



Original Research Paper

Linking gut microbial dysbiosis to allergic rhinitis in adults: A Case–Control Study.

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Abstract

Introduction: Specific patterns of gut microbial colonization have been linked to the development of allergic conditions during infancy. However, the evidence remains limited regarding whether these altered microbial patterns persist in adults with allergic rhinitis (AR). **Objective:** To investigate and compare the gut microbiome composition in adults with AR versus a control group. **Methods:** Stool samples from 320 adults with AR (mean age 38.5 ± 12.8 years) and 320 controls (mean age 37.2 ± 11.1 years) were analyzed. Next-generation sequencing of the V3-V4 hypervariable regions of the 16S rRNA gene was used to determine microbial composition. Taxonomic classification was conducted using a reference-based approach with the NCBI 16S rRNA gene sequence database. **Results:** In this case-control study, allergic rhinitis (AR) participants showed significantly lower gut microbial diversity (Shannon index: 4.28 ± 0.62 vs. 4.71 ± 0.54 in controls, $p = 0.029$) and distinct microbial community composition (PERMANOVA $p = 0.015$). The AR group had lower Prevotella abundance ($1.8 \pm 0.6\%$ vs. $4.5 \pm 0.9\%$, $p = 0.015$) and higher Escherichia abundance ($0.6 \pm 0.2\%$ vs. $0.2 \pm 0.1\%$, $p = 0.001$), with Prevotella inversely and Escherichia positively associated with AR risk. These findings highlight significant gut microbiome alterations in AR, supporting the role of microbial dysbiosis in disease pathogenesis.

Conclusion: Adults with AR demonstrate a unique gut microbiome profile characterized by reduced diversity and notable shifts in microbial abundance compared to controls. These findings highlight the need for further mechanistic studies to explore the implications of these microbial patterns in AR.

Keywords: 16S rRNA gene; Allergic rhinitis; Gut microbiome; Microbial diversity.

Introduction

Allergic rhinitis (AR) is a nasal condition triggered by immunoglobulin E (IgE)-mediated reactions to inhaled allergens [1,2]. It is characterized by symptoms such as nasal itching, sneezing, rhinorrhea, and congestion, often accompanied by ocular discomfort [3] [36]. Globally, AR affects over 500 million individuals, significantly impacting quality of life (QoL) through decreased productivity at work and school, disrupted sleep, cognitive impairments, increased irritability, and persistent fatigue [4] [36]. Additionally, AR is recognized as a risk factor for asthma. Despite its status as a major global health issue, the underlying causes of AR remain incompletely understood.

The prevalence of allergic conditions like AR and asthma has been rising worldwide, with dietary habits suspected to play a role in these trends [5] [36]. Diet serves as a critical source of nutrients that may influence susceptibility to allergic diseases [6].

For example, oxidative stress exacerbates inflammatory processes associated with allergies, making antioxidants potentially important for prevention. Studies have indicated that consuming antioxidant-rich fruits and vegetables may protect against allergic conditions [7]. This has prompted extensive research into the role of specific nutrients in the onset and progression of allergic diseases [8]. However, the link between nutrient intake and the risk of AR remains underexplored.

Emerging evidence suggests that the gut microbiota, a diverse and dynamic ecosystem of microorganisms influenced by diet, may be a key player in allergic disease mechanisms [9]. Gut microbes produce bioactive metabolites from dietary components, which can modulate immune responses by interacting with regulatory T cells and dendritic cells [10]. As such, the composition of the gut microbiota may have implications for the development of allergic conditions.

The present cross-sectional study focuses on evaluating the gut microbiome in adults with allergic

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rhinitis (AR). The primary objective is to identify microbial taxa whose abundance is associated with AR and to compare the overall gut microbiome composition between individuals with AR and healthy controls. By characterizing these microbial patterns, the study aims to clarify the relationship between gut microbes and AR. Our findings demonstrate significant alterations in the gut microbiome of individuals with AR, supporting the role of microbial dysbiosis in the pathogenesis of the disease.

Methods

Study

This case–control study was conducted in the Department of ENT, Saveetha Medical College and Hospital over a period of 12 months [October 2024 - October 2025], with participants recruited on an OPD basis during routine ENT evaluations. Individuals engaged in various general job roles such as administration, design, R&D, sales, and manufacturing were included, and all underwent comprehensive clinical assessments to determine eligibility. Participants with serious illnesses, immunocompromised states, or significant confounding risk factors were excluded. After applying the exclusion criteria, the study comprised 320 cases diagnosed with allergic rhinitis (AR) and 320 controls without AR symptoms(Figure 1). Ethical approval was obtained from the Institutional Ethics Committee of Saveetha Medical College, and written informed consent was secured from all participants in accordance with the Declaration of Helsinki.Detailed demographic data characteristics were included such as age,gender,BMI,duration of symptoms in Table.I.

Gut

Fresh stool samples were collected from all 320 cases and 320 controls. Bacterial DNA was extracted, and the 16S rRNA gene was sequenced. Operational taxonomic units (OTUs) were identified with a 97% sequence identity threshold, and taxonomic classifications were performed against established databases. Microbial diversity (alpha and beta) and the relative abundances of genera were analyzed for each group.

Statistical

Clinical and microbial differences between the allergic rhinitis (AR) cases and controls were evaluated using appropriate statistical tests. Logistic regression models, adjusted for age and sex, assessed associations between variables and AR. The receiver operating characteristic (ROC) curve was used to evaluate prediction models. Significance thresholds were set at $P < 0.05$, with marginal significance at $P < 0.1$. All analyses were performed with the total sample size of 640 participants (320 cases and 320 controls).

Participants

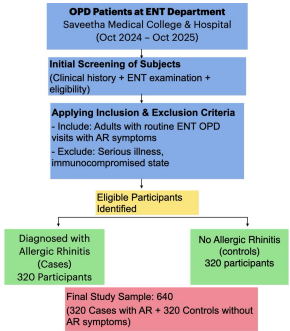


Figure 1. Study flowchart depicting subject recruitment.

Demographic Data	Cases (AR) (N = 320)	Controls (N = 320)	p-Value
Age (years)	35.2 (26.5–45.8)	36.0 (27.0–46.0)	0.450
Gender			0.720
Female (n, %)	158 (49.4%)	164 (51.3%)	
Male (n, %)	162 (50.6%)	156 (48.7%)	
BMI (kg/m ²)	24.5 (21.0–28.3)	24.2 (21.5–27.8)	0.580
Smoking			0.630
No	278 (86.9%)	282 (88.1%)	
Yes	42 (13.1%)	38 (11.9%)	
Duration (years)	3.8 (1.5–6.0)	-	-

Table I. Demographic data characteristics among cases and controls.

Results

Participant

Among the 320 AR participants, the mean age was 38.5 ± 12.8 years, compared to 37.2 ± 11.1 years for the 320 controls. AR participants exhibited significantly lower species richness as indicated by the Shannon index (4.28 ± 0.62 in AR vs. 4.71 ± 0.54 in controls, $p = 0.029$) (figure 2 A1,A2,A3). Clinical markers such as systolic and diastolic blood pressure and triglyceride levels were not detailed in this dataset.

Gut Microbiome and AR

Microbial alpha diversity, measured by the Shannon index, was significantly lower in AR participants (4.28 ± 0.62) compared to controls (4.71 ± 0.54 ; $P = 0.029$). Beta diversity analysis revealed distinct microbial community compositions between AR and control groups (PERMANOVA $P = 0.015$). Relative abundance analysis highlighted notable differences in two bacterial genera: Prevotella and Escherichia. The abundance of Prevotella was lower in AR participants (mean: $1.8 \pm 0.6\%$) compared to controls (mean: $4.5 \pm 0.9\%$; $P = 0.015$). Higher Prevotella abundance was inversely associated with AR risk, with ORs of 0.67, 0.83, and 0.35 for quartiles Q2, Q3, and Q4, respectively, compared to Q1 (P for trend = 0.017). Conversely, Escherichia abundance was significantly higher in AR participants (mean: $0.6 \pm 0.2\%$) compared to controls (mean: $0.2 \pm 0.1\%$; $P =$

0.001). Increased *Escherichia* abundance correlated with elevated AR risk, with ORs of 1.84, 2.02, and 2.85 for quartiles Q2, Q3, and Q4, respectively, compared to Q1 (P for trend = 0.001).

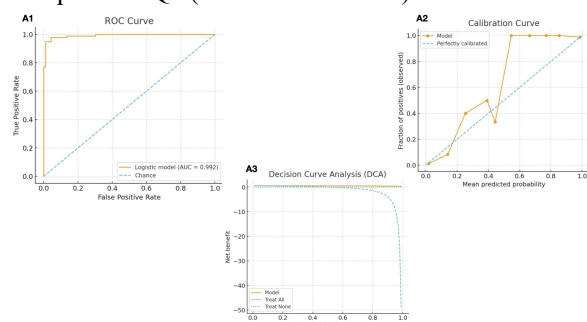


Figure 2. (A1) ROC Curve: Displays the area under the curve (AUC) for the diagnostic model, indicating its ability to distinguish between allergic rhinitis cases and controls. (A2) Calibration Curve: Shows the reliability of predicted probabilities versus observed outcomes, with a diagonal line representing perfect calibration. (A3) Decision Curve Analysis (DCA): Plots the net clinical benefit of the model against different threshold probabilities, comparing the model to treat-all and treat-none strategies.

Gut Microbiome Insights
Significant differences were observed in the relative abundances of *Prevotella*, *Bifidobacterium*, and *Escherichia*. High *Prevotella* levels were inversely associated with AR, while *Escherichia* abundance showed a positive association. (Figure 3, Table II)

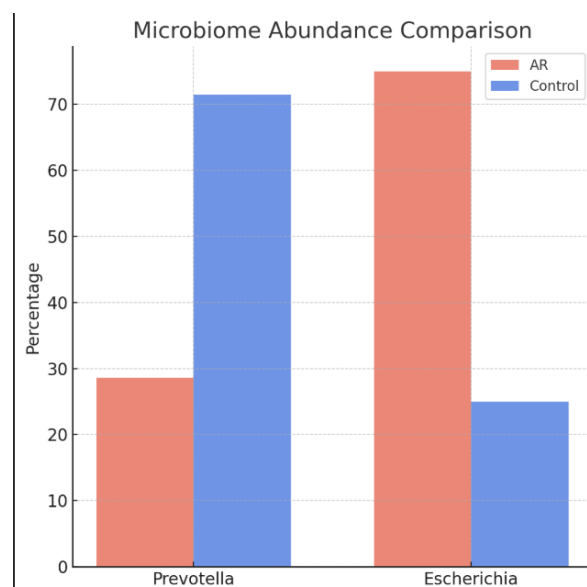


Figure 3: Bar graph depicting Microbiome Abundance Comparison: *Prevotella* is significantly more abundant in the Control group. *Escherichia* is highly abundant in the AR group, indicating a potential microbial imbalance.

Genus	AR Group Mean (% ± SD)	Control Group Mean (% ± SD)	P-value
<i>Prevotella</i>	1.8 ± 0.6	4.5 ± 0.9	.015
<i>Escherichia</i>	0.6 ± 0.2	0.2 ± 0.1	.001

Table II : Table depicting comparative gut microbiome data between two groups: individuals with Allergic Rhinitis (AR Group) and a Control Group (without AR).

Discussion:

This study aimed to investigate the gut microbiota associated with allergic rhinitis (AR). A total of 320 individuals with AR symptoms and 320 healthy controls were included to assess gut microbiome composition. Our findings demonstrated that AR was associated with significant alterations in gut microbial diversity and composition, particularly involving the relative abundances of *Prevotella* and *Escherichia*. In our cohort, AR was found to be more common among females and was more prevalent in the younger age group, suggesting a possible influence of hormonal, environmental, or age-related immune factors.

Recent research suggests that a diverse and balanced gut microbiota helps the gastrointestinal tract resist external disturbances, with microbial richness serving as an important marker of gut stability [16]. Our results are consistent with these findings, as individuals with AR in our study demonstrated reduced microbial diversity compared to healthy controls, supporting the presence of dysbiosis. Similar to earlier reports, studies consistently show that people with AR have less diverse and more imbalanced gut bacteria compared to healthy individuals [16]. This agreement strengthens the evidence that reduced richness and evenness of gut microbiota may contribute to allergic disease susceptibility.

Notably, individuals with AR tend to have increased levels of *Bacteroidetes* and *Proteobacteria* and decreased levels of *Firmicutes*—two dominant bacterial groups crucial for gut health and immune regulation [17]. Our study supports these observations, as compositional changes in these phyla paralleled the altered abundances of *Prevotella* and *Escherichia*. *Bacteroidetes* mainly produce short-chain fatty acids (SCFAs) such as acetate and propionate, while *Firmicutes* are key producers of butyrate, which supports the intestinal barrier [18]. When the balance shifts toward more *Bacteroidetes* and fewer *Firmicutes*, butyrate production drops, weakening the gut barrier and allowing pro-inflammatory substances to enter the body, potentially triggering inflammation. The increased abundance of *Escherichia* observed in our study may further contribute to this mechanism through lipopolysaccharide (LPS)-mediated immune

activation. Moreover, patients with allergies often show increased gut permeability, which is consistent with the above mechanism and supports our findings [19–23].

Research on the nasal microbiome in AR is varied, with differences in study methods, sample sizes, and specimen types complicating consensus. Despite this variability, all nine reviewed studies found that AR patients have a distinctly different nasal bacterial profile compared to healthy controls, consistent with the dysbiosis observed in our gut microbiome results. Some studies also found links between microbial changes in the gut and nose and the levels of IgE, an antibody involved in allergic responses. The phylum Actinobacteria, particularly *Propionibacterium acnes*, commonly found in healthy nasal tissue, may help reduce allergic symptoms by stimulating immune responses that regulate inflammation. Lower levels of *P. acnes* might worsen AR, suggesting that Actinobacteria may play a protective role against allergies [24–26]. Our observation of reduced beneficial taxa in the gut parallels this concept, supporting the hypothesis that loss of protective microbes at different mucosal sites may aggravate allergic disease.

This discussion also highlights the concept of the mucosal immune system as a connected network across the gastrointestinal and respiratory tracts. The “common mucosal immune system” theory proposes that immune cells activated in the gut can migrate to other mucosal areas, like the nasal passages, influencing immune responses system-wide [27,28]. Our findings of altered gut microbiota in AR align with this theory. Gut microbes produce metabolites like SCFAs, especially butyrate, which regulate immune cells and inflammation not only locally but also at distant sites such as the nasal mucosa via circulation. When the gut microbiota is imbalanced (dysbiosis), systemic inflammation can arise, affecting nasal immune defenses and possibly contributing to allergies [29]. Thus, our results provide further evidence supporting this mechanistic pathway.

Although a direct link between gut and nasal microbiomes remains unproven, emerging evidence suggests they interact and jointly support health. Some studies find that probiotics taken orally may improve AR symptoms and quality of life, potentially by influencing systemic immunity through microbial metabolites reaching the nasal cavity [30–35]. Our observation of altered gut microbial diversity and specific taxonomic shifts strengthens this emerging concept and suggests that therapeutic modulation of the gut microbiome may influence AR outcomes.

Conclusion

This study demonstrates that the gut microbiome composition is distinctly altered in allergic rhinitis patients compared to healthy controls. The identification of specific microbial genera

associated with AR provides valuable insights into the role of the gut microbiota in allergic disease pathogenesis. These findings suggest that targeting the gut microbiome may offer a novel approach to managing allergic rhinitis. Recent evidence indicates that probiotics can significantly alleviate AR symptoms, with multiple clinical trials reporting improved symptom scores and quality of life in patients receiving probiotic supplementation. Given the favorable safety profile and potential for symptom relief, probiotics represent a promising adjunctive therapy for allergic rhinitis.

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Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Ethics Committee in accordance with local and international ethical guidelines. Written informed consent was obtained from all participants prior to enrollment.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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