD lesions reveal T-lymphocyte and myeloid-derived CD11c+ dendritic cell infiltration exceeding 10-fold compared to healthy skin, predominantly comprising BDCA1⁺/CD11c+ inflammatory dendritic cell subsets.^{62,63} mRNA quantification and immunohistochemical assays^{64–66} identify three hallmark features in lesional regions: (1) Marked elevation of activated T-cell-associated factors; (2) Aberrant expression of dendritic cell-derived chemokines; (3) Epidermal reactive hyperplasia or re-emergent aberrant differentiation. These findings collectively elucidate the complex immune-epidermal interactome driving AD chronicity. Beyond the aforementioned three barrier abnormalities, AD patients exhibit significant chemical barrier dysfunction. Studies demonstrate markedly reduced β -defensin levels in AD skin compared to healthy controls,⁶⁷ suggesting diminished antimicrobial peptide (AMP) expression may underlie SA susceptibility. Notably, Th2-derived IL-4 in AD skin actively suppresses AMP production. Dermcidin, a broad-spectrum antibacterial and antifungal AMP secreted by eccrine sweat glands, provides critical defense against pathogens.⁶⁸ AD patients show pronounced dermcidin deficiency in sweat, with deficiency severity correlating with cutaneous infection intensity.⁶⁹ Thus, reduced AMP levels in AD skin may arise from both primary deficiencies and secondary suppression via Th2-mediated IL-4 inhibition.

In summary, the four cutaneous barriers—physical, microbial, immune, and neural—do not function in isolation but interact synergistically to drive AD pathogenesis. AD progression exhibits multi-layered regulatory coordination within the skin barrier system. Research delineates that these barriers collectively contribute to disease mechanisms through a multidimensional interaction network. Following epidermal permeability barrier disruption, exogenous antigens and pathogens activate immune responses via dual pathways: (1) antigen-presenting cell (APC)-mediated adaptive immunity and (2) direct microbial clearance by antimicrobial peptides (AMPs). Notably, barrier dysfunction induces microbial dysbiosis, promoting pathogenic dominance (eg, *Staphylococcus aureus*, SA) over commensals and exacerbating ecological imbalance. Immune barrier dysregulation, particularly Th2-skewed immunity, occupies a central role in AD pathology. Th2-derived IL-4/IL-13 drive IgE class-switching in B cells via the IL-4R α signaling pathway, mechanistically explaining elevated serum total IgE levels in AD patients. This immunophenotypic shift perpetuates a self-amplifying cycle: IgE-mediated hypersensitivity enhances barrier permeability, while Th2 cytokines (IL-4/IL-13/IL-31) further suppress AMP production and impair keratinocyte differentiation, thereby consolidating the barrier-immune-microbiome axis as a pivotal therapeutic target in AD. IL-4/IL-13 exhibit dual pathogenic effects in AD: they suppress FLG gene expression and ceramide synthesis in keratinocytes while inhibiting antimicrobial peptide production and dendritic cell function, establishing a microenvironment favorable for *Staphylococcus aureus* (SA) colonization. Recent studies demonstrate that synergistic activation of SA surface TLR-2 and IL-4 receptors downregulates anti-inflammatory IL-10 expression, amplifying inflammatory cascades in AD skin.^{70,71} AD progression exhibits phase-specific immune dynamics: non-lesional phases are characterized by Th2/Th22-dominant infiltration initiating barrier dysfunction; acute phases involve antigen-driven Langerhans cell activation, promoting Th2/Th22 expansion with mild Th1/Th17 upregulation; chronic lesions display progressive Th2/Th22/Th1 activation.⁷² This immune evolution forms a vicious cycle with delayed epidermal repair—IL-4 impedes barrier restoration by suppressing keratinocyte proliferation-associated signaling pathways, while persistent barrier defects exacerbate Th2 inflammation. These mechanisms elucidate the molecular basis of AD chronicity and provide a rationale for therapies targeting the IL-4/IL-13-immune-barrier axis.

Skin Barrier Repair Strategies in AD

In the treatment of AD, repairing the skin barrier function of patients is an extremely important measure, and there are currently multiple methods available to restore the skin barrier function in patients.

Basic Skincare and Moisturizers as the Cornerstone of Barrier Repair

Topical moisturizers represent a fundamental intervention in AD management, typically comprising emollients, occlusives, and humectants that delay transepidermal water loss (TEWL), promote lesion repair, and restore skin barrier integrity, making them widely utilized in clinical practice. Moisturizers enhance epidermal hydration, replenish intercellular lipids, and normalize stratum corneum structure and function. In AD care, barrier repair through consistent skincare and routine emollient application is universally endorsed. Multiple guidelines and reviews emphasize that moisturizers serve as both foundational therapy and first-line treatment for AD, requiring continuous and adequate use.^{73–75} Gentle bathing aids in skin cleansing, hydration, and reduction of *Staphylococcus aureus* colonization. Optimal efficacy is achieved by applying moisturizers within 3–5 minutes post-bathing, as this timing maximizes occlusion-induced TEWL reduction and exogenous lipid supplementation to directly repair the compromised physical barrier.⁷⁶ Emollients not only alleviate xerosis but also enhance topical drug efficacy, minimize adverse effects, and prolong disease remission. Furthermore, specialized formulations containing prebiotics, ceramides, hyaluronic acid, or non-proteinogenic ornithine-containing plant extracts demonstrate promising barrier-restorative properties, improving hydration, elevating natural moisturizing factors (NMFs), and mitigating symptoms. A 10-week prebiotic-enriched skincare regimen significantly reduced disease severity, increased skin hydration, and elevated NMF levels in mild AD patients.⁷⁷ Wollenberg et al reported that emollients with non-proteinogenic ornithine-containing plant extracts exhibit anti-inflammatory and barrier-repairing capacities.⁷⁸ Common moisturizers include petrolatum, lanolin, and urea, and clinicians should tailor product selection to individual skin types and disease severity.

Pharmacological Barrier Repair Therapies

Topical corticosteroids, as conventional therapeutic agents, exert anti-inflammatory, anti-allergic, and antipruritic effects, thereby indirectly promoting skin barrier repair. However, their formulation, dosage, and duration of application must be strictly regulated under medical supervision to minimize adverse events caused by prolonged or extensive use.⁷⁹ Topical calcineurin inhibitors, a novel class of non-steroidal anti-inflammatory drugs, enhance barrier repair by suppressing T-cell activation and inflammatory mediator release while increasing stratum corneum ceramide levels. Tacrolimus is a commonly prescribed agent in this category.⁸⁰ Oral antihistamines effectively alleviate pruritus, reducing physical trauma from scratching and indirectly supporting barrier restoration. Widely used agents such as loratadine and cetirizine require careful monitoring to manage potential side effects.

Targeted Biologics Improve Barrier Function at the Source

In recent years, targeted biologics directed against type 2 inflammatory pathways, particularly dupilumab, have demonstrated remarkable efficacy in restoring skin barrier function in AD. Their mechanism involves blocking IL-4 and IL-13 signaling, thereby suppressing inflammation at its source and indirectly restoring endogenous synthesis of barrier-associated proteins and lipids.^{81,82}

Multiple studies validate dupilumab's barrier-repairing effects. A 2022 study evaluating dupilumab's impact on skin barrier function in adults and adolescents with moderate-to-severe AD revealed that 16-week treatment significantly reduced transepidermal water loss (TEWL) in lesional skin to levels comparable with healthy controls. Mechanistically, dupilumab normalized skin lipid composition by inhibiting IL-4/IL-13 signaling, marked by reduced levels of non-hydroxy fatty acid- and C18-sphingosine-based ceramides, alongside increased levels of esterified ω -hydroxy fatty acid-containing ceramides (critical for barrier integrity) and elongation of ceramide chain lengths. These favorable changes emerged partially at 2 weeks and fully by 8 weeks.⁸³ Another study in children aged 6–11 years with severe AD showed that 16-week dupilumab treatment not only improved TEWL but also restored filaggrin (FLG) processing and its degradation products (precursors of natural moisturizing factors, NMF), with these improvements strongly correlating.⁸⁴ Cork et al further demonstrated that lesional and non-lesional skin TEWL normalized to healthy levels

after 16 weeks of dupilumab, accompanied by significant ceramide profile improvements.⁸⁵ Notably, dupilumab effectively reduced TEWL and normalized ceramide levels regardless of FLG gene mutation status, highlighting its broad therapeutic applicability. Collectively, these findings underscore that targeted inhibition of core inflammatory pathways can reverse AD-associated barrier defects at both molecular and functional levels, achieving “inside-out” repair.

Emerging Therapies

Microbial Intervention Therapy

Targeting the skin microbiome for AD management has emerged as a novel therapeutic approach in recent years. Microbial strain-based interventions may reduce antibiotic overuse and mitigate the development of antimicrobial resistance. AD patients exhibit dysbiosis of the cutaneous microbiome, characterized by *Staphylococcus aureus* overgrowth, which exacerbates skin barrier impairment. Consequently, microbiome modulation represents an innovative therapeutic strategy. Approaches including probiotics, prebiotics, postbiotics, and fecal microbiota transplantation aim to restore balance in both cutaneous and gut microbial ecosystems.^{86,87} Studies demonstrate that microbiome interventions improve disease status through multifaceted regulation of inflammatory pathways. Various modalities, such as single or combined probiotics, prebiotics, postbiotics, unmethylated CpG motifs, fecal microbiota transplantation, microbial fermentation technologies, and bacteriophages, have been investigated in preclinical and clinical settings. These interventions show potential in modulating Th1/Th2 balance with minimal adverse effects, ultimately aiding in controlling AD-associated inflammation. Oral probiotics, for instance, promote intestinal microbial homeostasis and may prevent AD onset. By regulating host immune responses and suppressing pathogenic bacterial growth, these strategies indirectly enhance skin barrier integrity.

Novel Topical Therapies

To enhance therapeutic efficacy and minimize adverse effects of topical treatments, researchers are developing advanced drug delivery systems and exploring novel small-molecule inhibitors with barrier-repairing potential. Technologies such as liposomes, nanoemulsions, hydrogels, and microneedles improve drug penetration and retention in the skin, enabling targeted and controlled release to optimize efficacy while reducing systemic exposure.^{88–91} These systems can encapsulate conventional corticosteroids or emerging targeted agents, facilitating more efficient repair of the compromised skin barrier.

Novel topical small-molecule inhibitors include Janus kinase (JAK) inhibitors, phosphodiesterase-4 (PDE4) inhibitors, and aryl hydrocarbon receptor (AhR) modulators.^{92–94} Topical JAK inhibitors such as ruxolitinib and delgocitinib act locally to suppress JAK-STAT signaling, thereby reducing inflammation, pruritus, and promoting barrier restoration. Topical PDE4 inhibitors like crisaborole and roflumilast exert anti-inflammatory effects by elevating intracellular cyclic adenosine monophosphate (cAMP) levels. These agents provide non-steroidal alternatives for mild-to-moderate AD, supporting long-term management and barrier repair. AhR modulators such as tapinarof represent another emerging non-steroidal option, suppressing inflammation and improving barrier function through AhR pathway modulation.

Physical Treatment Methods

Certain photoelectric therapies, including intense pulsed light (IPL), Q-switched lasers, radiofrequency (RF), and low-level laser therapy (LLLT), may aid in skin barrier restoration by enhancing metabolic activity, exerting anti-inflammatory effects, and stimulating collagen synthesis. However, studies caution that inappropriate parameter selection may exacerbate barrier damage.⁹⁵ Phototherapy, utilizing specific ultraviolet (UV) wavelengths, serves as a physical treatment modality to achieve anti-inflammatory, immunomodulatory, and antipruritic effects. Narrowband UVB (NB-UVB) and UVA1 are widely recognized as clinically effective and safe light sources for managing moderate-to-severe chronic lichenified lesions in adult patients, primarily for symptom control and maintenance therapy. Notably, phototherapy is not recommended during acute flares, and systemic UV therapy should be avoided in children under 12 years of age. Patients with symptom exacerbation upon sun exposure are also advised against this intervention.⁹⁶ Beyond conventional approaches, emerging adjunctive physical and behavioral interventions demonstrate potential utility. A 2022

study investigated magnetotherapy in pediatric and adolescent AD, suggesting that transcranial traveling variable magnetic fields and localized low-frequency magnetotherapy may reduce cutaneous hypersensitivity and pruritus by modulating central nervous system (CNS) and autonomic nervous system functions, thereby demonstrating promising therapeutic potential.⁹⁷

Moisturizer Selection and Care Applications in AD

Moisturizers generally contain humectants, occlusants, and absorbers as main components, can delay transepidermal water loss, promote repair of lesional sites, and improve the skin barrier, and are widely used in clinical practice. Moisturizers may include substances that reduce friction or reflective particles that cover the skin to make it smooth and potentially increase skin elasticity; in addition, moisturizers also exert anti-inflammatory, anti-proliferative, antipruritic, and photoprotective effects. A summary of moisturizer selection and care applications in AD (Table 1).

Ceramide-Containing Moisturizers

Ceramides, a core component of stratum corneum lipids, form the lipid bilayer structure alongside free fatty acids and cholesterol. The orthogonal packing between ceramides and free fatty acids is critical for maintaining barrier homeostasis, with ceramides constituting approximately 50% of stratum corneum lipids, thereby serving as a structural foundation.⁹⁸ Current evidence indicates abnormalities in skin barrier lipids in AD patients, particularly alterations in ceramide content and composition. While the mechanisms underlying these ceramide deficiencies remain incompletely understood, their association with Th1/Th2 cytokine dysregulation underscores their pivotal role in AD pathophysiology.⁹⁹

Two interventional studies in adults with moderate AD demonstrated the efficacy of ceramide-containing moisturizers in barrier restoration. Compared to placebo, ceramide-based formulations significantly reduced transepidermal water loss (TEWL), enhanced skin hydration, and improved patient-reported outcomes, including pruritus relief, xerosis mitigation,

Table 1 Classification of Moisturizers and Their Application in AD

Moisturizers	Key Advantages (Applications in AD)	Summary of Key Studies	Indications and Critical Usage Guidelines
Ceramide-based moisturizers	Restores lipid bilayer structure to enhance skin barrier function; alleviates pruritus and xerosis; demonstrates comparable efficacy to low-potency corticosteroids with fewer adverse effects	Significantly reduces transepidermal water loss (TEWL) and improves skin hydration, with high patient-reported satisfaction rates	Recommended for mild-to-moderate AD in daily care regimens. When used as adjuvant or alternative therapy to low-intensity corticosteroids, it reduces corticosteroid burden and improves barrier integrity
Licochalcone A	Exhibits anti-inflammatory, antimicrobial, and antioxidant properties; inhibits ERK/MAPK and PPAR- γ signaling pathways; demonstrates lower relapse rates compared to corticosteroid monotherapy	Shows comparable efficacy to hydrocortisone with significantly lower recurrence rates	Serves as an anti-inflammatory and antimicrobial moisturizer option. When utilized as an alternative or adjunctive therapy to corticosteroids, it may reduce disease recurrence
Colloidal oatmeal	Regulates epidermal differentiation genes; improves microbial composition and skin pH; enhances barrier function	Demonstrates comparable efficacy to prescription emollients with significant improvements in skin barrier integrity and hydration	Recommended for pediatric populations with mild-to-moderate AD in daily care. Serves as a key component for routine moisturization and barrier restoration
Aloe vera extract	Exhibits anti-inflammatory and immunomodulatory properties; suppresses Th2/Th17 immune responses; reduces serum IgE levels; demonstrates a favorable safety profile	Alleviates AD-like symptoms in murine models with efficacy comparable to corticosteroids. Mechanistically suppresses NF- κ B pathway activation and mitigates inflammatory cascades	Serves as a potential adjuvant therapy with immunomodulatory properties, offering a viable option to reduce dependency on conventional corticosteroid regimens
Traditional Chinese herbal moisturizers (eg, Portulaca oleracea extract, Prinsepia utilis oil, etc.)	Exerts anti-inflammatory and antiallergic effects; ameliorates pruritus and enhances skin hydration; Reduces recurrence risk and improves quality of life (QoL).	Demonstrates superior efficacy to mometasone furoate in maintenance therapy, significantly decreasing relapse rates and enhancing QoL in pediatric AD patients	Serves as an adjuvant therapy during both maintenance and remission phases. Synergistic effects observed when combined with topical corticosteroids (TCS)/calcineurin inhibitors (TCI), enhancing therapeutic outcomes and prolonging relapse-free intervals

and skin smoothness.¹⁰⁰ Multiple trials validate their therapeutic utility. A multicenter study involving 312 patients (109 with AD) revealed that a 4-week ceramide-enriched regimen reduced SCORAD scores by 61.2% and improved quality of life by 67.2%.¹⁰¹ A double-blind, self-controlled trial comparing a physiological lipid-based lotion (MVE+GL) containing ceramides with conventional glycerol-based emollients found that only MVE+GL significantly enhanced barrier integrity (reduced TEWL) and increased stratum corneum levels of ceramide NP and AP.¹⁰² These findings highlight the efficacy of ceramide-containing moisturizers in AD management, particularly in improving skin hydration, positioning ceramide supplementation as a vital adjunctive therapeutic strategy.

Plant-Derived Moisturizers

Moisturizers containing botanical extracts are widely used, with common ingredients including licochalcone A, colloidal oatmeal, and aloe vera extract.

Licochalcone A

The chemical composition of *Glycyrrhiza* spp. encompasses triterpenoid saponins (eg, glycyrrhizic acid), flavonoids (eg, licochalcones), polysaccharides, and coumarin derivatives, with triterpenoids and flavonoids constituting the primary bioactive components. Experimental studies demonstrate that total *Glycyrrhiza* flavonoids exert anti-inflammatory effects via dual mechanisms: (1) suppressing protein expression of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2), and (2) modulating extracellular signal-regulated kinase (ERK) phosphorylation, thereby interfering with ERK/MAPK signaling and peroxisome proliferator-activated receptor gamma (PPAR- γ) activity.^{103–105}

Among licochalcones, isomers A, B, and E exhibit broad pharmacological activities due to stereochemical configurations, including potent anti-inflammatory effects, oxygen free radical scavenging, and regulation of T-lymphocyte subsets. A 2025 study reported that licochalcone A demonstrated a minimum inhibitory concentration (MIC) of 7.5 $\mu\text{g}/\text{mL}$ against both *Listeria monocytogenes* and *Staphylococcus aureus*, significantly inhibiting mono- and dual-species biofilm formation,¹⁰⁶ suggesting its potential anti-infective applications.¹⁰⁶ A 2024 study developed a licochalcone A-loaded hydrogel, which effectively suppressed *S. aureus* activity and accelerated wound healing in a murine skin injury model.¹⁰⁷ In clinical translation, a randomized controlled trial by Udompataikul et al revealed that 0.5% licochalcone A formulation applied for 6 weeks in AD patients achieved comparable SCORAD score improvement to 0.1% hydrocortisone, with no significant difference between groups. Long-term follow-up showed a 42.9% relapse rate within 3 months in the steroid-treated group versus 17.6% in the licochalcone A group, with no severe adverse events reported in either group.¹⁰⁸ These findings provide evidence for botanical chalcones as alternatives to corticosteroids in mild-to-moderate AD management.

Colloidal Oatmeal

Colloidal oatmeal serves as a common component of moisturizers. Accumulated evidence indicates that colloidal oatmeal extract modulates the expression of genes associated with epidermal differentiation, tight junction proteins, and lipid metabolism, while exerting significant pH-buffering capacity to alleviate AD symptoms. In pediatric AD studies, over-the-counter (OTC) emollients containing colloidal oatmeal demonstrate comparable efficacy to prescription formulations.¹⁰⁹ For barrier repair, compared to conventional moisturizers, colloidal oatmeal-containing formulations not only improve cutaneous microbiome composition but also promote pH stabilization, enhance barrier function, and reinforce water retention capacity, thereby optimizing stratum corneum hydration.¹¹⁰

Aloe Vera Extract

Aloe vera exhibits multi-target regulatory benefits in dermatological management. Its bioactive constituents, including polysaccharides, anthraquinones, and mucopolysaccharides, exert anti-inflammatory effects by suppressing NF- κB pathway activation and modulating Th1/Th2/Th17 immune axis balance. Aloe vera extract reduces serum IgE levels in AD murine models while significantly inhibiting Th2-associated cytokines such as IL-4 and IL-13.¹¹¹

Mechanistically, Finberg et al¹¹² demonstrated that aloe vera gel suppresses Th2 cell hyperactivation via down-regulation of STAT6 phosphorylation, aligning with the immune dysregulation observed in AD chronicity. Further studies reveal that fermented aloe vera derivatives reduce *Staphylococcus aureus* colonization density and promote probiotic

proliferation (eg, *Staphylococcus epidermidis*), reprogramming the cutaneous microbiome. Panahi Y et al¹¹³ conducted a clinical trial involving 36 AD patients to compare a compound cream containing aloe vera and extra virgin olive oil with topical betamethasone. Results demonstrated significantly greater improvement in the experimental group, with a 64.5% reduction in AD severity scores versus 13.5% in the betamethasone group. Health-related quality of life, measured by the Dermatology Life Quality Index (DLQI), improved by 60.7% in the experimental group compared to 22.3% in the control group. Additionally, the experimental group exhibited reduced eosinophil counts and serum IgE levels. This study suggests that aloe-containing formulations may surpass mid-potency corticosteroids in improving AD severity and patient-reported outcomes.

Current advancements indicate that microbial fermentation technology enhances aloe vera extracts by generating novel bioactive peptides with superior stratum corneum penetration efficiency compared to conventional preparations, providing a rationale for transdermal delivery system development. These findings resonate with the traditional Chinese medicine theory of “Qingre Liangxue” (clearing heat and cooling blood), underscoring the unique advantages of natural compounds in managing chronic inflammatory dermatoses.

Traditional Chinese Medicine (TCM)-Based Moisturizers

Guided by the holistic therapeutic principle of “simultaneous internal and external regulation” in TCM, AD management strategies extend beyond classical herbal formulations and acupuncture to include systematic scientific reconstruction of external therapies. Clinical evidence highlights the advantages of TCM approaches, including low adverse event rates, high treatment adherence, and sustained long-term efficacy. Recent research has focused on developing TCM-derived moisturizers, which transcend conventional emollient functions by synergistically repairing the skin barrier and modulating immune responses. A 2023 randomized controlled trial investigating chronic hand eczema—a condition sharing pathological features with AD—provided indirect support for the clinical application of *Portulaca oleracea* extract. Involving 70 patients, the study demonstrated that four-week use of *Portulaca oleracea* extract significantly improved pruritus and xerosis while reducing physician-assessed fissuring scores compared to placebo.¹¹⁴ Beyond direct anti-inflammatory and barrier-restorative effects, *Portulaca oleracea* extract exhibits potent antioxidant and anti-photoaging activities, offering additional benefits for long-term AD skin care. Mechanistically, it mitigates UVB-induced oxidative stress and inflammation in skin cells by regulating the miR-138-5p/Sirt1 signaling axis, thereby counteracting photoaging.¹¹⁵ Given that AD skin, due to barrier defects and chronic inflammation, exhibits heightened sensitivity to environmental triggers (including UV radiation) and elevated oxidative stress (a recognized exacerbating factor),¹¹⁶ the antioxidant properties of *Portulaca oleracea* may protect vulnerable AD skin from exogenous insults. Pruritus, a hallmark of AD, has been addressed through TCM topical formulations. Huang JL et al¹¹⁷ demonstrated that the modified anti-allergic decoction (JWGMD) alleviates itching by suppressing the histamine/protease-activated receptor-2 (HIS/PAR-2) pathway and regulating mast cell degranulation. Similarly, a compound TCM ointment reversed substance P upregulation in pruritic skin lesions,¹¹⁸ providing pharmacological rationale for TCM-based antipruritic interventions.

Prophylactic use of emollients containing *Prinsepia utilis* oil during AD remission has proven effective in reducing relapse risk and prolonging disease-free intervals. A 2019 multicenter randomized parallel-controlled trial¹¹⁹ involving 309 infants with moderate AD (aged <2 years) evaluated *Prinsepia utilis* oil-based emollients during maintenance therapy. The treatment group (using desonide with *Prinsepia utilis* oil emollients) achieved a cumulative maintenance rate of 60.5%, significantly higher than the control group (23.5%). Median flare-free survival was 90 days in the treatment group versus 28 days in controls, confirming its relapse-preventive efficacy. A 2025 clinical study further validated the benefits of glycyrrhizate-containing emollients in adult AD, showing a 6.56-point reduction in SCORAD scores and significant pruritus relief after 12-week therapy, with complete remission rates markedly surpassing the non-emollient group.¹²⁰

In Summary, AD is a chronic, relapsing inflammatory dermatosis predominantly affecting patients with mild-to-moderate severity, necessitating topical therapies as the cornerstone of management. Advances in skin barrier research have highlighted the critical role of moisturizers in AD treatment. Based on their composition, moisturizers can be categorized into distinct classes, with certain formulations demonstrating comparable efficacy to low-potency corticosteroids while exhibiting fewer adverse effects. When combined with corticosteroids, moisturizers enhance therapeutic

outcomes through synergistic interactions, reduce corticosteroid-related toxicity, and serve as invaluable adjunctive therapies. These attributes underscore their expanding clinical utility in AD care.

The selection criteria for moisturizers and their nursing applications in AD are comprehensively summarized (Table 1).

Discussion

Skin barrier dysfunction constitutes a central pathogenic mechanism in AD. The interplay between genetic predisposition (eg, FLG mutations) and environmental triggers (eg, microbial colonization, chemical irritant exposure) compromises stratum corneum structural integrity, manifesting as lipid matrix disorganization, reduced filaggrin expression, and tight junction protein abnormalities. These defects drive increased transepidermal water loss (TEWL), antigen penetration, and sensory nerve hyperexposure. Although basic moisturization is widely accepted as first-line therapy, environmental factors may exacerbate barrier repair deficits through epigenetic regulation (eg, DNA methylation) or immune activation pathways (eg, IL-4/IL-13-driven Th2 inflammation), particularly in moderate-to-severe AD patients with elevated specific IgE [1,3]. Nevertheless, current evaluations of barrier repair strategies face methodological limitations: most evidence derives from short-term observational studies and small-sample RCTs with heterogeneous interventions/outcomes; research on phenotype-specific repair protocols remains scarce; and long-term impacts on microbiome homeostasis and recurrence rates are inadequately characterized. These gaps highlight the need for large-scale longitudinal cohort studies integrating multimodal barrier assessment (eg, reflectance confocal microscopy, dynamic TEWL monitoring) and omics platforms (lipidomics, metabolomics) to establish precision-guided repair algorithms.

The causal link between barrier restoration and AD progression underscores the necessity of early intervention, particularly for high-risk genetic phenotypes or environmentally exposed populations. Clinically, a tiered “prevention-repair-maintenance” framework should be implemented: acute-phase management combines ceramide-enriched barrier creams with anti-inflammatory agents, while remission phases prioritize personalized moisturization to sustain microbiome equilibrium. Research priorities include: 1) developing biomimetic delivery systems mimicking native lipid architectures to enhance repair efficiency; 2) elucidating microbe-barrier-immune crosstalk (eg, *S. aureus*-mediated tight junction disruption); 3) establishing AI-driven predictive models for dynamic barrier function monitoring. Additionally, environmental epigenetics (eg, humidity-regulated FLG expression) may unveil novel etiology-targeted therapeutic avenues.

Based on this, skincare for AD patients should shift from merely controlling symptoms to a system-wide management oriented toward barrier function. In treatment, an integrated approach that combines epidermal structure repair with modulation of the immune microenvironment should be formed—for example, in severe AD patients, using biologics to control inflammation while pairing pH-balanced moisturizers to restore the acidic skin surface, and reducing mechanical barrier damage through lifestyle adjustments (such as selecting cotton fabrics and using low-irritant cleansers). In research, a multidimensional skin barrier assessment system should be constructed (including stratum corneum integrity scoring, lipidomics profiles, and itch-related neural signaling biomarkers), and machine learning algorithms should be used to analyze their dynamic associations with disease activity. Establish an international AD registry platform based on electronic health records to integrate genetic testing data, environmental exposure histories, and treatment response patterns, which will drive evidence-based optimization of stratified care guidelines. Through interdisciplinary collaboration to decipher the molecular code of skin barrier repair, the ultimate goal is to transform AD from symptom-focused treatment to etiological prevention.

Conclusion

The core pathological features of AD are a vicious cycle of skin barrier dysfunction and immune imbalance. Existing evidence suggests that abnormalities in epidermal lipid metabolism, differentiation defects of keratinocytes, and microbial dysbiosis may constitute a “three-punishment” that jointly promotes barrier damage and conversion to chronic inflammation. Although studies have begun to reveal key mechanisms such as filaggrin deficiency and imbalanced ceramide ratios, methodological limitations—including reliance on cross-sectional studies, non-uniform assessment

standards, and the reductionist nature of in vitro models—mean that causal evidence for the temporal relationship between barrier repair and disease progression, and for microbe-host interaction pathways, remains lacking.

Future research should overcome bottlenecks through: first, establishing multicenter birth cohorts and longitudinal adult follow-up cohorts, integrating standardized barrier function assessments (such as transepidermal water loss monitoring), skin metagenomic analyses, and epigenetic marker tracking to decipher the spatiotemporal patterns of gene-environment interactions; second, using 3D skin organoid models and gene-editing technologies (such as CRISPR-Cas9-mediated repair of FLG mutations) to simulate barrier injury and restoration at the molecular cascade level under controlled conditions; furthermore, interventional trials are needed to validate the long-term efficacy of targeted lipid supplementation therapies, bacteriophage-directed clearance of pathogenic bacteria, and other innovative strategies, with single-cell spatial transcriptomics to elucidate heterogeneity in treatment responses.

Clinical translation should focus on three directions: first, develop personalized barrier repair strategies based on multi-omics features, such as lipid-based delivery systems targeting ceramide subtypes or microbiome-based tailored anti-infective and probiotic therapies; second, construct a synergistic “barrier repair-immune modulation” therapeutic paradigm, for example combining JAK inhibitors with epidermal lipid precursors to simultaneously block inflammatory signals and rebuild the physical barrier; third, implement early prevention for high-risk populations (such as FLG mutation carriers) through newborn moisturization interventions and intelligent environmental controls to reduce disease risk. Achieving these goals requires deep integration of dermatology, microbial ecology, biomaterials engineering, and digital health technologies, facilitated by interdisciplinary collaboration to translate mechanism research into clinical practice and ultimately drive AD management from symptom control toward barrier homeostasis-based precision medicine.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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