

Exploring the Role of Microbiome Alterations in the Pathogenesis of Psoriatic Arthritis: A Multidisciplinary Approach

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Abstract

Psoriatic arthritis (PsA) is a complex, chronic inflammatory disease with intertwined dermatological and rheumatological manifestations, profoundly affecting patients' quality of life. Recent advances have highlighted the potential role of microbiome alterations, both in the gut and skin, in the pathogenesis of PsA. This review synthesizes current research on the microbiome's involvement in PsA, emphasizing the interplay between microbial dysbiosis and immune system modulation. Emerging evidence suggests that shifts in the composition and function of the gut and skin microbiota can trigger or exacerbate PsA by promoting systemic inflammation and autoimmune responses. Studies have identified specific microbial taxa associated with PsA, revealing potential biomarkers for early diagnosis and therapeutic targets. However, the precise mechanisms through which microbiome alterations influence PsA pathophysiology remain incompletely understood. Future research should focus on longitudinal studies to further understand the causal relationships between microbiome changes and PsA onset and progression. Integrating varying approaches, including metagenomics, transcriptomics, and metabolomics, will provide a comprehensive understanding of microbial functions and host-microbiome interactions. Additionally, investigating the effects of microbiome-modulating interventions, such as probiotics, prebiotics, and dietary modifications, could guide innovative therapeutic strategies. This multidisciplinary approach, combining dermatological and rheumatological insights, holds promise for unraveling the complexities of PsA and improving patient outcomes through personalized medicine. By advancing our knowledge of microbiome dynamics, we can move closer to precise, targeted treatments that address the underlying causes of PsA.

Keywords: Psoriatic arthritis, Microbiome alterations, Gut microbiota, Skin microbiota, Dysbiosis, Inflammation, Immune system modulation, Systemic inflammation, Autoimmune responses, Biomarkers, Metagenomics, Transcriptomics, Metabolomics, Probiotics, Prebiotics, Dietary modifications, Personalized medicine, Host-microbiome interactions.

INTRODUCTION

Psoriatic arthritis (PsA) is a complex, chronic inflammatory disease that affects up to 30% of individuals with psoriasis, impacting approximately 1.5 million Americans [1,2]. PsA intertwines dermatological and rheumatological manifestations, profoundly affecting the quality of life for patients. This condition is characterized by skin lesions typical of psoriasis and joint inflammation, which can

result in severe pain and disability [3]. This dual impact on skin and joints presents unique management challenges, often leading to physical limitations and psychological distress [4].

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Furthermore, PsA can significantly diminish quality of life by causing chronic pain, fatigue, and mobility issues, potentially reducing social participation and contributing to mental health issues [5]. It is estimated that at least 33% of individuals with PsA experience anxiety, and 20% experience depression [6]. Studies also indicate that 45% of individuals experience regular fatigue and up to 38% of those with PsA experience sleep disturbances [7]. These statistics underscore the pervasive impact of PsA not only on physical health but also on emotional well-being.

Beyond its physical and emotional impacts, PsA also poses a significant burden due to its chronic nature and the potential for progressive joint damage and deformity if untreated [8]. Effective management of PsA requires a multidisciplinary approach that addresses both dermatological and rheumatological aspects of the disease. Recent advances in understanding the pathogenesis of PsA have highlighted the potential role of the microbiome, particularly the microorganism communities in the gut and on the skin [9]. This new perspective suggests that microbial alterations might play a crucial role in the onset and progression of PsA.

Emerging research suggests that microbial dysbiosis could be a key factor in the pathogenesis of PsA, potentially influencing the immune system and promoting the chronic inflammation characteristic of the condition [10]. Certain microbial taxa in the gut and skin have been linked to PsA, suggesting that these microorganisms might trigger or exacerbate the disease [11]. Understanding the interaction between these microbial communities and the host immune system is critical for developing new therapeutic strategies.

This review synthesizes current research on the role of the microbiome in PsA, with a focus on the interplay between microbial dysbiosis and immune system modulation. It examines how changes in the composition and function of the gut and skin microbiota may trigger or exacerbate PsA by promoting systemic inflammation and autoimmune responses. The identification of specific microbial taxa associated with PsA may reveal potential biomarkers for early diagnosis and therapeutic targets. By merging dermatological and rheumatological insights, this review aims to provide a comprehensive overview of the literature and explore the complexities of microbial involvement in PsA.

DISCUSSION

PsA has far-reaching implications for multiple domains of patients' lives and is intertwined in both the physical

and emotional disturbances of quality of life. Patients with PsA not only endure the substantial pain and physical debility related to joint disease but also face the additional burden of cutaneous disease. This dual impact is clearly reflected in their self-reported quality of life assessments. Compared to individuals with cutaneous psoriasis alone, those with PsA report markedly lower health-related quality of life across several survey instruments [12]. Furthermore, when compared to patients suffering from rheumatoid arthritis (RA), another chronic, progressive joint disease, those with PsA patients experience more bodily pain and significant limitations in life roles due to emotional disturbances [13].

Psychological distress is also notably prevalent among PsA patients, with a profound relationship between this distress and overall quality of life. Kotsis et al. found that anxiety was the most significant psychological symptom independently associated with physical health-related quality of life [14]. Additionally, they reported that 21.7% of PsA patients suffer from moderate to severe depression, but only two out of 18 of these individuals were receiving treatment for their depression. These findings highlight a critical gap in recognizing and addressing the psychological burdens of PsA, potentially leading to worsened outcomes across various domains of life.

Current research underscores the significant role of gut microbiota dysbiosis in influencing immune responses. Dysbiosis, characterized by a shift in microbiota balance, disrupts intestinal environments and immune system communication [15]. This altered state of the gut microbiome has been implicated in contributing to autoimmune conditions by promoting pro-inflammatory and immune deregulatory effects. Dietary changes can rapidly alter microbial composition; for example, changes can occur within 24 hours of switching to a diet solely based on plants or animals and revert within 48 hours of cessation [16]. The metabolic byproducts from diet, such as butyrate, propionate, and acetate, play a critical role in reinforcing the mucosal barrier. A diet rich in fats and carbohydrates, for example, can increase segmented filamentous bacteria, boosting Th17 cell numbers while reducing regulatory T cells, thus triggering autoimmune arthritis [17].

The intake of a Mediterranean diet, rich in fruits, vegetables, nuts, legumes, grains, and olive oil, while reducing the intake of red meat, dairy products, and processed foods enhances the production of short-chain fatty acids

(SCFAs) by promoting butyrate-producing bacteria like *Faecalibacterium prausnitzii* and *Akkermansia*. Probiotics, such as those found in fermented foods containing lactic acid bacteria including cultured milk products and yogurt, increase beneficial bacteria like *Bifidobacteria* and *Lactobacilli*, which produce beneficial end products such as SCFAs, particularly acetate and lactate [18]. These dietary approaches not only enhance the immune system but also inhibit pathogenic bacteria and potentially modulate the effects of dysbiosis in PsA.

Conversely, a high-fat, Western diet that is rich in animal protein and fat and low in fiber increases the incidence of bile-tolerant segmental bacteria such as *Bacteroides*, *Alistipes*, and *Bilophila*, with a reduced number of beneficial bacteria such as *Bifidobacterium*, *Eubacterium* species, *Akkermansia*, and *Lactobacillus* [16,18]. Therefore, psoriatic patients are advised to incorporate intake of probiotic-rich diets, low-FODMAP diets, and Mediterranean plant-based foods, which increase the beneficial gut microbiome and prevent most autoimmune pathologies. This dietary approach also supports innovative treatments such as IL-17 inhibitors and IL-12/23 inhibitors, which have been shown to increase the abundance of the phylum Proteobacteria and decrease *Bacteroides* and *Alistipes*, proven effective for the treatment of psoriasis and PsA [18].

Farache et al. noted that antigen-presenting cells (APCs), including dendritic cells and macrophages, play a pivotal role in initiating the inflammatory response by presenting antigens derived from the microbiome to effector T and B lymphocytes, further activating inflammation [19]. These actions by APCs could explain how changes in the gut microbiota in certain individuals, especially those with a genetic predisposition, can lead to a pathogenic state of autoimmunity.

Specific inflammatory pathways related to dysbiosis have been associated with the development and progression of PsA. Belkaid et al. postulated how dysbiosis could lead to inflammation and autoimmunity by stimulating mucosal systems to recruit Th17 lymphocytes and inhibit regulatory T cells, thus inducing local and potentially systemic inflammation [20]. This highlights the link between the intestinal microbiota and immune system dysregulation, suggesting an important role for microbial communities in driving inflammatory processes. Several studies have also reported a correlation between PsA and reported gut inflammation and complications.

Studies have shown that individuals with PsA often

have a distinct gut microbiota composition compared to healthy individuals. Ivanov et al. showed how segmented filamentous bacteria in the intestinal lamina propria impacted CD4+ helper T cells, causing a shift toward Th17-derived inflammation [21]. Individuals with PsA have decreased populations of beneficial bacteria species such as *Lactobacillus* and *Bifidobacterium*. Scher et al. found reduced numbers of certain gut bacteria (*Akkermansia*, *Pseudobutyrvibrio* species, *Ruminococcus*, *Alistipes*, *Coprococcus*) in fecal samples from PsA patients compared to controls along with increased levels of IgA, suggesting an inflammatory mucosal response [11].

Dysbiosis can lead to increased intestinal permeability, commonly known as "leaky gut syndrome." This condition allows microbial components and other antigens to enter the bloodstream, potentially triggering a systemic immune response and inflammation. Under normal circumstances, beneficial gut bacteria such as *Bacteroides fragilis* and *Clostridium* spp. produce short-chain fatty acids (SCFAs) like butyrate, acetate, and propionate. These compounds have anti-inflammatory properties and support regulatory T-cell responses, which are crucial for maintaining immune balance [22].

However, dysbiosis seen in individuals with PsA can lead to a diminished immune response. Ciccia et al. have documented how dysbiosis contributes to altered intestinal permeability in individuals with PsA, creating a proinflammatory environment. This environment is characterized by an expansion in the number of lymphoid and Th9 cells, and a reduction in T regulatory cells [23]. These changes to the gut microbiome influence the balance between effector Th1 and Th17 lymphocytes, which are essential for host defense and related to the pathogenesis of PsA [24].

Additionally, the presence of bacterial DNA in the blood of individuals with PsA and other autoimmune diseases highlights further complications arising from gut microbiota alterations. Jones et al. reported that this DNA, found in the synovial fluid, is due to increased permeability of intestinal epithelial barriers [25]. Furthermore, outer membrane vesicles, much smaller than the bacteria themselves and derived from gut bacteria, can carry bacterial DNA from the intestines into the bloodstream and then into the synovium. This mechanism illustrates the complex relationship between gut health and systemic autoimmune responses, demonstrating the importance of maintaining intestinal integrity in the management of autoimmune diseases.

Recent studies have explored how the gut microbiota can modulate the host's immune response. Links between altered miRNA expression in intestinal epithelial cells and the pathogenesis of autoinflammatory disorders have been identified. Nakata et al. highlighted the impactful effects on the expression of miRNA-21-5p induced by commensal bacteria, modulating intestinal epithelial permeability [26]. Machar et al. further demonstrated a direct correlation between miRNA-21-5p levels with IL-17 and IL-23, suggesting that miRNA-21-5p plays a role as a potential biomarker for PsA [27].

Specific microbial taxa have been linked to the development of PsA. Xiao et al. reported substantial reductions in *Eubacterium rectale* and *Alistipes spp.* in patients with PsA, indicating these microbiota as potential biomarkers of disease and targets for therapeutic intervention [28]. A pilot study by Lin et al. identified differences in gut microbiota between PsA patients and those with undifferentiated arthritis, finding a positive correlation with *Megasphaera elsdenii* in patients with PsA [29]. Another study reported a positive relationship between *Prevotella copri* and *P. stercorea* in patients with PsA, suggesting that these taxa could also serve as future potential biomarkers [30]. Fecal metabolites have also been studied in patients with PsA. Wang et al. revealed that 154 fecal metabolites were significantly altered in patients with PsA compared to controls, with five of these metabolites (α/B-turmerone, dihydrosphingosine, pantothenic acid, glutamine, and glycerol 1-hexadecanoate) indexed as potential biomarkers for PsA [31].

Understanding microbial alterations in PsA is crucial and leads to the development of targeted therapies such as probiotics, prebiotics, dietary interventions, and potentially fecal microbiota transplantation (FMT). These strategies aim to restore a healthier gut microbiome balance, reduce gut inflammation, and alleviate the symptoms of PsA. Several studies have shown the impact that dysbiosis has in patients being treated for PsA with standard therapy. Vallentini et al. reported an abundance of key gut microbial taxa, namely *Akkermansia muciniphilia* and *Bacteroides plebius*, in patients with PsA treated with biologic therapy, indicating these taxa could be used as an indicator of response to therapy [32]. An observational study by Yeh et al. investigated the effects of secukinumab, an (IL-17 inhibitor, and ustekinumab, an IL-12/23 inhibitor, on the gut microbiome in patients with psoriasis [33]. The study found that although both therapies altered the gut microbiome, secukinumab therapy was associated with a

more profound shift in the gut microbiome, which could serve as a potential indicator of response to secukinumab treatment.

FUTURE DIRECTIONS IN PSA RESEARCH

PsA is a chronic inflammatory musculoskeletal disease associated with psoriasis. Research indicates a 30% chance of patients developing arthritis within ten years of the onset of cutaneous symptoms [34]. The pathogenesis of PsA, while still not fully understood, has seen promising research on environmental factors and the pathological interactions among the skin, gastrointestinal tract, and joints in genetically predisposed individuals.

Despite multiple studies, ambiguities remain regarding the complete loss of protective taxa such as *Akkermansia* and *Echinococcus* in gut dysbiosis before or during the transition to PsA [11]. As microbiomes evolve over time, recent guidelines advocate shifting from metataxonomics, which uses 16S rRNA gene sequencing, to more advanced metagenomics. This shift enables species-level identification and functional analysis, offering insights that antiquated, culture-based methods fail to provide. Notably, a study by Scher et al. used a metataxonomics approach and observed a significant decrease in gut microbiota diversity in PsA patients compared to healthy controls, particularly in the levels of *Akkermansia* and *Ruminococcus* [11].

The ongoing longitudinal study Mi-PART (Microbiome in Psoriatic Arthritis) by Miguens Blanco et al. uses third-generation nanopore sequencing to explore the gut microbiome's role in PsA and identify specific biomarkers or signaling molecules [35]. This underscores the need for more longitudinal studies using high-throughput sequencing technologies to pinpoint microbiome changes that precede PsA onset in psoriasis patients. Identifying predictive biomarkers could significantly advance our understanding and management of PsA, especially given that 25-30% of patients are likely to develop arthritis over time.

The interplay between the host and microbiome is complex, requiring multi-omics approaches, including metagenomics, transcriptomics, and metabolomics. These techniques are invaluable in shaping our understanding of genetic makeup and metabolic functions that contribute to host-microbe interactions. Metagenomics provides insights into the taxonomic composition and functionality of organisms within specific environments, potentially revealing pathways of disease pathogenesis or treatment

responses [36]. Transcriptomics help in understanding gene expression patterns and identifying biomarkers and molecular pathways linked to inflammatory responses in diseases like psoriasis [37]. Metabolomics, on the other hand, offers a glimpse into the metabolic byproducts of microbial activity that influence host physiology and immunity [38]. Together, these approaches facilitate a more comprehensive understanding of the connections between genetic predispositions, microbial communities, and environmental factors, ultimately advancing personalized medicine, biomarker discovery, and targeted therapies [38].

MULTIDISCIPLINARY APPROACH AND PERSONALIZED MEDICINE

PsA is a complex condition that integrates both dermatological and rheumatological challenges, necessitating a collaborative, multidisciplinary team of dermatologists, rheumatologists, and other healthcare professionals. This integrated approach not only comprehensively assesses the patient's condition but also underscores the importance of each professional's role in the holistic understanding of the disease. Such collaboration is instrumental in making informed treatment decisions and in crafting personalized treatment plans that effectively address both the cutaneous manifestations of psoriasis and the joint inflammation characteristic of PsA [39]. Moreover, the potential of leveraging advancements in microbiome research to develop precise, targeted treatments for PsA is transformative. By exploring the complex interactions between the microbiome, immune system, and disease pathogenesis, researchers and healthcare providers are paving the way for more personalized and effective therapies that address the underlying causes of PsA, setting a new standard in patient care.

CONCLUSION

This comprehensive review underscores the significant impact of dysbiosis on the immune system and its association with autoimmune disorders such as PsA. Emerging microbiome research offers promising avenues for revolutionizing PsA management through personalized, targeted, and preventive strategies that address the underlying mechanisms of the disease. By leveraging the knowledge gained from the study of the gut microbiome, healthcare providers can optimize treatment outcomes and enhance the overall well-being of patients. Moving forward, it is crucial to emphasize the importance of continued research efforts, improve collaboration

across disciplines, advocate for necessary resources, and prioritize patient-centered care. By collectively advancing the frontier of microbiome research in PsA, researchers and healthcare providers can unlock the full potential of the gut microbiome as a key component in personalized therapeutic approaches. These efforts hold promise in significantly improving the lives of individuals affected by this complex autoimmune condition, paving the way for a more holistic approach to PsA care.

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