Unravelling the gut–skin axis: the role of gut microbiota in pathogenesis and management of psoriasis

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Inflammopharmacology

Aims and scope

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Arun Pachauri & Shweta Sharma

Abstract

Psoriasis is a chronic, multifactorial, inflammatory skin disease, increasingly recognized as a systemic disorder influenced by the gut–skin axis, which is a dynamic bidirectional communication between intestinal microbiome and cutaneous immune response. This narrative review explores the understanding of the gut–skin axis with the latest evidence on how gut dysbiosis occurs in psoriasis, characterized by reduced microbial diversity and its shifts, and how it contributes to pathogenesis and exacerbation of psoriasis. Notably, recent

scientific literature evidence suggests that the alteration of gut microbiome in psoriasis includes a decreased level of beneficial species like *Faecalibacterium prausnitzii* and a rise in the level of proinflammatory bacterial species like *Prevotella copri*. Mechanistic insights reveal that gut-derived metabolites, impaired barrier functions, and immune signaling, particularly involving IL-23 and Th17 cells, play a pivotal role in this axis, linking intestinal health to cutaneous manifestations. Both animal and human trials underscore the therapeutic potential of interventions targeting the gut microbiota, including prebiotics, probiotics, dietary modifications, and FMT, demonstrating some promising but variable effects on disease severity and systemic inflammation. Despite these advances, translating the gut–skin axis into clinical practice presents a notable challenge due to limited scientific evidence, a lack of standardised microbiome profiling, and the absence of universally accepted biomarkers to monitor and stratify therapeutic outcomes. These limitations hinder the development of personalised care approaches and the integration of the gut–skin axis as a promising frontier in many autoimmune diseases, where the gut–skin axis and the intestinal microbiome play a crucial role.

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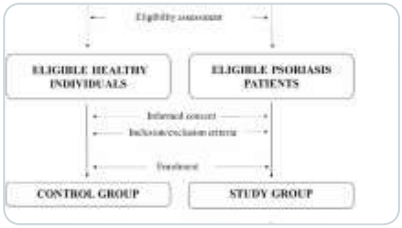

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Data availability

This review is based on previously published studies, which are cited throughout the article. No new dataset were generated.

Abbreviations

IL: Interleukin

TNF: Tumor necrosis factor

SCFAs: Short chain fatty acids

Th: T helper

DC: Dendritic cells

Tregs: Regulatory T cells

GI: Gastrointestinal

IBD: Inflammatory bowel disease

FMT: Fecal microbiota transplantation

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Author information

Authors and Affiliations

Parul Institute of Pharmacy, Vadodara, Gujarat, India

Arun Pachauri & Shweta Sharma

Corresponding author

Correspondence to Arun Pachauri.

Ethics declarations

Conflict of interest

The authors declare that there is no conflict of interest.

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