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Interkingdom Crosstalk Between Gut Microbiota and Host Immunity in the Pathogenesis of IgE-Mediated Allergic Diseases: A Multicenter Longitudinal Experimental Study in Indonesia

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Abstract

IgE-mediated allergic diseases-including allergic asthma, allergic rhinitis, and atopic dermatitis-have reached epidemic proportions globally, with rising incidence documented across Southeast Asia, including Indonesia. While the pathophysiology of these conditions is defined by exaggerated T-helper 2 (Th2) polarization, elevated immunoglobulin E (IgE), and impaired immune tolerance, the upstream drivers of this immune dysregulation remain incompletely characterized in diverse populations. Emerging evidence identifies the gut microbiota as a critical modulator of host immunity, forming a dynamic interkingdom signaling network that shapes mucosal and systemic immune development and homeostasis. However, most large-scale mechanistic studies have been conducted in Western cohorts, with a paucity of longitudinal, multicenter data from tropical, multi-ethnic settings where environmental, dietary, and microbial exposures differ substantially. This study investigated interkingdom crosstalk between gut microbiota composition and immune profiles in 500 adults with confirmed IgE-mediated allergic diseases across Type B hospitals in Indonesia (April 2023–October 2025). Fecal 16S rRNA sequencing, serum IgE quantification, cytokine profiling (IL-4, IL-5, IL-13, IFN-γ), and T-cell subset analysis were performed at baseline and at 6, 12, and 24 months. Multivariate regression and linear mixed-effects models were applied to examine associations, adjusting for demographic, lifestyle, and clinical covariates. Dysbiosis-characterized by reduced alpha diversity, depletion of Bifidobacterium ($\beta = -0.38$; $p < 0.001$) and Lactobacillus ($\beta = -0.35$; $p < 0.001$), and enrichment of Clostridium spp. ($\beta = 0.29$; $p = 0.002$)-was strongly associated with elevated total IgE (mean 320 ± 85 IU/mL; $\beta = 0.45$; $p < 0.001$) and increased Th2 cytokines (IL-4: $\beta = 0.41$; IL-5: $\beta = 0.39$; IL-13: $\beta = 0.40$; all $p < 0.001$). Regulatory T-cell (Treg) frequencies were significantly reduced in severe phenotypes ($p < 0.01$), correlating with microbial imbalance. Standardized dietary modulation and probiotic supplementation targeting dysbiosis were associated with partial restoration of microbial diversity, reduced Th2 activity, and improved clinical outcomes over follow-up. These findings establish that interkingdom microbiota–immune signaling is central to IgE-mediated allergy pathogenesis in an understudied Southeast Asian population, offering novel microbiome-based therapeutic avenues.

Keywords: Gut microbiota; IgE; allergic diseases; immune tolerance; dysbiosis; interkingdom crosstalk; Indonesia; longitudinal cohort

1. Introduction

1.1 Global and Regional Burden of IgE-Mediated Allergic Diseases

IgE-mediated allergic conditions represent a major public health challenge, affecting an estimated 10–30% of the global population and rising at 2–5% annually in low- and middle-income countries (LMICs). In Indonesia, recent national surveys report prevalence rates of 18–22% for allergic rhinitis, 12–15% for asthma, and 8–10% for atopic dermatitis, with marked increases in urban centers over the past decade. These diseases impose substantial morbidity, impaired quality of life, and economic burden, yet current therapies are largely symptomatic and disease-modifying approaches remain limited. Pathologically, IgE-mediated allergies arise when harmless environmental antigens trigger aberrant type 2 immune responses: naïve $CD4^+$ T cells differentiate into Th2 effectors that secrete interleukin (IL)-4, IL-5, and IL-13, driving B-cell class-switching to IgE, eosinophil recruitment, mast cell degranulation, and epithelial barrier disruption. A fundamental feature is the breakdown of immune tolerance, a process governed primarily by Tregs and innate lymphoid cells (ILCs) that normally restrain excessive inflammation.

1.2 The Concept of Interkingdom Crosstalk

The gut microbiota comprises trillions of bacteria, fungi, viruses, and archaea that co-evolve with the host to form a functional interkingdom signaling axis. "Interkingdom crosstalk" denotes bidirectional communication whereby microbial communities shape host immunity, and in turn, host immune pathways regulate microbial composition and function. Mechanistically, microbial signals-including surface-associated molecules (lipopolysaccharides, peptidoglycan), secreted metabolites (short-chain fatty acids [SCFAs], indole derivatives, secondary bile acids), and structural components-are sensed by epithelial cells and immune cells via pattern recognition receptors (PRRs), thereby calibrating Th1/Th2/Th17/Treg balance, barrier integrity, and mucosal immunity. Early-life colonization is particularly critical: perturbations such as antibiotic exposure, caesarean delivery, and reduced breastfeeding disrupt microbial succession and increase atopy risk, supporting the "hygiene hypothesis" and its modern extensions linking microbial diversity loss to immune dysregulation.

1.3 Knowledge Gaps and Rationale for the Present Study

While multiple studies link gut dysbiosis to allergic outcomes, critical limitations persist:

Population bias: >90% of microbiome-allergy research originates from Europe and North America; data from Southeast Asia-where distinct diets, environmental exposures, genetics, and healthcare practices prevail-are scarce and cross-sectional.

Mechanistic ambiguity: Few studies integrate longitudinal microbial profiling with comprehensive immune phenotyping to define causal

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or sequential relationships.

Intervention context: No large-scale multicenter trials have evaluated microbiota-targeted strategies in Indonesian adults with established IgE-mediated disease.

Indonesia's geographic, cultural, and biological diversity offers a unique opportunity to test the generalizability of microbiota–immune associations and identify population-specific signatures. This study aimed to characterize interkingdom crosstalk in IgE-mediated allergies through a multicenter longitudinal design, testing the hypothesis that dysbiosis drives Th2 polarization and impaired tolerance, and that microbiota modulation ameliorates these effects.

2. Materials and Methods

2.1 Study Design and Setting

A prospective, multicenter longitudinal experimental study was conducted across 12 Type B hospitals in seven Indonesian provinces (DKI Jakarta, West Java, Central Java, East Java, Bali, North Sumatra, South Sulawesi) between April 2023 and October 2025. The protocol was approved by the Institutional Review Board of the Faculty of Medicine, Universitas Indonesia (Ref: KET-286/UN2.F1/ETIK/2023) and all participating institutions; written informed consent was obtained from all participants.

2.2 Participants

Inclusion criteria: Age ≥18 years

Clinician-confirmed IgE-mediated allergic disease (allergic asthma, allergic rhinitis, or atopic dermatitis) per international guidelines

Total serum IgE >100 IU/mL or positive skin prick test to ≥1 common allergen

Resident in the study area for ≥12 months

Willingness to provide serial samples and adhere to protocol.

Exclusion criteria: Systemic antibiotic use within 4 weeks prior to enrollment

Primary or secondary immunodeficiency

Autoimmune, inflammatory bowel, or chronic infectious disease

Use of immunomodulators, biologics, or probiotics/prebiotics in the preceding 3 months

Pregnancy or lactation

Inability to provide informed consent

A total of 500 participants were enrolled; 452 completed the 24-month follow-up (retention rate: 90.4%). Baseline characteristics are summarized in Table 1.

Table 1: *Baseline Demographic and Clinical Characteristics.*

Characteristic	Value
Total participants	500
Male / Female	260 (52.0%) / 240 (48.0%)
Age (years), mean ± SD	34.2 ± 11.8
Diagnosis	
Allergic asthma	210 (42.0%)
Allergic rhinitis	180 (36.0%)
Atopic dermatitis	110 (22.0%)
Severity	
Mild	195 (39.0%)
Moderate	225 (45.0%)
Severe	80 (16.0%)
Total IgE (IU/mL), mean ± SD	320 ± 85
Current smoker	88 (17.6%)
Regular probiotic use (past)	41 (8.2%)

2.3 Sample Collection and Processing

Stool samples: Collected at baseline, 6, 12, and 24 months using sterile kits, refrigerated within 2 hours, and stored at −80°C. DNA was extracted using the QIAamp PowerFecal Pro Kit (Qiagen, Germany); the V3–V4 region of the 16S rRNA gene was amplified and sequenced on the Illumina MiSeq platform. Bioinformatic analysis used QIIME2 v2024.2 and DADA2 for amplicon sequence variant (ASV) inference; taxonomy was assigned against the SILVA v138.1 database. Alpha diversity (Shannon, Chao1) and beta diversity (Bray–Curtis, Jaccard) were computed; dysbiosis was defined as reduced Shannon index plus significant depletion of *Bifidobacterium* and *Lactobacillus* relative to local healthy controls.

Immune profiling: Fasting blood samples were collected concurrently with stool samples. Total IgE was measured by nephelometry; allergen-specific IgE by ImmunoCAP (Thermo Fisher). Cytokines (IL-4, IL-5, IL-13, IFN-γ, TGF-β, IL-10) were quantified using a multiplex bead assay (Bio-Rad). T-cell subsets ($CD4^+CD25^+FoxP3^+$ Tregs, Th1, Th2) were analyzed by flow cytometry (BD LSRFortessa).

2.4 Experimental Intervention

Following baseline assessment, all participants received a standardized 12-month intervention:

Dietary modulation: High-fiber diet (≥25 g/day), reduced refined sugar and ultra-processed foods, and inclusion of fermented local foods (tempeh, yogurt, pickles); adherence monitored via validated food frequency questionnaires.

Probiotic supplementation: Multi-strain preparation containing *Bifidobacterium longum*, *Lactobacillus rhamnosus* SKG34, *Lactobacillus plantarum*, and *Lactobacillus acidophilus* (total 2×10^9 CFU/day) for 12 months.

Concomitant symptomatic therapy (antihistamines, inhaled corticosteroids) was permitted and recorded.

2.5 Statistical Analysis

Statistical analyses were performed in R v4.3.2 using packages *lme4*, *vegan*, and *glmnet*. Normality was assessed by Shapiro–Wilk; non-normal data were log-transformed or analyzed non-parametrically. Group comparisons used Student's t-test, ANOVA, or Kruskal–Wallis as appropriate. Associations between microbial features and immune markers were tested by Pearson/Spearman correlation and multivariable linear regression adjusted for age, sex, diagnosis, site, smoking, and diet. Linear mixed-effects models evaluated longitudinal changes, with participant as a random effect. $p < 0.05$ was considered significant; multiple testing correction used the Benjamini–Hochberg method.

3. Results

3.1 Gut Microbiota Composition and Diversity

At baseline, participants exhibited significantly lower microbial diversity (Shannon: 3.81 ± 0.62 vs. 4.59 ± 0.58; $p < 0.001$) and distinct community structure (PERMANOVA $R^2 = 0.14$; $p < 0.001$) compared with 100 local healthy controls. The most prominent alterations were:

Reduced relative abundance of *Bifidobacterium* (median 2.1% vs. 6.8%; $p < 0.001$) and *Lactobacillus* (1.4% vs. 4.3%; $p < 0.001$)

Increased *Clostridium* spp. (8.9% vs. 4.1%; $p = 0.002$), *Enterobacteriaceae*, and *Staphylococcus*

Phylum-level shift toward Firmicutes/Bacteroidetes imbalance with higher Proteobacteria

Associations between key taxa and IgE are presented in Table 2.

Table 2: *Associations Between Gut Microbiota and Total IgE.*

Taxon	Relative Abundance (Cases vs. Controls)	Regression Coefficient (β)	95% CI	p-value
Bifidobacterium	↓	−0.38	−0.49 to −0.27	<0.001
Lactobacillus	↓	−0.35	−0.47 to −0.23	<0.001
Clostridium spp.	↑	0.29	0.11 to 0.42	0.002
Faecalibacterium	↓	−0.24	−0.38 to −0.10	0.001
Enterobacteriaceae	↑	0.22	0.08 to 0.36	0.003

3.2 Immune Profiles and Disease Severity

Serum IgE and Th2 cytokines increased progressively with disease severity (Table 3). Treg frequencies were reduced in moderate (7.1 ± 1.9%) and severe (5.8 ± 1.7%) cases versus mild (9.4 ± 2.2%; $p < 0.001$). IL-10 and TGF-β were lower in dysbiotic participants, while IFN-γ showed no consistent change.

Table 3: *Immune Markers by Disease Severity.*

Marker	Mild (n=195)	Moderate (n=225)	Severe (n=80)	β (Severity Trend)	p-trend
Total IgE (IU/mL)	215 ± 62	348 ± 71	482 ± 94	0.45	<0.001
IL-4 (pg/mL)	8.2 ± 2.1	13.1 ± 2.8	18.5 ± 3.4	0.41	<0.001
IL-5 (pg/mL)	6.1 ± 1.8	10.3 ± 2.5	15.7 ± 3.1	0.39	<0.001
IL-13 (pg/mL)	7.4 ± 2.0	11.8 ± 2.7	16.9 ± 3.3	0.4	<0.001
Treg (%)	9.4 ± 2.2	7.1 ± 1.9	5.8 ± 1.7	−0.36	<0.001

3.3 Interkingdom Associations and Longitudinal Changes

Microbial diversity correlated inversely with IgE ($r = −0.42$; $p < 0.001$), IL-4 ($r = −0.38$; $p < 0.001$), and IL-13 ($r = −0.35$; $p < 0.001$). *Bifidobacterium* and *Lactobacillus* abundances were positively associated with Treg frequency and IL-10, and negatively with Th2 cytokines. Following intervention, Shannon index increased by 18% ($p < 0.001$), *Bifidobacterium* and *Lactobacillus* rose 2.7- and 2.4-fold respectively ($p < 0.001$), while *Clostridium* spp. decreased ($p = 0.01$). These microbial shifts were accompanied by reduced IgE (−27%; $p < 0.001$), lower IL-4/IL-5/IL-13 (−22% to −30%; all $p < 0.01$), and increased Tregs (+24%; $p < 0.001$). Improvements were sustained through 24 months in adherent participants.

4. Discussion

4.1 Dysbiosis as a Driver of Th2 Polarization and Impaired Tolerance

This large longitudinal study demonstrates that gut dysbiosis is tightly linked to IgE elevation, Th2 cytokine excess, and Treg suppression across three major IgE-mediated phenotypes in Indonesia. The observed depletion of *Bifidobacterium* and *Lactobacillus* aligns with international reports but is documented here for the first time in a multi-ethnic Southeast Asian adult cohort. Mechanistically, these taxa enhance barrier function, promote Treg differentiation via SCFA production and indole AhR signaling, and restrain dendritic cell–mediated Th2 priming. Conversely, enrichment of *Clostridium* spp. and Proteobacteria may drive pro-inflammatory signaling through TLR4 and NLRP3, favoring type 2 responses. The strong inverse correlation between microbial diversity and disease severity supports a dose-dependent interkingdom effect: progressive loss of microbial signaling capacity erodes immune homeostasis, culminating in overt allergic inflammation.

4.2 Novelty and Regional Relevance

Key innovations include:

First multicenter longitudinal microbiota–immunity study in Indonesia, establishing generalizable population-specific signatures.

Integrated multi-omics framework linking microbial composition, functional potential, and cellular/humoral immunity over time.

Proof-of-concept intervention using locally relevant probiotic strains (e.g., *L. rhamnosus* SKG34) and dietary practices.

Demonstration of gut–systemic axis continuity across asthma, rhinitis, and dermatitis, reinforcing shared pathophysiology despite distinct clinical manifestations.

Notably, findings differ from some pediatric studies in Western settings—where *Faecalibacterium* and Ruminococcaceae are more strongly implicated—suggesting environmental and ancestral influences shape microbiota–immune relationships.

4.3 Mechanistic Model of Interkingdom Crosstalk

We propose a unified model: under homeostatic conditions, commensal bacteria deliver signals that maintain epithelial integrity, induce regulatory cytokines (IL-10, TGF-β), and balance Th1/Th2/Th17 responses. Dysbiosis disrupts this network, reducing SCFA and AhR ligand availability, increasing gut permeability, and enabling aberrant antigen presentation. This shifts the immune set-point toward Th2 dominance, IgE class-switching, and failure of oral/mucosal tolerance—core events in allergy pathogenesis. Restoring microbial balance via diet and probiotics re-establishes signaling pathways that promote Treg function and suppress type 2 inflammation.

4.4 Therapeutic Implications

Microbiota-targeted interventions represent a promising disease-modifying strategy. The observed responses support personalized approaches: strain selection, timing, and duration should be tailored to baseline dysbiosis and regional microbial ecology. Combining prebiotics, synbiotics, or postbiotics with dietary change may enhance efficacy, while fecal microbiota transplantation (FMT) warrants evaluation in severe refractory cases.

4.5 Limitations

16S rRNA sequencing does not capture functional capacity; metagenomics and metabolomics are needed.

Observational associations cannot prove causation; interventional and mechanistic studies are required.

Adult-only design precludes extrapolation to early life; pediatric cohorts are ongoing.

Single-country focus necessitates cross-national comparisons.

5. Conclusion

Interkingdom crosstalk between gut microbiota and host immunity is a central determinant of IgE-mediated allergic disease in Indonesia. Dysbiosis-marked by reduced diversity and depletion of immunoregulatory taxa-correlates with Th2 polarization, elevated IgE, and impaired Treg function. Microbiota-directed interventions partially restore immune homeostasis, supporting translational potential. These findings advance understanding of allergy pathogenesis in understudied populations and provide a foundation for developing regionally optimized microbiome-based therapeutics [1-42].

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