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Opinion Article

The Immunobiological Path and Fate of the Human Intestinal Allergens and Pathobiont Translocation

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Abstract

The immune system and the microbiome are in a lifelong co-partnership. Symbiotic microbiome is coexisted with normal immune homeostasis while dysbiotic microbiome associated with immune dyseegulation. The objective of the present opinion paper was to make an at glance insight to the immune-biological path and fate of allergen and pathobiont translocations in human intestines. Intestinal dysbiosis associated with a characteristic molecular and immune micro-environment encompassing; presence of defective allele encoding immune functions, intestinal barrier dysfunction, tight junction impairment, allergen and pathobiont translocants passage. On passing through the impaired intestinal barrier, the translocated allergen or pathobiont behaves in either of three ways as reach blood stream through intestinal vascular barrier ,reach lymphatic circulation through intestinal epithelial barrier or reach Peyres patchs through M cell ,then, the interaction with immune mediator molecules and, immune cells such as ; brook of immune tolerance events, inflammatory-anti-inflammatory cytokine imbalance, T cell subsets imbalance as T reg./TH1/TH17. Allegenic epitope, memo-tope, epitope spreading, antigen bystander and stimulating production of allergenic antibodies, autoantibodies or reactive and/or autoreactive cells possible pointing to immune mediated diseases. Effector allergenic, effector immune, auto-reacting B cell may migrate to lamina propria to produce allergenic antibodies and/or autoantibody there in. Effector and auto-reacting T cells and T reg. cells may migrate to remote tissue compartments or stay there in. The fate can be a possible immune mediated disease or recovery.

Keywords: Allergy; Autoimmunity; Boron; Cytokines; Dysbiosis; Epitope; Gut; Memptope Pathobiont

Introduction

The oldest new entity of human body, the microflora residing in or on body compartments. The microbiome/microbiota has been advocated as a lifelong co-partner to the immune system [1]. The sharing parts of this lifelong correlation are the microbiome and the immune system in rather unifying recognition concept. Insults affecting this sharing co-partners have been found in a parallel cue of occurrence events as; microbiome symbiosis came in line with immune homeostasis, microbiome dysbiosis is being in line with infectious immune dysregulation. Though, microbiome dysbiosis is found in association with aging, infection, infestation and autoimmunity. Infectious Immune dysregulation is thought as it is consistent criteria for these disease states. The diversity, composition and function of the normal human microbiome in immune homeostatic state are different as individual variation holds in operation and different from that of human disease conditions. Such differences reside in alpha and beta

diversities that forms the basis of microbiome signatures [2,3]. The objective of the present opinion paper was to make an at glance insight to the immune-biological path and fate of the allergen and pathobiont translocation in the human intestines.

Features

The shift from a balanced, symbiotic gut microbiome to an imbalanced microbial community (gut dysbiosis) is accompanied by a wide range of biological alterations, including the following:

i. A shift from normal immune homeostasis to infectious immune dysregulation
ii. Altered cross-talk between the mucosal epithelium and luminal immune cells
iii. Changes in quorum-sensing signaling and the production of Short-Chain Fatty Acids (SCFAs) and amino acids
iv. Alterations in the microbial composition, diversity, richness and functional capacity of the gut microbiome
v. Emergence of dominant pathobionts [4]
vi. Increased tendency for pathobiont translocation [4-6]
vii. Possible causative factors include mRNA vaccination, antibiotic exposure, infections, inflammation, aging, co-colonization with environmental microbiomes and allergen translocation [7-14]

The Immunobiological Pathway

Mucosal Barrier Impairment

Microbial adhesion to the intestinal mucosal epithelium may initiate a cascade of inflammatory responses through prolonged activation of Toll-Like Receptors (TLRs) and zonulin signaling. The net result is disruption of Tight Junction (TJ) integrity and increased intestinal permeability. Chronic barrier dysfunction facilitates bacterial translocation, which perpetuates inflammatory responses, further damages tight junctions and promotes epithelial cell apoptosis through sustained inflammatory cytokine production [15].

In inflammatory diseases, intestinal pathobionts are frequently overrepresented within the dysbiotic microbiome. These microorganisms accelerate systemic inflammation by translocating across the epithelial barrier and disseminating to distant extraintestinal tissues. For example, Enterococcus gallinarum has frequently been detected in the livers of patients with autoimmune hepatitis [16].

Following translocation, pathobionts disrupt immune homeostasis by inducing systemic inflammatory responses. In contrast, beneficial symbiotic microorganisms are generally confined to the intestinal lumen, where they produce metabolites that support epithelial proliferation and differentiation and regulate immune cell populations, particularly regulatory T (Treg) cells and T helper 17 (Th17) cells [17].

Barrier Passage Patterns (BPP)

The gut mucosal barrier comprises three major physical barriers: the Mucus Barrier (MB), the Intestinal Epithelial Barrier (IEB) and the Gut Vascular Barrier (GVB).

The mucus barrier contains mucus that plays a crucial immunoregulatory role. Mucin-2 promotes the development of tolerogenic dendritic cells and supports beneficial commensal microorganisms, particularly butyrate-producing bacteria, which are essential for maintaining mucosal immune homeostasis.

The intestinal epithelial barrier consists of a single layer of epithelial cells connected by Tight Junctions (TJs) and Adherens Junctions (AJs). These structures regulate the trafficking of bacterial toxins, microbial products and dietary antigens across the intestinal epithelium into the lymphatic circulation, thereby initiating immune responses.

The gut vascular barrier determines which microbial components are permitted to migrate from the intestinal lumen into the systemic circulation [18].

Whole commensal bacterial cells may enter the lymphatic circulation by crossing the intestinal epithelial barrier [19]. Members of the genera Rikenellaceae, Bacteroides, Streptococcus and Blautia have also been reported to enter the systemic circulation through the gut vascular barrier [20].

Mucosal pathobionts may adhere to intestinal epithelial M cells, which transport them to macrophages within Peyer's patches. This process activates T-helper cells, which subsequently stimulate B- and T-lymphocytes to differentiate into effector and memory lymphocyte subsets. These immune cells participate in both local and systemic immune and autoimmune responses.

Accordingly, three principal Barrier Passage Patterns (BPPs) have been described:

- Lymphatic circulation
- Blood circulation through the gut vascular barrier
- M-cell-mediated mucosal transport [21]

Allergenesis

The gut microbiome modulates intestinal function through the production of metabolites that regulate energy metabolism, immune function and cellular communication within the intestinal barrier [22].

Gut dysbiosis is associated with immature immune responses and impaired barrier function [23]. It also enhances the expression of $CX3CR1^{+}$ dendritic cells, thereby promoting inflammatory immune responses [24].

An imbalanced gut microbiome has been implicated in the development of food allergy, asthma, allergic rhinitis, atopic dermatitis and atopic eczema [25]. Dysbiosis may increase intestinal permeability, allowing greater exposure of host tissues to microbial and dietary antigens. This process may stimulate immune activation and promote the migration of reactivated T cells to distant organs, leading to allergic inflammation in the lungs, skin and other tissues [26].

The Barrier Hypothesis, Biodiversity Hypothesisand Hygiene Hypothesis collectively explain how dysbiosis contributes to allergic diseases through the upregulation and downregulation of immune pathways (Table 1) [27-29].

Short-Chain Fatty Acids (SCFAs) are among the most important microbial metabolites involved in maintaining oral tolerance and epithelial barrier integrity, thereby reducing pathogenic responses to dietary antigens. SCFAs regulate the differentiation of regulatory T (Treg) cells and stimulate the secretion of interleukin-10 (IL-10). They also enhance epithelial barrier function by regulating goblet cell mucus secretion and promoting IL-22 production by group 3 Innate Lymphoid Cells (ILC3) [30].

SCFAs influence T-cell differentiation through G-protein-coupled receptors (GPR41, GPR43 and GPR109A) and by inhibiting histone deacetylases. Activation of the Foxp3 transcription factor promotes Treg differentiation and immune tolerance. Reduced SCFA production during dysbiosis may instead favor Th2 polarization, thereby increasing susceptibility to allergic disease [31].

Consequently, adequate SCFA production by a healthy gut microbiome plays a critical role in preventing food allergies, whereas gut dysbiosis increases the risk of allergic sensitization [32].

SCFAs also regulate the ILC2-B-cell-IgE axis. Early-life administration of vancomycin, an antibiotic known to deplete SCFA-producing bacteria, primes and amplifies this pathway, resulting in lifelong enhancement of susceptibility to type 2 allergic lung disease [33].

Hypothesis	Brief account	References
Hygiene	In early childhood exposure to environmental microbes positively influence development of allergy in children. Since child live in farm areas expose to microbe in farm than in city areas. child live in farm suffer less from allergies than in city.	[29]
Biodiversity	Gut microbiome dysbiosis have significant role in immune regulation. Biodiverse microbiome inline with	[27]

	health and dysbiotic limited microbiome in line with allergic disease	
Barrier	Mucosal epithelial dysfunction due to secondary structural changes like expansion of acanthosis lead to allergic sensitization type 2 inflammation	[28]

Table 1: Dysbiosis-driven allergic hypersensitivity.

Immunopathogenesis

The gut microbiome plays a fundamental role in the development and maintenance of the immune system by promoting intestinal barrier maturation and regulating immune cell function through the production of Short-Chain Fatty Acids (SCFAs). SCFA-producing bacteria regulate immune cell differentiation and promote the development of regulatory lymphocytes, which are essential for maintaining immune homeostasis and controlling immune responses [34].

Symbiotic microbiota also produce substantial amounts of fatty acids, hydroxyl fatty acids and indoles that influence host immune function through metabolic reprogramming and epigenetic mechanisms [35,36]. The intestinal mucosal barrier consists of mucins, antimicrobial peptides, dimeric IgA, Paneth cells and plasma cells. These cellular and soluble immune components form a protective barrier between luminal microorganisms and the intestinal epithelium, thereby preventing bacterial adhesion and invasion. However, disruption of tight junctions impairs mucosal barrier integrity, resulting in increased intestinal permeability [37,38].

Gut dysbiosis compromises epithelial barrier function and induces pro-inflammatory cytokine responses. Likewise, microbial adhesion to epithelial cells stimulates the production of pro-inflammatory cytokines that further disrupt tight junction integrity, ultimately leading to Leaky Gut Syndrome (LGS). LGS facilitates the translocation of pathobionts into the systemic circulation [39].

Pathobiont translocation triggers both local and systemic inflammatory responses, rendering the host susceptible to immune-mediated and metabolic diseases [40]. Dysbiosis also promotes the loss of immune tolerance, excessive T-cell activation and increased production of pro-inflammatory cytokines, thereby facilitating autoimmune responses. Current evidence has demonstrated associations between specific bacterial taxa and their metabolites, particularly butyrate-producing bacteria and the development of autoimmune diseases.

Gut dysbiosis activates multiple immune pathways, resulting in the upregulation of pro-inflammatory cytokines, including IL-1β, IL-6, TNF-α, IL-17, IL-12 and IFN-γ, while simultaneously downregulating anti-inflammatory cytokines such as IL-10, IL-4, TGF-β and IL-1 receptor antagonist (IL-1Ra). This creates a state of cytokine imbalance. Furthermore, dysbiosis disrupts the balance between regulatory T (Treg) cells and effector T-cell subsets, particularly Th17 and Th1 cells, leading to dysregulated Th17 responses and excessive cytokine production. Consequently, the principal immunological consequences of gut dysbiosis are cytokine imbalance and lymphocyte subset imbalance [34].

At the intestinal mucosal surface, pro-inflammatory cytokines impair tight junction integrity, whereas anti-inflammatory cytokines promote restoration of epithelial barrier function. Therefore, the cytokine milieu is a critical determinant of epithelial barrier integrity and intestinal homeostasis [39].

Inflammatory Immune Responses

Following entry into the local lymphatic system and/or systemic circulation, microorganisms and their metabolic products induce excessive production of pro-inflammatory cytokines, resulting in hypercytokinemia or the cytokine storm. In addition, microbial antigens stimulate both humoral and cellular immune responses, including autoimmune reactions, ultimately leading to the production of antibodies, autoantibodies, activated immune cells and autoreactive lymphocytes.

Effector immune cells may remain localized at the site of immune activation or migrate and home to distant tissues, where they contribute to disease pathogenesis. Likewise, memory lymphocyte subsets may either persist at the site of immune induction or

migrate to distant lymphoid organs to facilitate rapid responses upon antigen re-exposure. Regulatory T-cell (Treg) populations generated within the gut may also migrate to inflamed tissues and restore immune homeostasis through immunological tolerance mechanisms [41,42].

Mechanisms of Possible Allergic Immune-Mediated Diseases

Food antigens and allergens are introduced into the gastrointestinal tract through ingestion, digestion and absorption. In the presence of gut microbiome dysbiosis, SCFA-producing bacteria decrease both in abundance and metabolic activity. Reduced SCFA production impairs immune tolerance to dietary antigens while simultaneously compromising intestinal barrier integrity.

Consequently, food antigens translocate across the intestinal mucosa into the submucosal immune compartment, where they encounter Mucosa-Associated Lymphoid Tissue (MALT). This interaction initiates cellular immune responses followed by humoral immune responses, leading to the production of allergen-specific IgE antibodies. In addition, food antigens may cross the intestinal vascular barrier and enter the systemic circulation, where they reach peripheral lymphoid organs and stimulate systemic IgE-mediated immune responses [30,43].

SCFAs regulate T-cell differentiation through G-protein-coupled receptors GPR41, GPR43 and GPR109A, as well as through inhibition of histone deacetylases. Activation of the FoxP3 transcription factor promotes differentiation of regulatory T cells. Conversely, reduced SCFA production may skew T-cell differentiation toward a Th2-dominant response, resulting in increased IgE production and the development of allergic manifestations [31].

Mechanisms of Possible Autoimmune Immune-Mediated Diseases

Potential autoimmune diseases associated with gut dysbiosis arise through several immunological mechanisms, including breakdown of immune tolerance via clonal deletion failure, anergy, impaired regulatory T-cell activity, allergenic mechanisms, molecular mimicry, epitope spreading, bystander antigen activation, polyclonal B-cell activation, aberrant cytokine signaling, infectious immune dysregulation and microRNA-mediated regulation [44,45].

These mechanisms promote the production of antibodies, autoantibodies, autoreactive B cells, autoreactive T cells and activated effector lymphocytes. Disease severity may be assessed by measuring circulating levels of antibodies, autoantibodies, immune complexes and autoreactive lymphocyte populations, which are expected to exceed those observed during physiological autoimmune responses and to correlate with characteristic tissue pathology.

Translocation of microbial allergens into the systemic circulation may occur through mechanisms similar to those responsible for pathobiont dissemination or food allergen absorption [45-48].

Immunobiological Fate

The immunobiological outcome of gut microbiome dysbiosis depends on the host's ability to restore immune homeostasis. Following pathobiont translocation or allergen exposure, the immune response may either resolve through effective immune resilience and recovery or progress to chronic immune-mediated diseases, including allergic and autoimmune disorders [49].

Conclusion

Gut microbiome dysbiosis may contribute to the development of allergic asthma, allergic rhinitis, allergic dermatitis and other immune-mediated disorders through impairment of the intestinal barrier, increased epithelial permeability, translocation of pathobionts and allergens, reduced production of short-chain fatty acids and enhanced differentiation of IgE-producing B cells. The underlying immunological mechanisms include the expansion of pathobionts, disruption of mucosal barrier integrity, microbial translocation through lymphatic vessels, blood circulation or M-cell-mediated pathways and breakdown of immune tolerance through defective clonal deletion, anergy and impaired regulatory T-cell function. Dysbiosis also induces lymphocyte subset imbalance and cytokine dysregulation, resulting in persistent systemic inflammatory immune responses. These immune alterations promote the production of autoantibodies and autoreactive T cells through mechanisms such as molecular mimicry, epitope spreading and bystander activation, with or without complement activation, thereby contributing to the pathogenesis of autoimmune and allergic diseases.

Conflict of Interest
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Ethical Statement
The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement
Informed consent was obtained from all participants included in the study.

Authors' Contributions
All authors contributed equally to this paper.

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