Exploration of Musculoskeletal Diseases

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Open Access Letter to the Editor

Dysbiosis as a common pathogenetic mechanism in psoriasis and gastrointestinal diseases

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Academic Editor: Fernando Pérez-Ruiz, Cruces University Hospital, Spain

Received: November 5, 2024 **Accepted:** December 18, 2024 **Published:** January 10, 2025

Cite this article: Zavos C. Dysbiosis as a common pathogenetic mechanism in psoriasis and gastrointestinal diseases. Explor Musculoskeletal Dis. 2025;3:100778. https://doi.org/10.37349/emd.2025.100778

Keywords

Psoriasis, gastrointestinal diseases, inflammatory bowel disease, Crohn's disease, dysbiosis

Dr. Sabooniha's recent review [1] in Exploration of Musculoskeletal Diseases provided a detailed overview of the overlapping epidemiological, genetic, and immunological factors affecting the intriguing and complex relationships between psoriasis and gastrointestinal diseases, particularly inflammatory bowel disease (IBD) and Crohn's disease (CD). However, the paper does not address an increasingly recognized factor that may be relevant to psoriasis pathogenesis: dysbiosis of the gut microbiota. Emerging evidence indicates that imbalances in gut microbial communities are frequently observed in patients with psoriasis and related gastrointestinal disorders. Factors like aging, diet, environment, and genetics may all cause gut dysbiosis and increase both the risk of developing psoriasis and impairing recovery.

Extensive research demonstrates that patients with psoriasis exhibit distinct microbial profiles compared to healthy individuals. Specifically, studies have shown that psoriasis patients often present with increased levels of *Firmicutes* and *Actinobacteria* and reduced levels of *Bacteroidetes* [2, 3]. This altered microbial composition correlates with reduced microbial diversity and changes in specific bacterial genera, such as an increased presence of *Faecalibacterium* and *Megamonas*. Notably, *Faecalibacterium prausnitzii*, which has anti-inflammatory properties, plays a key role in maintaining gut homeostasis [4].

The ecological imbalance of the gut microbiota is linked to inflammatory pathways associated with psoriasis and gastrointestinal diseases. Shifts in microbial composition may disrupt the intestinal mucosal barrier, potentially triggering local and systemic immune responses. Variations in the abundance of certain genera, such as *Phascolarctobacterium* and *Dialister*, have been correlated with inflammatory markers in psoriasis patients. For instance, *Phascolarctobacterium* levels show a positive correlation with interleukin (IL)-2R, an immune activation marker, while *Dialister* exhibits an inverse relationship, suggesting these bacteria may serve as indicators of inflammatory activity and disease progression [5]. Additionally, studies report lower levels of medium-chain fatty acids (MCFAs) such as caprylic and capric acids in the fecal samples of patients with psoriasis and psoriatic arthritis. Given the antimicrobial properties of MCFAs, their

reduction could contribute to an imbalanced gut microbiome, potentially impacting gut health and inflammation [6].

Genetic research has also identified associations between specific bacterial taxa and psoriasis risk, suggesting that the presence of certain microbial communities could influence disease susceptibility. For example, genera such as *Prevotella*, *Eubacterium*, *Lactobacillus*, *Odoribacter*, and *Slackia* have been genetically associated with an increased risk of psoriasis [7]. While these associations do not imply causation, they support the notion of interconnected genetic and microbial pathways in psoriasis and gastrointestinal conditions.

Dr. Sabooniha's discussion of immunological pathways, specifically the IL-17/IL-23 axis, highlights the role of immune dysregulation in these diseases. Dysbiosis could be an additional factor contributing to inflammatory responses in these pathways. While causal relationships remain to be definitively established, changes in gut microbiota may amplify the pro-inflammatory environment seen in patients with psoriasis and IBD. Integrative approaches that address both microbial imbalances and immune dysregulation, such as microbial restoration and immunomodulation therapies, could offer a more comprehensive approach to treatment.

To further enhance the discussion in Dr. Sabooniha's review, future research may benefit from a focus on the gut microbiota's role in shaping the inflammatory profiles of psoriasis and gastrointestinal diseases. By examining dysbiosis alongside genetic and immunologic factors, future studies could provide a more holistic understanding of the mechanisms that underpin psoriasis and its association with gastrointestinal comorbidities. Such approaches could open avenues for innovative treatments aimed at restoring microbial balance, potentially offering new hope for patients with psoriasis, IBD, and related conditions. To date, strategies focus on manipulating the microbiome via fecal microbiota transplantation, administration of prebiotics and probiotics, and dietary interventions.

Abbreviations

IBD: inflammatory bowel disease

IL: interleukin

MCFAs: medium-chain fatty acids

Declarations

Acknowledgments

Dr. Zavos can be found at https://peptiko.gr/en/ for more information.

Author contributions

CZ: Conceptualization, Investigation, Writing—original draft, Writing—review & editing.

Conflicts of interest

The author declares that he has no conflicts of interest.

Ethical approval

Not applicable.

Consent to participate

Not applicable.

Consent to publication

Not applicable.

Availability of data and materials

Not applicable.

Funding

Not applicable.

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