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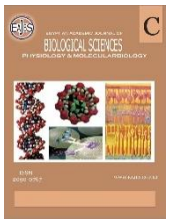

Gut Microbiota and Food Allergy: Mechanistic Pathways, Early-Life Programming, and Microbiota-Targeted Strategies

Hadeer Mattar
Department of Laboratory Medicine, Faculty of Applied Medical Sciences, Al-Baha University, Al-Baha - 65779, Saudi Arabia
E-mail: hmatar@bu.edu.sa

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ABSTRACT

Food allergies have emerged as a pressing global health concern, particularly in industrialized countries. Recent advances in immunology and microbiome research highlight the critical role of gut microbiota in modulating immune responses and shaping susceptibility to food allergies. Dysbiosis in microbial composition is increasingly associated with severe allergic responses and reduced immune regulatory activities. This review paper thoroughly examines the complex association between gut microbiota and food allergy, using latest human cohort research, animal models, and molecular insights. Key aspects addressed include early-life microbial colonization, immune system imprinting, and the role of microbial metabolites in immune tolerance. In addition to short-chain fatty acids (SCFAs), emerging modulators such as bile acids (acting through FXR and TGR5 pathways) and indole derivatives (activating the aryl hydrocarbon receptor to promote IL-22 production) are evaluated for their immunoregulatory potential. Emerging microbiome-targeted therapies, including probiotics, prebiotics, synbiotics, faecal microbiota transplantation (FMT), and tailored dietary regimens, are examined comprehensively for their therapeutic potential. Informed by a critical appraisal of 172 peer-reviewed studies published through 2025, this review identifies key microbial signatures of tolerance and proposes microbiota-targeted strategies for prevention and treatment. Emphasis is placed on early-life interventions and the integration of multi-omics platforms to translate microbial insights into clinically actionable solutions for managing food allergies.

INTRODUCTION

Food allergies (FAs) are an increasingly prevalent immunological disorder, affecting an estimated 32 million people in the United States alone, including approximately 8.0% of children and 5.0% of adults (Elghoudi and Narchi, 2022). The global incidence of food allergies has escalated in the last twenty years, especially in developed countries, prompting significant concerns about public health, nutritional habits, and immune system development (Rennie *et al.,* 2023). Food allergies are defined by inappropriate immune responses to food antigens, mostly mediated by immunoglobulin E (IgE), which is responsible for the majority of clinically severe reactions, including anaphylaxis (Carnazza *et al.,* 2025). Genetic predisposition plays a role in susceptibility, but environmental variables, particularly those that impact the early-life gut microbiota, are increasingly acknowledged as crucial determinants of immunological tolerance.

A primary growing emphasis is the function of gut microbiota in influencing food allergy sensitivity and response (Fofanova *et al.,* 2016; Hoskinson *et al.,* 2024). The gut microbiome is a complex and dynamic community of bacteria that plays a role in metabolic control, epithelial integrity, and immunological conditioning (Zhao *et al.,* 2023). Recent research indicates that disturbances in microbial diversity, especially during infancy, are significantly linked to the onset of allergic disorders, including food allergies (Khalil *et al.,* 2024). In addition to fundamental allergy sensitization, there is increasing interest in the correlation between gut microbiota and food allergen cross-reactivity, a phenomena where structurally similar antigens from diverse foods elicit overlapping immune responses.

This cross-reactivity is believed to arise from both common amino acid sequences (>70%) and conserved three-dimensional structures and physicochemical characteristics (Trier and Houen, 2023). It is to mention that microbiota-derived antigens, such as lipopolysaccharides (LPS), may mimic dietary proteins, triggering or modulating these cross-reactive responses through molecular mimicry and immune priming mechanisms (Rojas et al., 2018). Commensal microbes such as *Bifidobacterium* and *Lactobacillus* have been shown to support the development of regulatory T cells (Tregs) and enhance production of immunoglobulin A (IgA), both of which are crucial for promoting immune tolerance and maintaining intestinal homeostasis (Mazziotta *et al.,* 2023). Dysbiosis, characterized by an imbalance in microbial populations, has been linked to enhanced gut permeability, known as 'leaky gut,' impaired epithelial barrier function, and increased antigen exposure to mucosal immune cells (Christovich and Luo, 2022). A significant molecular connection between microbiota and immunological regulation is found in the function of microbial metabolites, especially short-chain fatty acids (SCFAs) including acetate, propionate, and butyrate. SCFAs are generated from bacterial fermentation of dietary fibers and have shown the ability to enhance Treg development by inhibiting histone deacetylases (HDACs) and activating G-protein coupled receptors (e.g., GPR43, GPR109A) (O'Riordan *et al.,* 2022; Silva *et al.,* 2020). In mouse models, SCFA supplementation has shown protective benefits against allergen-induced anaphylaxis by reinstating immunological tolerance.

Furthermore, early-life exposures, delivery method, nursing practices, antibiotic use, and the timing of food introduction may profoundly influence microbial succession and immune imprinting (Stephen-Victor *et al.,* 2020). Vaginal birth and exclusive breastfeeding correlate with enhanced colonization by advantageous anaerobes such as *Bifidobacteria*, while Cesarean sections and early antibiotic treatments are related with dysbiosis and a heightened risk of atopic illnesses (Davis *et al.,* 2022; Inchingolo *et al.,* 2024). Numerous studies indicate that microbial immaturity during these crucial periods might disrupt oral tolerance and bias immune responses towards a Th2 profile, which is fundamental to allergy illness (Dogra *et al.,* 2021). Probiotic and microbiome-targeted therapeutics are emerging as possible interventions, with clinical studies using *L. rhamnosus* GG, *B. longum*, and FMT demonstrating efficacy in modifying immune function and restoring microbial equilibrium. Nonetheless, heterogeneity in strain effectiveness, host response, and long-term safety continues to pose a barrier (Gulliver *et al.,* 2022; Hitch *et al.,* 2022). This review offers an in-depth examination of the relationship between gut microbiota and food allergy, focusing on mechanisms of cross-reactivity, microbial diversity, and immunological tolerance. This review seeks to connect essential microbiological discoveries with clinical approaches to tackle the pressing immunological issues of the 21st century by synthesizing results from murine and human investigations and emphasizing current progress in microbiota-targeted therapies.

Methodology:

This narrative review was based on a systematic search and critical appraisal of 172 peer-reviewed studies published up to July 2025. Literature was identified using PubMed, Web of Science, and Scopus, employing Boolean combinations of keywords such as *gut microbiota*, *food allergy*, *immune tolerance*, *early-life programming*, and *microbial metabolites*. The search strategy prioritized studies published up to 2025, with special emphasis on research from 2021 onward with inclusion criteria as: (1) original research or systematic reviews, (2) relevance to gut microbiota and food allergy, (3) studies involving humans, animal models, or immunologically relevant in vitro systems, and (4) English language whereas the exclusion criteria included non-peer-reviewed items, insufficient methodology, inaccessible full texts, or lack of relevance to immune-microbiota mechanisms. Articles were screened in two phases: (1) title/abstract screening and (2) full-text review. A total of 376 records were initially identified, of which 172 were deemed eligible and included. The selection process is illustrated in Figure 1 (PRISMA-style summary).

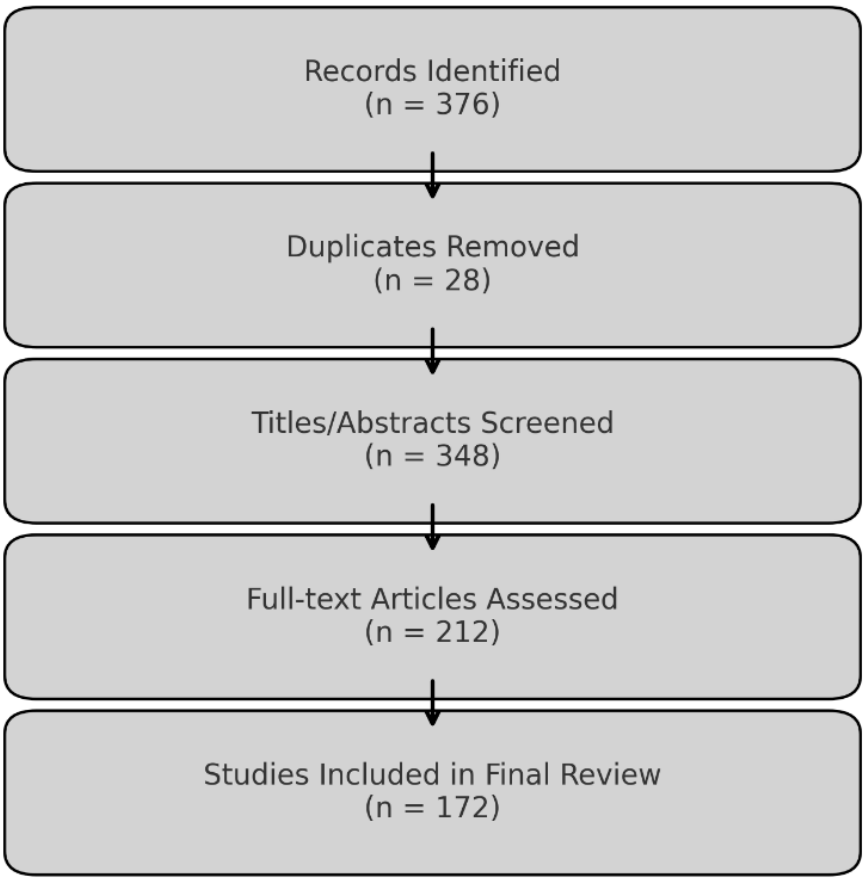

Fig. 1: PRISMA-style summary of literature selection. Flow of studies included in this narrative review, from initial identification to final inclusion.

Food Allergy: Epidemiology, Mechanisms, and Clinical Spectrum:

Clinically, there are three different categories of food allergies (FA) depending on the immunological processes involved, which include a range of immune-mediated adverse responses to dietary proteins: types of allergies: IgE-mediated, non-IgE-mediated, and mixed-type (Calvani *et al.,* 2020; Meyer and Palmer, 1934). Activation of mast cells and basophils by allergen-specific IgE antibodies characterizes IgE-mediated

responses, which comprise most acute clinical presentations and occur within minutes to hours after allergen exposure. Urticaria, angioedema, gastrointestinal problems, or even anaphylaxis might be symptoms of these responses (Anvari *et al.,* 2019). For example, in food protein-induced enterocolitis syndrome (FPIES), where T-cell driven inflammation produces vomiting, diarrhoea, and failure to thrive without IgE involvement, symptoms often manifest delayed in the gastrointestinal tract (2-48 hours post-exposure) (Nowak-Węgrzyn *et al.,* 2017). Eosinophilic esophagitis (EoE) is a mixed-type response that combines elements of both routes. It causes persistent inflammation and stricture development in the oesophagus via eosinophilic infiltration and occasional IgE sensitization (Anvari *et al.,* 2019; Dellon *et al.,* 2018; Vitte *et al.,* 2022). The intricacy of FA categorization is highlighted by emerging research that implies other subtypes, such as delayed IgE-mediated responses requiring basophil priming (Shah et *al.,* 2021). Especially in developed countries, the incidence of FAs has been steadily rising; recent estimates put the global prevalence at 8-10% in children and 4-6% in adults (Gupta *et al.,* 2018). Peanut allergies impact less than half of children in rural African communities, whereas 3% of children in the US have challenge-confirmed allergies (Joyce *et al.,* 2018).

Due to the inexplicable quick increase, this epidemiological trend rules out genetic predisposition as the only cause. The hygiene theory has given way to the "microflora hypothesis," which states that the immune system is impaired during development because of diminished microbial exposures caused by factors like as early antibiotic usage (OR=1.4), ultra-processed foods, and Caesarean deliveries (which are linked to a 30% increased risk of FA) (Marenholz *et al.,* 2017). This review article adds to our understanding of the mechanisms at work by demonstrating that delayed oral allergen introduction, in conjunction with cutaneous sensitization due to poor skin barriers (as seen in eczema patients), enhances Th2-skewing (Gray, 2020). Children residing in urban areas have FA rates that are two to three times greater than those in rural areas, indicating a clear correlation between urbanization and FA prevalence (Maestre et al., 2024; Morandini *et al.,* 2023). The disintegration of oral tolerance mechanisms is at the heart of food allergy pathophysiology, with various FA subtypes using separate pathways. Dietary antigens undergo processing by dendritic cells that go to mesenteric lymph nodes in IgE-mediated responses; this process is facilitated by dysbiosis-induced barrier failure (Chinthrajah *et al.,* 2016). By secreting IL-4, IL-5, and IL-13, these antigen-presenting cells drive B-cell class flipping to IgE production, promoting Th2 differentiation (Wang *et al.,* 2019). Acute symptoms are caused by histamine, leukotrienes, and prostaglandins released when mast cells degranulate in response to allergen cross-linking of FcεRI-bound IgE upon re-exposure. EoE is characterized by fibrosis generated by TGF-β and eosinophil recruitment mediated by IL-5/IL-13, while non-IgE processes in FPIES include T-cell activation driven by IL-17A/IL-15 (Dellon *et al.,* 2018). New evidence suggests that the IL-33/ST2 axis has a key role in dysbiosis, especially in the absence of butyrate-producing bacteria, in enhancing Th2 responses (Ge *et al.,* 2025). Exercise, nonsteroidal anti-inflammatory drugs (NSAIDs), and alcohol all have a role in increasing the absorption of allergens, which may lead to a wide range of clinical manifestations, from acute urticaria to chronic oesophageal strictures.

Modulating FA risk is a complicated interaction of hereditary, environmental, and behavioural variables. Twin concordance rates of 30–50% and relationships with filaggrin mutations (OR=3.2 for peanut allergy) are indicators of a hereditary susceptibility (Gupta and Margolis, 2020). The critical interaction between genes and the environment may, however, be epigenetic alterations caused by metabolites produced by microbes. Factors during pregnancy greatly

affect the course of risk; for example, the chance of FA increases by 30% with a caesarean section, while the chances of protective *Bifidobacterium* colonization increase with a vaginal birth and breastfeeding (Rautava *et al.,* 2012). Equally important are dietary habits during weaning; a research called LEAP found that high-risk babies whose peanuts were introduced early on (between four and six months) had an 81% reduction in peanut allergy symptoms (Lankireddy and Hopkins, 2024). There is a correlation between a rise in FA prevalence with modern lifestyle factors such as eating ultra-processed foods, which is linked to a 40% reduction in microbiota diversity, not getting enough vitamin D, and being exposed to particulate matter (Nascimento *et al.,* 2023). During the most malleable stages of development, from prenatal up until about the age of three, these risk factors seem to have the greatest impact on the immune system and microbial programming (Zhang et al., 2024).

The Human Microbiota: Composition, Function and Development:

The human gastrointestinal tract harbours an extraordinarily complex microbial ecosystem, comprising approximately 100 trillion microorganisms that collectively weigh up to 2 kg in adults (Yarahmadi *et al.,* 2024). Most of this community consists of bacteria, specifically *Firmicutes* (60–65%) and *Bacteroidetes* (20–25%). However, new multi-omics methods have shown that archaea, viruses (mainly bacteriophages), and fungi (*Saccharomyces* and *Candida* species) also play a significant role (Aggarwal *et al.,* 2022). Through complex interactions across kingdoms, these organisms carry out essential functions. For example, butyrate and other SCFAs are produced by microbes' fermentation of indigestible polysaccharides; these can supply up to 10% of the body's daily caloric needs. Additionally, these organisms regulate the host's metabolism through G-protein coupled receptors (GPR41/43) (Thulasinathan *et al.,* 2025). The microbiota also synthesizes essential vitamins (B12, K2) and bioactive compounds (tryptophan metabolites) that modulate systemic immunity (Shaw et al., 2023). Spatial metatranscriptomics was used to demonstrate that the mucosal-associated microbe (*Akkermansia muciniphila*) directly regulate epithelial tight junction proteins (claudin-3, occludin) through extracellular vesicle signalling, while luminal communities specialize in dietary fiber breakdown (Melo-Marques *et al.,* 2024). Modern, contamination-controlled research has disproved the long-established "sterile womb" theory by finding trace amounts of microbial DNA in amniotic and placental fluid, mostly from *Lactobacillus* and *Propionibacterium* species (Milani *et al.,* 2017). *Prevotella* and *Lactobacillus* are common in the microbiota of babies born vaginally, whereas *Staphylococcus* and *Corynebacterium* are more common in those born via caesarean section, and immunomodulatory *Clostridia* take longer to colonize these babies (Coscia et al., 2021; Suárez-Martínez *et al.,* 2023). Human milk oligosaccharides (HMOs) play a significant role in breastfeeding by feeding *Bifidobacterium longum* subsp. *Infantis*, a kind of bacteria that has specific clusters of genes that metabolize HMOs and improve the function of the intestinal barrier (Masi and Stewart, 2022). While the microbiota settles into an adult-like composition by the time a child reaches the age of 3, the first thousand days of life are crucial for immunological programming, during which microbial metabolites, especially butyrate, epigenetically control regulatory T cell FoxP3 expression by inhibiting histone deacetylase (Tang *et al.,* 2025). By the time they reach school age, children whose microbial succession patterns are ideal i.e., when *Bifidobacterium* predominates early on and *Clostridia* acquires the majority gradually have 60% reduced rates of food sensitivity, according to longitudinal cohort studies (Yao *et al.,* 2021). Several intrinsic and extrinsic factors influence the composition and stability of the gut microbiota. These include:

- **Diet:** The dietary factors are now known to have the greatest impact on the

ecology of microbes. In comparison to Western diets, the polyphenol and fiber-rich Mediterranean diet enhances microbial gene richness by 30% and raises SCFA production proportionally (Barber *et al.,* 2023). Omega-3 fatty acids increase the number of *Lachnospiraceae* (which is negatively related to peanut allergy, p = 0.002), and artificial sweets, such as saccharin, boost the proliferation of Bacteroides, which is connected to Th2 skewing (Del Duca *et al.,* 2022).

- **Antibiotics:** The use of antibiotics throughout a baby's first six months of life leads to a condition called chronic dysbiosis. New research shows that macrolides select for *Enterobacteriaceae* bacteria resistant to antibiotics, while simultaneously reducing *Bifidobacterium* abundance for two years or more (Shayista *et al.,* 2025). According to adjusted analysis, there is a threefold greater incidence of food allergies up to age 6 in children who were exposed to systemic antibiotics during the neonatal period (OR = 2.89, 95% CI = 1.34-6.92, P = 0.01) (Ofri *et al.,* 2025).
- **Environmental exposures:** Exposures to the environment: Environmental factors have effects that depend on the dose, but the "farm effect" is still strong; for example, compared to their urban peers, Amish children have 40% more microbial diversity and 5-fold more *Faecalibacterium prausnitzii*, a butyrate producer, which correlates with 70% lower rates of food allergies (Stein *et al.,* 2016). As a result of improved TGF-β signalling, having a pet lowers the incidence of eczema (OR=0.6) in newborn intestines by increasing *Lactobacillus* and lowering *E. coli* (Tun *et al.,* 2017).
- **Breastfeeding vs. Formula Feeding:** The methods of feeding, such as breastfeeding or formula feeding, leave permanent marks on the immune system. *Bifidobacterium*, accounting for 90% of all bacteria in a breastfed infants microbiome, transforms HMOs into galacto-oligosaccharides, which have an immunoregulatory effect (Wong *et al.*, 2024). On the other hand, babies that are given formula have more variety in their microbiome but more *Proteobacteria*, which is linked to 2.3 times more food sensitivity in at-risk populations (Chong *et al.,* 2022). In cases when breastfeeding is not an option, there is some evidence that pasteurized donor milk may be a good substitute, helping to maintain some of the microbiome advantages (Cacho *et al.,* 2017).

Microbiota–Immune System Interactions:

Gut–Immune Axis:

In a well-coordinated network known as the gut-immune axis, components of the host immune system interact directly with commensal microbiota to promote tolerance and immune surveillance (Zheng et al., 2020). Intestinal mucosa cells, especially those in the gut-associated lymphoid tissue (GALT), are responsible for this dynamic communication via molecular and cellular signalling (Fig. 2). More than 70-80% of the body's immune cells are in GALT, which includes Peyer's patches, isolated lymphoid follicles, and mesenteric lymph nodes. GALT is essential for antigen sampling and immune conditioning (Forchielli and Walker, 2005). Lymphoid regions activate T and B cell responses after M cells in the follicle-associated epithelium transport dietary and luminal microbial antigens to underlying dendritic cells (DCs) (Nakamura *et al.,* 2018). Functional heterogeneity in gut DC subsets has been shown by single-cell RNA sequencing. While $CD11b^{+}CD64^{+}$macrophages sustain tolerance by means of IL-10 signalling, $CD103^{+}CD11b^{+}$DCs are concentrated in the lamina propria and stimulate peripheral Tregs by means of retinoic acid and TGF-β production (Bain *et al.,* 2017). Intestinal epithelial cells (IECs) and innate lymphoid cells (ILCs) work together with these myeloid subgroups to adjust mucosal immunity. To detect microbes, pattern recognition receptors (PRRs) such toll-like receptors (TLRs) and receptors that are like NODs play an essential role. For instance, as shown in Figure 2, TLR5 can identify bacterial flagellin and stimulate Treg development; in contrast,

TLR4, which can detect LPS, may moderate immunological tolerance at low concentrations but can start inflammation when strongly activated (Yu *et al.,* 2024). Crucially, cytokines including IL-25, IL-33, and thymic stromal lymphopoietin (TSLP) are secreted by epithelial cells, which actively influence immunological tone. These variables, especially when present in dysbiosis, might cause immune responses to become more polarized toward Th2, which in turn can lead to allergy sensitization (Stanbery *et al.,* 2022). Duodenal biopsies from children with IgE-mediated food allergies demonstrate increased production of epithelial-derived alarmins (TSLP and IL-33), which correlates with greater eosinophil counts and mucosal inflammation (Schmidt *et al.,* 2014).

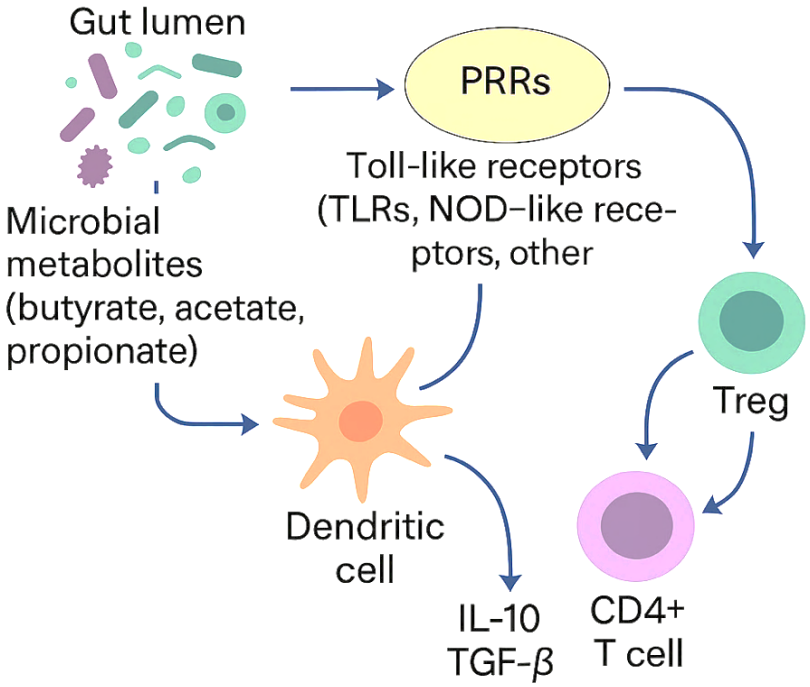

Fig. 2: Interaction between gut microbiota, microbial metabolites, and immune regulation.

Tolerance vs. Sensitization: Role of Microbiota and Immune Regulations:

Especially throughout development, the commensal microbiota is crucial in determining immunological tolerance. In the absence of microbiota, germ-free (GF) animal models show a 90% decrease in FoxP3^{+} regulatory T cells in the colon and an imbalanced Th2 response to allergens (Belkaid and Hand, 2014). Restorative colonization with certain microbial taxa may bring the immune system back into balance. As an example, the growth of Tregs is enhanced by *Clostridia* clusters IV and XIVa via the induction of TGF-β and IL-10 through pathways that are confined to MHC-II (Kamada and Núñez, 2014). Likewise, a multi-center metagenomic study indicated that infants with food allergies by the age of one had lower levels of faecal *Clostridia* (-55% reduction) and higher levels of *Enterobacteriaceae* (+180%) in the first 100 days of life, lending credence to the idea that *Bifidobacterium infantis* promotes PD-L1 expression on dendritic cells and encourages the formation of tolerogenic immune circuits (Azad *et al.,* 2015; Zhou *et al.,* 2022). In addition, daily treatment with *Bifidobacterium* breve BB99 resulted in a substantial rise in Treg counts (+47%) and a 30% decrease in oral food challenge reactivity, according to a randomized controlled experiment that included 88 children with proven egg allergy (Palmer *et al.,* 2017). It has also been shown that dysbiosis, an abnormality in the normal microbial ecology, may worsen sensitivity. The release of IL-33 and the activation of

basophils via the ST2 receptor are both increased when pathobionts like *E. coli* and *S. aureus* overgrow (Teufelberger *et al.,* 2018). This, in turn, enhances IgE production and allergen responsiveness. A decreased microbial diversity (Shannon index −38%) and higher levels of inflammatory markers, such as faecal calprotectin and β-defensins, are routinely seen in stool samples from persons with allergies (Heinzel et al., 2024). As a potential treatment option, FMT has become more popular. Anaphylaxis severity was decreased by 70% in mouse models of the disease when FMT was performed on peanut-sensitized animals from healthy donors. This reduction was mainly due to the development of colonic Tregs driven by IL-10 (Chernikova *et al.,* 2022).

Researchers are now studying (GUT-ALL: NCT05872144) the effectiveness of encapsulated FMT in alleviating symptoms and sensitization in children with food allergies (Huang *et al.,* 2024). Diet, microbiota, and immunological function are metabolically connected via microbial metabolites, particularly SCFAs. The fermentation of dietary fibers by anaerobic bacteria, such as *Faecalibacterium prausnitzii* and *Roseburia* species, produces SCFAs such acetate, propionate, and butyrate (O'Riordan et al., 2022). Butyrate has the strongest immunoregulatory characteristics of any of them. By promoting mucus formation and controlling the expression of tight junctional proteins, butyrate helps keep the intestinal epithelial barrier intact (Siddiqui and Cresci, 2021). It inhibits histone deacetylase 3 (HDAC3) at physiological concentrations (>1 mM in the colon), which increases Treg activity by increasing acetylation of the FoxP3 gene locus. To further modulate SCFA signalling and regulate myeloid and T cell lineages, GPR43 and GPR109A receptors are involved (Bakshi and Mishra, 2025). Reduced faecal butyrate has been associated in human trials with elevated blood IgE levels and more severe allergy reactions (Wang *et al.,* 2024). Propionate, on the other hand, influences bone marrow haematopoiesis, which in turn has systemic effects (Liu *et al.,* 2023; Lucas *et al.,* 2018). In the research work on mice given a high-propionate diet, Tan *et al.* (2016) examined the improvement in oral tolerance to β-lactoglobulin as a result of a 15% increase in tolerogenic $CD103^+$ DCs (Tan *et al.,* 2016).

The immune system is influenced by additional new metabolites. Epithelial integrity is improved by 12,13-diHOME, which is generated by certain strains of *Lactobacillus*, via the upregulation of tight junction proteins (claudin-4, occludin) (Lynes *et al.,* 2017). In a similar vein, indole-3-aldehyde, which is produced by *Bifidobacterium*, enhances IL-22 production and promotes mucosal healing by activating the aryl hydrocarbon receptor (AhR) (Abdulqadir *et al.,* 2023). Similarly, bile acids, particularly secondary bile acids, interact with host receptors such as farnesoid X receptor (FXR) and Takeda G-protein receptor 5 (TGR5), modulating dendritic cell activation and promoting regulatory T cell (Treg) differentiation. These findings underscore the broader landscape of microbiota-derived metabolites in shaping host immune responses and tolerance mechanisms relevant to food allergy. Contrary to what one would expect, low levels of systemic butyrate (<0.5 mM) might paradoxically activate mast cells and amplify allergic inflammation, even as high levels of luminal butyrate (>2 mM) promote barrier function and Treg induction (Folkerts *et al.,* 2020). These results provide credence to the idea that butyrate-conjugated nanoparticles should be targeted for distribution; in mouse models, they decreased peanut-induced anaphylaxis by 80%, and studies are now underway to determine their efficacy in people (Wang et al., 2023).

Dysbiosis and Food Allergy:

Evidence from Animal Models and Human Studies:

There is strong evidence that dysbiosis of the gut microbiota contributes to the pathophysiology of food allergies, as shown in experimental research using germ-free and antibiotic-treated animal models

(Uzbay, 2019; Zhang *et al.,* 2021). When exposed to food antigens, germ-free (GF) mice show an overactive Th2-type immune response, defined by increased levels of IL-4, IL-5, and IL-13 (Rivas *et al.,* 2013). Additionally, these mice have a significant decrease in the quantity of $FoxP3^+$ regulatory T cells (Tregs), suggesting that their ability to tolerate oral substances is impaired. A return to immunological homeostasis was shown when GF mice were colonized with certain microbial consortiums, most notably Clostridia clusters IV and XIVa. These species cause the cells in the colon to release TGF-β and encourage the formation of Tregs, which in turn reduces the synthesis of IgE specific to allergens and the degranulation of mast cells. Polukort *et al.* (2016) conducted groundbreaking research where they found that when *Anaerostipes caccae* colonized GF mice, the allergic reactions to ovalbumin were reduced by 75%. At the same time, the levels of IL-10 and TGF-β in the mesenteric lymph nodes were elevated (Polukort *et al.,* 2016). In a similar vein, an increased incidence of food allergies has been associated with antibiotic-induced microbiome depletion. Peanut protein challenge revealed elevated serum IgE levels and intestinal permeability in mice treated with metronidazole, neomycin, or vancomycin. These effects were minimized by restoring microbial balance using FMT from healthy donor mice, highlighting the causative role of microbial dysbiosis (Guo *et al.,* 2019; Ray *et al.,* 2021). More and more evidence from human cohort research links certain microbial profiles to food allergy susceptibility and symptoms. Fewer helpful commensals like *Bifidobacterium*, *Faecalibacterium*, and *Clostridia* and more pro-inflammatory taxa like *Escherichia coli*, *Klebsiella*, and *Enterobacter* are often seen in infants with IgE-mediated food allergies (Farnetano *et al.,* 2024; Koc *et al.,* 2025).

Nooij *et al.* (2025) conducted recent multicentre metagenomic profiling research of 350 children. They found that the abundance of *Ruminococcus gnavus* was the microbial risk score that predicted the emergence of food allergies with >90% sensitivity (Nooij *et al.*, 2025). Additional longitudinal data from the CHILD cohort research showed that being colonized by B. breve and *A. muciniphila* in early childhood was negatively linked to developing numerous food allergies by the age of three (Ismail *et al.,* 2016; Saturio *et al.,* 2021). Allergenic newborns have a diminished ability for ecological resilience, as seen by persistently lower levels of alpha diversity measurements (Shannon and Chao1 indices). In addition, metagenomes of children who suffer from long-term food allergies reveal an increase in bacterial virulence genes and LPS manufacturing pathways while decreasing the amount of SCFA-producing enzymes (Zhao *et al.,* 2023; Zhu *et al.,* 2024). When comparing allergic individuals to healthy controls, functional metatranscriptomic studies showed that the former had reduced expression of butyryl-CoA:acetate CoA-transferase while the latter had elevated levels of nitrate-reducing enzymes (Zhang *et al.,* 2020). Table 1, summarizes major microbial taxa reported in the literature as being either protective against or promotive of food allergy.

Table 1: Key Microbial Taxa Implicated in Food Allergy Protection or Risky

S. No.	Micrbial Taxon	Immune role	Implication in FA	References
1	*Clostridia* (Cluster IV, XIVa)	Promotes Treg differentiation via TGF-β and IL-10	Protective	Kamada & Núñez, 2014; Abdel-Gadir et al., 2019
2	*Bifidobacterium longum* ssp. *infantis*	Enhances epithelial barrier; induces PD-L1 on dendritic cells	Protective	Azad et al., 2015; Palmer et al., 2017
3	*Faecalibacterium prausnitzii*	Butyrate producer; reinforces tight junction proteins	Protective	Stein et al., 2016; Mo et al., 2024
4	*Lactobacillus* spp.	Produces indole derivatives; activates AhR pathway	Protective	Lynes et al., 2017; Abdulqadir et al., 2023
5	*Enterobacteriaceae* (e.g., *E. coli*)	Promotes Th2 skewing; increases IL-33 and TSLP	Risk	Teufelberger et al., 2018; Heinzel et al., 2024
6	*Ruminococcus gnavus*	Associated with increased LPS biosynthesis and antigen presentation	Risk	Nooij et al., 2025
7	*Staphylococcus* spp.	Pathobionts linked to dysbiosis and epithelial disruption	Risk	Beharry et al., 2023

Mechanistic Insights: Barrier Function, Inflammation, And Antigen Presentation:

The disruption of mucosal immunity and the facilitation of allergen sensitization are two of the many ways in which gut dysbiosis leads to food allergies. Intestinal permeability may rise, the gut's barrier function can change, and the immune cell balance can shift, especially about T regulatory cells (Tregs), which play a key role in preserving tolerance.

- **Barrier dysfunction:** When it comes to protecting the host immune system against luminal antigens, the intestinal epithelial barrier is the first line of defence. Goblet cells that secrete mucus, epithelial tight junctions, intraepithelial lymphocytes, and antimicrobial peptides make up this multilayered structure (Faderl e*t al.,* 2015). The production of mucins (like MUC2) and AMPs (such RegIIIγ and defensins) is improved by the microbiota, which plays a crucial role in controlling barrier function via microbial-epithelial interaction. It is well-documented that commensals such as *Faecalibacterium prausnitzii* and *Akkermansia muciniphila* enhance the production of tight junction proteins (such as claudin-1, occludin, and ZO-1) and the integrity of the epithelium (Fig. 3) (Mo *et al.,* 2024; Wade *et al.,* 2023).

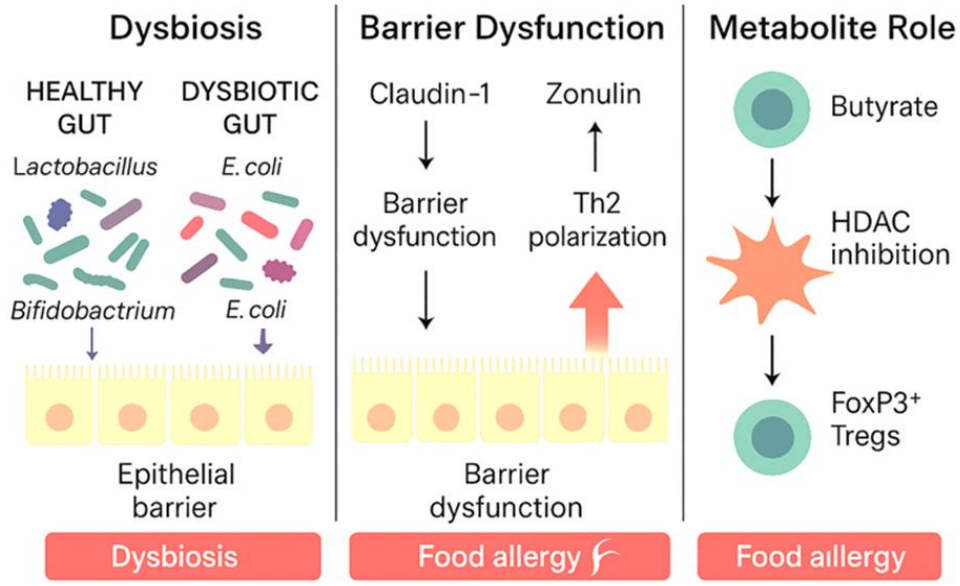

Fig. 3: Mechanistic pathways linking dysbiosis to food allergy.

Reduced SCFA synthesis and mucosal barrier breakdown are symptoms of dysbiosis, an imbalance in the microbiome that allows food antigens to translocate into the lamina propria. The stimulation of antigen-presenting cells and the skewing of naïve T cells toward Th2 differentiation, which is hallmark of allergic disease, are both caused by this increased permeability, sometimes known as "leaky gut" (Christovich and Luo, 2022). Tight junction proteins like claudin-1, occludin, and ZO-1 are compromised when dysbiotic changes occur, especially when butyrate-producing organisms like *Faecalibacterium prausnitzii* and *Roseburia* disappear. When antigens from the luminal compartment are able to translocate into the subepithelial tissues, a condition known as "leaky gut" occurs (Bowie *et al.,* 2012). In comparison to controls who did not have food allergies, individuals with food allergies had faecal zonulin levels that were three times higher and claudin-1 levels that were 2.8 times lower (Bergmann *et al.,* 2020; Bhat *et al.,* 2020). Restoring barrier function, suppressing serum IgE, and reducing anaphylaxis severity by 60-70% were all outcomes of colonizing germ-free or antibiotic-treated mice with butyrate-producing *Clostridia* (Abdel-Gadir *et al.,* 2019).

- **Inflammatory signalling:** The development and severity of food allergies may be influenced by dysbiosis, an imbalance in the gut microbiota, which can lead to inflammatory signalling. This link emphasizes the role of gut bacteria in immunological regulation, which in turn may impact the way the body reacts to food allergies. The immune system relies on pattern recognition receptors (PRRs) to detect microbial-associated molecular patterns (MAMPs) (Carding *et al.,* 2015). Toll-like receptors (TLRs) particularly TLR2, TLR4, and TLR5 and NOD-like receptors (NLRs) mediate microbial signal transduction and shape mucosal immunity. Overrepresentation of Proteobacteria, particularly *E. coli* and *Klebsiella*, promotes IL-33 and TSLP secretion from intestinal epithelial cells. These cytokines promote IgE class flipping in B cells and push dendritic cells toward a Th2-polarizing phenotype by boosting IL-4/IL-5 production. An even stronger reaction is elicited by ILC2s with high levels of ST2 receptor expression (Stanbery *et al.,* 2022). The activation of tolerogenic dendritic cells (tDCs) to secrete retinoic acid and IL-10 by commensal flagellin enhances regulatory T cells (Treg) responses (Hoang *et al.,* 2019).

Evidence suggests that TLR4 has a dual regulatory function; although low-dose LPS exposure increases tolerance via IL-10 induction, high-dose LPS exposure promotes Th2 sensitization. For example, *Bacteroides fragilis* secretes polysaccharide A (PSA), which acts via processes reliant on Toll-like receptor 2 (TLR2) to generate IL-10+ regulatory T cells (Tregs) (Ramakrishna *et al.,* 2019). Early-phase studies have shown that FMT from healthy donors into peanut-sensitive patients may restore Treg profiles and decrease allergen reactivity (Gray, 2020). Clinical studies found that children with FA exhibit elevated TLR4 and TLR9 expression on intestinal monocytes, associated with reduced Treg:Th2 ratios (Szebeni *et al.,* 2008).

- **Altered antigen presentation:** Dysbiosis can alter antigen presentation, increasing the risk of food allergies. These changes in the gut's microbial composition, can lead to heightened IgE responses and skewed T-cell differentiation towards a Th2 response, which is characteristic of allergic reactions. Dysbiosis alters dendritic cell maturation and antigen presentation profiles. In allergic individuals, DCs in lamina propria show elevated OX40L expression and decreased PD-L1, favouring effector T cell activation over tolerance (Iriki *et al.,* 2023; Wythe et al., 2012). SCFA deficiency also downregulates Aldh1a1, impairing retinoic acid synthesis, a factor for Treg induction.
- **SCFAs and postbiotics in tolerance induction:** SCFAs such as butyrate, acetate, and propionate are produced via anaerobic fermentation of non-digestible polysaccharides by gut microbiota. SCFAs function as potent immune-modulatory molecules that support epithelial repair, modulate immune cell activity, and induce tolerance to food antigens. Butyrate, for instance, inhibits histone deacetylases (HDACs), thereby enhancing the acetylation of histone H3 at the FOXP3 locus, promoting regulatory T cell (Treg) development (Siddiqui and Cresci, 2021). Recent trials have shown that children with milk or peanut allergy have significantly lower faecal SCFA levels compared to healthy controls ($p < 0.01$), especially butyrate and propionate (Goldberg *et al.,* 2020). Supplementation with butyrate-releasing nanoparticles in murine models restored Treg frequency in the mesenteric lymph nodes by over 40%, reduced serum IL-4 and IL-13, and conferred protection from allergen-induced anaphylaxis (Tan *et al.,* 2022). Beyond SCFAs, other postbiotics such as indole derivatives (e.g., indole-3-aldehyde) produced by *Bifidobacterium* spp. can activate the aryl hydrocarbon receptor (AhR), promoting IL-22 secretion from innate lymphoid cells (ILCs), which reinforces epithelial integrity and mitigates allergic priming (Sajiir *et al.,* 2024).

Cumulatively, these mechanisms create a feed-forward loop of barrier dysfunction, microbial imbalance, and immune dysregulation, all contributing to food allergen sensitization (Ali *et al.,* 2020). Targeted microbiome therapies, such as precision probiotics or SCFA-boosting interventions, are being explored as viable strategies to restore tolerance.

Early-Life Microbiota and Immune Imprinting:

Microbial Seeding During Birth:

The method of delivery is a pivotal determinant of an infant's initial microbial landscape. Vaginally delivered neonates are inoculated with beneficial commensals from the maternal vaginal and faecal microbiota including *Lactobacillus*, *Bifidobacterium*, and *Bacteroides*. These play essential roles in early immune education and mucosal tolerance (Milani *et al.,* 2017; Tang *et al.,* 2025; Wong *et al.,* 2023). In contrast, infants delivered via Cesarean section (C-section) are predominantly colonized by skin and environmental microbes, including *Staphylococcus*, *Corynebacterium*, and *Propionibacterium* species, and show delayed colonization by anaerobic bacteria critical for immune modulation (Beharry *et al.,* 2023). Recent multicenter cohort studies link C-section delivery to a higher risk of IgE-mediated food allergies, independent of

antibiotic use and feeding practices (Yao *et al.,* 2021). Infants born via C-section show reduced Clostridial diversity in the first six months, a pattern linked to increased sensitization to milk, egg, and peanut proteins (Rutayisire et al., 2016).

Impact of Infant Feeding and Weaning:

Breastfeeding is a crucial postnatal determinant of gut microbial colonization and immune system priming. Human milk is a bioactive matrix that supplies not only nutrients but also immunomodulatory components, including secretory IgA, lactoferrin, cytokines, and live maternal bacteria, along with human milk oligosaccharides (HMOs). The growth of beneficial bacteria in the gut is facilitated by HMOs, which are categorized as prebiotics. HMOs selectively promote the growth of *Bifidobacterium longum subsp. infantis*, a keystone taxon known to support Treg induction and reduce intestinal inflammation (Yang, Shuo *et al.,* 2024). Many studies have demonstrated the changes in the gut microbial makeup between breastfed and formula-fed babies. Nursing supplies for the newborn are high in prebiotics, fatty acids, lactoferrin, and other essential nutrients that protect the infant against pathogenic infections, enhance barrier function, and boost immune function. Nursing is a great way to ensure that the infant receives all these benefits (Ballard and Morrow, 2013). Breast milk stimulates the development of the gut microbiota by delivering probiotics and prebiotics and giving protection against infections. The bacteria that are prominent in breast milk include *Bifidobacterium*, *Lactobacillus*, *Staphylococcus*, *Bacteroides*, *Enterococcus*, *Streptococcus*, and *Clostridium* (Collado *et al.,* 2009). Preterm infants fed their own mother's milk showed greater gut microbiome diversity, with more *Clostridiales* and *Lactobacillales*, than those given donor or formula milk. This is regardless of whether the children were breastfed or fed formula (Cong *et al.,* 2016). Recent study has revealed the exclusive breastfeeding for at least 4–6 months is linked with a 30-40% reduction in food allergy risk, especially in infants with a family history of atopy. Breastfed infants show higher faecal concentrations of acetate and butyrate, which are known to enhance barrier integrity and immune tolerance (Danielewicz, 2022).

Maternal Microbiome and Prenatal Influence:

Beyond delivery and postnatal nutrition, the maternal microbiome exerts pre-birth effects on the developing immune system of the newborn. Dysbiosis in the maternal gut or vaginal tract caused by diet, antibiotics, obesity, or gestational diabetes can influence foetal immune imprinting via microbial metabolites, transplacental cytokine signalling, and even microbe-derived extracellular vesicles (Nyangahu and Jaspan, 2019). Other factors, such as the mode of delivery, breastfeeding, geographical location, living with siblings and furry pets, antibiotic treatment, and assisted reproductive technology, were found to have a significant influence on neonatal gut colonization, according to the findings of the TEDDY study, which involved six institutions in the United States and Europe and collected 12,500 stool samples from over 900 infants (Stewart *et al.,* 2018). A more recent prospective cohort (MELODY) study reported that maternal abundance of SCFA-producing bacteria during the third trimester was inversely associated with neonatal cord blood Th2-skewing cytokines (IL-4, IL-13), suggesting transplacental tolerance imprinting (Peter *et al.,* 2020). Furthermore, maternal intake of a high-fiber diet in late pregnancy was associated with a 25% lower incidence of food allergy in infants at 18 months (Palmer *et al.,* 2025).

Antibiotic Exposure and Microbial Perturbation:

Early-life antibiotic exposure, particularly in the first 100 days, has profound and often long-lasting effects on microbial succession (Fig. 4). Antibiotics are more often given to premature and C-section babies, which raises their chance of developing conditions including obesity, inflammatory bowel disease, and asthma in the future. Antibiotic exposure during the perinatal

period may delay the maturation of microbial activity until around 6 to 12 months after birth (Chong *et al.,* 2018). Broad-spectrum antibiotics reduce *Bifidobacterium* and *Lachnospiraceae* abundance, impair butyrate production, and alter Treg/Th17 balance (Fallani *et al.,* 2010). Meta-analyses indicate that antibiotic exposure before age 1 is associated with a 1.8–2.5 times higher risk of food allergy, especially when combined with C-section or formula feeding (Ahmadizar *et al.,* 2018). Conversely, environmental exposures such as pet ownership, farm residence, daycare attendance, and siblings promote microbial diversity and exposure to non-pathogenic endotoxins that help train the immune system. *Peptostreptococcus* bacteria are more prevalent, and *Bifidobacterium* bacteria are less prevalent in the gut microbiota of children who grow up with cats (Adamek *et al.,* 2019). Infants reared in pet-owning households had greater abundances of *Peptostreptococcaceae*, low levels of *Bifidobacteriaceae*, and animal-derived *B. pseudolongum* (Azad e*t al.,* 2013; Nermes *et al.,* 2015). A recent study (FARMFLORA birth cohort; N = 65) showed that farm-dwelling children had higher faecal microbial diversity and significantly lower rates of food sensitization under the age of 2 (12 months appropriate) (Ljung *et al.,* 2024). Children with siblings likely to have more *Bifidobacterium* and less *Peptostreptococcus* bacteria. The KOALA Birth Cohort Study in the Netherlands indicated that children with older siblings had a greater abundance of *Bifidobacteria* and enhanced gut microbial variety and richness compared to newborns without siblings (Penders *et al.,* 2007). A lack of older siblings was also related with early colonization by *B. adolescentis*, *Clostridium*, and *C. difficile*, whereas colonization by *Bifidobacteria*, *Bacteroides*, and *Lactobacillus* increased with larger number of siblings (Laursen *et al.*, 2015).

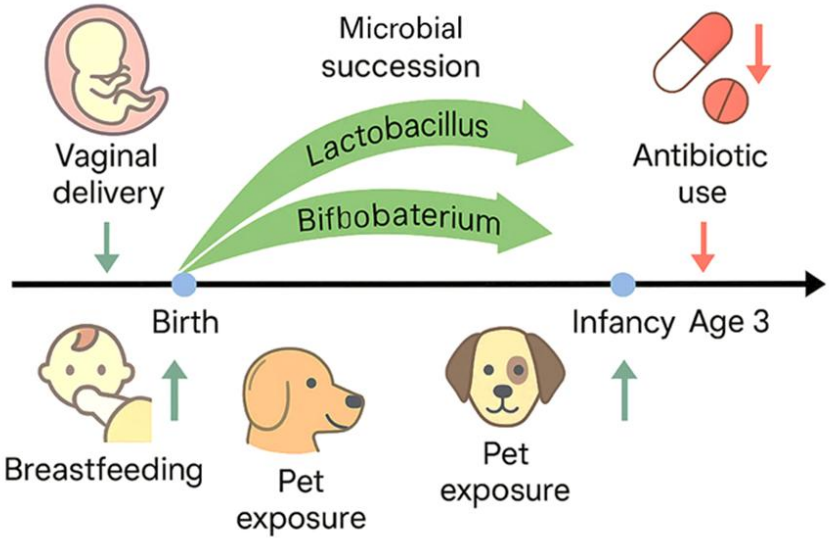

Fig. 4: Early-life microbial colonization and critical windows for immune development.

The timing of introducing allergenic foods is a critical window for immune education. Contrary to earlier beliefs advocating delayed introduction, landmark studies and trials have shifted paradigms by demonstrating that early introduction (between 4–6 months) of allergenic proteins like peanut, egg, and milk significantly reduces the risk of sensitization (Trogen *et al.,* 2022). Newer studies have shown that the benefits of early food introduction are amplified in the presence of a healthy, *Bifidobacterium*-rich microbiome (Beharry *et al.,* 2023; Chan *et al.,* 2018; Gupta and Sicherer, 2017). Infants introduced to peanut before 6 months who also had high faecal

butyrate levels were 80% less likely to develop peanut allergy at age 3 (Li *et al.,* 2025). Although further research is required to determine if this technique is applicable to low-risk groups, the early introduction of allergenic foods seems to be a successful strategy for reducing the public health burden of food allergies.

Therapeutic and Preventive Strategies: Microbiota Modulation Approaches:

Advances in our knowledge of the gut microbiota's involvement in immune modulation have led to the development of numerous microbiota-targeted therapies aimed at avoiding or reducing food allergies. These techniques vary from live microbial supplementation to dietary and faecal therapies, each aimed to change microbial composition and functional outputs in ways that enhance immunological tolerance (Fig. 5). Probiotics confer a health benefit when administered in adequate amounts such as *L. rhamnosus* GG and *B. breve*, which have shown promising clinical effects by promoting Treg differentiation, reducing inflammatory cytokines, and strengthening epithelial barrier integrity, thereby reducing allergic symptoms (Eslami *et al.,* 2020; Gill et al., 2009; Mazziotta et al., 2023). Complementing this, Prebiotics such as inulin and fructo-oligosaccharides are non-digestible fibers which serve as substrates that stimulate the growth of beneficial gut microbes and enhance SCFA production and improve gut barrier function, indirectly contributing to immune modulation (Kaur et al., 2021; Obayomi et al., 2024). When used in combination, Synbiotics aim to maximize these effects by simultaneously introducing beneficial microbes and supporting their growth (Bhatia et al., 2025; Markowiak and Śliżewska, 2017). While Postbiotics defined as non-viable microbial metabolites or components (e.g., SCFAs, bacteriocins) offer a promising alternative that exert health-promoting effects. The emerging research suggests postbiotics may offer safer and more stable alternatives for allergy prevention (Rafique *et al.,* 2023; Thorakkattu *et al.,* 2022).

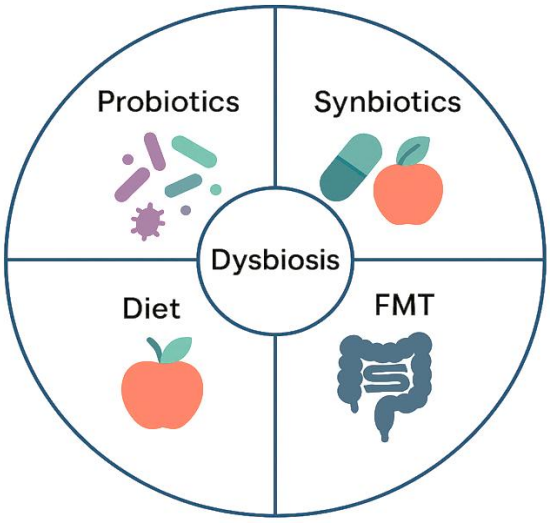

Fig. 5: Microbiota-targeted therapeutic strategies for food allergy prevention and treatment.

One particularly promising alternative/ method is faecal microbiota transplantation, which involves the transfer of healthy donor stool to restore microbial diversity. Though primarily used in *Clostridioides difficile* infections, preclinical studies in allergic mouse models have shown that FMT can reduce food allergy severity by restoring SCFA-producing bacterial populations (Cha and Sonu, 2025; Moya Uribe *et al.,* 2025; Novelle *et al.,* 2024). Challenges remain regarding donor selection, safety, and standardization for allergy applications, but FMT remains a promising avenue for further exploration (Karimi *et al.,* 2024). Alongside these microbial strategies, dietary modulation

remains a cornerstone of prevention: diets rich in fiber, polyphenols, and fermented foods promote microbial diversity and increase levels of SCFAs, thereby enhancing immune tolerance (Aziz *et al.,* 2024; Wastyk *et al.,* 2021). While Western dietary patterns, characterized by high fat and low fiber, are linked to dysbiosis and increased allergy risk (Clemente-Suárez *et al.,* 2023). Notably, early-life nutritional interventions such as exclusive breastfeeding and the adoption of Mediterranean dietary patterns are increasingly recognized for their capacity to influence microbiota development and lower the incidence of food allergies. Together, these microbiota modulation strategies represent a dynamic and evolving frontier in allergy prevention and therapy, with growing clinical evidence supporting their efficacy and expanding interest in personalized, microbiome-informed interventions.

Advances and Limitations in Microbiome-Allergy Research:

Methodological Limitations:

One major limitation in the study of microbiota and food allergy is the small cohort size and heterogeneity among participants. Many studies are underpowered to detect meaningful associations, especially given the complex and dynamic nature of the microbiome. Also, the variations in age, geography, diet, delivery mode, and antibiotic exposure introduce significant confounding factors (Berg *et al.,* 2020). Human microbiota composition is highly individualized, influenced by genetics, environment, and lifestyle. This variability makes it difficult to generalize findings and identify universal microbial biomarkers of disease (Xia *et al.,* 2025). Moreover, temporal variability even within the same individual complicates longitudinal assessments and causal inference. Establishing causality remains a major hurdle in microbiome research. Most human studies are observational, making it difficult to determine whether dysbiosis is a cause or consequence of food allergy (Flores *et al.,* 2014). While animal models offer more controlled environments, translating findings to humans remains a challenge due to interspecies differences in microbiota composition and immune response. The methodologies used for microbiota profiling, such as 16S rRNA sequencing and metagenomic shotgun sequencing, introduce analytical biases. For example, 16S rRNA sequencing may lack resolution at the species level and is influenced by primer selection and database accuracy (Humphries and Daud, 2018).

Emerging Research Tools:

Multi-omics approaches integrate genomics, transcriptomics, proteomics, metabolomics, and microbiomics to provide a comprehensive understanding of host-microbiota interactions. Metagenomic shotgun sequencing allows for functional profiling of microbial communities beyond taxonomic classification (Mohamed *et al.,* 2023; Pérez-Cobas et al., 2020). Metabolomics, particularly profiling of SCFAs and other microbial metabolites, enhances our ability to link microbiota composition with immune modulation and disease phenotypes (O'Riordan *et al.,* 2022). Gnotobiotic models, including germ-free and selectively colonized mice, offer controlled systems to examine causal relationships between specific microbes and host immunity. These models have been instrumental in demonstrating that colonization with *Clostridia* species can induce Treg development and protect against food allergy (Faith *et al.,* 2014). Humanized mouse models, colonized with human faecal microbiota, allow for the study of patient-specific microbial influences on allergic outcomes (Arrieta *et al.,* 2016; Moya Uribe *et al.,* 2025; Yang, Shuai *et al.,* 2024). Artificial intelligence (AI) and machine learning (ML) techniques are increasingly used to identify microbial patterns predictive of disease, classify complex microbiome data, and uncover hidden relationships between microbial features and clinical phenotypes (Dhaarani and Reddy, 2025; Li *et al.,* 2024; Rozera *et al.,* 2025). ML models can integrate multi-omics datasets to generate hypotheses, predict treatment response, and stratify patients based on microbial risk profiles.

Translating Insights to Clinical Practice: From Bench to Bedside:

Translating the expanding knowledge of microbiota-allergy interactions into effective clinical strategies represents a pivotal step toward personalized and preventive healthcare in allergy management. As the comprehension of host-microbiota-immune interactions expands, several potential therapeutic and preventative strategies are advancing from experimental models to clinical use. A particularly exciting advancement is the emergence of tailored microbiome-based medicines. Progress in microbial sequencing, metabolomic profiling, and immunological testing is enabling customized dietary treatments that promote the proliferation of beneficial bacteria and the synthesis of immune-modulating metabolites, including short-chain fatty acids (SCFAs) (Hitch *et al.,* 2022). These tailored techniques, including customized dietary regimens and specialized probiotic and prebiotic supplements, seek to enhance mucosal immunity, stimulate regulatory T cell (Treg) responses, and diminish the vulnerability to food allergies by rectifying certain microbial imbalances (Ashique *et al.,* 2024; Ma *et al.,* 2024). Longitudinal cohort studies are essential for delineating crucial periods of immunological imprinting and microbial growth. These studies provide insights on the optimal timing and methods for intervention by monitoring microbiome development from infancy to early childhood. The notion of early-life microbiome engineering, including maternal gut optimization during gestation and newborn supplementation with advantageous bacteria, has significant potential in mitigating the risk of atopic diseases, especially in genetically susceptible groups (Nunez *et al.,* 2025). As microbiota-based treatments approach clinical implementation, regulatory and ethical issues become more vital. The lack of standardization in probiotic formulations, variability in FMT donor screening procedures, and uncertainties in long-term outcomes demand stringent clinical trial designs and clear guidelines (Parigi *et al.,* 2023). Paediatric applications need further vigilance owing to the susceptibility of growing immune systems. Additionally, ethical concerns like informed consent, patient data confidentiality, equitable access to microbiome therapeutics, and the commercialization of tailored interventions must be resolved to guarantee safe and equitable implementation. These translational projects highlight the significance of a multidisciplinary approach integrating microbiology, immunology, clinical research, ethics, and health policy to connect laboratory findings with practical applications in food allergy prevention and treatment.

Conclusion

The growing prevalence of food allergy worldwide presents an urgent need to understand its underlying mechanisms and develop effective preventive strategies. Recent advances in microbiome science have highlighted the pivotal role of gut microbial communities in shaping immune development and oral tolerance. Evidence from animal models, human observational studies, and mechanistic investigations has demonstrated that alterations in the composition and function of the gut microbiota referred to as dysbiosis, are closely linked to the pathogenesis of food allergies. Key microbial metabolites, particularly short-chain fatty acids (SCFAs) like butyrate, play crucial roles in promoting regulatory T cell responses and maintaining epithelial barrier integrity. These findings have led to the development of microbiota-targeted interventions including probiotics, prebiotics, synbiotics, faecal microbiota transplantation (FMT), and diet-based strategies. Despite significant progress, several challenges remain. These include methodological limitations in microbiome research, the need for larger and more diverse cohort studies, and the difficulty in establishing causality. Nevertheless, the integration of multi-omics approaches, gnotobiotic models, and machine learning technologies offers promising avenues for future exploration. Moving forward, personalized microbiome modulation and

early-life interventions hold potential to transform the landscape of allergy prevention and treatment. A multidisciplinary effort involving clinical research, microbiology, immunology, and data science is essential to fully harness the therapeutic potential of the gut microbiota in combating food allergies.

Declarations:

Ethical Approval: This study did not involve human participants or animals. The research was limited to in vitro laboratory analyses of plant extracts and thus did not require ethical approval.

Informed Consent: Not applicable. The study did not involve human participants, patient data, or biological samples.

Declaration of Generative AI and AI-assisted Technologies in the Writing Process: The author has not used any of the generative AI or AI-assisted tool in the writing of this article.

Competing interests: The author declares no conflict of interest of any kind

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