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Association Between Psoriasis and Gut Microbiota: A Review

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ABSTRACT

Psoriasis is a systemic immune-mediated inflammatory disease that affects target organs and manifests primarily through skin lesions resulting from keratinocyte hyperproliferation. This condition can significantly reduce the quality of life. Psoriasis is widespread: according to the scientific data, it affects approximately 2% of the global population. Research in the psoriasis pathogenesis and its systemic effects remains a relevant focus in dermatology. Numerous recent studies have identified a correlation between psoriasis and inflammatory bowel diseases.

Psoriasis has been found to reduce both the qualitative and quantitative diversity of the gut microbiota, as well as to increase in opportunistic bacteria such as *Escherichia coli*, *Helicobacter* spp., and *Mycobacterium* spp. In addition, patients with psoriasis exhibit an increased abundance of *Firmicutes* and a decrease in *Bacteroides*. A decrease in *Bacteroides*, in turn, reduces the production of butyrate, which plays a key role in protecting the intestinal epithelium. Alterations in the gut microbiota may contribute to the stimulation of autoimmune inflammation in psoriasis. Many researchers also agree that severe psoriasis is characterized by significantly altered microbiota in the study groups compared with controls. Intestinal dysbiosis may serve as a trigger for psoriasis relapse. Increased intestinal epithelial permeability contributes to the entry of a greater number of bacterial metabolites into the bloodstream, which in turn aggravates the course of psoriasis.

Clinical observations confirm the improvement of psoriatic skin lesions following the use of antibiotics, probiotics, or fecal microbiota transplantation. A detailed investigation of the relationship between psoriasis and the gut microbiota may serve as a potential marker for therapy assessment, improve treatment quality, and enhance the quality of life in affected patients.

Keywords: psoriasis; intestinal microbiota; inflammatory bowel diseases; intestinal dysbiosis; immune-mediated inflammatory diseases; keratinocyte hyperproliferation; immune response.

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Взаимосвязь псориаза и микробиоты кишечника: обзор литературы

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АННОТАЦИЯ

Псориаз — системное иммуновоспалительное заболевание, поражающее органы-мишени и проявляющееся главным образом образованием дефекта на коже вследствие гиперпролиферации кератиноцитов. Это заболевание может значительно снижать качество жизни. Псориаз распространён повсеместно: по литературным данным, он затрагивает около 2% населения планеты. Изучение патогенеза псориаза и его влияния на организм является актуальным направлением в дерматологии. Многочисленные работы последних лет обнаруживают определённую корреляцию между псориазом и воспалительными заболеваниями кишечника.

Установлено, что при псориазе наблюдается снижение качественного и количественного разнообразия микрофлоры кишечника и увеличение числа условно-патогенных бактерий, например *Escherichia coli*, *Helicobacter* spp., *Mycobacterium* spp. Наряду с этим у больных псориазом выявляется увеличение количества бактерий типа *Firmicutes* и снижение — *Bacteroides*. Уменьшение количества *Bacteroides* в свою очередь ведёт к уменьшению образования бутирата, который играет роль в защитной системе эпителия кишечника. Изменение микробиоты кишечника может являться причиной стимуляции аутоиммунного воспаления при псориазе. Многие исследователи также сходятся во мнении, что тяжёлое течение псориаза характеризуется значительно изменённой микробиотой у исследуемых групп по сравнению с контрольными. Дисбиоз кишечника может являться стимулом рецидива псориаза. Повышение проницаемости кишечного эпителия способствует проникновению большего количества бактериальных метаболитов в кровь, что в свою очередь усугубляет течение псориаза.

Клинические наблюдения подтверждают улучшение псориатических поражений кожи после применения антибиотиков, пробиотиков или трансплантации фекальной микробиоты. Детальное изучение взаимоотношения псориаза и микробиоты кишечника может служить потенциальным маркером оценки проводимой терапии, улучшать её качество и повышать уровень жизни пациентов с данной патологией.

Ключевые слова: псориаз; микробиота кишечника; воспалительные заболевания кишечника; дисбиоз кишечника; иммуновоспалительные заболевания; гиперпролиферация кератиноцитов; иммунный ответ.

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INTRODUCTION

Psoriasis is a multifactorial T cell–mediated immune-inflammatory disease. The inflammatory process develops as a result of the formation of autoreactive T lymphocytes that damage target organs [1–3]. In the past, psoriasis was often confused with other dermatological conditions, particularly leprosy, due to the similarity of symptoms—patches and scaling of the skin. However, in 1841, the dermatologist Ferdinand von Hebra proposed classifying psoriasis as a separate disease entity [4].

The prevalence of psoriasis ranges from 0.1% to 8%, depending on the geographical region and the patient's ethnic background. It affects 125 million people worldwide and more than 2% of the population in Russia. Psoriasis is less common in certain regions of Asia and Africa, and more prevalent in Scandinavian countries. Since 2013, an increasing incidence of psoriasis has been noted among individuals over the age of 18 years [1, 3, 4]. Recent studies indicate that the core pathogenetic mechanisms of psoriasis involve a triad of elements: dendritic cells producing interleukin (IL)-23, Th17 cells secreting IL-17, and activated keratinocytes. Proinflammatory cytokines and proliferation-stimulating factors in psoriasis are synthesized by T cells (IL-17, IL-21, IL-22, interferon-gamma), dendritic cells (tumor necrosis factor-alpha, IL-6, IL-20, IL-23, nitric oxide), and keratinocytes (antimicrobial peptides, IL-20, chemokines) [1, 5, 6]. The formation of the immune response in the epidermis begins in the areas of keratinocytes sensitive to deoxyribonucleic acid. The activation signal from keratinocytes is transmitted to the cells of the myeloid lineage—Langerhans cells, which are present not only in the epidermis but also in other barrier tissues such as the intestinal and bronchial epithelium. Langerhans cells have dendritic processes through which they connect with one another, forming a barrier to the penetration of pathogenic molecules. Foreign antigens or autoantigens are taken up by Langerhans cells, undergo subsequent processing, and are presented as antigenic structures together with major histocompatibility complex class I–II molecules on the cell surface. Concurrently, these cells migrate into the dermis, where they recruit plasmacytoid dendritic cells into the inflammatory process [2]. It has been established that the expression of Ki-67 and keratins 6, 16, and 17 contributes to epidermal hyperplasia, impaired epithelial differentiation, and inflammation in the dermis. These molecules serve as indicators of keratinocyte hyperproliferation in psoriatic lesions. Keratinocytes mature in the basal layer and reach the skin surface within 6–8 days, whereas in healthy skin, the keratinization process takes approximately 40 days [5].

ALTERATION OF MICROBIOTA COMPOSITION

When studying gut microbiota in psoriasis, it is important to consider the intestinal microbial status of patients with diseases associated with psoriasis [7]. The obligate microbiota involved in metabolic processes and intestinal protection against pathogens consists primarily of anaerobic bacteria such as *Propionibacterium*, *Bacteroides*, and *Bifidobacterium.* Although these bacteria exhibit low diversity, their population density is consistently high. The facultative microbiota includes conditionally pathogenic bacteria such as *Escherichia*, *Fusobacterium*, *Peptostreptococcus*, *Enterococcus*, *Clostridium*, *Eubacterium*, and others. Due to the influence of various factors, the qualitative and quantitative composition of this flora may change throughout life. Transient flora accounts for less than 1% and consists of microorganisms that temporarily enter the gastrointestinal tract from the external environment, including *Clostridium*, *Staphylococcus aureus*, *Proteus*, *Candida*, and others [8]. Maintaining a healthy immune system depends on a balanced composition of commensal gut bacteria. Human immune cells—such as T-lymphocyte subpopulations, neutrophils, natural killer cells, and macrophages—are sensitive to changes in microbiota composition. Both the loss of beneficial microbiota and the overgrowth of pathogenic microorganisms exert a direct negative effect on intestinal immune cells [9]. The relationship may also be mediated by immunological disorders, primarily involving an imbalance between pro- and anti-inflammatory cytokines [10].

Patients with psoriasis exhibit lower gut microbiota diversity compared with the control group [11]. Researchers have noted an association between intestinal dysbiosis and some comorbidities commonly observed in psoriasis, including metabolic syndrome, arthritis, depression, cardiovascular diseases, and inflammatory bowel diseases (IBD) [12, 13]. Intestinal microbiota dysbiosis may act as a trigger or even a cause of psoriasis relapses. It has been observed that patients with plaque psoriasis more frequently present with increased intestinal permeability, which leads to the translocation of bacterial DNA from the gut into the bloodstream. The confirmation of microbiota alterations has become possible through the use of genome sequencing techniques. This method allows for the identification of cases where the gut microbiota serves as a pathogenic factor in the course of psoriasis.

It has been established that dysbiosis in patients with IBD shows similarities to that found in patients with psoriatic arthritis [9]. Studies indicate that intestinal microbiota dysbiosis results in gut barrier disruption and bacterial translocation. This can contribute to the development of chronic systemic inflammatory diseases, positioning dysbiosis as a potential trigger of psoriasis due to the release of inflammatory mediators such as lipopolysaccharides and lipoteichoic acid, as well as due to the activity of both Gram-negative and Gram-positive bacteria [14]. The IL-23/IL-17 signaling pathways play a key role in the pathogenesis of both skin and intestinal inflammation. Therefore, alteration of the gut microbiota through systemic regulation of the immune system may help

reduce cutaneous inflammation [15]. The gut immune system closely interacts with its resident microbiota. The immune response involves T and B lymphocytes, T regulatory cells, dendritic cells, and macrophages. Antigen presentation is mediated by microfold cells located between epithelial cells. Toll-like receptors (TLRs) located on the epithelial membrane recognize components of microbial cells. Dendritic cells present antigens to T cells, thereby initiating the adaptive immune response [16]. In dysbiosis, the permeability of the intestinal wall increases. Once bacterial antigens enter liver tissue through the portal vein and biliary tract, antigen-presenting cells (such as Kupffer cells and macrophages) recognize foreign agents and stimulate the production of pro-inflammatory cytokines, leading to active immune-mediated inflammation [17].

When examining the relationship between the course of psoriasis and alterations in the gut microbiota, it is important to consider the increased translocation of bacterial metabolites across the intestinal barrier. This phenomenon is also observed in IBD [12]. Psoriasis can be regarded as a systemic condition, as it affects multiple organ systems, including the cardiovascular system (e.g., ischemic heart disease, arterial hypertension), the endocrine system (e.g., type 2 diabetes mellitus, obesity, non-alcoholic fatty liver disease), the musculoskeletal system, the gastrointestinal tract (e.g., IBD), and the urinary system. However, the most visible manifestations of psoriasis are typically skin changes resulting from impaired keratinocyte proliferation and differentiation. The above-mentioned comorbidities often accompany psoriasis, as they share common pathogenetic mechanisms driven by a systemic immune-inflammatory response [6,13]. There is a high incidence of psoriasis associated with IBD, particularly Crohn disease and ulcerative colitis [18]. IBD are caused by reduced tolerance of the intestinal immune system to the gut microbiota, which under normal conditions does not cause harm but rather benefits from coexistence with the host organism. IBD are accompanied by dysbiosis, as evidenced by elevated levels of plasma immunoglobulins against bacteria. IgA and IgG antibodies to *Saccharomyces cerevisiae* are found in 29%–69% of patients with Crohn disease, whereas 24%–55% have antibodies to the transport protein OmpC of *E. coli* and to Cbir1 flagellin [7]. The existence of a gut–microbiome–skin axis has been confirmed in both psoriasis and IBD. Compared with healthy controls, patients with psoriasis exhibit lower fecal concentrations of *Faecalibacterium prausnitzii* and elevated levels of *E. coli*. Similar results have been reported in patients with IBD. Notably, patients with IBD-associated psoriasis show the greatest decrease in *F. prausnitzii* and the greatest increase in *E. coli* concentrations [19]. Thus, alterations in the gut microbiota may serve as a potential biomarker for evaluating treatment efficacy in psoriasis.

Similar gut microbiota alterations have been observed in patients with IBD and psoriasis. Both conditions are associated with decreased levels of *Bifidobacterium* spp., *F. prausnitzii*, *Lactobacillus* spp., *Coprobacillus*, and *Parabacteroides*, along with increased levels of *Campylobacter* spp., *E. coli*, *Salmonella* spp., *Helicobacter* spp., and *Alcaligenes* spp. [20–22].

It has been shown that an unbalanced diet and excessive body weight lead to the development of a systemic inflammatory response. This factor plays a significant role in the comprehensive management of psoriasis [23].

In a case-control study by Schade et al., patients with psoriasis were found to have a reduced count of the genera *Ruminococcus*, *Lachnospira*, *Blautia*, and the species *Akkermansia muciniphila*. An increased abundance of the genus *Dialister* and the species *Prevotella copri* was also observed [11]. Studies of the gut microbiota in patients with psoriasis revealed a decreased number of members of the family *Ruminococcaceae* and of the genera *Coprococcus* and *Blautia*, compared with the control group [24, 25]. Shapiro et al. found that patients with psoriasis had an increased abundance of bacteria from the phyla *Firmicutes* and *Actinobacteria* compared with the controls. Specifically, in patients with psoriasis compared with the control samples, the number of *Collinsella aerofaciens*, *Dorea formicigenerans*, and *Ruminococcus gnavus* was significantly increased, whereas the number of *Parabacteroides distasonis* and *Prevotella copri* was decreased [26]. In a study by Dei-Cas et al., alterations in the gut microbiota composition were associated with the condition of patients with psoriasis. The study was conducted using the weighted UniFrac analysis. In patients with psoriasis, an increased abundance of bacteria of the *Firmicutes* phylum and a decreased abundance of *Bacteroides* was observed. In patients with psoriasis, there was also a higher abundance of bacteria of the genera *Faecalibacterium* and *Blautia*, whereas in individuals from the control group without psoriasis, *Bacteroides* and *Paraprevotella* predominated [27]. In the study by Yegorov et al., an increased abundance of *Faecalibacterium* and a decreased abundance of *Oscillibacter* and *Roseburia* were observed in patients with psoriasis [28]. The study showed that imiquimod-induced psoriasis was more severe in mice that had received a fecal microbiota transplant from the patients with psoriasis compared with those that received microbiota from healthy donors. Differences were also observed in gut and skin microbiota between mice receiving transplants from healthy and psoriatic donors. 16S rRNA sequencing revealed that *Lactobacillus reuteri* was significantly more abundant in the gut and skin microbiota of mice that received healthy donor microbiota. Interestingly, supplementation with *L. reuteri* enhanced the expression of the anti-inflammatory gene *IL-10*, reduced the number of Th17 cells, and provided resistance to imiquimod-induced inflammation in the mice with disrupted gut microbiota [29]. Abnormal colonization of the gut by *C. albicans* and *S. aureus* can alter the microbiota and potentially act as pathogenic factors in psoriasis [30]. Imiquimod-induced psoriasis is accompanied by alterations in the composition of the gut microbiota. The altered microbiota produces higher levels

of succinate, which induces proliferation of CX3CR1hi macrophages in the colon and enhances cytokine synthesis. In mice with imiquimod-induced psoriasis, administration of dextran sulfate sodium into the intestine resulted in more severe colitis compared with the controls. These findings showed that the altered intestinal environment in psoriasis facilitates the development of IBD [31]. According to a study by Li et al., oral administration of abietic acid to mice reduced imiquimod-induced psoriasiform inflammation. Sequencing also showed a decreased relative abundance of inflammation-associated gut bacteria (such as *Anaerotruncus* and *Christensenella*) [32]. The studies have demonstrated that microbiota alterations correlate with elevated inflammatory marker levels observed in patients with psoriasis. IL-2 receptor levels were positively correlated with *Phascolarctobacterium* and negatively with *Dialister*. Therefore, the relative abundance of *Phascolarctobacterium* and *Dialister* may serve as predictive markers of psoriasis activity. Comparison of the gut microbiota between healthy individuals and patients with psoriasis revealed significant differences at the family and genus levels. The microbiota of patients with psoriasis is characterized by lower levels of the *Lachnospiraceae* family bacteria and higher levels of *Veillonellaceae* and *Ruminococcaceae* bacteria. Differences at the genus level included *Faecalibacterium* and *Megamonas* bacteria, which were more abundant in psoriasis patients compared with the controls. The central complement system component C3 was noted to negatively correlate with *E. coli* abundance [33]. Patients with psoriasis typically have increased levels of *E. coli* in their microbiota [19]. *E. coli* is a facultative, conditionally pathogenic microorganism, normally accounting for no more than 10% of the gut flora [8]. An analysis using the LEfSe method showed that the abundance of *E. coli* in patients with psoriasis was significantly higher than in healthy individuals [34].

CORRECTION OF GUT MICROBIOTA

It has been demonstrated that the use of probiotics in psoriasis treatment reduces keratinocyte hyperproliferation and alleviates psoriatic skin manifestations. Thus, the use of probiotics exerts a protective effect against the development of clinical signs of psoriasis. A reduction in proinflammatory biomarkers has been observed in 75% of patients after daily intake of probiotics for 6–8 weeks [21]. It is important to note the potential anti-inflammatory role of probiotics, as their intake has been shown to reduce plasma levels of C-reactive protein and proinflammatory cytokines [22].

Balneotherapy has been found to increase the abundance of bacterial taxa that are reduced in psoriasis. The composition of gut microbiota in treated patients contains more bacteria associated with favorable metabolic health and fewer bacteria linked to adverse metabolic profiles. This suggests that balneotherapy may help improve the course of psoriasis [24].

The microbial community was found to be more diverse in the group of the patients with psoriasis who showed a positive response to secukinumab, compared to the patients not receiving genetically engineered biological therapy and healthy individuals. In the group receiving genetically engineered biologic therapy, the gut microbiota profile was characterized by an increased abundance of bacteria of the phylum *Firmicutes* and the family *Ruminococcaceae*, and a decreased abundance of the phylum *Bacteroidetes*. Metagenomic analysis demonstrated that the metabolic functional pathway was altered following secukinumab therapy and contributed to a more stable homeostasis of the gut microbiota [35]. *Firmicutes* and *Bacteroidetes* are known to participate in the synthesis of propionic and butyric acids, promote the production of anti-inflammatory cytokines (IL-10), and—through the interaction of their cell wall polysaccharides with antigen-presenting cells—activate regulatory T cells [36]. Butyrate plays a crucial role in maintaining the epithelial barrier and mediating anti-inflammatory effects. It can also suppress oxidative stress and regulate the Th17/Treg cell balance [37]. The severity of the inflammatory response can be reduced by establishing new dietary habits. A high-fiber diet increases the proportion of bacteria capable of fermenting plant fibers into butyrate: *F. prausnitzii*, *Blautia*, *Eubacterium rectale*, *Roseburia*, as well as bacteria of the phyla *Actinobacteria* (*Bifidobacteria*, *Lactobacilli*) and *Bacteroidetes*. A diet rich in animal products favors the growth of bile acid-resistant bacteria, including *Bacteroides*, *Bilophila*, *Clostridium*, and *Alistipes*, which also produce butyrate [8]. Administration of a calorie-restricted diet resulted in a 75% improvement from baseline in the Psoriasis Area and Severity Index (PASI) score [38]. Psoriasis worsening in the setting of gut dysbiosis may be attributed to the following factors: disruption of immune homeostasis; altered quantitative balance of bacteria producing short- and medium-chain fatty acids; and increased intestinal permeability [12]. A general decrease in microbial diversity may affect the balance between individual short-chain fatty acids: acetate synthesis increases, whereas butyrate synthesis decreases. As acetate concentration is positively correlated with ghrelin levels and insulin resistance, whereas butyrate levels are negatively associated with inflammation, changes in the abundance of *Firmicutes* and *Bacteroidetes* in the intestine—which influence short-chain fatty acid production—may represent another common pathway in the pathogenesis of psoriasis and obesity, which are frequently linked [37]. A Brazilian study found that *Akkermansia muciniphila* and *Lachnospira* were less abundant in patients with psoriasis compared with the controls [11]. A reduction in *A. muciniphila* in patients with psoriasis was confirmed by 16S rDNA sequencing of gut microbiota [39]. These changes are associated with disrupted butanoate metabolism and decreased production of butyrate by the colonic microbiota. Butyrate plays a significant role in regulating various inflammatory factors, including

lipopolysaccharides, tumor necrosis factor-alpha, IL-10, and IL-1β [40]. Keratinocytes contain inflammasomes—protein complexes that trigger cascades of inflammatory responses. One of the key proteins involved is caspase-1. In a study by Okada et al., mice with keratinocyte-specific caspase-1 exhibited higher levels of *S. aureus* and *Streptococcus danieliae* in their microbiota. Oral administration of these bacteria led to exacerbation of skin lesions. In addition, elevated concentrations of pro-inflammatory cytokines were detected in blood serum [41].

The studies indicate a beneficial effect of antibacterial and immunomodulatory therapy in psoriasis. The use of broad-spectrum antibiotics reduces the Th1 immune response, thereby alleviating cutaneous inflammation. A pronounced anti-inflammatory effect has also been observed with immunologic agents such as secukinumab and ustekinumab [13]. Clinical observations confirm a reduction in psoriatic skin lesions following the use of antibiotics, probiotics, or fecal microbiota transplantation, the essence of which lies in transferring intestinal bacteria contained in the stool of a healthy donor into the recipient's gut to normalize microbiota function and restore bacterial balance by competitively displacing pathogenic microorganisms [13]. It is suggested that streptococcal degradation products in the gut may contribute to psoriatic eruptions. Due to impaired intestinal motility, the small intestine may become colonized by *Streptococcus pyogenes* antigens, which in turn activates the immune response and induces epidermal cell proliferation [42].

A study by Buhaș et al. demonstrated that probiotics and prebiotics containing *Bacillus* spp. may serve as an effective and safe adjunct to conventional psoriasis therapy. In patients receiving standard treatment in combination with dietary supplements, lower PASI scores and body mass index values were observed compared with the control group. Improvements were also associated with modulation of cytokine activity (including decreased levels of tumor necrosis factor alpha, IL-6, and interferon gamma, and increased IL-10 expression). Supplementation enhanced gut microbiota diversity by increasing the *Firmicutes*/*Bacteroidetes* ratio, decreasing the *Prevotella*/*Bacteroidetes* ratio, and reducing the abundance of lipopolysaccharide-producing bacteria. A decrease in *Prevotella* spp., *Bacteroidetes* spp., *Clostridium difficile*, and *P. copri* was observed, whereas the abundance of *Verrucomicrobia*, *A. muciniphila*, and *Ruminococcus* spp. increased [43].

In another study, patients receiving capsules containing *L. acidophilus*, *Bifidobacterium bifidum*, *B. animalis subsp. lactis*, and *Bifidobacterium longum* at concentrations of no less than 1.6×10^9 CFU/g exhibited reductions in serum lipopolysaccharide and IL-1β levels in serum, as well as significant improvements in the quality of life and PASI scores compared with the placebo group [44]. A systematic review and meta-analysis confirmed that probiotic supplementation alleviates the severity of psoriasis symptoms by improving skin condition and reducing inflammation. The authors also emphasize the need for further research with larger sample sizes and clearly standardized protocols to confirm and clarify these findings [45].

CONCLUSION

The immune responses in the gut and skin are mediated to some extent by the presence of their unique microbial communities. In psoriasis and in inflammatory bowel alterations of various origins, identical qualitative and quantitative changes in the gut microbiota have been observed. Several studies have demonstrated consistent patterns, including an increased abundance of *Campylobacter* spp., *E. coli*, *Salmonella* spp., and *Helicobacter* spp., and a decreased abundance of *Paraprevotella*, *Oscillibacter*, and *Roseburia*. UniFrac-based analyses have revealed an increased abundance of bacteria of the phylum Firmicutes and a decreased abundance of *Bacteroides*. Excessive colonization of the gut by opportunistic pathogens such as *C. albicans* and *S. aureus* has been shown to contribute to the pathogenesis of IBD. These conditions can act as triggers for the development of psoriasis, exacerbate its course, or lead to recurrence. Conversely, the generation of an immune response in psoriasis may provoke damage to the intestinal barrier and translocation of bacterial flora, thereby disturbing the gut microbiota. Some patterns have been identified to indicate the involvement of common IL-23/IL-17 signaling pathways in both skin and intestinal inflammation, as well as an increased risk of IBD associated with altered gut microbiota in psoriasis. It has been found that the IL-2 receptor is positively correlated with *Phascolarctobacterium* and negatively correlated with Dialister. Accordingly, these bacterial genera may serve as indicators of inflammation severity in psoriasis.

A detailed understanding of the relationship between the gut microbiota and psoriasis provides a rationale for assuming that pharmacological interventions targeting the microbiota to improve its qualitative and quantitative composition may also reduce the clinical manifestations of cutaneous inflammation in psoriasis. Oral administration of probiotics (such as *Bacillus* spp., *Lactobacillus acidophilus*, *B. bifidum* at concentrations of at least 1.6×10^9 CFU/g) is most commonly reported to be effective, as it reduces the activity of proinflammatory cytokines and increases that of anti-inflammatory cytokines. Although modulation of the gut microbiota is not a primary treatment for psoriasis, it continues to demonstrate its effectiveness as an adjunct therapy, lowering the PASI score, thus easing patients' daily life and enhancing their chances of remission.

Analyzing the gut microbiota during treatment with various drug classes is important for predicting therapeutic efficacy and risks associated with pharmacological interventions in psoriasis. Thus, the gut microbiota may serve as a potential biomarker for assessing the effectiveness of the primary therapy.

ADDITIONAL INFORMATION

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