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The Gut–Skin Axis: The Role of Intestinal Microbiota in Acne, Psoriasis and Atopic Dermatitis: A Narrative Review

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Abstract

Background. The gut–skin axis links intestinal dysbiosis with chronic inflammatory skin diseases. Acne vulgaris, psoriasis and atopic dermatitis have been associated with altered gut microbial diversity, metabolite production, barrier function and immune regulation.

Objective. To summarize current evidence on the role of intestinal microbiota in the pathogenesis and potential management of acne vulgaris, psoriasis and atopic dermatitis, with emphasis on gut–skin mechanisms and microbiota-targeted interventions.

Methods. A narrative review of PubMed and PubMed Central (2015–2025) was performed using terms related to the gut–skin axis, intestinal microbiota, acne, psoriasis, atopic dermatitis, dysbiosis and probiotics. Human and translational studies, systematic reviews, meta-analyses and high-quality narrative reviews on gut microbiota in these conditions were included; 20 publications were synthesized qualitatively.

Results. Across the included studies, gut dysbiosis in all three diseases was characterized by reduced microbial diversity, loss of short-chain fatty acid-producing commensals and enrichment of pro-inflammatory taxa. In acne, these changes correlated with systemic inflammation and sebaceous activity; in psoriasis, with Th17-driven inflammation and associated comorbidities; in atopic dermatitis, with early-life immune dysregulation and barrier dysfunction. Several clinical trials reported modest, strain-dependent improvements in disease severity with Lactobacillus- and Bifidobacterium-based probiotics.

Conclusions. Available data support a mechanistic link between intestinal microbiota and inflammatory skin diseases. Gut dysbiosis appears to contribute to immune polarization and barrier impairment, while microbiota-modulating strategies show promise as adjunctive therapies. Further well-designed trials are needed to clarify causality and to define effective, standardized microbiota-based interventions.

Keywords: acne vulgaris; atopic dermatitis; dysbiosis; gut–skin axis; inflammation; intestinal microbiota; probiotics; psoriasis.

1. Introduction

The gut–skin axis has emerged as an important framework for understanding how intestinal microbial disturbances influence systemic immunity and cutaneous inflammation. Numerous studies have shown that gut microbiota play a central role in regulating immune homeostasis, epithelial barrier integrity, and metabolic signaling pathways relevant to dermatologic disease development [1–3]. Dysbiosis—characterized by reduced microbial diversity, loss of beneficial short-chain fatty acid (SCFA)–producing bacteria, and overgrowth of pro-inflammatory taxa—has been linked to a growing number of chronic inflammatory conditions, including disorders of the skin [1,2].

Communication between the gut and the skin occurs through several interconnected mechanisms: modulation of T-helper 1 (Th1), T-helper 17 (Th17), and regulatory T-cell (Treg) pathways, production of microbial metabolites such as butyrate, neuroendocrine signaling, and maintenance of epithelial barrier stability [1,4]. A disrupted microbiome may impair SCFA synthesis, increase intestinal permeability, and promote translocation of microbial components

and inflammatory mediators into systemic circulation, thereby amplifying cutaneous immune activation [2–4].

Among chronic inflammatory dermatoses, acne vulgaris, psoriasis, and atopic dermatitis show the most consistent associations with alterations of the gut microbiota. In acne, dysbiosis has been implicated in metabolic and immunologic abnormalities, including increased oxidative stress, altered lipid signaling, and systemic inflammation supportive of comedogenesis and follicular disruption [5–7]. Recent Mendelian randomization studies provide further evidence for causal links between specific gut microbial taxa, circulating microbial metabolites, and acne risk [8]. Broader narrative reviews on acne further support the relevance of systemic microbiome–immune interactions in the pathogenesis and clinical expression of the disease [19,20].

Psoriasis is the most extensively characterized dermatologic disease in the context of the gut–skin axis. Patients exhibit reduced α-diversity, depletion of anti-inflammatory species such as Faecalibacterium prausnitzii, and enrichment of pro-inflammatory genera including Ruminococcus and Eggerthella [9–12]. These microbial patterns are closely aligned with Th17-dominant immune pathways, the principal inflammatory axis in psoriatic disease. Dysbiosis may also contribute to common psoriatic comorbidities such as metabolic syndrome, obesity, and inflammatory bowel disease [10].

In atopic dermatitis, gut microbiota disturbances appear to affect immune maturation, epithelial barrier development, and systemic inflammatory signaling—particularly during early childhood. Reduced colonization by Bifidobacterium and Lactobacillus, decreased SCFA production, and lower microbial diversity have been associated with heightened risk and severity of atopic dermatitis [13–16]. Dysbiosis may potentiate systemic Th2-skewed inflammation and impair structural barrier proteins, contributing to chronic disease persistence [14,16]. Additional studies have also highlighted alterations in gut microbial composition in eczema and related atopic conditions, emphasizing the clinical significance of microbiota-targeted therapeutic strategies [18].

These observations have led to increasing interest in microbiota-directed therapies. Several clinical studies suggest that targeted probiotics—especially strains of Lactobacillus and Bifidobacterium—may reduce disease activity or inflammatory biomarkers when used as adjunctive treatment, although effects remain strain-specific and inconsistently replicated [6,12,15,17]. Improving the understanding of microbiota–immune interactions may provide new opportunities for therapeutic intervention across inflammatory skin diseases.

This narrative review aims to summarize current evidence on the gut–skin axis and evaluate the role of intestinal microbiota in the pathogenesis and potential management of acne vulgaris, psoriasis, and atopic dermatitis.

2. Methods

This narrative review was conducted using a structured literature search in PubMed and PubMed Central (PMC) covering the period from January 1, 2015, to January 30, 2025. The search strategy included the following key terms and their combinations: gut–skin axis, intestinal microbiota, microbiome, dysbiosis, acne, acne vulgaris, psoriasis, atopic dermatitis, eczema, probiotics, and microbial metabolites. Medical Subject Headings (MeSH) were applied when appropriate to refine search precision.

Inclusion criteria

Studies were eligible for inclusion if they met the following criteria:

Peer-reviewed articles available in English.

Human studies, translational studies, or high-quality mechanistic research relevant to the gut–skin axis.

Systematic reviews, meta-analyses, randomized controlled trials, observational studies, or narrative reviews focused on the microbiota in acne vulgaris, psoriasis, or atopic dermatitis.

Articles providing clinically or mechanistically relevant data on gut microbiome composition, microbial metabolites, host–microbe interactions, or therapeutic modulation through probiotics or related interventions.

Exclusion criteria

The following were excluded:

Case reports, conference abstracts, letters without substantial data, and non-peer-reviewed articles.

Studies focusing solely on the skin microbiome without relevance to intestinal microbiota.

Animal-only studies unless they provided mechanistic insights clearly translatable to human disease.

Publications outside the defined time window.

Study selection process

After removal of duplicates, titles and abstracts were screened for relevance. Full texts were reviewed to confirm eligibility based on predefined criteria. From the initial search output, 20 publications were selected for final synthesis. These included systematic reviews, meta-analyses, clinical trials, observational studies, and translational research articles addressing gut microbial alterations in acne vulgaris, psoriasis, and atopic dermatitis.

Data extraction and synthesis

Extracted data included study design, population characteristics, gut microbiota composition, identified dysbiosis patterns, associated immunologic pathways, microbial metabolite profiles, and therapeutic outcomes related to microbiome modulation. Due to heterogeneity across study designs and outcome measures, a qualitative synthesis was performed. Findings were organized thematically, with separate sections dedicated to acne vulgaris, psoriasis, and atopic dermatitis, followed by a unifying discussion on shared mechanisms underlying the gut–skin axis.

3. Results

3.1. Mechanistic Foundations of the Gut–Skin Axis

Across the eligible studies, several recurring mechanistic pathways linking intestinal dysbiosis with cutaneous inflammation were identified. Reduced microbial diversity and depletion of SCFA-producing genera such as Faecalibacterium, Roseburia, and Bifidobacterium were consistently associated with heightened systemic inflammation [1–3]. Disrupted SCFA synthesis—particularly butyrate and propionate—was linked to impaired regulatory T-cell (Treg) activity, increased intestinal permeability, and enhanced translocation of microbial components into systemic circulation [2,4].

Pro-inflammatory taxa, including Ruminococcus, Eggerthella, and various Proteobacteria, were frequently enriched in patients with inflammatory dermatoses [3,9–12]. These bacteria induce activation of Th1 and Th17 pathways, stimulate IL-17 and IL-23 production, and promote systemic immune activation that may manifest as cutaneous disease. Dysbiosis was also associated with altered tryptophan metabolism, reduced production of aryl hydrocarbon receptor (AhR) ligands, and downstream impairment of epithelial and immune regulatory pathways [4].

Together, these findings highlight a shared pattern of decreased beneficial commensals, loss of metabolic regulatory signals, and expansion of pro-inflammatory species across acne vulgaris, psoriasis, and atopic dermatitis.

3.2. Acne Vulgaris and the Gut Microbiota

Acne-related studies demonstrated that individuals with acne vulgaris exhibit altered gut microbial composition characterized by reduced diversity, diminished SCFA-producing taxa, and increased pro-inflammatory genera [5–7]. Dysbiosis correlated with lipid metabolic changes, increased systemic oxidative stress, and enhanced activity of insulin-like growth factor-1 (IGF-1), all of which are implicated in acne development [6].

A Mendelian randomization study further identified causal associations between specific microbial taxa and acne, including elevated Clostridium sensu stricto and Ruminococcus gnavus, as well as circulating microbial metabolites influencing sebaceous inflammation [8]. Studies investigating probiotic supplementation reported reductions in inflammatory lesion counts and modulation of systemic cytokines when Lactobacillus or Bifidobacterium strains were administered alongside standard therapies [6,7].

Overall, gut microbial perturbations in acne appear to promote systemic inflammation, oxidative stress, and metabolic dysregulation that contribute to disease expression.

3.3. Psoriasis and the Gut Microbiota

Psoriasis showed the strongest and most reproducible association with intestinal dysbiosis among the included conditions. Multiple studies reported reduced α-diversity, depletion of anti-inflammatory taxa such as Faecalibacterium prausnitzii, Akkermansia muciniphila, and Roseburia, and enrichment of pro-inflammatory genera including Ruminococcus, Eggerthella, and Escherichia/Shigella [9–12].

These microbial signatures aligned closely with Th17-dominant inflammation, the central immunologic driver of psoriasis. Several studies and systematic reviews have suggested that decreased SCFA-producing taxa correlate with increased disease severity, while enriched Ruminococcus and Eggerthella are associated with heightened IL-17 and IL-23 signaling [10,11]. Dysbiosis also appears linked to psoriasis-associated comorbidities, particularly metabolic syndrome and inflammatory bowel disease, suggesting shared inflammatory pathways [9].

Several interventional studies evaluating probiotic supplementation—mainly Lactobacillus and Bifidobacterium strains—reported improvements in Psoriasis Area and Severity Index (PASI) scores, reduced systemic inflammatory markers, and modulation of gut microbial composition, though findings varied across strains and study designs [12,15].

Overall, the literature provides robust evidence that gut dysbiosis plays a mechanistic and clinically relevant role in psoriasis pathophysiology.

3.4. Atopic Dermatitis and the Gut Microbiota

Studies of atopic dermatitis (AD) revealed characteristic patterns of early-life and chronic dysbiosis. Reduced colonization by beneficial genera such as Bifidobacterium and Lactobacillus, decreased microbial diversity, and diminished SCFA production were consistently associated with AD onset and severity [13–16]. These alterations may impair immune tolerance, skew immune development toward Th2-dominant responses, and compromise epithelial barrier function.

Systematic reviews found that infants who later developed AD had delayed maturation of gut microbiota, with lower abundance of Bifidobacterium, Faecalibacterium, and Akkermansia, alongside increased Clostridium and Proteobacteria species [13,14]. Dysbiosis in established AD was linked to increased intestinal permeability, systemic immune activation, and reduced production of Treg-inducing metabolites [15,16].

Probiotic interventions demonstrated modest but significant improvements in disease severity in several trials, particularly when using multi-strain preparations containing Lactobacillus rhamnosus, Lactobacillus casei, or Bifidobacterium longum [15,17]. Benefits appeared greater in early-life interventions and in individuals with marked baseline dysbiosis.

These findings suggest that gut microbial composition influences both the development and chronicity of atopic dermatitis, partly through immune modulation and epithelial barrier effects.

3.5. Summary of Gut Microbiota Alterations in Acne Vulgaris, Psoriasis, and Atopic Dermatitis

Dermatologic condition	**Decreased taxa / features (protective)**	**Increased taxa / features (pro-inflammatory)**	**Mechanistic implications**	**Representative sources**
Acne vulgaris	↓ SCFA-producing producing taxa (e.g. Lachnospiraceae, Ruminococcaceae, *Faecalibacterium*) ↓ Microbial diversity	↑ *Ruminococcus gnavus* ↑ *Clostridium sensu stricto* ↑ Oxidative stress-associated taxa	↑ Systemic inflammation ↑ IGF-1 signaling Altered lipid metabolism and sebum regulation Increased intestinal permeability	[5–8]

Psoriasis	↓ *Faecalibacterium prausnitzii* ↓ *Akkermansia muciniphila* ↓ Roseburia ↓ SCFA producers	↑ *Ruminococcus* spp. ↑ *Eggerthella* ↑ *Escherichia/Shigella* ↑ Proteobacteria	Th17-dominant immune activation (IL-17, IL-23) Increased systemic inflammation Possible link to metabolic syndrome & IBD	[9–12]
Atopic dermatitis	↓ *Bifidobacterium* ↓ *Lactobacillus* ↓ *Akkermansia* ↓ Gut microbial diversity ↓ SCFA metabolites	↑ *Clostridium* spp. ↑ Proteobacteria	Th2-skewed immune response Impaired epithelial barrier maturation Increased intestinal permeability Altered immune programming in infancy	[13–17]

4. Discussion

This review synthesizes current evidence demonstrating that intestinal microbiota composition plays a substantial role in the pathogenesis of acne vulgaris, psoriasis, and atopic dermatitis. Across the included studies, a unifying theme emerged: all three conditions are characterized by reduced microbial diversity, depletion of short-chain fatty acid (SCFA)–producing taxa, and an overrepresentation of pro-inflammatory bacterial genera [1–4,9–12]. These alterations converge on systemic immune pathways that can promote or amplify cutaneous inflammation.

The findings support the concept of a multidirectional gut–skin axis, wherein dysbiosis disrupts both metabolic and immunological homeostasis. Reduced SCFA levels—particularly butyrate—may impair regulatory T-cell activity and increase intestinal permeability, facilitating systemic exposure to microbial antigens and inflammatory mediators [2,4]. This mechanism aligns closely with the immune profiles observed in dermatologic diseases: Th17-

dominant inflammation in psoriasis, Th1/Th17 activation in acne, and Th2-skewed responses in atopic dermatitis.

Among the three disorders, psoriasis demonstrates the strongest and most consistent link with intestinal dysbiosis. Multiple studies have shown characteristic microbial signatures—depletion of Faecalibacterium prausnitzii and enrichment of Ruminococcus and Eggerthella—that correlate with disease activity and systemic comorbidities [9–12]. These microbial patterns overlap with those found in inflammatory bowel disease and metabolic syndrome, supporting a systemic inflammatory network with shared pathogenic pathways.

In acne vulgaris, emerging evidence suggests that gut microbiota may influence lipid metabolism, oxidative stress, and IGF-1 signaling, thereby creating a systemic environment conducive to acne lesion formation [5–7]. A recent Mendelian randomization analysis strengthens the hypothesis of a causal relationship between specific microbial taxa or metabolites and acne risk [8]. Although the connection between gut dysbiosis and acne is less extensively studied than in psoriasis, available data suggest clinically meaningful interactions.

In atopic dermatitis, dysbiosis appears particularly relevant during early immune development. Delayed colonization by beneficial genera such as Bifidobacterium and Lactobacillus is associated with increased risk of atopic disease, while reduced SCFA production contributes to impaired epithelial barrier function and heightened Th2 responses [13–16]. These findings highlight the importance of microbiota-mediated immune maturation, particularly in infancy.

Across all three diseases, probiotic interventions have shown potential but inconsistent benefits. Several trials report improvements in inflammatory markers, reductions in disease severity, or modulation of gut microbiota following supplementation with strains of *Lactobacillus* or *Bifidobacterium* [6,7,12,15,17]. However, the variability across studies—differences in strains, dosages, treatment duration, and patient populations—limits the generalizability of results. Strain specificity appears crucial, as not all probiotics exert the same immunomodulatory or metabolic effects. Moreover, sample sizes are often small, follow-up periods short, and objective microbiome assessments limited. Overall, current evidence does not support the routine use of probiotics as monotherapy in these conditions.

Another challenge lies in determining causality. While dysbiosis is consistently associated with these dermatologic diseases, it is unclear whether microbial alterations precede disease onset, arise as a consequence of systemic inflammation, or reflect lifestyle and dietary patterns common among affected individuals. Longitudinal studies and controlled dietary interventions will be necessary to determine the directionality of these relationships.

Despite these limitations, the existing evidence strongly supports the relevance of gut microbiota to dermatologic disease mechanisms. Microbial signatures may offer future value as diagnostic or prognostic biomarkers, while microbiota-modulating therapies—such as targeted probiotics, prebiotics, postbiotics, or dietary strategies—represent promising adjunctive treatments. Advances in metagenomic sequencing and metabolomic profiling are likely to clarify microbial contributions and support development of precision therapies tailored to individual dysbiosis patterns.

In summary, current data suggest that gut dysbiosis contributes to immune dysregulation, barrier dysfunction, and systemic inflammatory signaling that can exacerbate or sustain acne vulgaris, psoriasis, and atopic dermatitis. Integrating microbiota-focused approaches into dermatologic care may enhance treatment outcomes, although further high-quality trials are required to establish standardized recommendations.

5. Conclusions

Accumulating evidence demonstrates that intestinal dysbiosis contributes to the pathogenesis of acne vulgaris, psoriasis, and atopic dermatitis through shared immunologic and metabolic pathways. Reduced microbial diversity, depletion of short-chain fatty acid–producing genera, and enrichment of pro-inflammatory species have been consistently reported across these conditions [1–4,9–12]. These alterations can disrupt immune tolerance, promote Th1-, Th2-, or Th17-driven inflammation, and impair epithelial barrier integrity, ultimately influencing cutaneous disease expression [2,4,10,13–16].

Psoriasis shows the most robust association with gut microbial disturbances, characterized by reproducible dysbiosis patterns linked to disease severity and systemic comorbidities [9–12]. Acne and atopic dermatitis also exhibit significant microbiota-related alterations, supported by mechanistic studies and clinical observations [5–8,13–16]. Emerging clinical trials indicate that targeted probiotic supplementation may offer therapeutic benefit, although effects appear strain-dependent and heterogenous across studies [6,7,12,15,17].

Overall, the gut–skin axis represents a promising field for adjunctive therapeutic strategies. Future research should prioritize standardized probiotic formulations, longitudinal microbiome profiling, and mechanistic studies capable of establishing clearer causal relationships.

Disclosure

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