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REVIEW ARTICLE

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Gut microbiota-skin axis in the treatment of psoriasis and atopic dermatitis with the promotion of gut health: a systematic review

Tania Parra Vazquez[1,2,*], Elias Naim Kassis[2]

[1] IESS - Ecuadorian Social Security Institute, Type B Health Center Cañar, Ecuador.
[2] University Center of Northern São Paulo, Dermatology department, São José do Rio Preto, São Paulo, Brazil.

*Corresponding author: Tania Parra Vazquez.
IESS - Ecuadorian Social Security Institute, Type B Health Center Cañar, Ecuador.
E-mail: tanyparra_abril@hotmail.com
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Abstract

Introduction: Psoriasis is a chronic inflammatory dermatological condition that affects approximately 60 million individuals worldwide, with recurrence rates between 0.1% and 1.5% after conventional treatments. Atopic dermatitis (AD) is a prevalent chronic inflammatory skin disease associated with genetic and environmental factors, characterized primarily by an allergic immune response to environmental antigens with increased levels of immunoglobulin E (IgE). The gut microbiota is essential for immune system activation and the treatment of these diseases. **Objective:** To investigate the gut microbiota-skin axis in the treatment of atopic dermatitis and psoriasis through the promotion of gut health. **Methods:** The PRISMA Platform systematic review rules were followed. The search was carried out from July to August 2025 in the Scopus, Embase, PubMed, Science Direct, Scielo, and Google Scholar databases. Study quality was based on the GRADE instrument, and the risk of bias was analyzed according to the Cochrane instrument. **Results and Conclusion:** A total of 131 articles were found, and 28 articles were evaluated in full, and 17 were included and developed in the present systematic review study. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 24 studies with a high risk of bias and 25 studies that did not meet GRADE and AMSTAR-2. Most studies showed homogeneity in their results, with X^2=77.6 %>50%. It was concluded that the gut microbiota has enormous metabolic capacity along the gut-skin axis. Dietary or microbiota metabolites are accessible to the skin. Therefore, after defining key open questions about the nature of these metabolites, their detection, and the skin changes they can induce, understanding these pathways will lead to new therapeutic strategies that target one organ to improve the health of another. Probiotics and prebiotics are microbiota management tools for treating psoriasis and atopic dermatitis.

Keywords: Skin. Psoriasis. Atopic dermatitis. Gut microbiota. Treatment. Gut healthy.

Introduction

Psoriasis is a chronic inflammatory dermatological condition affecting approximately 60 million individuals worldwide, with recurrence rates ranging from 0.1% to 1.5% after conventional treatments **[1]**. The disease is characterized by rapid keratinocyte proliferation and aberrant differentiation, clinically manifesting as erythematous plaques covered by silvery-white scales, with pruritus intensity varying based on the severity and location of the lesion. Microscopic examination shows evidence of microvascular bleeding and significant infiltration of inflammatory cells, highlighting an inflammatory process in the deeper layers of the skin **[1,2]**.

In psoriasis, T cells are overactivated, leading to the abundant release of pro-inflammatory cytokines, thus perpetuating the dermal inflammatory environment **[1]**. Furthermore, there is an association between psoriasis and obesity and hypertension. Disease onset is characterized by the activation of

plasmacytoid dendritic cells through antimicrobial peptides, which activate Toll-like receptors (TLRs) and facilitate antigen presentation to CD8+ T cells. This cascade triggers the production of interferons α and β, leading myeloid dendritic cells to secrete a suite of inflammatory cytokines, including IL-12, IL-23, and tumor necrosis factor, which are key to Th cell differentiation, particularly promoting the emergence of Th1, Th17, and Th22 cells. Within the pro-inflammatory environment of psoriasis, Th1 cells further amplify the inflammatory cycle, stimulating myeloid dendritic cells to release tumor necrosis factor and interferon γ, thus maintaining a positive feedback loop in the inflammatory response and worsening disease severity **[3-6]**.

Infectious agents also contribute significantly to the pathogenesis of psoriasis. Streptococcal infections have been implicated in the etiology of guttate psoriasis, with persistent or recurrent infections increasing the risk of disease flare-ups. Furthermore, colonization by pathogens such as *Staphylococcus aureus* and *Candida albicans* can exacerbate psoriasis. Environmental and lifestyle factors such as air pollution, ultraviolet radiation exposure, smoking, alcohol consumption, and dietary patterns have been identified as factors that influence the manifestation and severity of the disease **[5,6]**.

Atopic dermatitis (AD) is a prevalent chronic inflammatory skin disease associated with genetic and environmental factors, characterized primarily by an allergic immune response to environmental antigens with increased levels of immunoglobulin E (IgE) in the bloodstream. A notable feature of AD pathogenesis is the reduced production of filaggrin, which is critical for skin barrier integrity, allowing allergens to penetrate and activate immune responses that further weaken the epithelial barrier and potentially lead to intestinal permeability **[1,5,6]**.

In this context, the normal human skin microbiome was found to exhibit high diversity and high interpersonal variation. The lesional skin microbiota compositions in AD and psoriasis showed distinct differences compared to healthy skin **[1-4]**. The role of microbial colonization in establishing immune system homeostasis has been reported, while host-microbe interactions and genetic variation may be linked to skin dysbiosis. Both are relevant to skin disorders with aberrant immune responses and/or disturbed skin barrier function. Modulating the skin microbiota composition to restore host microbiota homeostasis may be a future strategy for treating or preventing the disease **[7-9]**.

In the human microbiota, a symbiotic relationship exists between the human body and microorganisms **[4,5]**. The adult body sustains a healthy community of microorganisms, including bacteria, viruses, and fungi, as well as the genetic elements that constitute the human microbiota, where all these microorganisms, both beneficial and potentially pathogenic, coexist **[10]**.

Microorganisms perform important functions such as preserving and promoting the development of immune defenses, exerting considerable influence on a series of host biochemical reactions, such as the transformation of dietary fiber into simple sugars, the transformation of short-chain fatty acids and other nutrients for absorption, the production of vitamin K, vitamin B12, and folic acid, participation in the metabolism and recirculation of bile acids, the transformation of potential carcinogens, and the activation of bioactive compounds **[6,7]**. An imbalance in the gut microbiota can promote the onset and progression of human diseases **[10]**.

Thus, the presence of bacteria in the intestine is mandatory for the development of various gut microbiota functions. Moreover, the gut microbiota is essential for activating the immune system, particularly *Lactobacillus acidophilus*, *Lactobacillus bulgaricus*, and *Lactobacillus casei*, increasing IgA for antigen removal through a non-inflammatory pathway and increasing T and B lymphocytes. In other words, in the absence of gut microbiota, intestinal motor function is compromised **[10]**. Lactobacilli and Bifidobacteria inhibit the growth of exogenous and/or harmful bacteria, stimulate immune functions, aid in the digestion and/or absorption of food ingredients and minerals, and contribute to vitamin synthesis **[11]**.

In this sense, one of the first skin diseases in which the positive influence of probiotic use was observed was AD. Recent studies have shown that concomitant use of probiotics with specific treatment in atopic individuals helps reduce disease flare-ups **[7]**. Two other diseases have received some research regarding the use of probiotics: inflammatory acne and psoriasis. In the former, bacteria are very important, and when lesions worsen, an imbalance in the microbiota has been detected. In psoriasis, the use of probiotics appears to help by reducing skin inflammation **[11]**.

Thus, the present study investigated the gut microbiota-skin axis in the treatment of atopic dermatitis and psoriasis by promoting gut health.

Methods

Study Design

This study followed the international systematic

review model, following the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) guidelines. Available at:http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1. Accessed on: September 12, 2025. The AMSTAR-2 (Assessing the Methodological Quality of Systematic Reviews) methodological quality standards were also followed. Available at: https://amstar.ca/. Accessed on: September 12, 2025.

Data Sources and Search Strategy

The literature search process was conducted from July to August 2025 and was based on Scopus, Embase, PubMed, Lilacs, Ebsco, Scielo, and Google Scholar, covering scientific articles from various periods to the present day. The following descriptors (DeCS/MeSH Terms) were used: "*Skin. Psoriasis. Atopic dermatitis. Gut microbiota. Treatment. Gut healthy*" and the Boolean expression "and" was used between MeSH terms and "or" between historical findings.

Study Quality and Risk of Bias

Quality was classified as high, moderate, low, or very low based on the risk of bias, clarity of comparisons, precision, and consistency of analyses. The most prominent factors were systematic reviews or meta-analyses of randomized controlled trials, followed by randomized clinical trials. Low-quality evidence was attributed to case reports, editorials, and brief communications, according to the GRADE instrument. Risk of bias was analyzed according to the Cochrane instrument by analyzing the funnel plot (sample size versus effect size), using Cohen's d test.

Results and Discussion

Summary of Findings

A total of 131 articles were found and submitted for eligibility analysis, with 17 final studies selected to comprise the results of this systematic review. The selected studies were of medium to high quality (Figure 1), considering the level of scientific evidence from studies such as meta-analysis, consensus, randomized clinical trials, prospective, and observational studies. Biases did not compromise the scientific basis of the studies. According to the GRADE instrument, most studies presented homogeneity in their results, with X^2 = 77.6% > 50%. Using the Cochrane risk of bias tool, the overall assessment resulted in 24 studies with a high risk of bias and 25 studies that did not meet the GRADE and AMSTAR-2 criteria.

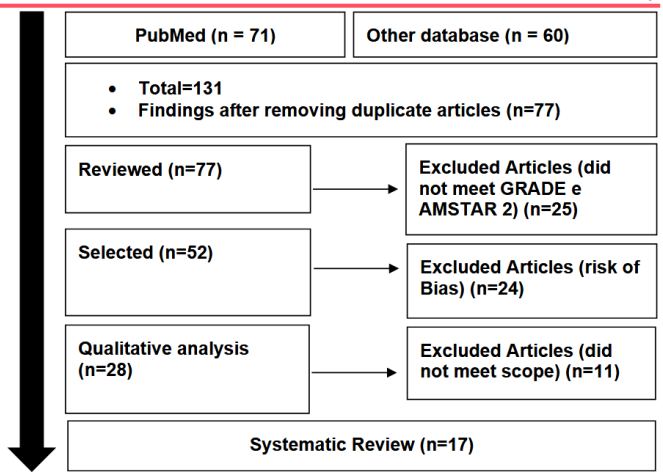

Figure 1. Flowchart showing the article selection process. Source: Own authorship.

Figure 2 presents the results of the risk of bias of the studies using the funnel plot, showing the calculation of the effect size (magnitude of the difference) using Cohen's d test. Precision (sample size) was determined indirectly by the inverse of the standard error (1/Standard Error). This graph displayed symmetrical behavior, suggesting no significant risk of bias, either among studies with small sample sizes (lower precision), shown at the bottom of the graph, or among studies with large sample sizes, shown at the top.

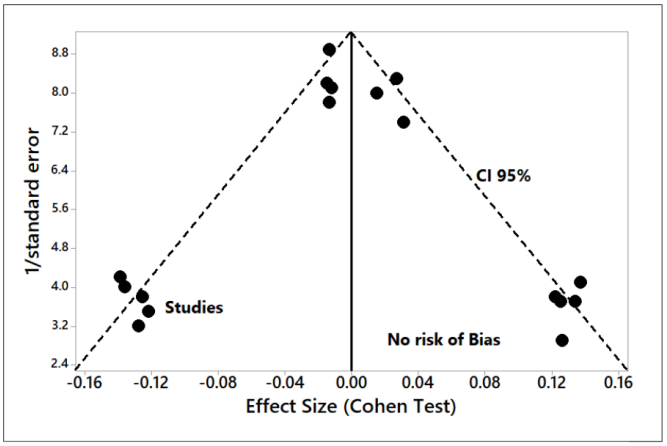

Figure 2. The symmetrical funnel plot suggests no risk of bias among the studies with small sample sizes, shown at the bottom of the graph. High-confidence and highrecommendation studies are shown above the graph (n=17 studies). Source: Own authorship.

Major Outcomes and Considerations

In the context of psoriasis and atopic dermatitis (AD), the gut microbiota interacts with the host primarily through structural components and metabolic outcomes. Specific gut bacteria, such as Clostridia species, are critical for promoting the development of regulatory T (Treg) cells, thus maintaining immune

homeostasis by balancing the activities of Th1, Th2, and Th17 cells. Gut microbiota dysbiosis, characterized by a decrease in short-chain fatty acid (SCFA)-producing bacteria, plays a key role in the development of AD **[1-3]**.

In this regard, colonization by *Staphylococcus aureus* is closely associated with the enhancement of several metabolic pathways in psoriasis, including the tricarboxylic acid (TCA) cycle, tryptophan metabolism, butyric acid metabolism, and other amino acid metabolic pathways. S. aureus can regulate energy homeostasis by participating in the TCA cycle **[6]**.

A healthy gut microbiota can protect the skin, while an imbalanced gut microbiota can exacerbate psoriatic symptoms. Increasing evidence suggests that gut dysbiosis can trigger T cell differentiation, with overactivation of Treg/Th17 cells regulating the immune response by releasing various inflammatory factors (such as IL-23, IL-17A, IL-22, IL-6, and IFN-α), thus promoting the progression of psoriasis and AD **[5,6]**.

Bacteroides fragilis and segmented filamentous bacteria have also been shown to positively impact the maturation of regulatory T cells (Tregs) and Th17 cells, increasing the host's ability to fight infections and overall immune stability. Chronic inflammation is a crucial factor in the development of autoimmune diseases **[1-3]**. Specifically, pathological T cells residing in the skin of psoriasis patients produce excess IL-17 in response to IL-23, triggering the production of pro-inflammatory mediators IL-1β, IL-6, IL-8, TNF-α, and keratinocyte chemoattractants. These signaling molecules sustain chronic skin inflammation and cause epidermal hyperplasia, the hallmark of psoriatic plaques. In this context, psoriasis patients who consumed *B. infantis* 35624 experienced a decrease in the pro-inflammatory markers IL-6, and TNF-α. These effects can be attributed to probiotic-induced Treg proliferation **[12]**.

The interplay of hormonal, neuronal, and inflammatory signaling has a significant impact on skin health **[4-6]**. Psychological distress alters skin physiology, stimulating pro-inflammatory responses. Indeed, acne, a common skin condition among adolescents and young adults, is correlated with neurogenic skin inflammation, which alters mast cell function and survival and induces the production of vasodilatory and pro-inflammatory factors. Psychological stress upregulates prolactin secretion, which in turn determines keratinocyte proliferation and sebum production by the sebaceous glands. Similarly, the onset of autoimmune skin diseases, such as psoriasis, and allergic disorders, such as AD, is correlated with chronic inflammation and mast cell degranulation **[12,13]**.

Probiotic bacteria with anti-inflammatory properties have the potential to bring therapeutic benefits to people suffering from neurogenic skin inflammation or autoimmune skin diseases **[7]**. However, more clinical evidence is needed to support their routine use in medical practice. Similarly, probiotics that protect keratinocytes from oxidative stress or induce skin re-epithelialization may be invaluable for non-healing wounds **[11]**.

Also, AD is the result of an imbalance in the Th1/Th2 leukocyte population, leading to excessive mast cell degranulation and a Th2-mediated allergic response. Phenotypically, this translates into skin erythema, hemorrhage, and itching, which can be triggered by genetic and environmental factors. Most studies on the alleviation of AD focus on two parameters: inflammation and the composition of the gut and skin microbiota **[8]**. Probiotic supplementation has been shown to shift T cell differentiation toward Th1 and Treg populations, and concomitantly, the microbiota composition was altered, favoring the reduction of type I hypersensitivity. Similarly, *L. plantarum* IS-10506 attenuated the levels of specific inflammation markers, such as IL-4, IL-17, and interferon-γ (IFN-γ), and increased the expression of immunomodulatory factors Forkhead box P3 (Foxp3+) and IL-10 in pediatric patients with AD who orally received this probiotic strain **[12,13]**.

Thus, a low-glycemic diet, rich in plant fiber and low in processed foods, has been associated with an improvement in acne, possibly through gut changes or attenuated insulin levels. While there is much interest in the human microbiome, much more remains unknown, especially along the skin axis **[3]**. Collectively, the evidence suggests that approaches such as plant-based foods and supplements may be a viable alternative to the current first-line standard of care for moderate acne, which typically includes antibiotics. Although patient adherence to major dietary changes is likely much lower than with medications, it is a treatment avenue that deserves further study and development **[14-17]**.

Psoriasis patients often have distinct microbial compositions in their skin and gut, particularly within the major bacterial phyla Firmicutes, Bacteroidetes, and the *Akkermansia genus* **[18]**. Furthermore, bacterial DNA has been found in patients with psoriasis, both locally and systemically, suggesting a crucial role for bacteria in psoriatic disease and future studies in this field **[19]**.

Finally, current scientific evidence reveals the existence of an important skin-gut microbiota axis, highlighting the management of dermatoses through probiotics and prebiotics, as well as lifestyle changes **[20]**.

Limitations

Additional research is needed to address existing gaps and explore the full therapeutic potential of probiotics and prebiotics for the treatment of psoriasis and atopic dermatitis.

Conclusion

It was concluded that the gut microbiota has enormous metabolic capacity along the gut-skin axis. Dietary or microbiota metabolites are accessible to the skin. Therefore, after defining key open questions about the nature of these metabolites, their detection, and the skin changes they can induce, understanding these pathways will lead to new therapeutic strategies that target one organ to improve the health of another. Probiotics and prebiotics are microbiota management tools for treating psoriasis and atopic dermatitis.

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It was applied by Ithenticate®.

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It was performed.

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