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THE IMPORTANCE OF MICROORGANISMS IN PSORIASIS

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Abstrac: Recent advances (2024–2025) have fundamentally reshaped our understanding of the role of microorganisms in psoriasis pathogenesis. Cutting-edge studies using Mendelian randomization have provided the first genetic evidence of bidirectional causality between the skin microbiome and psoriasis, demonstrating that dysbiosis not only triggers but is also perpetuated by the disease. Integrated microbiome–metabolome analyses revealed novel inflammatory loops driven by increased Staphylococcus aureus, Corynebacterium spp., arachidonic acid pathway metabolites, and oxidative stress markers, alongside depletion of short-chain fatty acid-producing bacteria. Biologic therapies show differential effects on gut microbiota: secukinumab restores a healthier Firmicutes/Bacteroidetes ratio and boosts Faecalibacterium prausnitzii, whereas ustekinumab may favor Enterobacteriaceae expansion. Emerging preclinical data highlight the therapeutic potential of topical microbiome transplantation and engineered strains (e.g., modified Roseomonas mucosa). Additionally, urban air pollution was shown to exacerbate psoriasis risk up to 3.6-fold through microbiome disruption.

Keywords : Psoriasis, skin microbiome, gut microbiota, bidirectional causality, Mendelian randomization, microbiome–metabolome integration, Staphylococcus aureus, Corynebacterium, Firmicutes/Bacteroidetes ratio, secukinumab, ustekinumab, topical microbiome therapy, engineered Roseomonas mucosa, arachidonic acid metabolism, environmental pollution, leaky gut syndrome, superantigen, guttate psoriasis, Faecalibacterium prausnitzii, personalized medicine, microbial therapeutics

Psoriasis is a chronic, relapsing inflammatory disease that mainly affects the skin. The clinical picture is red, often sharply demarcated, plaques with a whitish-silver coating; sometimes changes are observed in the nails, in the dermis and under the nails. The etiopathogenesis of the disease is multifactorial and is a complex interaction between immunological dysregulation, genetic predisposition and environmental factors (infections, stress, drugs, etc.). In recent years, a lot of evidence has been accumulated that microorganisms, including the skin and intestinal microbiota, are important modulators of the onset and course of the disease. This article

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examines the role of microorganisms in the process of psoriasis and how this knowledge can affect diagnostic and treatment strategies. Initially, small, pink or reddish nodules form on the skin, which are covered with a flaky, silvery crust. The nodules gradually increase in size and merge with each other. When the disease is severe, P. rashes completely cover the body (psoriatic erythroderma); in this case, the patient feels unwell, has a fever, and the lymph nodes enlarge. In some types of P., nails and joints are also affected, their shape changes, and movement causes severe pain, and the surface of the nails thickens and becomes dull (resembles bird claws). The disease usually flares up in the fall and winter months, and eases slightly in the summer (remission). P. rashes reappear as a result of nervous trauma, alcohol consumption, exhaustion, and dietary disorders. When symptoms characteristic of a skin disease appear, it is necessary to see a doctor and undergo treatment according to his instructions. In addition to special treatment measures, dietary meals made from dairy and vegetable products, especially cottage cheese, vegetable oil, and foods rich in vitamins (A, B. C) are recommended for obese patients. Depending on the type and course of P., the doctorWhen prescribed, treatment with climate brings good benefits.

Epidemiology

Global prevalence: Recent analyses suggest that psoriasis affects approximately 1–5% of the world population; new large-scale analyses suggest a global prevalence of ~4.4%, but there are regional differences (e.g., relatively high in Asia and Europe, lower in some regions). Age and gender: The disease can begin at any age; most often it begins between the ages of 20 and 40, but a second peak is observed in the 50s and 60s. The prevalence is approximately equal in men and women. Economic and social burden: Psoriasis can cause serious psychological (depression, social isolation) and economic consequences for patients; the cost of chronic therapy and the use of health care resources is high. In the last decade, it has been recognized that the skin and gut microbiota (a collection of bacteria, fungi, viruses, and other microorganisms) are in a dynamic relationship with the immune system. The composition of the microbiota in psoriasis patients differs from that of healthy individuals, and these differences may be a factor that supports or mediates inflammation. The microbiota plays a role in the onset and progression of psoriasis:

1. Direct local effect (on the skin surface): some bacteria and fungi can impair the skin barrier function and activate inflammation.

2. Systemic modulation (gut-skin action): dysbiosis of the gut microbiota can trigger or exacerbate skin inflammation through immune signals.

Below, we will review in detail the changes in the skin and gut microbiota, their mechanisms, and therapeutic prospects. Skin microbiota and psoriasis Changes in the skin microbiota. Differences in bacterial and fungal composition have been observed in psoriasis-affected areas compared with normal skin. For example, concentrations of Staphylococcus aureus and other opportunistic species may be higher in some patients; the role of Malassezia and other fungi has also been described. Such changes contribute to skin inflammation and barrier disruption. and Mechanisms (how skin microbiota may promote psoriasis)

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1. Antigen presentation and inflammation induction Some bacterial antigens or toxins activate keratinocytes and antigen-presenting cells, increasing the production of cytokines (e.g., through the IL-23, IL-17 pathway).

2. Impaired barrier function: Microorganisms affect skin lipids and structures, weakening the physiological barrier and facilitating the entry of antigens.

3. Biofilm and chronic infection Some bacteria can form biofilms that can lead to local invasiveness or chronic inflammation, which worsens the disease. (This feature can be observed especially in wounds, cavities or heavy plaques. Recent studies have shown that there are certain changes in the intestinal microbiota (dysbiosis) in psoriasis patients: various studies have reported an increase in Bacteroides and a decrease in genera such as Prevotella. Such changes may contribute to the activation of the Th17 pathway by altering immune regulation.

Mechanisms

Short-chain fatty acids (SCFAs) and other metabolites produced by gut bacteria regulate T reg (regulatory T cells) and other immune signals; dysbiosis can disrupt or alter these signals.

2. Immune enhancement can stimulate antigen-motivated systemic inflammation by pathogenic or opportunistic species; this increases inflammatory mediators such as IL-17, IL-23 in the skin. Bidirectional causality between skin microbiome and psoriasis New finding: A 2025 Mendelian randomization analysis showed that the skin microbiome (especially in dry skin areas) contributes to the onset of psoriasis, but psoriasis also alters the skin microbiota. aggravates the disease. For example, increased levels of asv002 (an unknown bacterial clade) and the genus Haemophilus (OR-1.364, 95% CI: 1.065-1.746) can increase the risk of psoriasis by up to 36%. This is the first bidirectional association proven based on genetic data, indicating the skin microbiome as both a “trigger” and a “consequence”. Significance: This finding highlights that psoriasis is not just related to the gut, but also to the skin microbiome. In the future, analyzing the skin microbiota could help predict the disease early.

Rare: Standard dermatology textbooks only report a unilateral effect, but this study highlights differences in skin regions (dry vs. moist). 2. Integrated analysis between microbiome and metabolome: New metabolic interactions New finding: 2025 study of skin samples from psoriasis patients (29 patients vs. 31 healthy controls) The microbiome and metabolome (metabolism) were studied together. As a result, in psoriasis lesions, along with the increase in Staphylococcus aureus and Corynebacterium, an increase in arachidonic acid (eicosanoid family) and oxidative stress metabolites (e.g. lipid peroxides) was observed. These metabolites are formed by the action of microbial enzymes (especially Firmicutes species) and increase inflammation (IL-17 and TNF-α). At the same time, a decrease in short-chain fatty acids (SCFAs, e.g. butyrate) increases gut-skin permeability. Significance: This is the first evidence of a microbiome-metabolome "loop" in psoriasis, suggesting novel biomarkers (e.g., eicosanoids). Modulating metabolites in treatment (e.g., omega-3 supplements) may normalize the microbiota.

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Rare feature: While traditional research focuses solely on the microbiome, this integrated approach identifies metabolic pathways (e.g., arachidonic acid metabolism) as novel targets that have not yet been applied in the clinic.

3. Effects of biological treatment on the microbiota: Differences between secukinumab and ustekinumab New finding: Clinical trials conducted in 2024-2025 showed that biological drugs (secukinumabanti-IL-17 and ustekinumab anti-IL-12/23) differently alter the intestinal microbiota. In patients receiving secukinumab, the Firmicutes/Bacteroidetes ratio approaches normal and beneficial Faecalibacterium prausnitzii increases, but ustekinumab can increase pathogenic Enterobacteriaceae. In addition, the effectiveness of treatment (PASI index) is associated with changes in the microbiota: remission is 30% higher in patients with a high content of beneficial bacteria. Importance: This suggests that biologic therapy should be selected after microbiota analysis, e.g. secukinumab is preferred in patients with dysbiosis. In the future, combination with probiotics may increase efficacy.

Rare feature: Much is said about the effectiveness of biologics, but their specific effects on the microbiota (especially differences) are new and personal based on medicine.

4. Topical microbiome therapy and engineered bacteria: Preclinical advances New findings: In 2024, a study tested topical microbiome therapy (MBT) in psoriasis. For example, engineered bacteria such as Roseomonas mucosa (a natural skin bacterium) modulated TNF-α signaling and reduced psoriasis lesions by 50% in mouse models. In addition, in the face of vaccine overexposure (e.g., COVID-19) vaccine) Preliminary evidence has been found that depletion of Bifidobacterium may trigger psoriasis and depression.

Importance: Unlike oral probiotics, topical MBT acts directly on the skin and reduces side effects. This is a new direction for severe psoriasis.

Rare aspect: The vaccine-microbiome-psoriasis connection and engineered bacteria are still rarely mentioned in the media, but clinical trials are expected to begin in 2025.

5. The combined role of environmental pollution and the microbiome

New finding: A 2024 study found that urban air pollutants (CO, NO2, PM) increase psoriasis incidence by 3.61 times and disrupt skin microbiota (specifically, a decrease in Staphylococcus epidermidis). This extends the “hygiene hypothesis,” highlighting the dual effects of excessive cleanliness and pollution.

Importance: For urban residents, protecting the microbiota (e.g. probiotics + clean air) can be preventive.

Rarely: Environmental factors beyond the microbiome have been poorly studied in psoriasis, but this finding explains the global epidemiology.

Clinical experiences and therapy prospects

Probiotics, prebiotics, and even fecal microbiota transplantation (FMT) are being explored as possible therapies. Some small studies and preclinical studies have shown improvement in patients' skin conditions, but there are no comprehensive, validated clinical guidelines yet and

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further studies are needed. If strep throat is detected, treatment with antibiotics (penicillin, erythromycin) may prevent flare-ups of guttate psoriasis. If gut dysbiosis is present, probiotics, prebiotics, and a diet (low FODMAP, gluten-free) may be helpful in some patients.

Pathogenesis of psoriasis (with a microbial context)

The pathogenesis of psoriasis consists of interconnected steps: genetic predisposition, external trigger (e.g. infection, injury, stress), innate immune activation, activation of the IL-23/Th17 pathway, keratinocyte proliferation, and a cyclical decrease in inflammatory mediators. The microbiota is can act as a trigger or modulator at one or more stages of the process. The IL-23 and Th17 pathways are particularly important because this pathway also plays a central role in the immune response to bacteria and fungi, and therefore changes in the microbiota can directly or indirectly disrupt this pathway.

Diagnostics

The diagnosis of psoriasis is primarily based on clinical findings, but microbiological and other ancillary methods may be useful in evaluating the disease and identifying infectious comorbidities.

Clinical assessment

History: onset of disease, family history (psoriasis has a genetic component), triggers (causes of exacerbations) - infections, medications, stress. Physical examination: plaques, spread, nail changes, ligament/joint pain (possible psoriatic arthritis). Laboratory and microscopic methods Dermoscopy: specific in psoriatic plaques . Dotted/glomerular vessels and a white mucus coating are observed. The diagnosis can be supported based on dermatoscopic signs. Biopsy: In severe or atypical cases, a skin biopsy is taken for histopathological confirmation (features such as epidermal hyperplasia acanthosis, parakeratosis, neutrophilic infiltrates Munro microabscesses). Microbiological tests: if superinfection is suspected (e.g., exudate, pus on plaque), bacteriological or fungal cultures and, if necessary, PCR are used. If Malassezia and other fungi are detected in skin or scalp samples, local or systemic antifungal therapy is considered. Gut microbiota analysis: For research purposes, the composition of the gut microbiota can be determined by 16S rRNA sequencing, but this is not widely used as a practical clinical diagnosis and remains in a research context for now.

Treatment (conventional and microbiota-targeted approaches)

The severity of the disease (mild/moderate/severe), the patient's age, comorbidities, and family preferences are taken into account when determining the treatment plan. International guidelines include a range of options, from local therapy to systemic and biological therapy. There are recommendations for therapies.

Local therapy (topical)

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Relevant drugs: corticosteroids (in various strengths), vitamin D analogues (calcipotriol), combinations (corticoid + vitamin D analogue), therapeutic retinoids (tazarotene), keratolytics (salicylic acid), emollients. This therapy is the basis for mild and some moderate diseases.

Phototherapy

NBUVB and PUVA methods are used in moderate and some severe cases. Phototherapy reduces cell proliferation and has an immunosuppressive effect.

Systemic therapy

Conventional immunosuppressive or retinoid therapies such as methotrexate, cyclosporine, acitretin are used in moderate to severe cases. It is important to note that each has side effects and requires monitoring.

Biological therapy

TNF-α blockers, IL-12/23 inhibitors, IL-17 inhibitors, and IL-23-specific inhibitors have revolutionized psoriasis over the past decade. These agents directly target the IL-23/Th17 pathway and are highly effective. Biologics are indicated for severe disease or cases refractory to systemic therapy.

Antimicrobial or microbiota-targeted therapies Antibiotics/antifungal therapy: if there is superinfection or if pathogens are found in specific locations such as the scalp, an appropriate antibiotic or antifungal is used. However, the prophylactic use of broad-spectrum antibiotics may be detrimental to the microbiota. Probiotics and prebiotics: are being studied to restore or modulate the gut microbiota: some small trials have shown positive results, but there is currently insufficient evidence for broad clinical guidelines. FMT (Fecal Microbiota Transplantation): currently mainly in a research context; evidence on the value and safety of widespread use in psoriasis is limited. Future high-quality, randomized studies are needed. Integrated approach: individualization and combination. In current clinical practice, a combination of therapies is often used: for example, topical therapy + phototherapy or a biological topical regimen. If microbiota dysbiosis is detected (as a result of a secondary infection or a study), Microbiota-targeted approaches may also be included, but these are complementary and still in the research phase. Because biologic therapies have shown high efficacy by targeting the IL-23/IL-17 pathway, biologics are often effective even in patients with microbiota alterations, but microbiota treatments may be helpful in improving the efficacy of therapy in the future.

Disease course and complications

Psoriatic arthritis: some patients may develop joint inflammation (psoriatic arthritis) along with skin inflammation, in which case collaboration with a rheumatologist is necessary. Nail changes, leading to atrophy, superinfections, psychological complications (depression, social isolation). There are studies that suggest that microbiota dysbiosis may exacerbate these complications in some cases, but the cause-and-effect relationship is not clear.

Summary

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Psoriasis is a complex, multifactorial chronic inflammatory disease, and recent studies suggest that the skin and gut microbiota may play a key role in the onset and progression of the disease. The microbiota interacts with key inflammatory pathways, such as the IL-23/Th17 pathway, as an immune modulator. In practice, the main treatment modalities are topical therapy, phototherapy, conventional systemic drugs, and biologics, while microbiota-targeted approaches (probiotics, FMT) are still under investigation. In the future, the possibility of individualized therapy based on the microbiota profile seems promising. These new data encourage us to view psoriasis not only as a skin disease, but also as a microbiome-metabolome-environment system. In the future, genetic testing and individual probiotics (e.g., Bifidobacterium supplements) may revolutionize treatment. If you have psoriasis, consider getting a microbiome test (such as the Viome kit) with your doctor's advice, as it is more effective than a simple blood test. Keep an eye on the journals Frontiers in Microbiology or BMC Microbiology for new research.

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