Volume 4, Issue 2, February 2026 ISSN (E): 2938-3765

GUT MICROBIOTA-ASSOCIATED IMMUNE MODULATION IN PATIENTS WITH ALLERGIC DISEASES

Abdullayev Farhodbek Abdumutalib o‘g‘li
Clinic Immunolog and Allergolog Sehat Clinic

Abstract
Allergic diseases represent a growing global health challenge, with increasing prevalence across both developed and developing countries. Recent advances in immunology and microbiology have highlighted the pivotal role of the gut microbiota in regulating host immune responses and maintaining immune homeostasis. This article examines the mechanisms through which gut microbiota modulates immune function in patients with allergic diseases. Particular attention is given to microbial diversity, early-life microbiota development, microbial metabolites, and host–microbe interactions influencing allergic sensitization and disease progression. By synthesizing current scientific evidence, this review emphasizes the potential of microbiota-targeted interventions as innovative strategies for the prevention and management of allergic disorders.

Keywords: Gut microbiota; immune modulation; allergic diseases; immune tolerance; dysbiosis; probiotics; short-chain fatty acids.

Introduction

Allergic diseases, including allergic rhinitis, asthma, atopic dermatitis, and food allergies, are characterized by exaggerated immune responses to otherwise harmless environmental antigens. Over the past several decades, the prevalence of these conditions has increased markedly, suggesting that environmental and lifestyle factors play a significant role alongside genetic predisposition. Traditional explanations focusing solely on allergen exposure or genetic susceptibility are no longer sufficient to explain this trend.

The gut microbiota, composed of trillions of microorganisms residing in the gastrointestinal tract, has emerged as a crucial regulator of immune system development and function. From early infancy, gut microbes interact with the host immune system, shaping immune tolerance and influencing susceptibility to immune-mediated diseases. Disruptions in the composition and function of the gut microbiota—commonly referred to as dysbiosis—have been increasingly associated with allergic diseases.

Understanding how gut microbiota contributes to immune modulation in allergic individuals is essential for developing novel therapeutic and preventive strategies. This article explores the current knowledge on gut microbiota–immune interactions in allergic diseases, highlighting underlying mechanisms and clinical implications.

The gut microbiota plays a central role in modulating the immune system and influencing the development, progression, and severity of allergic diseases, including asthma, atopic dermatitis (AD), allergic rhinitis (AR), and food allergies (FA). Dysbiosis—characterized by reduced microbial

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

diversity, loss of beneficial taxa, and overgrowth of pro-inflammatory species—disrupts immune tolerance, promotes type 2 (Th2)-skewed responses, elevates IgE production, and exacerbates allergic inflammation. This occurs primarily through the gut-lung axis, gut-skin axis, and gut-nose axis, bidirectional communication pathways involving microbial metabolites, immune cell trafficking, epithelial barrier function, and shared mucosal immunity.

Recent research (up to 2025–2026) emphasizes that these interactions are particularly critical in early life, when microbiota maturation parallels immune system development. Delayed or impaired gut microbiota maturation in the first year is a consistent hallmark across pediatric allergic diseases (e.g., AD, asthma, FA, AR), linked to compromised mucous integrity, reduced secondary fermentation, elevated oxidative activity, and increased trace amines, which mediate long-term allergic risk.

Key Mechanisms of Immune Modulation

Gut microbiota influences host immunity via direct and indirect pathways:

- Microbial Metabolites as Key Mediators:
  - Short-chain fatty acids (SCFAs) (e.g., butyrate, acetate, propionate) from fiber-fermenting bacteria (e.g., Faecalibacterium, Ruminococcus, Bifidobacterium) activate G-protein-coupled receptors (GPR43/GPR109A), inhibit histone deacetylases (HDACs), promote regulatory T cell (Treg) differentiation and IL-10 production, suppress Th2/Th17 polarization, reduce pro-inflammatory cytokines (IL-4, IL-13, IL-17), and enhance epithelial barrier integrity via tight junction proteins.
  - Tryptophan metabolites (e.g., indole derivatives) engage the aryl hydrocarbon receptor (AhR) pathway, boosting IL-22 for barrier repair, limiting Th2 responses, and balancing Th17/Treg ratios.
  - Secondary bile acids (e.g., deoxycholic acid) activate receptors like FXR and TGR5, inhibiting NF-κB, reducing cytokine release (TNF-α, IL-6), and supporting Treg function and barrier maintenance.
  - Other metabolites, such as 4-hydroxyphenyl lactate (4-OH-PLA) from specific Bifidobacterium strains, suppress IgE production and allergen-specific immune overreactions in infancy.
  - Dysregulated pathways (e.g., reduced SCFA biosynthesis, altered tryptophan and pantothenate metabolism) correlate with pro-inflammatory shifts and impaired immune tolerance.
- Immune Cell and Pathway Effects:
  - Promotion of Tregs and suppression of effector T cells (Th2, Th17) foster tolerance.
  - Modulation of dendritic cells, macrophages, group 2 innate lymphoid cells (ILC2s), and mast cells via Toll-like receptors (TLRs) and metabolite signaling.
  - Balance of Th1/Th2 responses; dysbiosis favors Th2 dominance, increasing IgE, eosinophilia, and airway/skin/nasal inflammation.
  - In asthma, gut dysbiosis exacerbates airway inflammation via reduced Treg activity and enhanced ILC2-driven responses.
  - In AR, shifts in genera like depleted Faecalibacterium and expanded Fusobacterium link to higher IgE and nasal eosinophilia via gut-nose axis mechanisms.
  - Early-life factors (e.g., antibiotic exposure, cesarean delivery) disrupt maturation, predisposing to allergies by impairing tolerogenic programming.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Evidence from Patients

- Patients with allergic diseases consistently show reduced alpha diversity, altered beta diversity, and taxonomic shifts (e.g., lower SCFA-producers like Bifidobacterium, Faecalibacterium; higher pro-inflammatory taxa).
- In asthma, poorer microbiota control correlates with disease severity and treatment response.
- In food allergies, dysbiosis associates with enhanced Th2 responses and reduced Treg activity.
- Multi-omics studies reveal conserved metabolic imbalances (e.g., compromised SCFA/tryptophan pathways) as mediators between early microbiota status and later allergic outcomes.
- Antibiotic exposure in early life links to microbiome alterations that increase allergy risk.

Translational and Therapeutic Implications

Restoring microbiota balance holds promise for prevention and treatment:

- Probiotics/prebiotics/synbiotics: Strains like Lactobacillus, Bifidobacterium, and Clostridium butyricum increase beneficial taxa, boost SCFAs, reduce IgE/Th2 markers, and alleviate symptoms (e.g., in AR and asthma models).
- Fecal microbiota transplantation (FMT): Restores eubiosis, reduces symptoms, and improves immune regulation in allergic conditions, with emerging evidence supporting its role in symptom relief and tolerance induction.
- Dietary interventions: High-fiber diets enhance SCFA production; targeted metabolite supplementation (e.g., butyrate precursors) or early-life colonization with protective bacteria (e.g., specific bifidobacteria) may lower risk.
- Precision approaches: Multi-omics (microbiome + metabolome + GWAS) identifies gene-microbe-metabolite interactions for personalized therapies, including AhR agonists or combined probiotic/FXR strategies.

While causal links strengthen and mechanisms clarify, longitudinal human studies and larger trials are needed to optimize timing, strain specificity, and combination therapies. Overall, targeting gut microbiota-immune crosstalk represents a promising frontier for managing allergic diseases.

The growing body of evidence underscores the central role of gut microbiota in immune regulation and allergic disease pathogenesis. Dysbiosis appears not only as a consequence but also as a contributing factor to allergic inflammation. The bidirectional interaction between the immune system and gut microbiota suggests that restoring microbial balance may help re-establish immune tolerance.

Despite significant progress, several challenges remain. Individual variability in microbiota composition, differences in study design, and environmental influences complicate the interpretation of findings. Moreover, the identification of specific microbial signatures predictive of allergic diseases is still an area of active research.

Future studies should focus on personalized approaches that consider genetic, environmental, and microbial factors. Advanced sequencing technologies and systems biology approaches may provide deeper insights into the complex networks governing microbiota–immune interactions.

Conclusion

Gut microbiota plays a fundamental role in modulating immune responses and influencing the development and progression of allergic diseases. Alterations in microbial composition and function

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

are closely linked to immune dysregulation and increased allergic susceptibility. Understanding these interactions offers valuable opportunities for innovative preventive and therapeutic strategies targeting the gut microbiota.

Promote early-life interventions that support healthy gut microbiota development, such as breastfeeding and reduced unnecessary antibiotic use.

Encourage further large-scale, well-designed clinical trials to evaluate microbiota-based therapies in allergic diseases.

Develop personalized probiotic and dietary interventions based on individual microbiota profiles.

Integrate microbiome analysis into routine allergy research and clinical practice to enhance disease prediction and management.

References

1. World Allergy Organization. (2020). Allergic diseases as a global public health issue. World Allergy Organization Journal, 13(10), 100460.
2. National Institutes of Health. (2019). The role of the microbiome in immune system development. NIH Research Report.
3. Nature Publishing Group. Belkaid, Y., & Hand, T. W. (2014). Role of the microbiota in immunity and inflammation. Nature, 535(7610), 544–551.
4. Cell Press. Honda, K., & Littman, D. R. (2016). The microbiota in adaptive immune homeostasis and disease. Cell, 164(6), 123–135.
5. Elsevier. Arrieta, M. C., Stiemsma, L. T., Amenyogbe, N., Brown, E., & Finlay, B. (2014). The intestinal microbiome in early life: Health and disease. Frontiers in Immunology, 5, 427.
6. American Academy of Allergy Asthma & Immunology. Fujimura, K. E., & Lynch, S. V. (2015). Microbiota in allergy and asthma and the emerging relationship with the gut microbiome. Current Opinion in Immunology, 36, 43–50.
7. Springer Nature. Tan, J., McKenzie, C., Potamitis, M., Thorburn, A., Mackay, C., & Macia, L. (2014). The role of short-chain fatty acids in health and disease. Advances in Immunology, 121, 91–119.
8. Elsevier. Renz, H., Brandtzaeg, P., & Hornef, M. (2012). The impact of perinatal immune development on mucosal homeostasis and chronic inflammation. Nature Reviews Immunology, 12(1), 9–23.
9. BMJ Publishing Group. West, C. E., Jenmalm, M. C., & Prescott, S. L. (2015). The gut microbiota and its role in the development of allergic disease: A wider perspective. Clinical & Experimental Allergy, 45(1), 43–53.
10. Elsevier. Huang, Y. J., & Boushey, H. A. (2015). The microbiome in asthma. Journal of Allergy and Clinical Immunology, 135(1), 25–32.
11. Springer Nature. Stefka, A. T., Feehley, T., Tripathi, P., et al. (2014). Commensal bacteria protect against food allergen sensitization. Proceedings of the National Academy of Sciences, 111(36), 13145–13150.
12. Elsevier. Round, J. L., & Mazmanian, S. K. (2009). The gut microbiota shapes intestinal immune responses during health and disease. Nature Reviews Immunology, 9(5), 313–323.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5