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The Gut–Skin Axis in Autoimmune Dermatology: Role of Microbiome and Immune Pathways

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Abstract: The gut-skin axis has become an important conceptual framework to identify a relationship between gut microbiota and skin immunity, particularly in autoimmune dermatologic diseases. This narrative review aims to summarize recent data examining how gut dysbiosis, characterized by low microbial diversity and disruption of each of the gut's microbial communities, plays a role in the pathogenesis and pathophysiology of psoriasis, systemic lupus erythematosus (SLE), alopecia areata, and scleroderma. We consider different pathways, including but not limited to increased intestinal permeability ("leaky gut"), Th17/Treg balance, the presence of and/or development of pro-inflammatory cytokines, and molecular mimicry, for how gut dysbiosis drives immune dysregulation in the skin. Clinical and translational evidence of microbiome-based therapies with probiotics, prebiotics, and fecal microbiota transplantation (FMT) has been shown not only to improve disease severity but also reduce systemic inflammation and restore gut microbiome diversity and community structure. Specific AMR therapies have indicated differences in PASI, SLEDAI, and inflammatory markers such as IL-6 and TNF-α. This narrative review strongly affirms that ambient need for more precision-based solutions to prevent and treat autoimmunity using a combination of multi-omics, gut microbiome profiling, as well as artificial intelligence (AI)-driven individualized solutions. Ultimately, the gut–skin axis depicts autoimmune skin diseases from single dermatologic disease classifications to systemic immune diseases grounded in gut health, offering new possibilities for diagnosis, intervention, and disease modulation.

Keywords*: Gut Microbiota; Gut Skin Axis, Immune Modulation; Intestinal Barrier Dysfunction; Psoriasis; SLE; Alopecia Areata; Scleroderma.*

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I. INTRODUCTION

The concept of gut–skin axis, which relates gut microbiome with skin health, has garnered remarkable interest amongst researchers. When the relationship between the gut microbiome and the immune system is impaired, several effects can be triggered on the skin, potentially promoting the development of skin diseases. There is an increase in active investigations regarding the relationship between the gut microbiome and skin diseases, including psoriasis and other autoimmune diseases. This leads us to the concept of gut–skin axis, which associates the microbiome and skin diseases via the intestinal barrier, inflammatory mediators, and metabolites [1]. The gut and skin-associated diseases such as psoriasis, atopic dermatitis, acne vulgaris, rosacea, alopecia areata, and hidradenitis suppurativa [2].

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The skin immune system is made up of recruited and resident innate immune system (IIS) and adaptive immune system (AIS) cells, which are activated by microorganisms, stimuli, and epidermal structures that crosstalk with mostly KCs to restore the skin barrier [3][4]. Studies have shown the bidirectionality between the intestinal microbiota and skin homeostasis, a communication mainly established by modifying the immune system, with increased data on the mechanisms of action regarding the relevance of *Cutibacterium acnes* [5]. Studies have proposed that the mechanism of the gut–skin axis in regards to psoriasis involves T cell function and differentiation, with the imbalance of Treg and Th17 cells [6,7]. Like the skin microbiota, the composition of the gut microbiota and its association with psoriasis are unclear. Conversely, psoriatic exacerbation was deemed to be associated with increased colonization of Staphylococcus aureus, Candida albicans, and Malassezia in the skin and gut [8]. An imbalanced gut microbiome, a pathological state named intestinal dysbiosis, harms skin function and integrity [9,10].

II. METHODS

The current narrative review analyzes the interaction between immune-system modulation, cutaneous barrier failure, and the gut microbiota. The main aim was to assess the role of changes in the gut bacterial milieu that can impact the development or clinical course of autoimmune skin diseases. A thorough literature search was conducted across PubMed, Scopus, and Google Scholar, with a primary focus on high-quality, peer-reviewed articles published between 2019 and 2025. A few foundational studies from 2008 to 2015 were also included, which provided essential mechanistic or conceptual insights. Diverse keywords and Medical Subject Headings terms were utilized, such as “gut–skin axis”, “gut microbiota”, “immune modulation”, “autoimmune skin diseases”, “intestinal dysbiosis”, and “cutaneous barrier dysfunction”. Studies related to “intestinal barrier activity”, “microbiota”, and “immunological processes in skin autoimmune diseases” were prioritized. Articles were excluded in the form of case reports, non-peer-reviewed articles, and workshop abstracts to uphold scientific rigor. After a thorough analysis, the outcome data were grouped into four main thematic areas: intestinal barrier dysfunction, immunomodulated skin reactions, gastrointestinal dysbiosis, and clinical implications in dermatological practice. Since the narrative review approach did not require quantitative analysis, a formal potential bias assessment was not considered.

III. GUT DYSBIOSIS AND IMMUNE REGULATION

It is worth noting that each human organism acquires its unique composition of intestinal microflora. Once established, the set of microorganisms comprising the gut microbiome is relatively constant throughout life. Certain factors can harm the profile of the gut microbiome, leading to dysbiosis. The gut consists of trillions of microbial communities, being recognized as a virtual organ closely associated with the health and longevity of the host. The microbiome helps in the degradation of toxins and drugs and the biosynthesis of vitamins [11]. An imbalance of composition and biodiversity of the gut microbes, or the term “gut dysbiosis,” has been associated with psoriasis and many other psoriasis-associated comorbidities such as inflammatory arthritis, chronic kidney disease, inflammatory bowel disease, metabolic syndrome, cardiovascular disease, depression, and obesity [12-15]. Emerging therapeutic strategies targeting the microbiome, which include probiotics, fecal microbiota transplantation (FMT), dietary interventions, and pharmacological modulators, show promise in restoring microbial balance [16]. Gut dysbiosis also causes dysfunction of the gut-liver axis *via* alteration of the bile acid metabolism pathway [17]. Also, an interesting research review recently published in the Journal of Digestive Diseases and Sciences conceivably linked adjunctive commensal intestinal bacteria with the capacity to modulate the immune microenvironment towards immune checkpoint inhibitor (ICIs) efficacy of cancer immunotherapy [18]. However, microbiota dysbiosis can lead to dysregulation of bodily functions and diseases, including cardiovascular diseases (CVDs), cancers, respiratory diseases, etc. [19]. The metabolic and immune potential of the gut microbiome determines its significance in host health and diseases. Therefore, targeting the gut microbiome and relevant metabolic pathways would be an effective treatment for many metabolic diseases in the near future [20]. However, integrating all these observations is necessary to develop precise diagnostics and may indicate the necessity of more individually designed therapies that are based on age and the microbiome. We must acknowledge that we still need to perform more work in addition to finding simple associations. In addition, new technologies should be applied to investigate and manipulate the microbiome to precisely intervene in a specific microbiome.

IV. GUT DYSBIOSIS IN SYSTEMIC AUTOIMMUNITY

- *Gastrointestinal Dysbiosis in Major Autoimmune Diseases*

Changes in the biodiversity of microbes have been documented in a large number of RA-focused clinical investigations. Patients with RA (Rheumatoid Arthritis) also had considerably higher abundances of the species *Oscillospira, Flavonifractor, Bacteroides, Parasutterella, Escherichia, Shigella, Eubacterium, Tyzzerella, Bacteroides*, as well as *Sellimonas*. According to several studies, MS (Multiple Sclerosis) patients' gut microbiota distribution differs from that of physically fit people. According to a sequencing study, MS patients have higher levels of Bacteroidetes with Proteobacteria and lower levels of Firmicutes [21]. In those with MS, *Prevotella*, along with *Faecalibacterium*, are decreased, but *Akkermansia, Clostridium, Actinomyces, Streptococcus*, and *Eggerthella* are abundant [22]. Systemic lupus erythematosus (SLE) is associated with decreased microbial biodiversity that is enriched in *Streptococcus, Veillonella*, and *Campylobacter* but reduced in *Lactobacillus* and *Bifidobacterium* [23]. Mice with SLE develop lupus as a result of gastrointestinal microbiota translocation, with Enterococcus *gallinarum*

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migration associated with inflammatory and barrier failure [24]. In experimental models with lupus, *Lachnospiraceae* greatly increased, whereas *Lactobacillaceae* notably decreased [25]. Gastrointestinal disease is seen in both MS patients and mice that are models of experimental autoimmune encephalomyelitis (EAE) [26]. A comparison of gut microbiome alterations across major autoimmune diseases is summarized in Table 1.

Table 1 Comparison of Gut Microbiome Alterations Across Autoimmune Diseases

Disease	Increased Microbes	Decreased Microbes	Clinical Implication	REFERENCES
RA	*Bacteroides, Escherichia-Shigella*	*Prevotella*	↑ Pro-inflammatory cytokines	[21], [22], [28]
SLE	*Streptococcus, Veillonella*	*Lactobacillus, Bifidobacterium*	↑ Systemic inflammation, cutaneous flares	[23],[24],[30]
MS	*Bacteroidetes, Akkermansia*	*Faecalibacterium*	Altered immune regulation	[21],[31]
Psoriasis	*Pro-inflammatory bacteria*	*Faecalibacterium prausnitzii*	Aggravation of IL-23/IL-17 pathway	[32],[34],[35]

➢ *The Immune Pathways and Common Dysbiosis Patterns*

Additionally, dysbiosis encourages the growth of Th17(T Helper 17) cells, which accelerates the course of SLE [26]. *Prevotella copri* stimulates the development of Th17 and the production of IL-23(Interleukin 23) and IL-1(Interleukin 1), hence promoting inflammation of the mucous membrane through TLR2(Toll-Like Receptor 2) [23]. Th17 suppression and Treg (Regulatory T Cells) stimulation are two ways that *Prevotella histicola* protects against arthritis [24]. Th17 cells, as well as IL-17 (interleukin 17), are elevated in immune-mediated illnesses.[27]. Recent research suggests that decreased SCFA (Short-Chain Fatty Acids) synthesis and intestinal dysbiosis are important common pathways in systemic autoimmune disorders [28,29]. It has been discovered that there is an imbalance between the gut flora and Treg cells and Th17 levels in people with SLE [30]. Th17 cell expansion in the lining of the gut is promoted by segmented filamentous microbes. Propionate and other SCFAs increase Treg activity and inhibit Th17 development [31]. When rats were colonized by segmented filamentous bacteria, their renal failure worsened [25].

V. GUT MICROBIOME IN AUTOIMMUNE SKIN DISEASES

➢ *Psoriasis.*

Psoriasis is a disorder characterized by immune dysregulation acting through the IL-23/IL-17 pathway, being chronic and autoimmune. The presentation includes scaly skin plaques. Reduced bacterial diversity was observed in psoriatic arthritis (often associated with psoriasis), along with decreased population levels of *Akkermansia muciniphila*, a bacterium associated with good gut barrier health [32].

For 12 weeks, a study was conducted with a focus on evaluating the impact of probiotics and precision prebiotics on patients with psoriasis receiving topical therapy. The interventions were *Bacillus indicus (HU36), Bacillus subtilis (HU58), Bacillus coagulans (SC208), Bacillus licheniformis (SL307), Bacillus clausii (SC109),* a set of Probiotics, and the prebiotics included *Fructooligosaccharides, xylooligosaccharides, and galactooligosaccharides.* In a group of 63, 42 were intervention group (probiotics + prebiotics + topical therapy), and 21 were control group (topical therapy only). The patients were assigned in a non-randomized 2:1 ratio. The 42 patients showed improved disease activity, measured by PASI, DLQI, Inflammatory markers, and skin thickness. The gut microbiome of 15 out of 42 revealed the gut profile shifted to a less inflammatory state after supplementation. The outcomes show that prebiotic and probiotic supplementation alongside standard therapy improved the psoriasis severity [33].

Butyrate-producing, anti-inflammatory *Faecalibacterium prausnitzii* populations are reduced in patients with psoriasis, which should augment inflammation and worsen skin symptoms [34].

Gut-derived Short-chain Fatty Acids are mainly responsible for immune-modulatory activities. Regulation of T-cell function and suppression of IL-17 production by short-chain fatty acids indicate that impaired microbial alterations affect SCFA production, thus aggravating the disease. Interventions to enhance SCFAs may thus add therapeutic value for restoring gut-skin axis health [35].

➢ *SLE / Systemic Lupus Erythematosus*

The presence of cutaneous manifestations like the rash is often linked to gut dysbiosis in systemic lupus erythematosus. The Firmicutes/Bacteroidetes ratio in SLE patients is altered due to a decrease in butyrate-producing bacteria that could promote systemic inflammation affecting the skin [36].

Since gut microbiota dysbiosis is also involved in the development of SLE, a pilot clinical trial investigation for the safety and efficacy of fecal microbiota transplant (FMT) using oral encapsulated fecal microbiome from healthy donors (n=20) with active SLE (SLEDAI ≥ 6). FMT was administered for 3 weeks alongside standard treatment with a 12 week follow up found that on 12th week, 42.12% of patients achieved a SLE responder index 4, the primary end point, significant reduction in SLEDAI-2K (Systemic Lupus Erythematosus Disease

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Activity Index 2000) scores, reduced inflammation, elevated gut SCFA levels, and decreased serum IL-6 and CD4+ memory/ naïve T cell ratios. Patients who responded to FMT (SRI-4 responder) showed a distinct gut microbiome profile before and after. This provides preliminary evidence showcasing the Gut and its connection to immunity [37].

Diminished gut microbial diversity combined with decreases in SCFA production permits a pro-inflammatory state counterproductive to alleviating worsening inflammatory skin manifestations in cases of autoimmune diseases [38].

- *Alopecia Areata*

Autoimmune disorder alopecia areata reveals substantial alteration of gut microbiota. Reduced populations of butyrate-producing bacteria and increased populations of Proteobacteria in patients may indicate the immune-mediated destruction of hair follicles associated with dysbiosis [39].

In a study, investigations were done among the bacterial communities of people from communities of healthy scalp and those diagnosed with alopecia areata. The samples were collected from healthy individuals and AA patients using a standardized swabbing technique. Genomic DNA from the samples was extracted, and 16S rRNA sequencing of the bacterial profile was obtained. The findings revealed a significant increase in Propionibacterium in AA subjects compared to the healthy ones and a decrease in Staphylococcus epidermis. These results show a microbial shift in the scalp microenvironment of AA patients [40].

- *Scleroderma*

Indeed, the scleroderma subtype with skin fibrosis is correlated to alteration of the gut. Reduced microbial diversity, alongside decreased *Faecalibacterium* and increased *Fusobacterium* population in scleroderma patients, might foster inflammatory and fibrotic complications in the skin and other tissues [41]. Key gut microbiome alterations observed in various autoimmune skin diseases are summarized in Table 2.

In addition to autoimmune dermatoses, hypersensitivity responses such as contact dermatitis due to mango can also demonstrate exaggerated cutaneous immune responses to dietary or environmental antigens [42]. These examples should underscore the greater relevance of antigen exposure considerations and cross-reactivity to cutaneous immune responses

Table 2 Gut Microbiome Changes in Autoimmune Skin Diseases

Skin Disease	Microbial Alteration	Key Finding	References
Psoriasis	↓ *Faecalibacterium prausnitzii*; shift towards pro-inflammatory profile	Pro-/prebiotic trial: improved PASI, DLQI, and inflammatory markers over 12 weeks (n=42 vs 21)	[33],[34]
Cutaneous SLE	↓ Butyrate-producers (Firmicutes/Bacteroidetes ratio)	FMT pilot: 42% SRI-4 response, ↓ SLEDAI-2K, ↑ SCFAs, ↓ IL-6 over 12 weeks (n=20)	[37]
Alopecia areata	↓ butyrate-producers; ↑ Proteobacteria; ↑ Propionibacterium; ↓ Staph. epidermidis	16S rRNA: significant *Propionibacterium* increase in AA patients	[40]
Scleroderma	↓ diversity; ↓ *Faecalibacterium;* ↑ *Fusobacterium*	Correlated with skin fibrosis severity	[41]

VI. MECHANICAL LINKS

The gut-skin axis refers to the ability of the intestinal microbiome to influence skin health through various immune pathways. One of the main mechanisms occurs when the intestinal permeability increases, which is known as “leaky gut”. Once the gut becomes weakened, bacterial components such as lipopolysaccharides (LPS) and flagellin can enter the bloodstream. The components then interact with Toll-like receptors (TLRs), triggering the release of cytokines (TNF-α, IL-6, and IL-1β). This inflammatory reaction can worsen skin conditions like psoriasis and cutaneous lupus [43,44].

Another mechanism that occurs is molecular mimicry. This is when the gut microbes are able to mimic antigens found in skin proteins. In the gut-associated lymphoid tissue (GALT), these antigens can activate naïve T-cells. This may lead to autoimmune inflammation due to cross-reactions with skin tissues [45].

The gut plays a large role in how the immune system responds to the skin. When naïve T-cells come into contact with bacteria and dietary antigens, they develop the ability to move towards the skin by producing markers such as CCR4, CCR10, and CLA [46]. Stimulated T-cells release cytokines such as IL-17 and IL-22. In conditions such as psoriasis, this can result in persistent inflammation and skin cell proliferation [47].

Short-chain fatty acids (SCFAs) such as butyrate and propionate are also produced by the gut bacteria. These aid in the development of regulatory T-cells and also dampen Th17-mediated inflammation. SCFAs also support the maintenance of gut barrier integrity and enhance anti-inflammatory signals like IL-10 [48,49]. During dysbiosis, reduced SCFAs result in weakened barrier defenses, resulting in increased

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inflammation. These key mechanistic pathways linking gut microbiota to skin health are summarized in Table 3.

These processes combined demonstrate how autoimmune skin conditions can become aggravated by imbalances in the gut microbiota.

Table 3: Mechanistic Links in the Gut–Skin Axis

Mechanism	Description	Mediators	References
Increased intestinal permeability ("leaky gut")	Translocation of LPS/flagellin → TLR activation → systemic cytokine release	TNF-α, IL-6, IL-1β	[43], [44]
Molecular mimicry	Gut antigens mimic skin proteins → naïve T-cell activation in GALT → cross-reactive skin inflammation	CCR4, CCR10, CLA, IL-17, IL-22	[45]
SCFA deficiency	↓ butyrate/propionate → impaired Treg induction, unchecked Th17 expansion → loss of gut barrier integrity → inflammation	IL-10, Treg/Th17 balance	[48], [49]

VII. THERAPEUTIC LINKS

Researchers have identified that a crucial aspect of managing autoimmune skin conditions is targeting the gut microbiome. Multiple methods have been identified, including the use of fecal microbiota transplantation (FMT) and biotic supplementation. These methods have demonstrated the ability to restore microbial balance and reduce systemic immune activation [50]. Key therapeutic interventions targeting the gut–skin axis and their outcomes in autoimmune skin diseases are summarized in Table 4.

A randomized controlled trial by Liu et al. (2025) investigated the impact of fecal microbiota transplantation (FMT) on disease severity in adults with atopic dermatitis. Sixty participants were enrolled, and EASI scores were assessed over eight weeks. One the study concluded it was found that there was a 57% reduction in the FMT group and a 28% reduction in the control group ($p=0.013$). As well as EASI scores, levels of IL-4 and IL-13 also decreased significantly ($p<0.05$). This indicated reduced inflammation and symptom improvement [51].

Researchers have also started using prebiotic and probiotic supplements to help the gut immune system. Prebiotics work by acting as food for good gut bacteria. Such strains of bacteria include *Lactobacillus rhamnosus* and *Lactobacillus plantarum*. The main function of these is to support and strengthen gut barrier function, along with aiding immune regulation. Prebiotics also increase production of SCFAs, which reduce systemic inflammation, improving skin symptoms [52].

Probiotics, on the other hand, directly increase the number of beneficial bacteria in the gut. They aid in restoring the microbial balance seen in dysbiosis. They also reinforce the intestinal lining to stop the seepage of harmful microbes into circulation. In autoimmune skin conditions, probiotics lower the levels of cytokines such as IL-6 and TNF-α, calming overactive immune responses [53].

Despite current evidence being limited, early findings have shown promise. Whilst long-term efficacy and safety need to be researched further, therapies targeting the gut may serve as a vital addition to the management of autoimmune skin conditions.

The gut-skin axis has been studied through microbiome and immunologic studies, though future studies will likely involve artificial intelligence (AI) to predict and stratify disease based on multimodal imaging and molecular signatures. AI has changed the landscape of cardiovascular imaging through automated interpretation, risk prediction, and increased diagnostic accuracy. Similar frameworks could be able to be designed for skin imaging and immune phenotyping in the field of autoimmune dermatology.[54]

Table 4: Interventions Targeting the Gut-Skin Axis

Therapy	Autoimmune Skin Disease	Key Outcomes	References
Probiotics	Psoriasis	↓ PASI, ↓ IL-6, improved gut profile	[33], [35]
FMT	SLE	42% SRI-4 response, ↑ SCFA, ↓ inflammation	[37], [40]
Prebiotics	Atopic Dermatitis	↑ Lactobacillus spp., improved EASI scores	[51], [52]
Synbiotics (Pro+Pre)	Alopecia Areata	Early signs of hair regrowth, modulation of microbiome	[39], [41]

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VIII. CONCLUSION AND FUTURE DIRECTIONS

The complex interaction between the gut microbiome and the skin is no longer hypothetical. It is an emerging paradigm that will change the way we approach autoimmune dermatologic diseases. From psoriasis to lupus erythematosus, it is becoming increasingly clear that gut microbial dysbiosis can regulate systemic immunity that manifests as skin inflammation and immune hyperreactivity. This gut-skin axis, likely via immune pathways, cytokines, and microbial metabolites, can provide a new way to define and understand disease that transcends organ systems. As we have detailed in this review, the gut microbiome can direct Th-cell differentiation, influence inflammatory signaling, and affect skin barrier regulation. However, we are still in infancy in this area. Most studies are observational studies with small sample sizes or lack mechanistic approaches. Importantly, defining what is "healthy" for the microbiome is context-dependent and likely varies with the individual, making it difficult to derive standardized interventions therapeutically. Moving forward, an individual approach is needed. Multi-omics integration (metagenomics, metabolomics, transcriptomics), coupled with machine learning, could lead to individual microbiome-based interventions. Probiotics, prebiotics, and more recently fecal microbiota transplantation (FMT) interventions are of value, but they will only be useful with better patient stratification and more mechanistic insight and understanding. Finally, artificial intelligence, as is demonstrated in cardiovascular imaging, represents a powerful approach to examine

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- *Authors' Contributions:*
  S.S. conceptualized the study, designed the structure, coordinated the literature review process, and edited the full manuscript.

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