

VITAMIN D IN IMMUNOMODULATION: A FACTOR IN THE PATHOGENESIS OF FOOD ALLERGIES

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ABSTRACT

Background: Vitamin D is known for its regulatory effects on both the innate and adaptive immune systems. Its potential role in modulating allergic responses, including the pathogenesis of food allergies, has gained increasing scientific attention.

Objective: To review and synthesize the current evidence on the immunomodulatory effects of vitamin D in the context of food allergy, with a focus on proposed mechanisms and clinical observations.

Methods: This narrative review was based on a structured literature search conducted in PubMed, Scopus, and Web of Science, covering 32 publications from January 2010 to April 2024. The search strategy used keywords and combinations such as "vitamin D", "food allergy", "immunomodulation", "gut immunity", "epithelial barrier", and "regulatory T cells". The selection emphasized research exploring immunological pathways, epithelial integrity, and microbiota-related mechanisms.

Results: The reviewed literature suggests that vitamin D may contribute to immune tolerance through upregulation of regulatory T-cell responses, suppression of Th2-mediated cytokines, reinforcement of epithelial barrier function, and modulation of gut microbiota composition. Several observational and preclinical studies report associations between vitamin D deficiency and increased susceptibility to food sensitization, particularly in early childhood. However, findings remain inconsistent across populations and study designs, and many results derive from animal models or small-scale human cohorts. There is a lack of large, well-controlled clinical trials.

Conclusions: Vitamin D appears to modulate immune pathways relevant to food allergy, including cytokine regulation, mucosal barrier integrity, and microbiota balance. However, current evidence is heterogeneous and largely preclinical, limiting firm conclusions. Future studies should focus on human clinical trials and mechanistic research to clarify causality. Individuals at high risk of vitamin D deficiency may benefit from targeted screening and correction, though clinical guidelines require further validation.

Keywords: vitamin D, food allergy, immunomodulation, mast cells, eosinophils

INTRODUCTION

Food allergies (FA) represent a significant public health issue, affecting up to 10% of the population. Over the past two to three decades, their prevalence has been increasing, particularly in highly industrialized and urbanized countries. Demographic analyses confirm that children are more predisposed to developing FA than adults, a phenomenon that may be attributed to environmental influences and immunological processes occurring during immune system maturation [1]. The essence of FA lies in the loss or inability to develop oral tolerance—a controlled suppression of immune responses to typically harmless dietary antigens. This predisposition results from dysregulation of immunological mechanisms and a predominance of the T helper 2 (Th2) cell phenotype, which leads to excessive production of immunoglobulin E (IgE) antibodies directed against allergens [2]. Eggs and peanuts are among the most common food allergens. The Australian HealthNuts study, based on oral provocation tests, reported confirmed FA prevalences of 9% for eggs and 3% for peanuts. Other frequently allergenic foods include milk, tree nuts, fish, shellfish, wheat, soy, and seeds, all of which play a key role in the epidemiology of FA [3]. Moreover, numerous scientific reports indicate that vitamin D possesses immunomodulatory properties in the context of FA, and its deficiency may contribute to the dysregulation of immunological mechanisms, thereby increasing the risk of developing food-induced allergic reactions [4].

EPIDEMIOLOGICAL CONTEXT

Food allergy is a growing health concern in Poland, particularly among children. The Polish arm of the EuroPrevall study reported food-related symptoms in 24.6% of children, with 5.6% classified as probable food allergy after standardized diagnostics [5]. These rates are comparable to those observed in Western European countries such as the United Kingdom (5–7%) and Germany (4–6%), though slightly lower than those in North America, where prevalence estimates often exceed 8–10% in children. In parallel, vitamin D deficiency remains widespread across Europe, but appears particularly severe in Poland. National studies indicate that over 80% of the Polish population has serum 25(OH)D levels below optimal thresholds, especially during winter. This contrasts with some Western European countries like Germany, France, or the Netherlands, where deficiency rates typically range from 50% to 70%[6,7]. The higher latitude of Poland and limited fortification policies may contribute to this difference. These data underscore the potential immunological relevance of vitamin D in the context of rising allergic diseases.

A comparative overview of the estimated prevalence of food allergy in children and the corresponding prevalence of vitamin D deficiency across selected European countries is presented in Table 1.

Table 1. Prevalence of Food Allergy and Vitamin D Deficiency

Country	Food Allergy Prevalence in Children (%)	Vitamin D Deficiency Prevalence (%)
Poland	5.6	>80
United Kingdom	5–7	50–70
Germany	4–6	50–70
France	4–5	50–60
Netherlands	4–6	50–65

OBJECTIVE

The aim of this study is to analyze the role of vitamin D in the immunological mechanisms associated with the development of FA. The study will delineate the potential mechanisms by which vitamin D influences immune system function, its role in the modulation of immune responses, and review recent scientific research exploring its relationship with the prevalence of FA.

METHODS

This narrative review was based on a structured literature search conducted in PubMed, Scopus, and Web of Science, covering publications from January 2010 to April 2024. The search strategy used keywords and combinations such as “vitamin D”, “food allergy”, “immunomodulation”, “gut immunity”, “epithelial barrier”, and “regulatory T cells”. Eligible studies included clinical trials, observational studies, animal models, and mechanistic reviews published in peer-reviewed English-language and Polish journals. Case reports, editorials, and articles lacking immunological focus were excluded. The selection emphasized research exploring immunological pathways, epithelial integrity, and microbiota-

RESULTS AND DISCUSSION

COMMON FOOD ALLERGENS

Food allergens are standardized within the WHO/IUIS allergen nomenclature database. This classification system is based on Linnaean taxonomy principles, and each newly defined allergen must meet specific criteria, including a confirmed IgE-mediated reaction in sensitized individuals. The ongoing updates of the WHO/IUIS database reflect advances in allergy research and the growing number of reported allergenic sources. Accurate classification is critical for the diagnosis and treatment of food allergies, particularly in the context of the increasing variety of plant- and animal-derived allergens[8].The most common food allergens include peanuts, tree nuts (e.g., almonds, walnuts, pecans, hazelnuts, macadamia nuts, Brazil nuts, pistachios, cashews), milk and dairy products, eggs, soy, wheat, certain fish (e.g., salmon, tuna), shellfish (e.g., shrimp, lobster, crab), and sesame [9]. In the United States, the Food and Drug Administration (FDA) mandates the labeling of nine major food allergens and their respective groups: peanuts, milk, shellfish, tree nuts, eggs, soy, fish, wheat, and sesame [10].

VITAMIN D METABOLISM

Vitamin D is a fat-soluble hormone with pleiotropic effects that plays a crucial role in regulating immune function, a key factor in the pathogenesis of FA [5]. In humans, sunlight represents the primary source for vitamin D synthesis. Although vitamin D is present only in limited quantities in selected foods, such as eggs, and is fortified in some countries, its endogenous synthesis in the skin remains the fundamental mechanism for maintaining optimal vitamin D levels [12]. Under ultraviolet B (UVB) radiation, 7-dehydrocholesterol undergoes a photochemical conversion to 25-hydroxyvitamin D₃, a critical step in the biosynthesis of vitamin D. Subsequently, vitamin D is bound by the vitamin D-binding protein (DBP), forming a complex that facilitates its transport in the bloodstream, though it remains biologically inactive in this form. The liver then converts it into 25-hydroxyvitamin D (25-OHD), an essential intermediate metabolite. Finally, in the kidneys, the enzyme 1 α -hydroxylase (CYP27B1) transforms 25-OHD into the active form, calcitriol (1,25-OHD), which enters cells and interacts with vitamin D receptors to initiate regulatory responses affecting various metabolic and immunological processes [13].

SIGNIFICANCE OF VITAMIN D IN THE DEVELOPMENT OF FOOD ALLERGIES

Vitamin D status in children is influenced by factors such as dietary habits, sun exposure, ethnicity, sex, and body mass, with up to 15% of children exhibiting a deficiency or insufficiency [14]. An increasing body of research examines the potential impact of vitamin D on FA development, particularly in relation to risk factors for deficiency such as limited sunlight exposure. Hwang et al. demonstrated that births during the winter season were associated with a higher incidence of FA symptoms over the following 12 months. Additionally, data analysis revealed that the prevalence of allergic diseases (excluding FA) was higher in children whose parents reported insufficient sun exposure before 24 months compared to those with adequate exposure [15]. An American study on acute allergic reactions documented geographical differences in emergency department visits, with rates of 5.5 per 1,000 cases in the northeastern United States versus 4.9 per 1,000 in the southern regions, and a stronger correlation between latitude and food allergen-induced anaphylaxis over other factors [16]. A meta-analysis by Psaroulaki et al. found that vitamin D deficiency significantly increased the risk of FA in children, with early childhood risk up to fourfold. In Australian cohorts, vitamin D deficiency nearly quadrupled the risk of egg allergy and doubled the risk of peanut allergy [17]. Furthermore, Matsui et al. reported that the season of birth might affect hypersensitivity to food antigens. Limited UVB exposure can disrupt Treg homeostasis, impairing immune tolerance mechanisms to dietary proteins irrespective of vitamin D levels. Additionally, diminished cutaneous synthesis of vitamin D may compromise intestinal barrier function and inhibit antimicrobial peptide production, thereby promoting gut dysbiosis and aberrant immune responses that increase the risk of FA [18].

In contrast, another meta-analysis did not confirm a protective effect of vitamin D against food allergy. Three randomized controlled trials evaluating supplementation during pregnancy or early infancy showed no impact on allergy incidence. Furthermore, six cohort studies found no association between maternal or infant 25(OH)D concentrations and the subsequent development of allergic disease [19].

POTENTIAL MECHANISMS OF VITAMIN D ACTION IN MODULATING THE IMMUNE RESPONSE IN FOOD ALLERGY

Vitamin D plays a pivotal role in modulating the immune system by influencing diverse regulatory mechanisms. It is essential for maintaining the integrity of the intestinal mucosal barrier, thereby limiting the exposure of the immune system to dietary allergens [20]. Deficiency in vitamin D may result in an imbalance of immune responses, compromised epithelial barrier function, and an increased susceptibility to infections and allergens. As dietary allergens breach the immune barrier, they stimulate B lymphocytes to produce elevated levels of IgE, which in turn exacerbates Th2-mediated allergic responses and disrupts innate epithelial defense mechanisms [21]. Moreover, vitamin D inhibits the proliferation of B and T lymphocytes while promoting the differentiation of regulatory T cells (Tregs). It reduces the levels of pro-inflammatory cytokines such as IL-17 and IL-21 and enhances the production of

anti-inflammatory cytokines, particularly IL-10. Vitamin D plays a critical role in regulating DNA methylation within Treg cells, contributing to their stability and population size. Deficient vitamin D levels lead to alterations in FOXP3 methylation, reducing the Treg cell pool and increasing the risk of food allergy. Interestingly, some reports also suggest that excessive vitamin D may limit Treg numbers through FOXP3-related methylation mechanisms, potentially exacerbating allergy risk. This implies that both vitamin D deficiency and excess can negatively affect immune tolerance [22]. Studies using animal models have demonstrated that vitamin D deficiency in the diet of pregnant females increases the susceptibility of their offspring to food allergy and reduces the proportion of regulatory T cells (Tregs) compared to control animals [23]. Moreover, vitamin D inhibits the proliferation of B and T lymphocytes while promoting the development of regulatory T cells; it decreases the levels of pro-inflammatory cytokines such as IL-17 and IL-21 and increases anti-inflammatory cytokine production, notably IL-10. Additionally, vitamin D attenuates the production of inflammatory mediators by monocytes [24] and suppresses the activity of CD69⁺ and CD4⁺ lymphocytes, potentially mitigating the severity of allergic reactions to food antigens. There is also emerging evidence that vitamin D₃ modulates mast cell activation, which may be relevant in controlling symptoms like allergic diarrhea [25,26]. Furthermore, Toll-like receptors (TLRs) have been implicated in the development of FA. Poole et al. conducted a cross-sectional study of 80 children, which revealed a direct association between TLR gene expression (including TLR2, TLR4, CD14, IL-5, IL-13) and the type of allergy, with TLR2 pathways playing a significant role in nut allergies [27]. The active metabolite 1,25(OH)₂D has been shown to inhibit eosinophil activation by inducing CXCR4 expression and reducing the release of mediators related to eosinophil activation, thereby alleviating inflammation [28]. Mast cells, fundamental players in allergic and inflammatory disorders, are critically regulated by vitamin D; its deficiency may lead to their uncontrolled, spontaneous activation, intensifying inflammatory processes [26]. Current evidence underscores the contribution of vitamin D to the immunomodulatory mechanisms underlying FA pathogenesis, although further detailed studies are required to elucidate these molecular pathways.

Table 2 summarizes selected immunological mechanisms by which vitamin D may influence food allergy pathogenesis. It highlights vitamin D's role in modulating regulatory T cell (Treg) activity, suppressing IgE synthesis by B cells, and attenuating mast cell degranulation. Each pathway is associated with specific immune cell types and linked to downstream effects on immune tolerance or allergic sensitization. The information is based on preclinical studies and mechanistic models, providing a conceptual framework for understanding vitamin D's immunomodulatory functions relevant to allergic disease. These pathways are under active investigation and warrant validation in human clinical studies.

Table 2. Immunological Pathways Influenced by Vitamin D in Food Allergy

Mechanism	Effect of Vitamin D	Related Immune Cells	Clinical Relevance
IL-10 expression	Increases	Treg (regulatory T cells)	Promotes immune tolerance
IgE synthesis	Reduces	B cells	May lower allergic sensitization
Mast cell degranulation	Inhibits	Mast cells	Reduces intensity of allergic response

An overview of representative studies selected for this review, categorized by study design (preclinical, observational, and clinical) is presented in Table 3. Each entry includes the study population or model used, the principal findings regarding the role of vitamin D in the development or modulation of food allergy, and the corresponding level of evidence.

Table 3. Summary of Key Studies Investigating the Relationship Between Vitamin D and Food Allergy by Study Type [29-32]

Author (Year)	Study Type	Population / Model	Main Findings	Evidence Level
Liu et al. (2017)	Preclinical	Mice	Vitamin D deficiency worsens Th2 immune response	Experimental (animal)
Smith et al. (2021)	Observational	430 children	Lower vitamin D levels associated with higher allergy risk	Cross-sectional

Jansen et al. (2019)	Clinical	100 patients with food allergy	Vitamin D supplementation reduced symptom severity	Randomized controlled trial
Mehrani et al. (2023)	Preclinical	Human mast cells (in vitro)	Vitamin D modulates mast cell activity, reducing allergic response and enhancing defense functions	Experimental (in vitro)

Preclinical studies offer mechanistic insights into immunomodulatory pathways, whereas observational and clinical trials contribute epidemiological and interventional perspectives. The diversity of methodologies highlights both the growing interest in this topic and the current limitations in translating mechanistic findings into clinical practice.

CONCLUSIONS

Vitamin D appears to influence multiple immune mechanisms relevant to the development and progression of food allergies, including effects on cytokine signaling, mucosal defense, and microbial balance. Nevertheless, the current evidence base is heterogeneous and limited by variable study designs, inconsistent outcome measures, and lack of interventional data. These limitations should be explicitly acknowledged. Future research should prioritize randomized controlled trials, longitudinal human studies, and mechanistic investigations to clarify causality and dose-response relationships. Special attention should be given to individual variability, including age, baseline vitamin D status, genetic predispositions, and environmental factors. The potential role of vitamin D in mucosal immunology and host-microbiome interactions also warrants deeper exploration.

PRACTICAL CONSIDERATIONS

Based on the literature, special consideration should be given to individuals at increased risk of vitamin D deficiency, including infants, pregnant and lactating women, people with limited time in the sun, patients with malabsorption syndromes, and dark-skinned individuals living in northern latitudes. In these populations, screening of vitamin D levels and correction of deficiency may be considered as an adjunctive strategy for allergy prevention until further clinical confirmation.

AUTHORS CONTRIBUTIONS

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The authors declare no conflict of interest

All authors have read and agreed with the published version of the manuscript.

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