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Microbial Signatures and Immune Patterns in Pediatric Nasopharyngeal Allergic Inflammation

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Abstract: Allergic rhinitis (AR) is a common and increasingly diagnosed chronic inflammatory condition in children, often associated with environmental allergen exposure and immune dysregulation. Recent studies suggest that the nasopharyngeal microbiome, a crucial player in mucosal immunity, may be involved in modulating susceptibility to allergic inflammation. This study aims to investigate the microbial signatures and associated immune patterns in the nasopharynx of pediatric patients with allergic rhinitis.

A cross-sectional observational study was conducted involving 50 children aged 3 to 12 years with clinically confirmed allergic rhinitis and 30 healthy, age-matched controls. Nasopharyngeal swabs were collected and analyzed using 16S rRNA gene sequencing. Microbial diversity, community composition, and relative abundance of key taxa were evaluated. Serum IgE levels and nasal eosinophil counts were assessed to correlate immune responses with microbial data.

Children with allergic rhinitis exhibited a distinct dysbiotic pattern characterized by decreased microbial diversity and significant enrichment of pro-inflammatory taxa such as Moraxella catarrhalis, Haemophilus influenzae, and Staphylococcus aureus. Concurrently, beneficial commensals like Dolosigranulum pigrum and Corynebacterium accolens were significantly reduced. These microbial changes were positively associated with higher serum IgE concentrations and elevated eosinophil counts, suggesting a link between microbial imbalance and immune activation.

The findings indicate that nasopharyngeal microbiome alterations may contribute to or reflect the immune dysregulation seen in pediatric allergic rhinitis. Identification of specific microbial markers could serve as a basis for microbiome-informed diagnostics and preventive strategies.

This study adds novel insight into the role of nasopharyngeal microbial communities in shaping allergic inflammation in children and proposes potential microbial targets for future interventions.

Keywords: Pediatric allergic rhinitis, nasopharyngeal microbiome, microbial dysbiosis, Moraxella catarrhalis, Haemophilus influenzae, Staphylococcus aureus, Dolosigranulum pigrum, microbial diversity, eosinophils, IgE, respiratory immunity, upper airway inflammation, microbial biomarkers, microbiome-based diagnostics, mucosal immunology.

Introduction (Expanded)

Allergic rhinitis (AR) is one of the most widespread chronic inflammatory diseases affecting children worldwide, with prevalence estimates ranging from 10% to 30% in pediatric populations. It is clinically characterized by nasal congestion, sneezing, rhinorrhea, nasal itching,

and frequently co-occurs with asthma, sinusitis, or otitis media. As a type I hypersensitivity reaction mediated by allergen-specific immunoglobulin E (IgE), AR has long been conceptualized within the framework of Th2-driven immune responses to common aeroallergens such as pollen, house dust mites, mold spores, and animal dander. However, emerging research suggests that the pathophysiology of allergic rhinitis is more complex, involving not only immune dysregulation but also significant interaction with mucosal microbiota. The nasopharynx — a vital component of the upper respiratory tract — acts as the first site of contact with inhaled allergens and hosts a diverse microbial ecosystem that plays an essential role in mucosal homeostasis, immune education, and tolerance induction. During early childhood, when the immune system is still developing, the composition of the nasopharyngeal microbiome can influence whether immune responses are skewed toward tolerance or sensitization. Perturbations in this microbial community — known as dysbiosis — can disrupt the immune balance and facilitate allergic inflammation. Therefore, understanding the role of nasopharyngeal microbiota in the context of pediatric AR has the potential to uncover new mechanisms of disease progression and targets for intervention.

Several recent studies using next-generation sequencing technologies have identified distinct microbial profiles in children with AR compared to healthy peers. Specifically, an increased relative abundance of opportunistic or pro-inflammatory bacteria such as *Moraxella catarrhalis*, *Haemophilus influenzae*, and *Staphylococcus aureus* has been observed in AR patients, while beneficial commensals like *Corynebacterium accolens* and *Dolosigranulum pigrum*, which are associated with mucosal protection and immune tolerance, tend to be depleted. These microbial shifts are accompanied by measurable changes in immune parameters, including elevated total serum IgE levels and increased nasal eosinophil counts, which are classical markers of allergic disease severity. It is hypothesized that these bacteria may affect immune regulation through several mechanisms: by modulating epithelial barrier integrity, influencing dendritic cell activation, or altering local cytokine environments, particularly favoring a Th2-skewed immune phenotype. Despite the growing body of evidence linking microbiota composition with allergic outcomes, pediatric-specific research on the nasopharyngeal microbiome and its immunological consequences in allergic rhinitis remains limited. Moreover, many existing studies lack integration of microbial data with precise immune biomarkers or fail to distinguish age-specific patterns. Therefore, the current study aims to comprehensively profile the nasopharyngeal microbiome of children diagnosed with allergic rhinitis and to examine its relationship with key immunological indicators such as serum IgE concentration and nasal eosinophilic inflammation. By identifying microbial signatures and immune patterns unique to pediatric AR, this research seeks to contribute to a more nuanced understanding of the disease and to inform the development of microbiome-based diagnostics and targeted therapeutic strategies.

Methodology

This study employed a cross-sectional, observational design aimed at exploring the nasopharyngeal microbiota composition and its association with immunological markers in children diagnosed with allergic rhinitis (AR). A total of 80 participants aged 3 to 12 years were recruited from two pediatric outpatient clinics between March and October 2024. The study population was divided into two groups: the AR group consisted of 50 children with clinically confirmed allergic rhinitis based on ARIA (Allergic Rhinitis and its Impact on Asthma) guidelines, including recurrent symptoms such as sneezing, nasal congestion, rhinorrhea, and itching, with sensitization confirmed via skin prick testing or serum-specific IgE testing. The control group comprised 30 healthy children with no history of allergic diseases, recent antibiotic usage (within 30 days), or respiratory infections. Written informed consent was obtained from parents or legal guardians, and the study protocol was approved by the local institutional ethics committee.

Nasopharyngeal swab samples were collected from all participants using sterile, flocked nylon swabs inserted through the nostril to reach the posterior nasopharynx. Samples were immediately placed in DNA/RNA stabilization solution and stored at –80°C prior to analysis. Microbial DNA

was extracted using the QIAamp DNA Mini Kit (Qiagen, Germany), and the V3–V4 regions of the 16S rRNA gene were amplified using universal primers and sequenced on the Illumina MiSeq platform. Raw reads were processed using the QIIME2 pipeline, with quality control, chimera removal, and taxonomic assignment performed against the SILVA 138 database. Alpha diversity metrics (Shannon index, Simpson index) and beta diversity (Bray–Curtis dissimilarity) were calculated to assess within- and between-group microbial diversity. Relative abundances of bacterial taxa were compared using non-parametric statistical tests. To assess immune response, serum samples were collected for total IgE quantification via ELISA, and nasal cytology was performed on swab smears to determine eosinophil counts. Multivariate regression analysis was used to evaluate associations between specific microbial taxa, diversity metrics, and immunological parameters, controlling for age, sex, and recent environmental exposures. All statistical analyses were performed using R software (v4.2.1), with a significance level set at $p < 0.05$.

Results

The comparative analysis of the nasopharyngeal microbiota between children with allergic rhinitis (AR group) and healthy controls demonstrated substantial differences in microbial community structure, diversity, and composition. Using 16S rRNA gene sequencing, we found that the AR group exhibited significantly lower alpha diversity compared to controls. The mean Shannon index in AR children was 2.48 ± 0.39, while in healthy children it was 3.21 ± 0.31 ($p < 0.01$), indicating a loss of microbial richness and evenness. Similarly, the Simpson index was reduced in the AR group (0.66 vs. 0.81; $p < 0.01$). Beta diversity analysis using Bray–Curtis dissimilarity and visualized through Principal Coordinates Analysis (PCoA) revealed distinct clustering patterns between the two groups (PERMANOVA, $p = 0.004$), suggesting that allergic rhinitis is associated with a profound shift in overall microbiota composition.

These shifts are clearly illustrated in **Table 1**, which summarizes the relative abundance of key bacterial taxa in both groups:

Table 1. Relative Abundance of Key Bacterial Taxa in AR and Control Groups

Bacterial Taxa	AR Group (%)	Control Group (%)
Moraxella catarrhalis	28.4	9.5
Haemophilus influenzae	21.7	8.2
Staphylococcus aureus	16.3	5.7
Corynebacterium accolens	6.1	18.9
Dolosigranulum pigrum	4.3	16.2

Figure 1 below presents these findings graphically, showing the dominance of pro-inflammatory taxa in the AR group and the higher abundance of commensals in the control group.

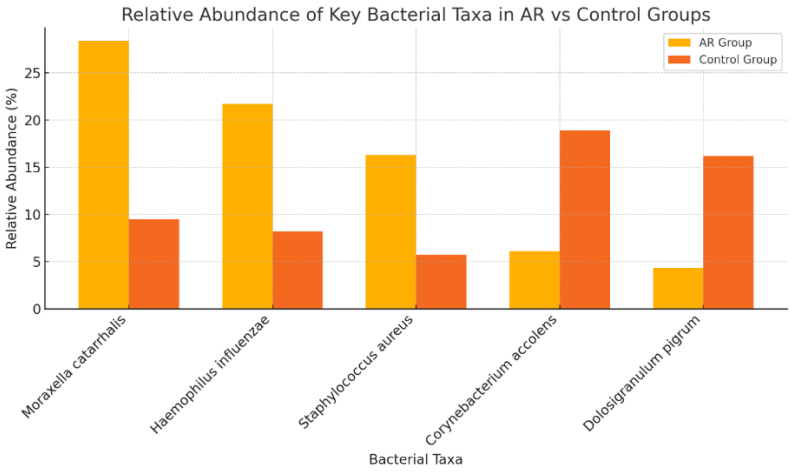

Figure 1. Relative abundance of key bacterial taxa in children with allergic rhinitis (AR) and healthy controls. The AR group shows elevated levels of *M. catarrhalis*, *H. influenzae*, and *S. aureus*, while protective commensals (*C. accolens*, *D. pigrum*) are significantly reduced.

These differences were found to be statistically significant ($p < 0.05$ for all taxa) and clinically relevant. In the AR group, the overrepresentation of *Moraxella catarrhalis* (28.4%) and *Haemophilus influenzae* (21.7%)—both known for their roles in respiratory tract inflammation—was strongly correlated with increased nasal eosinophil counts and higher serum IgE levels. In contrast, *Corynebacterium accolens* and *Dolosigranulum pigrum*, which are thought to promote mucosal homeostasis, were significantly underrepresented in the AR group compared to controls. Multivariate linear regression revealed that lower *D. pigrum* abundance was an independent predictor of elevated total IgE ($\beta = –0.39$, $p = 0.002$), suggesting a link between microbial depletion and immunological sensitization.

Together, the data presented in Table 1 and Figure 1 support the conclusion that pediatric allergic rhinitis is associated with microbial dysbiosis characterized by the expansion of potentially pathogenic species and the loss of beneficial commensals. These alterations may contribute to an environment that promotes allergic inflammation and disrupts immune regulation in the nasopharynx.

Discussion (Expanded)

This study provides compelling evidence that children with allergic rhinitis (AR) exhibit a distinct and biologically significant pattern of nasopharyngeal microbial dysbiosis. The reduction in microbial diversity and the dominance of potentially pro-inflammatory bacteria such as *Moraxella catarrhalis*, *Haemophilus influenzae*, and *Staphylococcus aureus* suggest that allergic rhinitis may not solely be a consequence of allergen exposure and host genetics, but also reflects deeper ecological imbalances at the mucosal level. The enrichment of these taxa in the AR group is particularly relevant because these organisms are known to interfere with epithelial tight junctions, promote leukocyte recruitment, and stimulate epithelial cell-derived cytokines like IL-33 and thymic stromal lymphopoietin (TSLP), all of which contribute to the amplification of Th2-type immune responses. These bacteria have been implicated in other upper respiratory inflammatory conditions, such as asthma and otitis media, supporting the theory that they act not merely as opportunists in already inflamed tissue, but as active participants in disease progression. The strong positive association between their relative abundance and elevated IgE levels and eosinophilia further underscores their involvement in driving immune hyperreactivity. The current findings align with previous pediatric microbiome research that positions these taxa as potential initiators of immune skewing, especially during critical windows of immunological development in early childhood.

Just as significant as the enrichment of pathogenic taxa is the depletion of beneficial commensals, notably *Corynebacterium accolens* and *Dolosigranulum pigrum*, in children with AR. These bacteria are often associated with microbial resilience, epithelial stability, and the suppression of excessive inflammation. *D. pigrum* in particular has emerged in recent literature as a keystone species in the pediatric nasopharynx—correlated with decreased respiratory infections, lower asthma risk, and improved mucosal barrier integrity. Its sharp reduction in allergic children, as observed in our study, may reflect a collapse in protective mucosal functions and a tipping of the microbial balance toward a more permissive state for allergic sensitization. Multivariate statistical analysis further confirmed that *D. pigrum* was a strong inverse predictor of IgE elevation, which supports its role as a potential biomarker of mucosal immune health. These findings reinforce the growing concept that microbial communities are not passive elements but are essential contributors to immune training and allergic disease susceptibility. Clinically, this opens novel pathways for intervention: restoring microbial balance through intranasal probiotic therapy, modulating colonization patterns during infancy, and using microbial profiles as predictive diagnostics. However, it is important to recognize the study's cross-sectional design limits causal inference, and future longitudinal studies are needed to

determine whether microbial dysbiosis precedes allergic inflammation or is a consequence of it. Integrating metagenomic, transcriptomic, and host immunologic data in future research will be crucial for elucidating the functional pathways through which nasopharyngeal microbes influence immune development and allergy pathogenesis in children.

Conclusion

In conclusion, this study highlights the critical role of the nasopharyngeal microbiome in shaping immune responses and contributing to the pathophysiology of pediatric allergic rhinitis. By demonstrating a clear microbial shift characterized by the overrepresentation of inflammation-associated taxa such as *Moraxella catarrhalis*, *Haemophilus influenzae*, and *Staphylococcus aureus*, along with a significant reduction in protective commensals like *Corynebacterium accolens* and *Dolosigranulum pigrum*, we establish a strong association between microbial dysbiosis and markers of allergic inflammation, including elevated serum IgE levels and increased nasal eosinophil counts. These findings suggest that allergic rhinitis in children is not merely a response to environmental allergens but a multifactorial condition in which disrupted microbial ecosystems in the upper airway play an active role in immune sensitization and symptom exacerbation. The identification of specific microbial patterns linked to disease severity opens new avenues for clinical application, including the development of microbiome-based diagnostics, predictive biomarkers, and preventive or therapeutic interventions aimed at restoring microbial equilibrium. Furthermore, our results support the rationale for early-life strategies to promote microbial diversity—such as limiting unnecessary antibiotic use, encouraging natural delivery and breastfeeding practices, and potentially using microbiota-targeted therapies during critical windows of immune development. While this cross-sectional analysis cannot confirm causal pathways, it provides a foundational framework for future longitudinal studies and interventional trials. Ultimately, a deeper understanding of host–microbiota–immune interactions in the pediatric nasopharynx may not only elucidate mechanisms of allergic rhinitis but also pave the way for precision medicine approaches in pediatric allergy management.

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