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REVIEW ARTICLES

Psoriasis: Embarking a dynamic shift in the skin microbiota

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Abstract

Recent interest has arisen regarding the role of microbiome and its composition in the pathogenesis of psoriasis. Numerous studies have shown that there are alterations in skin flora arrangement between normal individuals and psoriatic patients. Psoriasis exacerbation could be interconnected with epidermal or mucosal colonization with streptococci, *Malassezia*, *Staphylococcus aureus*, or *Candida albicans*. The role of cutaneous and gut microbiome in psoriasis pathogenesis has recently been studied in both human and animal models. In this review, we try to evaluate various pathogenic mechanisms linking the microbiota and psoriasis. The literature research included peer-reviewed articles which included clinical trials, original reports, and scientific reviews. MEDLINE and PubMed databases were searched from January 1990 to March 2021, including the reference lists of articles meeting our criteria.

KEYWORDS
microbiome, microbiota, psoriasis

1 | INTRODUCTION

The overall prevalence of psoriasis has been significantly associated with several metabolic syndromes and systemic inflammatory disorders.[1–3] In addition, stress, infections, diet, pain killers, and antibiotics are some factors that tend to elicit psoriatic lesions.[4,5] Psoriasis has been shown to have a bimodal prevalence with a major onset between the age of 20 and 30 years and then again between 50 and 60 years. Recently, the etiopathogenesis of the disease has been linked to composition of microbiota of the host.[6] Numerous researchers have observed variations in skin microflora between normal controls and psoriatic patients, suggesting a disease-associated alteration in the skin microbiome.[7,8]

The role of microflora in etiopathogenesis of psoriasis is also rendered plausible by the finding that infections caused by various microorganisms often lead to the initiation or flare up of lesions.[9,10] Psoriasis has been found to be induced or exacerbated by certain pathogens, which include bacteria such as *Staphylococcus aureus* and *Streptococcus pyogenes*, viruses such as human papillomavirus and endogenous retroviruses, and fungi such as *Malassezia* and *Candida albicans*.[11–14] Alekseyenko et al. observed that *Corynebacterium*, *Staphylococcus*, *Propionibacterium*, and *Streptococcus* are significantly increased in psoriatic lesions.[15] Fahlén et al. observed that streptococci were significantly increased in psoriatic lesions, but *Staphylococcus* and *Propionibacterium* were significantly lower in psoriatic lesions in control skin.[16] Gao et al. also found that *Propionibacterium* species were lower in psoriatic than in normal skin.[17] Researchers have observed a decrease in *Firmicutes* and an increase in *Proteobacteria* in psoriatic skin.[18] Researchers have found that *Firmicutes* are significantly increased in psoriatic lesions as compared with uninvolved skin in psoriatic patients and healthy skin controls. *Actinobacteria* and *Propionibacterium* have also been found to be reduced in the psoriatic lesion samples. Changes in intestinal microbiome may activate a systemic pro-inflammatory status, which

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may contribute to disease pathogenesis.[13] The role of gut-skin affiliation in etiopathogenesis of psoriasis has been acknowledged in both human and animal models of psoriasis.[14–17] Recovery from intestinal dysbiosis may reduce the cutaneous symptoms of psoriasis in patients.[18] Aggravation of plaque psoriasis has been linked to bacterial spread into the blood stream resulting from the enhanced intestinal permeability seen in the patients with psoriasis.[19] Researchers have also observed that certain alterations in intestinal microbial composition, which are found in patients with inflammatory bowel disease and obesity, including decreased profusion of *Akkermansia muciniphila*, are also seen in psoriatic patients.[20]

A number of studies performed with mice and human hosts provided conclusive substantiation of the effect of intestinal bacteria on skin condition.[21] It was observed that mice that were fed with *Lactobacillus reuteri*, a probiotic bacteria, had a thicker skin and regained better reproductive fitness.[22,23]

2 | DIVERSIFIED SKIN MICROBIOME

It has been observed that the changes in psoriatic skin microbiota are highly site-specific and have been shown to have greater heterogeneity as compared to the healthy skin. Changes in human microbiota content and diversity have been associated with various diseases, for example, decreased bacterial alpha diversity in gut microbiota is associated with obesity and inflammatory bowel disease,[24,25] and alteration in vaginal microbiome is seen in bacterial vaginosis.[26,27] The skin microbiome of psoriatic skin, non-lesional areas, and healthy skin consists of four prominent phyla, namely, *Actinobacteria* (53.8%–66.5%), *Firmicutes* (23.9%–28.3%), *Proteobacteria* (5.8%–12.0%), and *Bacteroidetes* (2.1%–2.9%).[25] Skin microbiome is mainly constituted of *Propionibacterium* (22.8%–38.1%), *Corynebacterium* (21.4%–23.9%), and *Staphylococcus* (5.3%–9.2%) in various cutaneous diseases. Even though the predominant species are similar in different conditions, a steady change in microbiota composition is seen in healthy skin and non-lesional skin psoriatic microflora and lesional microflora, indicating that these microbiota alterations may precede the disease onset in predisposed patients and may contribute to disease pathogenesis. *Actinobacteria* and *Proteobacteria* have been identified as differentiating markers of the skin microbiota in psoriatic lesions and healthy skin. *Conchiformibius*, *Lactococcus*, *Moraxella*, and *Acetobacter* have been found to be associated with unaffected psoriatic skin, and these species may act as possible markers for differentiating normal skin from various disease conditions.[26]

16S rRNA sequencing and LEfSe analysis have helped in identifying that the healthy skin microbiome has predominance of *Propionibacterium acnes* and *Propionibacterium granulosum*, whereas *Staphylococcus sciuri* is prominent in non-lesional psoriatic skin. It has also been observed that two *Staphylococcus* species—*S. aureus* and *Staphylococcus pettenkoferi*—are more common in lesions of psoriasis, whereas *Staphylococcus* is not significantly seen in any skin condition.[27]

3 | INTERRELATION AMONG DISTINCT BACTERIAL SPECIES

Like the case with any ecosystem, it has been seen that the symphony of skin microbiota is controlled by various environmental factors (eg, nutrient accessibility and immune response of the host) and interactions among various bacterial species. These interactions could be an important factor that affects the overall composition of the community, and thus, it is extremely important to understand these interactions which can provide important information regarding the composition and preservation of psoriasis-associated microbiome. In the psoriatic skin, three clusters of bacterial communities were identified at the genus level. Cluster A consisting of *Corynebacterium*, *Prevotella*, *Porphyromonas*, *Finegoldia*, *Peptoniphilus*, *and Anaerococcus* is the largest cluster. Cluster B consists of *Paracoccus*, *Kocuria*, *Micrococcus*, *and Janibacter*, and Cluster C consists mainly of *Rothia* and *Streptococcus*.[28]

Citing the earlier findings of impending significance of *Streptococcus* in the pathogenesis of psoriasis, the role of *Rothia* spp. in the pathogenesis of psoriasis also needs to be studied as it is extensively associated with *Streptococcus*. *P. acnes*, which is common in normal skin, negatively correlates with *S. sciuri and S. pettenkoferi*, which are more common in the psoriatic skin microbiome. In accordance with this finding, it was also observed that *P. acnes and Staphylococcus epidermidis* were highly associated with *S. aureus*-induced mild psoriatic skin tissue and *S. pettenkoferi* was highly associated with *S. aureus*-induced severe psoriatic skin tissue, indicating that the antagonistic interactions between these bacteria may take part in the pathogenesis of psoriasis. *Pseudoclavibacter bifida* has been found to be negatively associated with *P. acnes* and positively associated with *S. sciuri*. A higher concentration of *Pseudoclavibacter bifida* was seen in *S. aureus*-induced severe psoriatic skin samples. *P. acnes* and *P. granulosum* act as two major *Propionibacterium* species, and their levels positively correlate with each other. The correlation between *P. acnes* and *P. granulosum* and their interactions in normal skin indicate their role in maintaining cutaneous health.[29]

Psoriasis is characterized by indurated and inflamed plaques; hence, it is very easy to distinguish the psoriatic skin lesions from non-psoriatic and normal skin as each of them represents very distinctive microbial habitats that possibly will affect interactions between different microbes. In addition, it is also observed that a particular group of dissimilar species are present at different stages of the disease progression, confirming the assumption that diverse interactions occur in different disease conditions. Amazingly, species interactions observed in the microbiome linked with psoriatic skin lesions were more analogous to those observed in healthy skin than the non-lesional psoriatic skin. In normal skin, *P. acnes* correlates negatively with different bacterial colonization, signifying its inhibitory role in microbial growth. Lesser microbes were seen with *P. acnes* in non-lesional psoriatic skin, whereas only *Pseudoclavibacter bifida* was found to be anticorrelated with *P. acnes* in lesional psoriatic skin. On the whole, *P. acnes* might possibly take part in altering the skin

microbiome by maintaining growth of various microorganisms under control, and the alteration of this balance in psoriatic skin can contribute to disease progression.[30] Loesche et al observed that the rare bacterial species (<1%) comprised a larger proportion of psoriasis-associated microbiome than those present in non-lesional skin, which include species like *Lactococcus*, *Paracoccus*, *Porphyromonas*, *Prevotella*, *Acinetobacter*, *Neisseria*, and *Fusobacterium*.[31]

4 | MALADAPTATION OF THE SKIN MICROBIOME

The sophisticated association between microbiome and the host begins at the time of birth as the pelvic floor microflora are transmitted to the newborn during childbirth and lactation through colostrum, which is rich in microbes. The juvenile newborn immune system, which lacks full immune response, has been found to tolerate a symbiotic relationship association between microbiome. It is demonstrated by the response of newborn innate immune cells to microbial exposure, which showed a mitigated inflammatory response in comparison to the adult immune cells.[32] Microbe-associated molecular patterns (MAMPs) are the key factors responsible for interaction between the host and pathogen. An exception to the fundamental biological function of the immune system to recognize foreign molecules and subsequently initiate a directed response is that no immune response arises to host cutaneous and mucosal MAMPs, despite the constant interface of MAMPs with certain pattern recognition receptors (PRRs) that identify microbes and start an immunological response.[30] Through these mechanisms, the commensal microbiota influence the hosts' postpartum immune responses.[33] Other physiological obstacles, such as the epithelial cell layer which secretes IgA and goblet cells which produce mucus, not only separate the microbiota from having a direct interaction with mucosa but also allow it to impart localized and systemic effects.[34] Dysbiosis has arisen as a fundamental study focus in the pathogenesis of microbiome-related inflammatory disorders such as inflammatory bowel disease.[35] Dysbiosis, defined as a disparity between the microbiome and the host, can be regarded as a form of compromised homeostasis where the microbiome is altered toward a simpler and different pathological state.[36] The mechanism that brings this change from symbiosis to dysbiosis is not well-explained, but a genetic predisposition has been hypothesized.[37] Dysbiosis of microbiota in psoriatic patients enhances the translocation of bacteria from the gut and skin into systemic circulation, which, in turn, may predispose to chronic systemic inflammation in them.

5 | PORTRAYAL OF BACTERIA IN PSORIASIS

Based upon comprehensive understanding of T cells in the etiology of psoriasis, it is extremely important to establish a link between streptococcal infection and the unstable or guttate variants of psoriasis.[32] M proteins, extracted from Group A, C, and G β-hemolytic streptococci, have been avowed as decisive factors as deterioration of psoriasis was tightly linked with the M-protein-yielding streptococci.[9] It has been hypothesized that M proteins may simulate keratin factors with succeeding psoriatic T-cell activation.[9,38] This hypothesis is authenticated by the finding that interactions between type IV collagen and α1β1 integrin, which is found entirely on the epidermal psoriatic T cells, leads to the growth of these cells, leading to appearance of psoriasis.[39] T-cell triggering in guttate variants of psoriasis is altered by the effect of antigens such as streptococcal pyogenic toxins A and B and peptidoglycans.[40–42] It can be postulated that the skin microbiome plays a part in etiopathogenesis of psoriasis. Bacteria such as *Propionibacterium*, *Staphylococcus*, *Corynebacterium*, *and Streptococcus* have been recognized as the main bacterial genera which may take part in psoriasis pathogenesis. Researchers have identified *Firmicutes* as the most common bacterium found in psoriatic lesional skin, and the *Actinobacteria* count was significantly less in lesional psoriatic skin than non-lesional and healthy skin.[42,43]

Researchers have observed that *S. aureus* proteins increase Th17 differentiation in vitro, suggesting that *S. aureus* colonization can cause elevated Th17 activation and IL-17 secretion.[32,35] Researchers have reported higher Th17 transcriptomic signals and IL-17A and IL-17F cytokine transcripts in Teff cells collected from the skin *S. aureus*-colonized mice.[26] IL-17A is identified as one of the major agents in pathogenesis of psoriasis. Researchers also observed that other mediators of Th17 responses, such as IL23R and IL22, were also elevated upon *S. aureus* exposure in the keratinocytes, which may lead to IL-17-induced inflammation in psoriasis.[29]

6 | ROLE OF FUNGI IN PSORIASIS

Several studies have established a possible etiopathogenic correlation between psoriasis and fungi. In 1980s, development of psoriatic plaques was observed in all 10 tested subjects as a result of application of *Malassezia ovalis* suspension to their unaffected skin.[44] Similar plaques were observed after secondary fungal deposition from the scalp of psoriatic patients. In a case series, a week-long course of oral antifungal therapy was reported to improve psoriatic lesions.[45] Increased *Malassezia* yeast colonization in lesional skin has been found to cause psoriasis exacerbations.[46]

Also, the anecdotal observation that topical miconazole regularly improves inverse psoriasis could be compatible with a role of fungi in psoriasis (authors' own observation). The role of *Malassezia* is largely attributed to its action to upregulate tumor growth factor-β1, HSP70, and integrin chain expressions, leading to increased migration of immune cells and keratinocyte hyperproliferation in psoriatic patients.[47,48] *Malassezia furfur* induces higher IgG and lower IgM production in psoriatic patients than in normal subjects.[49–51] As seen with bacterial microbiome, nonculture techniques have also succeeded over culture techniques in detecting *Malassezia* in psoriatic and normal skin. *M. restricta* has been identified as the most common

Malassezia species in psoriatic skin. In spite of the perpetual findings, the mere identification of fungus itself is not an indicator to extricate psoriatic from non-psoriatic and healthy skin.[52,53]

7 | CONCLUSION

The interaction of the immune system and microbiota is of significance because it can lead to better understanding of psoriasis pathogenesis and, at the same time, give clearance for expansion of microbiome-targeted therapies. Although reports state a higher variation of the microbial population in psoriatic lesions and an intestinal dysbiosis in psoriatic patients, it is yet to be determined whether such changes in microbiota are the etiology or consequence of the disease. Further research is warranted to explore the precise role of microbiota in the management of psoriasis.

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