



Review Article

Modulating the Gut–Skin Axis in Dermatological Diseases: Mechanisms and Clinical Applications of Probiotics, Prebiotics, Synbiotics, and Postbiotics

Valentina Broshtilova¹ , Ivan Vasilevski² , Yoanna Velevska³ ,
Irina Yungareva⁴ , Yoanna Petkova¹ , Sonya Marina^{5,*}

¹Department of Dermatology and Venereology, Sofia University St. Kliment Ohridski, Sofia, Bulgaria

²Clinic of General, Abdominal and Vascular Surgery, Medical Institute of the Ministry of Interior, Sofia, Bulgaria

³Department of Dermatovenereology, Prof. Assen Zlatarov University, Burgas, Bulgaria

⁴Department of Skin and Venereal Diseases, Medical Institute of the Ministry of Interior, Sofia, Bulgaria

⁵Department of Research and Educational Activities, Medical Institute of the Ministry of Interior, Sofia, Bulgaria

Abstract

The gut–skin axis represents a bidirectional regulatory network linking intestinal microbiota composition and function to cutaneous inflammation and barrier integrity through interconnected immune, metabolic, barrier-related, and neuroendocrine pathways. Key mechanisms include microbiota-driven immune modulation, production of short-chain fatty acids (SCFAs), regulation of epithelial permeability and lipopolysaccharide (LPS) translocation, and modulation of the hypothalamic–pituitary–adrenal (HPA) axis and insulin-like growth factor-1 (IGF-1) signaling. Increasing evidence supports the role of these pathways in the pathogenesis and clinical course of inflammatory dermatological diseases, including atopic dermatitis, acne vulgaris, rosacea, and psoriasis. This narrative review critically evaluates current experimental and clinical evidence on probiotics, prebiotics, synbiotics, and postbiotics as therapeutic modulators of the gut–skin axis in dermatology. A targeted literature search covering the period 2000–2025 was conducted using PubMed/PMC, Scopus, Web of Science, and Google Scholar, prioritizing systematic reviews, meta-analyses, and randomized controlled trials reporting clinically relevant outcomes. Available data indicate that selected *Lactobacillus* and *Bifidobacterium* strains are associated with improvements in skin barrier function, modulation of inflammatory responses, and clinically meaningful benefits, most consistently in atopic dermatitis. The strongest evidence supports preventive and adjunctive use in pediatric atopic dermatitis. In acne vulgaris, psoriasis, and rosacea, reported efficacy appears moderate and heterogeneous, reflecting substantial variability in strains, formulations, dosing regimens, and study design. Prebiotics and synbiotics demonstrate favorable effects on microbial balance and inflammatory activity, while postbiotics and microbiota-modulating topical agents represent emerging therapeutic approaches with promising safety and tolerability profiles. Overall, microbiota-targeted interventions act on distinct nodes within the gut–skin axis and may serve as rational adjunctive strategies in selected dermatological conditions. However, clinical efficacy is strain- and formulation-specific, and further well-designed, adequately powered, and mechanism-driven clinical trials are required to define their precise role in routine dermatological practice.

*Correspondence: Sonya Marina (soniamarina1@yahoo.com)

Received: 30 January 2026; Accepted: 10 February 2026; Published: 25 February 2026



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Keywords

Gut–Skin Axis, Probiotics, Prebiotics, Synbiotics, Postbiotics

1. Introduction

The microbial communities of the skin and gastrointestinal tract constitute complex and dynamic ecosystems that play a pivotal role in epithelial barrier integrity, immune homeostasis, and metabolic regulation. The cutaneous microbiota—comprising bacteria, fungi, viruses, and archaea—forms stable symbiotic interactions with epidermal and immune cells. In healthy skin, the dominant bacterial phyla include Actinobacteria, Firmicutes, Proteobacteria, and Bacteroidetes, with pronounced variation according to anatomical site, moisture, and sebaceous activity [1]. Increased microbial diversity has been associated with improved barrier resilience, immune tolerance, and recovery following cutaneous injury.

Over the past decade, the concept of the gut–skin axis has emerged as a clinically relevant framework describing bidirectional communication between intestinal microbiota and skin physiology via immunological, metabolic, and neuroendocrine pathways. Intestinal dysbiosis may promote increased gut permeability, systemic low-grade inflammation, and altered cytokine signaling, thereby influencing both innate and adaptive immune responses in the skin [2-5]. These mechanisms are increasingly recognized as contributors to the pathogenesis of inflammatory dermatoses such as atopic dermatitis, acne vulgaris, rosacea, and psoriasis [6-9].

In parallel, growing interest has focused on microbiota-modulating therapeutic strategies, including probiotics, prebiotics, synbiotics, and postbiotics. While experimental and early clinical studies suggest potential benefits, translation into routine dermatological practice remains limited by substantial heterogeneity in strains, formulations, dosing regimens, study design, and clinical endpoints.

Against this background, the present article provides a critical narrative review of the gut–skin axis and microbiota-modulating therapies in dermatology, with particular emphasis on mechanistic pathways, disease-specific clinical evidence, and emerging therapeutic concepts such as postbiotics.

2. Methods

This narrative review was conducted using a structured literature search strategy to enhance transparency and reproducibility.

2.1. Data Sources and Search Strategy

A targeted search was performed in PubMed, Scopus, Web

of Science, and Google Scholar, covering publications from January 2000 to March 2024. Search terms included combinations of: “gut–skin axis”, “skin microbiota”, “intestinal microbiota”, “probiotics”, “prebiotics”, “synbiotics”, “postbiotics”, “dermatology”, “atopic dermatitis”, “psoriasis”, “acne”, “rosacea”, “seborrheic dermatitis”

2.2. Inclusion and Exclusion Criteria

Included studies:

- 1) Experimental (in vitro, animal) and clinical studies
- 2) Randomized controlled trials, cohort studies, systematic reviews, and meta-analyses
- 3) Studies evaluating microbiota-modulating interventions with dermatological relevance

Excluded studies:

- 1) Non-English publications
- 2) Case reports without mechanistic or clinical relevance
- 3) Studies lacking defined interventions or outcome measures

2.3. Evidence Assessment

Evidence was qualitatively assessed based on:

- 1) Study design and sample size
- 2) Consistency of findings
- 3) Clinical relevance and reproducibility

Formal guideline-based grading systems were not applied due to heterogeneity in study designs and endpoints; instead, levels of evidence were categorized descriptively (low, moderate).

3. Mechanisms of Interaction Within the Gut–Skin Axis

The gut–skin axis represents a bidirectional regulatory network integrating immunological, metabolic, barrier-related, and neuroendocrine pathways through which intestinal microbiota influences skin homeostasis and inflammatory activity [10, 11].

3.1. Immunological Regulation

Gut microbiota modulates T-cell differentiation and cyto-

kine balance via interactions with intestinal epithelial and antigen-presenting cells. Certain strains enhance IL-10 and TGF- β production while suppressing IL-6, IL-8, and TNF- α , promoting regulatory T-cell (Treg) activity and restoring Th1/Th2/Th17 balance [12-18].

3.2. Microbial Metabolites

Short-chain fatty acids (SCFAs)—acetate, propionate, and butyrate—exert systemic anti-inflammatory effects via NF- κ B inhibition, epigenetic regulation, and activation of G-protein-coupled receptors (GPR41/43), supporting epithelial integrity and skin hydration [19-21].

3.3. Barrier Integrity and Systemic Inflammation

Intestinal dysbiosis may disrupt tight junction proteins, facilitating translocation of microbial products such as lipopolysaccharides (LPS), thereby amplifying systemic and cutaneous inflammation [22, 23].

3.4. Neuroendocrine Pathways

Microbiota-mediated modulation of the hypothalamic-pituitary-adrenal axis influences cortisol, IGF-1, and stress responses, linking psychological stress to inflammatory skin disease exacerbation [19, 22].

4. Definitions and Therapeutic Categories of Microbiota-Modulating Interventions

4.1. Probiotics

Probiotics are defined as live microorganisms that, when administered in adequate amounts, confer a documented health benefit on the host. In dermatological research and clinical practice, the most extensively investigated probiotic strains belong to the genera *Lactobacillus* and *Bifidobacterium* [9]. Their mechanisms of action include modulation of innate and adaptive immune responses through enhanced production of anti-inflammatory cytokines such as interleukin-10 (IL-10) and transforming growth factor- β (TGF- β), suppression of pro-inflammatory mediators, and promotion of regulatory T-cell (Treg) differentiation [21].

Beyond immunomodulatory effects, probiotics contribute to the reinforcement of both intestinal and cutaneous barrier integrity by enhancing tight junction protein expression, reducing intestinal permeability, and limiting systemic inflammatory signaling. These mechanisms are of particular clinical relevance in inflammatory dermatoses such as atopic dermatitis, acne vulgaris, and psoriasis, in which barrier dysfunction

and immune dysregulation represent central pathogenic features.

4.2. Prebiotics

Prebiotics are non-digestible substrates that are selectively utilized by beneficial host microorganisms, resulting in a measurable health benefit. By promoting the growth and metabolic activity of commensal bacteria, prebiotics indirectly modulate microbiota composition and function. A key mechanism underlying their biological effects is stimulation of short-chain fatty acid (SCFA) production, which exerts anti-inflammatory, antioxidant, and barrier-protective actions. In dermatological contexts, prebiotics have been associated with improved skin hydration, reduced inflammatory activity, and stabilization of the cutaneous microbiota; however, direct clinical evidence remains more limited compared with that for probiotics [9, 11].

4.3. Synbiotics

Synbiotics are combined formulations containing probiotics and prebiotics designed to exert synergistic biological effects. The prebiotic component enhances probiotic survival, colonization, and functional activity, thereby amplifying immunomodulatory and barrier-restorative properties [14]. Compared with probiotic monotherapy, synbiotics may offer more pronounced modulation of intestinal microbiota composition and systemic inflammatory responses. This approach appears particularly promising in chronic inflammatory skin diseases characterized by significant dysbiosis; nevertheless, reported clinical outcomes remain heterogeneous due to variability in formulations, dosing regimens, and study design [15].

4.4. Postbiotics

Postbiotics are defined as non-viable microorganisms and/or their structural components and metabolites that confer a health benefit on the host. These include bacterial cell fragments, enzymes, peptides, organic acids, and other bioactive molecules [11, 15]. Postbiotics offer several practical advantages, including a favorable safety profile, improved physicochemical stability, and the absence of risks associated with microbial translocation. These properties make them particularly suitable for topical dermatological applications and for use in vulnerable patient populations. Experimental and early clinical data suggest that postbiotics exert immunomodulatory, anti-inflammatory, and barrier-restorative effects, positioning them as an emerging alternative or adjunct to live probiotic therapies.

Collectively, probiotics, prebiotics, synbiotics, and postbiotics represent complementary strategies for modulating the microbiota and the gut-skin axis. Their therapeutic efficacy in dermatology is highly dependent on strain-specific character-

istics, dosage, route of administration, and duration of intervention. Among these approaches, postbiotics are attracting increasing interest due to their enhanced safety profile and potential suitability for personalized dermatological therapies.

The principal mechanisms of action and clinically relevant effects of microbiota-modulating interventions as summarized in Table 1.

Table 1. Mechanisms of action and clinical effects of probiotics, prebiotics, synbiotics, and postbiotics in dermatological diseases.

Intervention	Main mechanisms of action	Reported clinical effects	Clinical relevance / comments
Probiotics	Immune modulation (↑ Treg activity, ↓ Th2/Th17 polarization); antimicrobial activity; enhancement of epithelial barrier integrity	Reduced disease severity; improved skin barrier function	Strongest clinical evidence in atopic dermatitis; effects are strain-specific
Prebiotics	Selective stimulation of beneficial intestinal microbiota; increased SCFA production	Improved intestinal barrier function; indirect anti-inflammatory effects	Limited direct dermatological evidence; supportive adjunctive role
Synbiotics	Synergistic probiotic–prebiotic interaction enhancing microbial balance and immune regulation	More pronounced clinical improvement in selected studies	Heterogeneous outcomes due to variable formulations and protocols
Postbiotics	Bioactive metabolites and microbial components with immunomodulatory and barrier-stabilizing properties	Anti-inflammatory effects; improved barrier function	High stability and favorable safety profile; emerging clinical data

Abbreviations: SCFA, short-chain fatty acids; Treg, regulatory T cells.

Note: Reported mechanisms and clinical effects are based on experimental, translational, and clinical studies and may vary depending on strain, formulation, dosage, and duration of administration.

5. Clinical Evidence by Dermatological Disease

Available clinical and experimental data on microbiota-modulating interventions—including probiotics, prebiotics, synbiotics, and postbiotics—outline a promising yet heterogeneous evidence base in dermatology. Reported outcomes are strongly influenced by disease-specific pathophysiology, strain- or formulation-specific characteristics, dosage, duration of administration, and selected clinical endpoints. Overall, the most consistent clinical benefits are observed in conditions in which epidermal barrier dysfunction and systemic low-grade inflammation represent central pathogenic drivers. In contrast, for several dermatological disorders, the available evidence remains limited to small, heterogeneous, and predominantly short-term studies.

5.1. Dermatological Diseases with a Moderate Level of Evidence

5.1.1. Atopic Dermatitis

The strongest and most consistent evidence for microbiota-modulating therapies is available in atopic dermatitis, particularly in preventive settings. *Lactobacillus rhamnosus* GG

(LGG) remains the most extensively studied probiotic strain, especially in high-risk infant populations, where randomized controlled trials and meta-analyses demonstrate a statistically significant—albeit population-dependent—reduction in the incidence of early-onset atopic eczema [24–27].

Therapeutic data in patients with established atopic dermatitis are more heterogeneous. Several clinical studies report moderate reductions in disease severity, as assessed by the SCORAD index, following administration of *Lactobacillus plantarum* and selected *Bifidobacterium* strains in both pediatric and adult populations. Improved outcomes are more frequently observed with multistrain formulations and longer treatment durations, supporting a cumulative and strain-dependent effect [28].

Postbiotic interventions, including heat-killed *Lactobacillus acidophilus* L-92 and *Lactobacillus paracasei* K71, have demonstrated favorable effects on pruritus and overall disease activity, combined with an excellent safety profile and sustained clinical responses in selected trials. These findings position postbiotics as a particularly attractive option for long-term disease management and for use in pediatric populations [29–31].

5.1.2. Psoriasis

In psoriasis, accumulating data of moderate-level evidence suggest a potential adjuvant benefit of oral probiotic supple-

mentation, particularly formulations containing *Bifidobacterium infantis* 35624. Reported clinical outcomes include modest improvements in the Psoriasis Area and Severity Index (PASI), enhanced quality-of-life measures, and reductions in selected systemic inflammatory markers [32].

The proposed biological mechanisms involve indirect modulation of the IL-23/Th17/IL-17 axis, SCFA-mediated anti-inflammatory pathways, and attenuation of NF- κ B-driven inflammatory signaling [33-35]. However, clinical efficacy appears highly dependent on probiotic composition and study design, and the limited number of large, well-controlled trials currently precludes generalized therapeutic recommendations [36-38].

5.1.3. Seborrhea and Seborrheic Dermatitis

Seborrhea and seborrheic dermatitis represent another disease group with relatively consistent clinical support for microbiota-modulating approaches, particularly topical formulations. Both probiotic and postbiotic interventions have demonstrated anti-inflammatory effects accompanied by improvements in epidermal barrier function [39-43].

The most convincing evidence is available for topical products containing *Lactobacillus pentosus* and *Streptococcus thermophilus*. Randomized controlled trials reporting reductions in *Malassezia* spp. colonization and partial restoration of microbial diversity lend additional credibility to these findings, despite the overall limited number of large-scale studies [44, 45].

5.1.4. UV-Induced Skin Damage and Photoaging

In the context of UV-induced skin damage and photoaging, oral probiotic interventions demonstrate a moderate level of evidence, particularly in controlled ultraviolet exposure models. Reported effects include stabilization of epidermal barrier function, attenuation of UV-induced immunosuppression, and improvements in clinical parameters such as erythema reactivity and minimal erythema dose [46, 47].

Among the best-studied strains is *Lactobacillus (Lacticaeibacillus) johnsonii*, for which controlled clinical data indicate beneficial effects on skin barrier integrity and local immune responses. In parallel, topical postbiotic ferments and lysates—such as *Galactomyces* ferment filtrate, *Saccharomyces* ferment lysate, *Bifida* ferment lysate, and *Lactobacillus* ferment lysate—have demonstrated antioxidant activity, stimulation of collagen synthesis, enhancement of barrier-related proteins (including filaggrin and involucrin), and suppression of UV-induced pro-inflammatory cytokines. Nevertheless, much of the available evidence remains derived from in vitro studies and small clinical series, underscoring the need for larger, well-controlled clinical trials [48, 49].

5.2. Dermatological Diseases with Low to Moderate Levels of Evidence

5.2.1. Acne vulgaris

In acne vulgaris, available clinical data support a low to moderate but statistically significant therapeutic effect of selected oral probiotic formulations. The most consistent evidence has been reported for preparations containing *Lacticaeibacillus rhamnosus* SP1, which have demonstrated reductions in both inflammatory and non-inflammatory lesion counts in controlled clinical studies [50-53]. Comparable effects have been observed with formulations combining *L. rhamnosus* (CECT 30031) and *Arthrospira platensis*, suggesting potential benefits on both inflammatory activity and sebaceous gland function.

Several studies also report favorable modulation of sebum secretion and acne-associated metabolic pathways, including regulation of the insulin-like growth factor-1 (IGF-1)/FOXO1 axis, providing a biologically plausible link between microbiota modulation and clinical improvement [54].

Topical postbiotic formulations containing *Lactobacillus paracasei* MSMC 39-1 have demonstrated additional therapeutic potential, particularly in patients with sensitive or reactive skin [55, 56]. Experimental and clinical data indicate inhibition of *Cutibacterium acnes* proliferation, suppression of pro-inflammatory cytokines (IL-6, IL-8, TNF- α), improvement of epidermal barrier function, and modulation of ceramide synthesis. Nevertheless, substantial heterogeneity in acne phenotypes, treatment regimens, and clinical endpoints underscores the need for larger, well-designed randomized controlled trials to confirm these preliminary findings [57, 58].

5.2.2. Rosacea

In rosacea, the current level of evidence for microbiota-modulating therapies remains low to moderate. Oral probiotic combinations containing *Lactobacillus acidophilus* and *Bifidobacterium bifidum*, as well as topical postbiotic formulations based on *Streptococcus thermophilus*, have been evaluated primarily in small pilot studies and limited randomized trials [59, 60]. Reported clinical benefits include improvement of epidermal barrier function, reduction in transepidermal water loss, attenuation of erythema, and modulation of cathelicidin–kallikrein 5 (KLK5)–mediated inflammatory pathways.

The frequent association of rosacea with gastrointestinal comorbidities, such as irritable bowel syndrome and *Helicobacter pylori* colonization, supports the pathophysiological relevance of the gut–skin axis. However, evidence supporting a direct and consistent clinical improvement of rosacea following correction of these gastrointestinal factors remains inconclusive. From a practical standpoint, topical postbiotic formulations—particularly *Streptococcus thermophilus* ferments—appear to represent the most reliable adjunctive approach, given their reproducible barrier-enhancing effects and favorable tolerability profile [61-63].

5.2.3. Cutaneous Infections and Chronic Wounds

In cutaneous infections and chronic wounds, including diabetic foot ulcers, available clinical evidence for microbiota-modulating interventions is limited but suggests potential benefit. Small clinical studies indicate that adjunctive use of topical probiotic or postbiotic formulations containing *Lactiplantibacillus plantarum* may reduce time to complete epithelialization and improve wound-healing parameters when added to standard wound care [64-67].

Experimental and early clinical data further suggest reduced biofilm formation and decreased colonization by multi-drug-resistant organisms, including methicillin-resistant *Staphylococcus aureus* (MRSA). Despite these encouraging findings, heterogeneity in wound populations, variability in clinical endpoints, and the limited number of multicenter trials currently restrict translation into routine clinical practice. Larger, well-controlled randomized studies are required to establish clinical efficacy and standardized treatment protocols [68, 69].

5.3. Dermatological Diseases with a Low Level of Evidence

5.3.1. Alopecia

In alopecia, current evidence supporting the efficacy of microbiota-modulating therapies remains limited and is derived predominantly from small, short-term clinical studies [70]. Available data suggest that topical postbiotic formulations containing lysates of *Lactobacillus*, *Lactiplantibacillus plantarum*, and *Bifidobacterium* spp. may exert biologically relevant effects, including stimulation of keratinocyte proliferation, upregulation of β -defensin-2 expression, and reduction of pro-inflammatory cytokines such as IL-1 α and TNF- α . These molecular effects have been associated with modest improvements in hair density and an increased proportion of anagen-phase hair follicles.

However, investigated patient cohorts remain small, follow-up periods are short, and reported outcomes frequently fail to reach statistical significance, substantially limiting clinical interpretability and generalizability [71]. Evidence supporting the use of oral probiotics across different forms of alopecia is currently insufficient, with the majority of available data restricted to experimental and preclinical models [72].

5.3.2. Chronic Spontaneous Urticaria

Preclinical evidence from in vitro systems and animal models indicates that postbiotic metabolites—including short-chain fatty acids and fermentative derivatives produced by *Lactobacillus* and *Bifidobacterium* species—may inhibit Fc ϵ RI-dependent mast cell degranulation, thereby reducing the release of histamine, tryptase, and pro-inflammatory cytokines such as IL-6 [73, 74]. In parallel, selected probiotic strains, including *Lactobacillus rhamnosus* GG and

Bifidobacterium longum, have demonstrated immunomodulatory effects through modulation of histidine decarboxylase expression, improvement of epithelial barrier function, and attenuation of Th2-associated cytokines (IL-4, IL-5, and IL-13), which are central mediators of chronic allergic inflammation [75].

In chronic spontaneous urticaria, clinical evidence for probiotic supplementation as an adjunct to standard therapy remains limited to small-scale clinical studies. These studies report modest reductions in disease activity scores, improvements in quality-of-life measures, and decreases in selected inflammatory biomarkers [76-78]. Evidence regarding the use of postbiotics in this clinical context is even more limited, and current data do not support standardized therapeutic recommendations or routine clinical use [79].

6. Discussion

This review highlights the gut-skin axis as a clinically meaningful regulatory system linking intestinal microbiota to inflammatory and barrier-related skin disorders. Across dermatological diseases, microbiota-modulating interventions consistently demonstrate adjunctive rather than standalone therapeutic value.

Several cross-cutting observations emerge:

- 1) Strain specificity is critical—clinical efficacy cannot be generalized across probiotic categories or commercial formulations.
- 2) Barrier dysfunction and low-grade inflammation predict better therapeutic responsiveness.
- 3) Postbiotics represent a particularly promising category, offering stability, safety, and suitability for topical and long-term use.
- 4) Evidence remains strongest in atopic dermatitis (prevention), psoriasis, seborrheic dermatitis, and UV-induced barrier dysfunction, while acne and rosacea show modest, variable responses.

Methodological heterogeneity, short follow-up periods, and lack of microbiome-based patient stratification remain major limitations.

6.1. Practical Interpretation and Key Limitations

The gut-skin axis constitutes a clinically relevant regulatory framework through which intestinal microbiota influences cutaneous inflammation, immune homeostasis, and barrier function across a broad range of dermatological conditions [3]. Current evidence indicates that probiotics, prebiotics, synbiotics, and postbiotics exert their most consistent clinical benefits when applied as adjunctive rather than standalone therapeutic interventions.

At present, the strongest evidence supports microbiota-modulating approaches in atopic dermatitis—particularly in preventive settings—as well as in plaque psoriasis, seborrhea

and seborrheic dermatitis (notably with topical postbiotic formulations), and UV-induced barrier and immune dysfunction. Across disease entities, clinical efficacy is highly strain-specific and dependent on dosage, formulation, and duration of administration, which substantially limits extrapolation between different products.

Among available strategies, postbiotics appear particularly promising due to their improved stability, favorable tolerability, and low risk of adverse effects. These properties render them especially suitable for topical dermatological use, long-term application, and for patients with multimorbidity or increased susceptibility to treatment-related adverse events.

Microbiota-modulating interventions should therefore be regarded as complementary tools within an integrated, evidence-based dermatological treatment strategy. In atopic dermatitis, preventive administration of selected probiotic or synbiotic formulations in high-risk pediatric populations is supported by the most consistent data, whereas therapeutic effects in established disease remain modest. In psoriasis, oral probiotics may be considered as adjunctive options in selected patients with moderate disease activity and systemic inflammatory features, but they do not replace conventional systemic or

biologic therapies.

Topical postbiotic formulations may be particularly appropriate for conditions characterized by barrier dysfunction and sensitive skin, including seborrheic dermatitis, rosacea, and acne vulgaris, given their favorable tolerability profile and minimal irritation potential. In acne and rosacea, reported clinical responses remain moderate and variable, underscoring the need for careful patient selection.

In chronic wounds, including diabetic ulcers, topical probiotic and postbiotic approaches show potential to support wound healing but should be implemented strictly within multidisciplinary wound care protocols. In contrast, evidence for efficacy in alopecia and chronic spontaneous urticaria remains preliminary and insufficient to support routine clinical use.

When selecting microbiota-modulating products, preference should be given to clinically evaluated strains, clearly defined dosages, and transparent formulations. Patient counseling regarding realistic expectations, limitations of the evidence, and the adjunctive nature of these interventions is essential. In immunocompromised individuals, postbiotics represent a safer alternative to live probiotic preparations.

An overview of clinical evidence is provided in [Table 2](#).

Table 2. Microbiota-modulating therapeutic approaches in dermatological diseases and level of clinical evidence.

Disease entity	Interventions with most evidence	Main clinical endpoints	Level of evidence	Key limitations
Atopic dermatitis	Probiotics (<i>L. rhamnosus</i> GG, <i>L. plantarum</i> , multistrain formulations), synbiotics; postbiotics (heat-killed <i>L. acidophilus</i> , <i>L. paracasei</i>)	↓ eczema incidence (prevention); ↓ SCORAD; ↓ pruritus; modulation of immune markers	Moderate (prevention); low–moderate (treatment)	Strong strain specificity; heterogeneous dosages, treatment durations, and study populations
Acne vulgaris	Oral probiotics (<i>L. rhamnosus</i> SP1); topical postbiotics (cell-free preparations)	↓ inflammatory and non-inflammatory lesion counts; ↓ inflammatory activity; ↓ sebum production; IGF-1/FOXO1 modulation	Low–moderate	Small RCTs; heterogeneous regimens; marked phenotypic variability
Rosacea	Oral probiotics (limited strain combinations); topical postbiotics (<i>S. thermophilus</i> ferments); GI-directed interventions	↓ erythema and burning; ↓ papulopustules (selected subgroups); ↑ epidermal barrier parameters	Low–moderate (topical/oral); low (GI correction)	Small, heterogeneous studies; non-uniform endpoints; frequent comorbidities
Seborrheic dermatitis / seborrhea	Topical probiotics (<i>L. pentosus</i>); topical postbiotics/ferments (<i>S. thermophilus</i>); postbiotic-containing shampoos	↓ erythema, scaling, and pruritus; ↓ <i>Malassezia</i> spp.; ↑ microbial diversity	Low–moderate to moderate (strongest for postbiotic shampoos)	Limited number of large RCTs; high product variability
Psoriasis (plaque type)	Oral multistrain probiotics (adjunctive therapy)	↓ PASI; ↑ DLQI; ↓ CRP and pro-inflammatory cytokines; ↓ relapse rate (selected studies)	Moderate	Limited number of RCTs; heterogeneous formulations; lack of multicenter data
Alopecia (AGA / TE)	Topical postbiotic lysates and ferments	↑ hair density; ↓ shedding; ↑ anagen-to-telogen ratio	Low–moderate (topical); low (oral)	Small cohorts; short follow-up periods; non-standardized outcome measures

Disease entity	Interventions with most evidence	Main clinical endpoints	Level of evidence	Key limitations
Chronic spontaneous urticaria	Oral probiotics (strain combinations); postbiotics (exploratory)	↓ UAS7; ↑ quality of life; ↓ CRP and IL-6; exploratory biomarkers	Low–moderate (probiotics); low (postbiotics)	Small studies; heterogeneous disease phenotypes; limited follow-up
Skin infections / chronic wounds	Topical probiotics (<i>L. plantarum</i>); cell-free postbiotic preparations	↑ epithelialization rate; ↓ healing time; ↓ biofilm burden	Low–moderate	Heterogeneous wound types; variable clinical endpoints; limited multi-center trials
UV-induced skin damage / photoaging	Oral probiotics (<i>L. johnsonii</i>); topical postbiotic ferments and lysates	↓ UV-induced immunosuppression; ↑ barrier stability; ↑ minimal erythema dose; ↓ erythema	Moderate (oral); low (topical)	Predominantly experimental data; small clinical series

Abbreviations: AGA, androgenetic alopecia; TE, telogen effluvium; SCORAD, Scoring Atopic Dermatitis; PASI, Psoriasis Area and Severity Index; DLQI, Dermatology Life Quality Index; UAS7, Urticaria Activity Score over 7 days; CRP, C-reactive protein.

Note: Levels of evidence reflect the overall quality, consistency, and sample size of available clinical studies rather than formal guideline-based grading.

6.2. Safety Considerations and Limitations

Microbiota-modulating therapies are generally well tolerated when used as adjunctive interventions in dermatological practice. However, several limitations merit consideration. Clinical effects are strictly strain-specific and influenced by formulation, dosage, and duration of administration, limiting generalizability across products and clinical settings. Furthermore, much of the available evidence derives from small, heterogeneous studies with variable endpoints and limited follow-up.

Live probiotic preparations require particular caution in immunocompromised patients and individuals with severe systemic disease, in whom rare cases of probiotic-associated infections have been reported. In contrast, postbiotics offer a favorable safety profile due to their non-viable nature, enhanced stability, and minimal risk of systemic adverse effects, making them particularly suitable for topical and long-term use.

Importantly, microbiota-modulating interventions should not substitute evidence-based dermatological therapies but should be incorporated as complementary options within an individualized, risk–benefit–oriented clinical approach. Lack of standardization in probiotic strains, formulations, and outcome measures remains a major challenge for future clinical research.

6.3. Future Perspectives

Future research should prioritize adequately powered, well-designed randomized controlled trials employing standardized strains, formulations, dosing regimens, and clinically meaningful outcome measures. Improved patient stratification based on age, disease phenotype, comorbidities, and baseline

microbiota profiles may facilitate identification of subgroups most likely to benefit from targeted interventions.

Integration of microbiome profiling and multi-omics approaches into clinical trial design is expected to advance mechanistic understanding and support the development of personalized therapeutic strategies. In parallel, postbiotic-based formulations with defined bioactive components represent a promising avenue for enhancing safety, stability, and regulatory clarity in future dermatological applications.

7. Conclusion

Accumulated experimental and clinical evidence over the past two decades firmly establishes the gut–skin axis as a biologically and clinically relevant regulatory system in dermatology, implicated in the pathogenesis and persistence of multiple inflammatory and functional skin disorders. Through interconnected immunological, metabolic, barrier-related, and neuroendocrine pathways, intestinal microbiota exerts a measurable and clinically meaningful influence on cutaneous homeostasis and inflammatory activity.

The most consistent and reproducible clinical evidence supports microbiota-modulating interventions in atopic dermatitis—particularly in preventive settings—as well as in plaque psoriasis, seborrheic dermatitis, and ultraviolet-induced barrier dysfunction. In acne vulgaris, rosacea, and chronic wounds, available data suggest potential therapeutic benefit, whereas evidence in alopecia and chronic spontaneous urticaria remains insufficient to support standardized clinical recommendations.

A central conclusion of this review is that therapeutic efficacy is strictly strain-specific and critically dependent on formulation, dosage, and duration of administration, which substantially limits extrapolation across products and commercial

preparations [11, 16, 80-82]. Methodological heterogeneity, limited patient stratification, and discrepancies between studied and marketed formulations remain key obstacles to broader clinical translation.

Within this evolving therapeutic landscape, postbiotics emerge as a particularly promising category owing to their enhanced physicochemical stability, favorable tolerability, and reassuring safety profile, especially for topical use and in vulnerable patient populations. Overall, microbiota-modulating therapies represent rational adjunctive strategies in contemporary dermatology. Their integration into clinical practice should remain cautious, individualized, and firmly grounded in robust, evidence-based principles, while future research should prioritize standardized, mechanism-driven clinical trials to define their precise therapeutic role.

Abbreviations

IL-10	Interleukin 10
TGF- β	Transforming Growth Factor Beta
TNF- α	Tumor Necrosis Factor Alpha
Tregs	Regulatory T Cells
SCFAs	Short-chain Fatty Acids
GPR41 / GPR43	G Protein-coupled Receptors 41 and 43
IGF-1	Insulin-like Growth Factor 1
ZO-1	Zonula Occludens 1
LPS	Lipopolysaccharide
HPA	Hypothalamic-pituitary-adrenal Axis
LGG	<i>Lactobacillus rhamnosus</i> GG
SCORAD	Scoring Atopic Dermatitis
PASI	Psoriasis Area and Severity Index
DLQI	Dermatology Life Quality Index
MRSA	Methicillin-Resistant <i>Staphylococcus Aureus</i>
FOXO1	Forkhead Box Protein O1
SIBO	Small Intestinal Bacterial Overgrowth
RCT	Randomized Controlled Trial
UAS7	Weekly Urticaria Activity Score
MED	Minimal Erythema Dose

Author Contributions

Valentina Broshtilova: Conceptualization, Supervision, Writing – review & editing

Sonya Marina: Supervision, Writing – original draft

Ivan Vasilevski: Data curation, Methodology

Yoanna Velevska: Data curation, Investigation

Irina Yungareva: Data curation, Resources

Yoanna Petkova: Data curation, Validation

Conflicts of Interest

The authors declare no conflicts of interest.

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