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REVIEW

Clinical Treatment and Clinical Application Research Progress of Psoriasis and Intestinal Microbiota Dysbiosis

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Abstract: Psoriasis is a prevalent chronic inflammatory dermatosis. Recent evidence indicates a significant association between gut microbiota dysbiosis and its pathogenesis, potentially mediated through immunoinflammatory modulation and skin barrier integrity. This article systematically reviews the mechanisms linking gut microbiota to psoriasis, with emphasis on clinical treatment strategies targeting microbiota modulation—including probiotics, fecal microbiota transplantation (FMT), dietary interventions, and antibiotic therapies. However, current research exhibits notable limitations: most evidence derives from small-scale studies or animal models, lacking validation via large-scale clinical trials; microbiota-targeted interventions are poorly standardized, and the impact of individual variability on therapeutic outcomes remains unclear; the long-term safety of antibiotics and FMT requires further assessment. While summarizing existing advances, this review presents an evaluative overview to highlight research gaps and proposes future directions, such as integrated multi-omics studies, development of standardized therapeutic protocols, and exploration of personalized microbiota-based strategies, to innovate clinical management of psoriasis.

Keywords: psoriasis, gut microbiota, clinical treatment, probiotics, fecal microbiota transplantation

Introduction

Psoriasis is a chronic immune-mediated inflammatory skin disease with a complex pathogenesis involving interactions among genetic, environmental, and immunological factors.[1] Globally, psoriasis affects approximately 2–4% of the population and is often accompanied by comorbidities such as arthritis, cardiovascular diseases, and metabolic syndrome. These complications not only exacerbate patients' suffering but also significantly impair their quality of life.[2]

In recent years, research has gradually revealed the critical role of gut microbiota dysbiosis in the development and progression of psoriasis. Patients with psoriasis exhibit significant differences in the diversity and composition of their gut microbiota compared to healthy individuals, typically characterized by a reduction in beneficial bacteria (eg, Faecalibacterium prausnitzii) and an increase in conditionally pathogenic bacteria.[3] This microbial imbalance influences the host's immune system and metabolic products via the "gut–skin axis", thereby regulating inflammatory responses and skin barrier function. For instance, gut microbiota dysbiosis can promote the differentiation and activation of Th17 cells, and the aberrant activation of the IL-23/IL-17 signaling pathway represents a core immune mechanism in psoriasis pathogenesis.[4] A deeper understanding of this mechanism has laid the groundwork for developing novel targeted therapeutic strategies.

In terms of clinical treatment, while conventional methods can alleviate symptoms, interventions based on modulating the gut microbiota are becoming a research focus. Probiotic supplementation (eg, Lactobacillus and Bifidobacterium) may improve the severity and quality of life in patients with mild to moderate psoriasis by modulating immune responses

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and increasing anti-inflammatory factors.[5] Meanwhile, emerging therapies such as fecal microbiota transplantation (FMT) have shown potential in significantly improving or completely clearing skin lesions in some patients, indicating that reconstructing a healthy gut ecosystem could become an important direction for future personalized treatment.[6] Additionally, risk factors such as obesity exacerbate psoriasis by promoting systemic inflammation, and weight management can enhance the efficacy of conventional treatments, further highlighting the synergistic role of lifestyle and microbiota modulation.

In summary, the pathological mechanisms of psoriasis involve complex interactions between immune regulation and gut microbiota. With the gradual elucidation of the gut–skin axis mechanism, future research will increasingly focus on optimizing clinical intervention strategies through targeted modulation of the gut microbiota, thereby providing more effective and personalized treatment options for patients with psoriasis. This review aims to summarize the latest advances in the relationship between psoriasis and gut microbiota dysbiosis, promoting innovative exploration in related clinical treatments.

Review and Integration

The Mechanism of the Association Between Gut Microbiota and Psoriasis

Gut Microbiota Dysbiosis and Immune System Disorder

The gut microbiota precisely regulates host immune homeostasis through a complex molecular network, and the dysbiosis of gut microbiota in psoriasis patients is manifested not only by changes in microbial composition but more critically by abnormalities in functional metabolites. Short-chain fatty acids (SCFAs), including butyrate, propionate, and acetate, exert immunomodulatory effects through multiple signaling pathways: on one hand, butyrate activates G protein-coupled receptors (GPCRs) such as GPR41, GPR43, and GPR109a, inhibiting histone deacetylase (HDAC) activity, thereby promoting the differentiation and function of regulatory T cells (Tregs) while suppressing pro-inflammatory Th17 cell differentiation.[7,] As is well known, the pathogenesis of psoriasis primarily involves the aberrant activation of the IL-23/IL-17 signaling pathway. On the other hand, butyrate, as an HDAC inhibitor, stabilizes the phenotype and function of Tregs through epigenetic mechanisms by enhancing histone acetylation at the Foxp3 gene locus. Additionally, SCFAs inhibit NF-κB, phosphorylation and degradation, preventing the nuclear translocation of nuclear factor kappa B (NF-κB), thereby reducing the production of pro-inflammatory cytokines such as IL-6, TNF-α, and IL-23. Beyond SCFAs, secondary bile acids transformed by the gut microbiota, such as lithocholic acid (LCA) and deoxycholic acid (DCA), also play important roles in regulating intestinal barrier function and immune cell differentiation by activating the farnesoid X receptor (FXR) and G protein-coupled bile acid receptor 1 (GPBAR1).[8,9]

The Role of the Gut-Skin Axis

The gut-skin axis functions as a multi-level, bidirectional regulatory network involving complex interactions among the nervous, endocrine, and immune systems. Increased intestinal permeability allows microbial-associated molecular patterns (MAMPs), such as lipopolysaccharide (LPS) and flagellin, to translocate into the circulation.[10] These MAMPs activate the innate immune system via Toll-like receptors (TLR4, TLR5), prompting dendritic cells and macrophages to produce IL-23, which in turn drives Th17 cell differentiation. Tryptophan metabolism represents another critical pathway: the gut microbiota metabolizes tryptophan via the indoleamine 2,3-dioxygenase (IDO) pathway, generating metabolites such as kynurenine and indole. For instance, indole-3-aldehyde promotes IL-22 production through the aryl hydrocarbon receptor (AhR) signaling pathway, influencing keratinocyte proliferation and differentiation, while kynurenine directly regulates the Th17/Treg balance via AhR signaling.[11] The neuroendocrine mechanism is equally important; neurotransmitters modulated by the gut microbiota, such as serotonin (5-HT) and norepinephrine, can influence neuropeptide release and neurogenic inflammation through the gut-brain-skin axis, indirectly regulating skin immune responses.

Reduced Microbial Diversity and Severity of Psoriasis

A profound association exists between reduced microbial diversity and the severity of psoriasis. The loss of diversity leads to vacancies in microbial ecological niches, allowing potentially pathogenic bacteria such as Enterococcus and Proteobacteria to gain a competitive advantage, thereby causing a systemic imbalance in the metabolic network: the

reduction of Prevotella and Faecalibacterium prausnitzii impedes the metabolic pathway from acetyl-CoA to butyryl-CoA and ultimately to butyrate production;[12] the depletion of Lactobacillus and Bifidobacterium affects the generation of indolelactic acid and indoleacrylic acid, weakening AhR signal activation; the decrease in Bacteroides reduces the conversion efficiency of primary bile acids to secondary bile acids.[13] Metagenomic studies further reveal that in psoriasis patients, the abundance of key enzyme-encoding genes, such as butyrate kinase and butyryl-CoA:acetate CoA-transferase, is significantly reduced in the gut microbiota. Concurrently, the expression of genes encoding β-glucuronidase, bile salt hydrolase, as well as polysaccharide utilization loci and vitamin B synthesis gene clusters, is downregulated, functionally explaining the metabolic basis of dysbiosis. Clinical observations reinforce these findings: the Psoriasis Area and Severity Index (PASI) score shows a significant negative correlation with the abundance of Faecalibacterium ($r = -0.68$, $p < 0.001$), disease duration is negatively correlated with microbial gene richness ($r = -0.52$, $p = 0.003$), while arthritic complications are positively correlated with the abundance of Ruminococcus gnavus and Klebsiella pneumoniae. Notably, gut microbiota characteristics demonstrate potential for predicting treatment response: a higher baseline abundance of Akkermansia and Faecalibacterium predicts a better response to methotrexate treatment (AUC = 0.79), and the diversity of butyrate-producing bacteria is an independent predictor of sustained remission with biologic agents (HR = 2.34, 95% CI 1.47–3.72).[14]

In summary, the gut microbiota deeply participates in the pathogenesis of psoriasis through multi-level mechanisms involving metabolite-mediated immune regulation, epigenetic modulation, and neuroendocrine interactions. Targeted interventions addressing specific microbial communities and their metabolic pathways hold promise for pioneering new strategies for the personalized treatment of psoriasis. Future research should focus on further elucidating the molecular details of microbiota-host interactions and advancing the development of microbiome-based diagnostic tools and innovative therapies.

The Application of Probiotics in the Treatment of Psoriasis

The Immune Regulatory Role of Probiotics

Probiotics show significant potential in regulating the immune system, especially in the treatment of psoriasis. Research has shown that certain probiotics, such as Lactobacillus and Bifidobacterium, can inhibit the differentiation of Th17 cells, a process closely related to the inflammatory response of psoriasis. The overactivity of Th17 cells is considered a key factor in the onset of psoriasis, as the cytokines they secrete (such as IL-17 and IL-23) promote inflammation and skin lesion formation. By modulating gut microbiota, probiotics can influence systemic immune responses, thereby alleviating skin inflammation and improving symptoms in psoriasis patients.[15] Additionally, probiotics can enhance gut barrier function, reduce intestinal inflammation, and further regulate immune responses, thus providing a new approach for the treatment of psoriasis[16.] Therefore, the immune regulatory effects of probiotics provide important scientific evidence for the clinical treatment of psoriasis.

Clinical Research Progress

In recent years, multiple clinical studies have demonstrated the effectiveness of probiotics in the treatment of psoriasis. For example, a systematic review and meta-analysis showed that probiotic supplementation significantly improved the psoriasis area and severity index (PASI) in patients with psoriasis, with a PASI score reduction of 1.40 (95% CI = −2.63 to −0.17, $p < 0.00001$) compared to placebo.[15] Another study also confirmed that probiotics can improve inflammation-related indicators, reducing levels of C-reactive protein (CRP) and tumor necrosis factor-alpha (TNF-α), indicating their positive role in the treatment of psoriasis.[17] Through a comprehensive analysis of multiple randomized controlled trials, probiotics are considered a safe and effective treatment option that can enhance the quality of life for patients and reduce the risk of disease recurrence.[18] The results of these clinical studies provide strong support for the inclusion of probiotics in the treatment of psoriasis.

Limitations of Probiotics

Although probiotics have demonstrated potential in psoriasis treatment, their clinical application faces several important limitations. First, the efficacy of probiotics is highly strain-specific, with significant variations observed among different strains. For instance, Lactobacillus rhamnosus GG and Bifidobacterium infantis 35624 have shown promising

immunomodulatory effects in specific patient subgroups, while other strains such as Lactobacillus acidophilus NCFM may exhibit minimal improvement in psoriasis patients.[19] This strain-dependent variability necessitates careful personalized selection of probiotic formulations.Second, current research remains predominantly focused on a limited number of probiotic species, primarily within the Lactobacillus and Bifidobacterium genera. Other potentially beneficial strains, including Faecalibacterium prausnitzii and Akkermansia muciniphila, have received relatively little attention in psoriasis clinical trials, resulting in significant gaps in our understanding of their therapeutic potential across diverse patient populations.Furthermore, the long-term sustainability of probiotic effects and potential adverse reactions require more comprehensive evaluation. Studies have reported that discontinuation of specific probiotic strains, particularly Lactobacillus casei Shirota, leads to rapid reversal of immunological benefits in psoriasis patients.[20] Additionally, documented evidence indicates that certain probiotic formulations containing Streptococcus thermophilus may temporarily exacerbate gastrointestinal symptoms in susceptible individuals with pre-existing impaired intestinal barrier function. Notably, the mechanisms underlying strain-specific effects are becoming increasingly clear. For example, Lactobacillus rhamnosus GG has been demonstrated to specifically enhance regulatory T cell function through the TGF-β signaling pathway, while Bifidobacterium longum BB536 primarily exerts its effects by modulating IL-10 production. This mechanistic diversity underscores why generic recommendations for "probiotics" without strain specification are insufficient for psoriasis treatment.

Therefore, although specific probiotic strains exhibit significant immunomodulatory properties, their clinical application requires precise strain selection, individualized dosing regimens, and long-term monitoring to ensure optimal therapeutic outcomes while minimizing potential risks. Future research should prioritize comparative effectiveness studies across different probiotic strains and establish biomarkers for patient stratification.

Fecal Microbiota Transplantation (FMT) Potential and Challenges

Mechanism of Action of FMT

Fecal microbiota transplantation (FMT), as an emerging therapeutic method, aims to restore and rebalance the gut microbiota by introducing healthy donor feces into the patient's gut. Studies have shown that the gut microbiota of psoriasis patients often exhibits dysbiosis, which may be closely related to abnormal immune responses. FMT can alleviate this immune dysregulation and improve psoriasis symptoms by introducing healthy gut microorganisms. Specifically, FMT regulates immune responses through multiple mechanistic pathways: for instance, by increasing the abundance of beneficial bacteria such as Lactobacillus reuteri, which produce anti-inflammatory metabolites like short-chain fatty acids (SCFAs). These SCFAs, particularly butyrate, can inhibit NF-κB signaling and promote regulatory T cell (Treg) differentiation, thereby rebalancing the Th17/Treg axis and reducing systemic inflammation in psoriasis patients.[11] Furthermore, FMT helps restore gut barrier integrity by enhancing tight junction protein expression, which reduces intestinal permeability ("leaky gut") and limits the translocation of pro-inflammatory molecules such as lipopolysaccharides (LPS). This, in turn, decreases the release of endogenous inflammatory factors and modulates dendritic cell and macrophage activity, which is crucial for improving the immune status in psoriasis.[21] In summary, FMT offers a new possibility for the treatment of psoriasis by reconstructing a healthy gut microbiota and influencing the host's immune response through these detailed mechanisms.

Clinical Cases and Efficacy

In recent years, a growing number of clinical cases have demonstrated that fecal microbiota transplantation (FMT) exhibits significant therapeutic efficacy in patients with refractory psoriasis who have not achieved satisfactory results following conventional systemic therapies (such as methotrexate or cyclosporine) or biologic treatments. In one study, a 36-year-old male patient showed marked improvement in psoriatic symptoms after undergoing two FMT procedures, with significant reductions in body surface area (BSA), Psoriasis Area and Severity Index (PASI), and Dermatology Life Quality Index (DLQI).[22] Similar clinical outcomes have been validated in other studies, where FMT not only alleviated cutaneous manifestations but also improved gastrointestinal health and mitigated concomitant irritable bowel syndrome in patients.[23] Furthermore, the therapeutic effect of FMT is closely associated with changes in the gut microbiota composition, as microorganisms from healthy donors can effectively replace the dysbiotic pathogenic flora in patients,

thereby promoting the reduction of inflammatory responses.[24] Although existing clinical data support the potential of FMT in psoriasis treatment, further large-scale randomized controlled trials are required to verify its efficacy and safety.

Safety and Ethical Issues

Despite the promising efficacy of FMT in treating psoriasis, safety concerns cannot be overlooked. Since FMT involves transplanting donor feces into the patient, the selection of donors and the standardization of procedural protocols are crucial. Research indicates that FMT may pose an infection risk, particularly if donors are not rigorously screened, which could lead to the transmission of pathogenic microorganisms and other adverse reactions.[9,24] Therefore, strict donor screening standards must be established in clinical practice, including comprehensive health assessments and medical history investigations to ensure the donor's health status. Additionally, the implementation process of FMT should follow strict operational standards to reduce the risk of infection and ensure patient safety. Regarding ethical issues, patients should be fully informed before undergoing FMT, understanding the potential risks and benefits to make informed treatment choices. Thus, although FMT offers new hope in the treatment of psoriasis, its application must be approached with caution and conducted under strict regulation.

Dietary Interventions on Gut Microbiota and Psoriasis

High-Fiber Diet and SCFAs

There is a close relationship between the intake of high-fiber diets and the generation of short-chain fatty acids (SCFAs), which play an important role in gut health and inflammation management. Studies have shown that dietary fiber can be fermented by gut microbiota to produce SCFAs, such as acetate, propionate, and butyrate, which help to suppress skin inflammation, thereby alleviating psoriasis symptoms. The gut microbiota of psoriasis patients often shows dysbiosis, while a high-fiber diet can help restore the balance of gut microorganisms, increase the production of SCFAs, and reduce skin inflammation through its anti-inflammatory effects.[25] Furthermore, some studies have indicated that increasing dietary fiber intake can significantly elevate SCFA concentrations, thereby affecting the host's metabolism and immune response, promoting overall health.[25] Therefore, developing a high-fiber dietary plan may become an effective adjunctive treatment strategy for psoriasis patients.

Benefits of the Mediterranean Diet

The Mediterranean diet, known for its richness in Omega-3 fatty acids and antioxidants, is believed to have significant benefits for psoriasis patients. Research has shown that the Mediterranean diet can improve the gut microbiota composition of psoriasis patients and reduce skin inflammation through its anti-inflammatory effects.[26] In particular, Omega-3 fatty acids, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), possess strong anti-inflammatory properties. These fatty acids can lower the levels of key pro-inflammatory factors involved in psoriasis pathogenesis, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β), while also promoting the production of anti-inflammatory mediators like resolvins and protectins. This modulation of the inflammatory cascade contributes to the improvement of clinical symptoms in psoriasis. Additionally, the high fiber content of the Mediterranean diet promotes the growth of beneficial gut bacteria and the subsequent production of SCFAs, which further support gut health and immune function regulation.[27] Therefore, it is recommended that psoriasis patients adopt the Mediterranean diet, which not only helps improve skin condition but also enhances overall health.

Clinical Practice of Dietary Interventions

In clinical practice, personalized dietary plans are receiving increasing attention. Research has shown significant differences in gut microbiota characteristics among different patients; therefore, developing personalized dietary intervention plans should consider the results of patients' gut microbiota testing. By analyzing the gut microbial composition of patients, doctors can design more targeted dietary plans to effectively improve psoriasis symptoms.[28] For example, patients with certain types of gut microbiota dysbiosis may need to increase their intake of specific dietary fibers or SCFA precursors to promote the growth and reproduction of beneficial bacteria, thus rebuilding microbial balance. Additionally, clinical studies have indicated that combining dietary interventions with traditional treatments can significantly improve

treatment outcomes and enhance patients' quality of life.[29] Therefore, it is recommended that clinicians consider dietary interventions as an effective adjunctive therapeutic approach in the treatment of psoriasis.

The Contradiction of Antibiotics and Psoriasis Treatment

The Destruction of Gut Microbiota by Antibiotics

Long-term use of antibiotics can significantly disrupt the gut microbiota, thereby exacerbating psoriasis symptoms. Studies demonstrate that antibiotic-induced dysbiosis not only compromises intestinal health but may also aggravate cutaneous inflammation through gut-skin axis mechanisms.[30] Specifically, psoriasis patients exhibit characteristic microbial alterations, with documented reductions in beneficial bacteria such as Faecalibacterium prausnitzii (a key butyrate producer) and Bacteroides species, while potential pathogens including Enterococcus faecalis and Klebsiella pneumoniae show relative enrichment.[31] These compositional changes drive functional metabolic disturbances, particularly through diminished production of anti-inflammatory short-chain fatty acids (SCFAs) like butyrate, along with altered bile acid metabolism and tryptophan-derived metabolite profiles. The resulting metabolic imbalance reinforces systemic inflammation through impaired regulatory T-cell differentiation and heightened IL-17 signaling, ultimately worsening psoriatic manifestations.

Furthermore, antibiotics frequently reduce microbial diversity, which has been independently associated with autoimmune disease development including psoriasis.[32] Although antibiotic therapy remains necessary for managing concurrent infections, its collateral damage to gut ecosystems may paradoxically aggravate psoriatic disease. This duality necessitates careful risk-benefit assessment when prescribing antibiotics to psoriasis patients, particularly given the established relationship between streptococcal infections and psoriasis pathogenesis, where antibiotic efficacy remains controversial.[33]

Potential Benefits of Specific Antibiotics

Despite the potential negative impacts of antibiotic use, some studies suggest that certain specific antibiotics may have positive therapeutic effects on psoriasis patients. For instance, antibiotics such as doxycycline, due to their anti-inflammatory properties, are thought to possibly help alleviate psoriasis.[34] These antibiotics may improve patients' skin symptoms to some extent by reducing skin inflammatory responses.

In some clinical studies, the use of antibiotics has been explored as an adjunctive treatment for psoriasis, particularly in patients with clear infections. For example, the close relationship between streptococcal infections and acute guttate psoriasis suggests that using antibiotics in such cases may help control the infection and improve psoriasis symptoms.[35] However, despite some studies showing potential benefits of antibiotics for psoriasis, more randomized controlled trials are needed to verify their long-term effects and safety.

Additionally, the impact of antibiotics on psoriasis may be influenced by individual differences; therefore, in clinical practice, treatment should be individualized based on the patient's specific situation and infection risk. While certain antibiotics may be effective in specific contexts, the risks and benefits of using antibiotics in the absence of clear infection need to be carefully assessed to avoid possible side effects and exacerbation of dysbiosis. Hence, comprehensive treatment strategies for psoriasis should consider both the use of antibiotics and their potential impact on gut microbiota.

Other Clinical Treatment Strategies

Targeted Treatment of Microbiota Metabolites

In recent years, short-chain fatty acids (SCFAs) and tryptophan metabolites have received widespread attention in gut microbiota research. SCFAs are metabolic products generated by gut microbes through the fermentation of dietary fibers, such as acetate, propionate, and butyrate, which play crucial roles in maintaining gut health and immune regulation. Studies have shown that SCFAs can regulate immune responses by activating G protein-coupled receptors (such as GPR41 and GPR43). Specifically, SCFA-mediated activation of these receptors enhances intestinal epithelial barrier integrity by upregulating tight junction proteins (e.g,occludin and zonula occludens-1) and promoting mucin production. This strengthened barrier reduces systemic translocation of pro-inflammatory antigens and cytokines, which is

particularly relevant in psoriasis where disrupted gut permeability may exacerbate cutaneous inflammation through systemic immune activation.[36] Additionally, SCFAs have been confirmed to play a role in regulating inflammatory responses, inhibiting the production of pro-inflammatory cytokines, thereby alleviating chronic inflammatory states.[37]

At the same time, tryptophan is metabolized by gut microbes into various bioactive products, such as indole and its derivatives, which are also believed to play important roles in immune regulation and inflammation control. Research has shown that tryptophan metabolites – particularly indole derivatives such as indole-3-aldehyde – activate the aryl hydrocarbon receptor (AhR) signaling pathway. AhR activation directly modulates T cell differentiation by promoting the development of regulatory T cells (Tregs) while simultaneously suppressing the differentiation of pro-inflammatory Th17 cells, which are central to psoriasis pathogenesis. This shift in the Treg/Th17 balance toward immune regulation represents a key mechanism through which tryptophan metabolites influence gut barrier function and overall immune status.[38] By targeting these metabolites, new clinical treatment strategies can be developed to improve the clinical manifestations and quality of life of psoriasis patients.

Although targeted treatment of SCFAs and tryptophan metabolites shows promising potential in experimental studies, challenges remain for clinical application. Issues such as how to effectively deliver these metabolites to target sites and how to overcome individual differences in treatment response need further exploration in future research.[39] Therefore, while targeted treatment strategies for gut microbiota metabolites hold significant clinical potential, more clinical trials are needed to verify their effectiveness and safety.

Combined Treatment Models

Combined treatment represents a therapeutic strategy that integrates agents with distinct mechanisms of action to enhance clinical efficacy. In psoriasis management, the combination of probiotics with conventional systemic agents—including traditional immunosuppressants (eg, methotrexate, cyclosporine), biologics, and small molecule inhibitors—has demonstrated improved treatment outcomes. Probiotics contribute to immune system homeostasis and inflammatory response reduction through modulation of gut microbiota composition and function, thereby playing an adjunctive role in psoriasis therapy.[40] Studies indicate that probiotic supplementation not only potentiates the efficacy of these therapeutic agents but also mitigates their adverse effects.[41]

For instance, clinical investigations have revealed that when probiotics are co-administered with corticosteroids or methotrexate, patients exhibit significantly greater improvement in skin lesion severity and quality of life compared to those receiving immunosuppressant monotherapy.[42] Similarly, emerging evidence suggests that probiotics may enhance treatment response to biologics (eg, anti-TNF-α and anti-IL-17 agents) by maintaining microbial diversity and reinforcing intestinal barrier function, potentially extending therapeutic durability. The combination with small molecule inhibitors (such as PDE4 and JAK inhibitors) is also being explored, with preliminary data indicating that probiotics may help preserve gut mucosal immunity during targeted therapy.

Through these combinatorial approaches, probiotics promote gut barrier restoration and microbial diversity, subsequently enhancing systemic immune regulation and anti-inflammatory capacity. It is noteworthy that the effects of combined treatment exhibit significant interindividual variation, influenced by baseline gut microbiota composition, immune status, and treatment responsiveness. Future research should therefore focus on developing personalized combination strategies tailored to patients' specific microbial and immunological profiles.[43] In summary, the integration of probiotics with various psoriasis treatment modalities provides novel insights for therapeutic optimization and is expected to improve clinical outcomes across different patient subgroups.

Future Research Directions and Challenges

Precision Medicine and Gut Microbiota Testing

In the context of precision medicine, the testing and analysis of gut microbiota is becoming an important component of personalized treatment. The rapid development of metagenomics technology has enabled researchers to deeply analyze the composition and functional characteristics of individual gut microbiomes, thereby providing tailored treatment plans for patients. Existing studies have shown that the composition of gut microbiota is closely related to the onset and progression of various diseases. For example, in patients with psoriasis, dysbiosis of gut microbiota may exacerbate skin lesions, leading to

disease deterioration[44.] Through metagenomics technology, scientists can identify microbial biomarkers associated with specific diseases, thus enabling more accurate disease prediction and therapeutic interventions in clinical settings.

Moreover, the implementation of personalized treatment also relies on dynamic monitoring of gut microbiota. As our understanding of gut microbiomes deepens, future research should focus on how to combine patients' genomic information, lifestyle, and environmental factors to optimize microbiome intervention strategies. For instance, by analyzing changes in the abundance of specific microbes within patients, we can better understand how microbes influence drug metabolism and efficacy, thus achieving precision medication.[45] However, achieving this goal still faces several challenges, including how to standardize testing processes, establish a universal microbial biomarker database, and develop effective interventions to address the differences in microbiomes among individuals.

Construction and Application of Mechanistic Diagrams

The interaction between gut microbiota, the immune system, and the skin has gradually gained attention, particularly in the research of skin diseases such as psoriasis. Future research will need to explore the specific mechanisms by which gut microbiota regulates immunity, particularly how the composition of gut microbiota can influence skin inflammatory responses and immune status.[46] Constructing a mechanistic diagram of the gut microbiota-immune-skin axis will help visualize these complex biological processes and provide researchers with clear ideas to guide subsequent experimental design.

To achieve this goal, researchers need to integrate various omics data, including metagenomics, transcriptomics, and metabolomics, to comprehensively analyze the interactions between gut microbiota and the host. For example, by analyzing how the metabolic products of gut microbes affect the activity of host immune cells, a theoretical basis for developing new therapeutic strategies can be provided.[45] At the same time, establishing relevant flowcharts not only helps researchers understand the relationship between gut microbiota and skin diseases but also guides clinicians in formulating personalized treatment plans.

In summary, future research should aim to reveal the role of gut microbiota in disease occurrence and its underlying molecular mechanisms, while constructing actionable mechanistic diagrams to facilitate in-depth research and the practical application of clinical treatments.

Conclusion

This review systematically elucidates the central role of the gut microbiome in the pathogenesis of psoriasis and its clinical translational potential, revealing the complex mechanisms by which gut microbiota participate in disease development through multiple pathways including metabolites (such as short-chain fatty acids, bile acids, and tryptophan metabolites), immune regulation (particularly the IL-23/Th17 axis and Treg cell differentiation), and bidirectional communication along the gut-skin axis. Studies indicate that changes in microbiome characteristics, such as the reduction of Faecalibacterium prausnitzii and decreased microbial diversity, are not only significantly correlated with disease severity but also demonstrate potential value in predicting treatment response. At the level of therapeutic strategies, microbiome-based interventions show a trend toward diversification: specific probiotic strains exhibit potential for clinical improvement by modulating the Th17/Treg balance; high-fiber and Mediterranean diets offer new adjunctive therapeutic directions by promoting short-chain fatty acid production and anti-inflammatory mediators; fecal microbiota transplantation demonstrates breakthrough potential in refractory cases; while antibiotic therapy must be strictly limited to infection-related subtypes to balance its anti-infective benefits against the risk of microbiome disruption. However, current research still faces numerous challenges, including limited sample sizes in most clinical studies, incomplete understanding of microbiota-host interaction mechanisms, and the lack of standardized biomarker systems for personalized treatment strategies. Future research should focus on integrating multi-omics technologies to construct precise classification systems, conducting large-scale randomized controlled trials to validate the long-term efficacy and safety of microbiome interventions, establishing dynamic microbiome monitoring platforms for early prediction and adjustment of treatment responses, and accelerating the translation from mechanistic discoveries to clinical applications by promoting interdisciplinary collaboration among dermatology, microbiology, and bioinformatics. Ultimately, this will enable a paradigm shift from traditional immunosuppression to eco-immunological reconstruction, ushering in a new era of precision medicine for psoriasis based on individual microbial characteristics.

AI Declaration

The authors utilized DeepSeek (OpenAI, Version May 29) for language polishing throughout the manuscript, primarily to improve academic writing precision. Authors assume full responsibility for all content generated by or edited with AI assistance.

Abbreviations

FMT, Fecal Microbiota Transplantation; SCFAs, Short-Chain Fatty Acids; GPCRs, G Protein-Coupled Receptors; HDAC, Histone Deacetylase; Tregs, Regulatory T cells; LCA, Lithocholic acid; DCA, Deoxycholic acid; FXR, Farnesoid X receptor; GPBAR1, G protein-coupled bile acid receptor 1; Th17, T Helper 17 Cells; TNF-α, Tumor Necrosis Factor Alpha; MAMPs, Microbial-Associated Molecular Patterns; LPS, Lipopolysaccharide; AhR, Aryl Hydrocarbon Receptor; PASI, Psoriasis Area and Severity Index; CRP, C-reactive protein; BSA, body surface area; PASI, Psoriasis Area and Severity Index; DLQI, Dermatology Life Quality Index; EPA, eicosapentaenoic acid; DHA, docosahexaenoic acid; TNF-α, tumor necrosis factor-alpha; IL-6, interleukin-6; IL-1β, interleukin-1β.

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Disclosure

The authors report there are no competing interests to declare.

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