The Role of Inhalant and Food Allergens in Atopic Dermatitis Exacerbations

ROLA ALERGENÓW WZIEWNYCH I POKARMOWYCH W ZAOSTRZENIACH ATOPOWEGO ZAPALENIA SKÓRY

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Streszczenie
Wprowadzenie: Atopowe zapalenie skóry (AZS) jest przewlekłą chorobą zapalną skóry, charakteryzującą się zaburzeniem funkcji bariery naskórkowej, dominacją odpowiedzi immunologicznej typu 2 oraz nawracającymi zaostrzeniami. Zarówno alergeny wziewne, jak i pokarmowe mogą wpływać na aktywność choroby, jednak ich rzeczywista rola pozostaje przedmiotem dyskusji, ponieważ uczulenie immunologiczne nie jest równoznaczne z klinicznie istotną alergią.
Cel: Celem pracy było podsumowanie aktualnej wiedzy dotyczącej udziału alergenów wziewnych i pokarmowych w zaostrzeniach atopowego zapalenia skóry, ze szczególnym uwzględnieniem mechanizmów patofizjologicznych, diagnostyki oraz implikacji terapeutycznych.
Metody: Przeprowadzono narracyjny przegląd piśmiennictwa z wykorzystaniem baz PubMed, Scopus i Web of Science. Uwzględniono badania oryginalne, przeglądy systematyczne, metaanalizy oraz wytyczne opublikowane w latach 2015–2025, a także wcześniejsze prace o fundamentalnym znaczeniu.
Wyniki: Uszkodzenie bariery naskórkowej sprzyja przenikaniu alergenów, indukcji odpowiedzi Th2 oraz produkcji swoistych przeciwciał IgE. Najczęstszymi alergenami wziewnymi są roztocza kurzu domowego, pyłki roślin, sierść zwierząt i pleśnie, natomiast do głównych alergenów pokarmowych należą mleko krowie, jaja, orzeszki ziemne, pszenica, soja, orzechy i ryby. Dodatnie wyniki testów skórnych oraz oznaczeń swoistych IgE świadczą jedynie o uczuleniu i wymagają interpretacji w kontekście obrazu klinicznego. Rutynowe diety eliminacyjne bez potwierdzonej alergii nie są zalecane.
Wnioski: Skuteczne leczenie AZS wymaga odbudowy bariery naskórkowej, kontroli zapalenia typu 2 oraz indywidualnej oceny znaczenia alergenów. Rozwój diagnostyki molekularnej i biomarkerów może poprawić personalizację postępowania terapeutycznego.

Słowa kluczowe: atopowe zapalenie skóry, alergeny wziewne, alergeny pokarmowe, IgE, bariera naskórkowa

Summary
Background: Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by epidermal barrier dysfunction, type 2 immune dysregulation, and recurrent exacerbations. Both inhalant and food allergens have been implicated in disease activity; however, their precise role remains controversial because immunological sensitization does not necessarily indicate clinically relevant allergy.
Objective: This narrative review summarizes current evidence regarding the contribution of inhalant and food allergens to AD exacerbations, with particular emphasis on pathophysiological mechanisms, diagnostic challenges, and therapeutic implications.
Methods: A literature search was performed using PubMed, Scopus, and Web of Science databases. Original studies, systematic reviews, meta-analyses, clinical guidelines, and position papers published between 2015 and 2025 were reviewed, with landmark earlier publications included where appropriate.
Results: Impaired epidermal barrier integrity facilitates transcutaneous allergen penetration, promoting Th2-mediated inflammation and allergen-specific IgE production. Common inhalant allergens include house dust mites, pollens, animal dander, and molds, whereas cow's milk, eggs, peanuts, wheat, soy, tree nuts, and fish represent the most frequent food allergens, particularly in children. Positive skin prick tests and serum-specific IgE identify sensitization but should not be interpreted as evidence of clinically relevant allergy without correlation with patient history. Current evidence discourages routine elimination diets in the absence of confirmed food allergy because of the risks of nutritional deficiencies and impaired oral tolerance. Emerging diagnostic approaches, including component-resolved diagnostics, basophil activation tests, and molecular biomarkers, may improve the distinction between sensitization and clinically significant allergy.
Conclusions: Effective management of AD requires restoration of epidermal barrier function, targeted control of type 2 inflammation, and individualized assessment of allergen relevance. Future research should focus on precision diagnostics and biomarker-guided therapeutic strategies to optimize personalized care.

Key words: atopic dermatitis, inhalant allergens, food allergens, IgE sensitization, skin barrier

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Wykaz skrótów:

AD – atopic dermatitis

AD/AE – atopic dermatitis / atopic eczema

IgE – immunoglobulin E

IL-4 – interleukin 4

IL-5 – interleukin 5

IL-13 – interleukin 13

IL-31 – interleukin 31

IL-33 – interleukin 33

TSLP – thymic stromal lymphopoietin

FLG – filaggrin gene

Th2 – T helper type 2

SPT – skin prick test

sIgE – specific immunoglobulin E

SCORAD – Scoring Atopic Dermatitis

1. Introduction

Atopic dermatitis (AD) is a chronic, itchy skin disease often starting in childhood and affecting 15–20% of children globally. Its cause is complex, involving genetic factors, skin barrier dysfunction, immune system imbalance, and environmental triggers like allergens and pollution. Key to AD is a damaged skin barrier, leading to allergen penetration and type 2 inflammation driven by cytokines IL-4 and IL-13. Sensitization to inhalant and food allergens varies among patients. Effective management requires personalized approaches focusing on restoring the skin barrier, controlling inflammation, and addressing allergen exposure through targeted therapies and diet.

Brief description of the state of knowledge

Inhalant and food allergens influence atopic dermatitis (AD) through skin barrier impairment and type 2 immune responses. Common triggers include dust mites, pollens, animal dander, molds, and foods like milk, eggs, and peanuts. Sensitization promotes inflammation and disease exacerbation but does not always correlate with severity. Early oral exposure may reduce allergy risk. Diagnosis requires distinguishing sensitization from true allergy. Treatment involves targeted avoidance, immunotherapy, and biologics, with ongoing research into personalized approaches and biomarkers for better management.

Atopic dermatitis (AD), also referred to as atopic dermatitis/atopic eczema (AD/AE), is a chronic, relapsing, and pruritic inflammatory dermatosis with an eczematous presentation, most commonly beginning in infancy or early childhood, although in a proportion of patients it persists into adulthood [1–3]. The disease is among the most common chronic dermatological conditions in the contemporary pediatric population [4]. Owing to its prevalence, AD constitutes a significant public health concern [5]. Its prevalence is particularly high in industrialized countries and shows an increasing trend [6].It affects approximately 15–20% of children and 3–5% of adults worldwide [1,2,7]. In the pediatric population, symptom onset most frequently occurs between the 3rd and 6th month of life, with a substantial proportion of cases manifesting in the first year and the majority by 5 years of age [4]. A consequence of the high prevalence and chronicity of the condition is its considerable impact on patient functioning, including a marked reduction in quality of life [7]

The pathogenesis of AD is multifactorial, not fully elucidated, and is associated with a feedback loop between an epidermal barrier defect and immune dysregulation, modified by environmental and infectious factors [1,8]. A key role is attributed to skin barrier dysfunction, which facilitates the penetration of environmental antigens and the initiation and maintenance of inflammation [2,3]. Of particular importance is filaggrin, an epidermal protein associated with barrier integrity as well as its hydration and resilience, and mutations in the FLG gene, the function of the filaggrin gene, which increase the risk of developing AD [2,3,9]. Damaged keratinocytes may initiate inflammatory cascades through the release of cytokines, thereby creating a bridge between structural defects and immune activation [10].

Recent evidence indicates that epidermal barrier dysfunction plays a central role in the development of food allergy in patients with atopic dermatitis [9]. According to the dual allergen exposure hypothesis, disruption of the skin barrier facilitates the penetration of environmental food proteins into the epidermis, where they are captured by antigen-presenting cells and promote a type 2 immune response leading to allergen-specific IgE production[9,11] Beyond cutaneous sensitization, accumulating evidence suggests that skin inflammation may also influence intestinal immune homeostasis through the skin-gut axis, thereby impairing the establishment of oral tolerance[9]

Importantly, the presence of allergen-specific IgE indicates immunological sensitization but should not be considered synonymous with clinically relevant food allergy, emphasizing the need for careful interpretation of allergy test results in the context of the patient's clinical history [9,12].

From an immunological perspective, it is associated with type 2 inflammation in the skin, encompassing overexpression of genes encoding, inter alia, IL-4, IL-5, IL-13, IL-31 and IL-33, as well as TSLP, with the involvement of multiple cell populations, including T lymphocytes, mast cells, and keratinocytes. [3] Particular importance is attributed to IL-4 and IL-13, whose activity drives the inflammatory response and impairs skin barrier function, thereby creating a self-perpetuating mechanism that sustains the disease [3,10]. From an allergological perspective, it is important that a leaky barrier facilitates antigen penetration and presentation, which may promote Th2 polarization and immunoglobulin class switching toward IgE, thereby linking allergen exposure with exacerbations and the coexistence of hypersensitivity [3].

An important component of the pathophysiology of AD is also the role of the environment and allergen exposure, particularly under conditions of an impaired epidermal barrier. It is emphasized that the defective barrier in AD enables the penetration of allergens, irritant substances, and bacteria, which promotes sensitization and may represent an initiating stage in the subsequent development of atopic diseases [2,13]. Among environmental determinants that may modulate the course of AD, the role of microorganisms and dysbiosis, aeroallergens, air pollution, diet, and climatic factors is highlighted, while it is underscored that the disease develops against a background of genetic susceptibility in an unfavorable environment [14,15].

Environmental factors not only trigger disease exacerbations but may also directly impair epithelial barrier function and enhance allergic sensitization [11].

Exposure to indoor and outdoor air pollutants, including particulate matter (PM2.5), nitrogen dioxide, ozone, volatile organic compounds, and tobacco smoke, has been associated with increased oxidative stress, disruption of epithelial tight junctions, and activation of epithelial-derived cytokines such as thymic stromal lymphopoietin (TSLP), IL-25, and IL-33 [10,16].

These mechanisms promote type 2 inflammation and may increase susceptibility to both inhalant and food allergen sensitization in genetically predisposed individuals[10,11,16].

Recent advances in allergy research have led to the development of the epithelial barrier hypothesis, which proposes that the skin, respiratory tract, and gastrointestinal tract constitute an interconnected barrier system. Structural damage to epithelial barriers facilitates the penetration of allergens, microorganisms, and environmental pollutants, promoting chronic inflammation and type 2 immune responses. In patients with atopic dermatitis, impaired epidermal barrier function may therefore represent not only a local skin abnormality but also part of a broader epithelial dysfunction contributing to the development of food allergy, allergic rhinitis, and asthma. This concept provides a biological framework for understanding the progression of the atopic march and highlights the importance of strategies aimed at preserving epithelial barrier integrity[11,12,17].

The aim of this paper is to discuss the relationship between exposure to inhalant and food allergens and exacerbations of AD, taking into account both pathophysiological mechanisms and clinical evidence regarding the significance of IgE sensitization, as well as practical diagnostic and therapeutic implications. The scope of the review includes the identification of typical sources of exposure, a description of the biological pathways through which allergens influence the course of the disease, and a concise synthesis of data indicating in which situations allergens may act as exacerbating factors in the heterogeneous population of patients with AD.

1.1. Methods of Literature Review

This narrative review was conducted to summarize current evidence regarding the role of inhalant and food allergens in the pathogenesis and exacerbation of atopic dermatitis. A comprehensive literature search was performed using the PubMed, Scopus, and Web of Science databases. Articles published in English between January 2015 and March 2025 were considered. Earlier publications were included only if they represented landmark studies essential for understanding the pathophysiology or clinical management of atopic dermatitis and food allergy [18,19].

The following keywords and their combinations were used: "atopic dermatitis", "atopic eczema", "food allergy", "inhalant allergens", "aeroallergens", "IgE sensitization", "skin barrier", "epicutaneous sensitization", "gut–skin axis", "microbiome", and "atopic march". Original studies, systematic reviews, meta-analyses, clinical guidelines, and position papers published by international scientific societies, including the European Academy of Allergy and Clinical Immunology (EAACI) and the American Academy of Allergy, Asthma & Immunology (AAAAI), were preferentially included [18,19].

Studies unrelated to atopic dermatitis, articles without available full texts, duplicate publications, conference abstracts without peer-reviewed manuscripts, and publications not written in English were excluded. Priority was given to recent high-quality evidence, including international clinical guidelines, systematic reviews, and meta-analyses [18,19].

2. Inhalant allergens and AD

In the course of AD, inhalant allergens are described as factors contributing to the development of sensitization within the atopic disease spectrum and as potential modulators of the clinical activity of skin lesions [15,20]. Among inhalant allergens, house dust mites are most frequently discussed, including *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae*, which appear to be particularly relevant due to their year-round presence, in the context of chronic exposure and possible modulation of disease activity [21–23]. The literature also draws attention to plant pollens, especially grasses and trees, which is reflected both in clinical observations and in measurements of specific IgE against major pollen allergens (e.g., birch and timothy) [21,24,25]. Significant roles are also attributed to animal-derived allergens and to mould fungi, including Alternaria alternata (formerly known as Alternaria tenuis), Penicillium notatum, and Aspergillus fumigatus [4,26].

From a practical standpoint, aeroallergens are categorized as indoor and outdoor allergens; specifically, the former group includes, inter alia, house dust mites, animal dander and epidermal allergens, and moulds, whereas the latter comprises primarily plant pollens [14].

The mechanism by which inhalant allergens influence the course of AD results from the pivotal role of epidermal barrier impairment and from the immunological consequences of allergen penetration into tissues. These effects are closely dependent on the condition of the epidermal barrier and on the manner in which the allergen comes into contact with the skin [14]. It is emphasized that factors such as air pollution may act as adjuvants for airborne allergens, increasing their allergenicity and promoting exacerbations [16]. It is also important that some aeroallergens exhibit proteolytic activity, e.g., certain pollens and house dust mite allergens, which may disrupt the integrity of epithelial barriers and sustain a vicious cycle of inflammation, dysbiosis, and epithelial leakiness [16,23]. Weakening of core barrier components, including reduced filaggrin, hydration, and stratum corneum ceramides, as well as tight junction dysfunction, increases skin permeability [15,26]. This leads to the initiation of a type 2 response cascade: an allergenic stimulus and/or mechanical damage results in keratinocyte activation and the secretion of mediators that promote recruitment and polarization of T-cell responses, with predominance of the cytokine axis characteristic of Th2, while in chronic lesions other components of the infiltrate and cytokine profile also coexist [15,24] Allergen-specific IgE associated with aeroallergens may activate effector cells via high-affinity receptors, leading to a mediator phase of inflammation, including histamine involvement [14]. Patients with atopic diseases and a predominant type 2 immune response may exhibit not only the expected immediate IgE-dependent type I reactions, but also delayed, cell-mediated type IV reactions in response to aeroallergens. This, in turn, indicates an overlap of two

mechanisms: IgE-dependent reactions that may manifest rapidly after exposure (immediate reactions) and delayed reactions that promote the persistence of chronic cutaneous inflammation and more difficult disease control [3,7,14,27].

Supportive observational evidence is provided by data in which AD severity was positively correlated with pollen levels [20,28,29]. At the same time, no association was demonstrated between AD severity assessed by the SCORAD scale and the presence of sensitization, indicating that sensitization alone does not necessarily translate linearly into disease severity in cross-sectional terms, even if it constitutes a risk component or a feature of a concomitant atopic phenotype [20]. Increased exposure to cat allergens has also been linked to greater AD intensification in adults [29] In a cohort study, it was demonstrated that a history of early-childhood AD was associated with a significantly more frequent presence of specific IgE against a broad spectrum of inhalant allergens, including animal allergens (cat, dog, horse), pollens (birch, timothy, mugwort), house dust mites, and moulds, assessed at successive time points throughout childhood and adolescence [2]. It was also indicated that among the most common allergens in skin prick tests in children with AD were, inter alia, dog, tree, cat, ragweed, cockroach, mould, house dust mite, and grass allergens, and that children sensitized to peanut, egg, cat, and dog were characterized by greater baseline skin barrier dysfunction (higher transepidermal water loss and lower filaggrin expression in non-lesional skin) and greater AD severity than children sensitized to other allergens or without sensitization [30]. In studies by Seo et al. [31] early-childhood AD phenotypes were analyzed, and attention was drawn to a subgroup of patients with later disease onset within this age category, characterized by high total IgE concentrations and high rates of sensitization to inhalant allergens, which reinforces the clinical perception that the relevance of aeroallergens increases with age.

3. Food allergens and AD

Atopic dermatitis is an autoimmune disease characterized by persistent itching and skin lesions. The pathophysiology of atopic dermatitis is complex and multifactorial, involving genetic abnormalities, a defect in the epidermal barrier, aberrant immune responses, and skin microbial imbalances[32]. Food allergy represents one of the most frequently discussed comorbidities in patients with atopic dermatitis, particularly in infants and children with moderate-to-severe disease.

The increased prevalence of food allergy among patients with atopic dermatitis is largely explained by impaired epidermal barrier function. Defects in structural proteins such as filaggrin facilitate the penetration of food allergens through inflamed skin, leading to transcutaneous sensitization. After crossing the damaged epidermis, allergens are captured by antigen-presenting cells, which promote a type 2 immune response and the production of allergen-specific IgE antibodies. This mechanism is particularly important during infancy, when skin barrier dysfunction often precedes the clinical manifestation of food allergy. Conversely, early oral exposure to food proteins promotes the development of immune tolerance, supporting the dual allergen exposure hypothesis as a key mechanism linking atopic dermatitis with food allergy[9,33].

Although sensitization to food allergens is frequently detected in patients with atopic dermatitis, particularly in those with moderate-to-severe disease, positive skin prick tests (SPT) or serum allergen-specific IgE results alone do not confirm clinically relevant food allergy. Both tests demonstrate sensitization rather than clinical reactivity and should always be interpreted together with a detailed medical history and, when indicated, oral food challenge results. Unnecessary dietary elimination based solely on laboratory findings may lead to nutritional deficiencies and, particularly in children, may adversely affect the development of oral tolerance [3,34].

Increasing evidence indicates that communication between the skin and the gastrointestinal tract plays an important role in the pathogenesis of atopic dermatitis and food allergy. Chronic skin inflammation may be associated with alterations in gut microbiota composition, which can impair the development of oral immune tolerance. Conversely, a diverse and balanced intestinal microbiota supports immune homeostasis and may reduce the risk of allergic sensitization. Although the precise mechanisms remain under investigation, the skin–gut axis is increasingly recognized as an important link between epidermal barrier dysfunction and food allergy [9,35]

In addition to allergen exposure, dietary patterns may influence the course of atopic dermatitis. Increasing evidence suggests that frequent consumption of ultra-processed foods rich in refined carbohydrates, saturated fats, and food additives may promote low-grade systemic inflammation and adversely affect gut microbial diversity. These alterations may impair immune regulation and oral tolerance, potentially contributing to allergic diseases, including atopic dermatitis. Although further studies are needed to establish causality, promoting a balanced diet rich in minimally processed foods may support both gut microbiota composition and immune homeostasis [11,12].

Food allergies are well documented in approximately one-third of children with moderate to

severe atopic dermatitis. Many foods, such as cow's milk, eggs, peanuts, wheat, soy, tree nuts, and fish, account for more than 90% of food allergies in children with atopic dermatitis. Their prevalence and the type of food may vary by age. These allergens can be categorized into age groups, as the most common food allergens in infants are cow's milk, eggs, peanuts, and soy, while in older children, they are fish, tree nuts, and shellfish [36].The prevalence of food allergies is increasing worldwide, affecting approximately 5% of adults and 8% of children in Western countries. The clinical response depends on factors such as the patient's sensitivity, the type of allergen, and how the food is processed. Adverse reactions in sensitive individuals can be mild, moderate, or even severe, such as anaphylaxis. Currently, there is no causal treatment for food allergies, so the mainstay of treatment is avoidance of the offending food [37].An important aspect of food allergy development is the dual exposure theory. This hypothesis posits that low-dose exposure to allergens through damaged skin promotes sensitization, while early, high-dose oral exposure leads to the development of immunological tolerance, reducing the risk of food allergy. Studies have shown that early introduction of allergens such as peanuts and eggs into infants' diets may reduce the risk of food allergy, although

the effectiveness of this strategy for other foods has not yet been clearly demonstrated [33].Food allergies can be divided into two categories: IgE-dependent and IgE-independent. Theformer is characterized by a simplified immunopathogenesis consisting of three phases: sensitization, challenge (re-exposure to the antigen), and the subsequent appearance of clinical symptoms. During the sensitization phase, IgE is produced and mast cells and basophils are sensitized by binding antibodies to FcεRI receptors. Subsequent exposure to the allergen causes degranulation of these cells, triggering a rapid allergic reaction with the release of potent inflammatory mediators. This reaction manifests itself with symptoms such as hives, itching, swelling, and wheezing, and is associated with reactions to specific food proteins such as milk, eggs, wheat, peanuts, tree nuts, fish, seafood, soy, and sesame [38]. Unlike IgE-mediated allergies, non-IgE-mediated reactions are characterized by a slower progression and are primarily associated with mechanisms of innate immunity that are not yet fully understood. An IgE-mediated reaction is characterized by a severe allergic reaction, a serious complication of which is anaphylaxis, whereas non-IgE-mediated allergies are subacute or chronić inflammation and primarily affect the gastrointestinal tract. These include food protein-induced enteritis syndrome, food protein-induced enteropathy, allergic proctocolitis of infancy, and eosinophilic gastrointestinal diseases such as eosinophilic esophagitis [38].Food allergy diagnosis is based on skin prick testing, specific IgE testing, and in some cases, atopy patch testing. The gold standard for diagnosis remains the double-blind food challenge,which is time-consuming, expensive, and carries the risk of severe reactions [39].

While patients with atopic dermatitis may choose to implement an elimination diet, such measures may increase the risk of developing food allergy, particularly IgE-mediated allergy, as confirmed by studies such as LEAP and retrospective cohort analyses. In a retrospective study of 298 patients undergoing food elimination for atopic dermatitis, 19% developed IgE-mediated allergy, 30% of whom experienced anaphylaxis. In other cohorts, children with atopic dermatitis developed IgE-mediated allergy after a median of 2.3 years of elimination dieting, and in one group of 30 patients suspected of having food allergy, 23 were confirmed by challenge testing, with one mortality due to cow's milk and 52% of anaphylaxis [3]. A systematic analysis of 10 randomized trials found that an elimination diet may modestly improve eczema severity, pruritus, and sleep problems in children with mild to moderate atopic dermatitis, although the evidence is low-certainty. The use of an elimination diet in patients with atopic dermatitis is controversial. The effects are independent of the elimination method or documented food allergy, but there is a risk that non-selective elimination diets may promote the development of IgE-mediated allergy. Therefore, the potential benefits of the diet should be considered in the context of possible risks and alternative treatments [40].Elimination diets also carry the risk of nutritional deficiencies, including calcium, protein, fatty acids, and vitamins, which can lead to growth retardation in children. The 2023 guidelines clearly demonstrate that the risks of an elimination diet outweigh its benefits. In patients with atopic dermatitis who do not experience immediate food reactions, implementing food avoidance may increase the risk of allergies. Therefore, most foods should remain in the diet to prevent serious food allergies and to maintain important nutrients in the growing child's diet without causing deficiencies [3].

Taken together, current evidence indicates that the relationship between atopic dermatitis and food allergy extends beyond simple allergen exposure and involves complex interactions between epidermal barrier dysfunction, immune dysregulation, environmental factors, and the gut microbiome. Consequently, the diagnosis of food allergy in patients with atopic dermatitis should always be based on a comprehensive clinical assessment rather than sensitization tests alone. Appropriate skin barrier care, avoidance of unnecessary elimination diets, and adherence to current recommendations regarding the timely introduction of complementary foods represent important components of evidence-based management aimed at reducing the burden of allergic disease [9,33].

4. Interaction between inhalant and food allergens

In the course of AD, inhalant and food allergens should be regarded as a single, shared immunological landscape of atopic disease, in which a predisposition to produce allergen-specific IgE antibodies and an established type 2 polarization promote multifocal sensitization and clinical variability of the disease phenotype [15,20]. It is suggested that the typical sequence of atopic disease manifestations includes early development of sensitization to food allergens accompanied by AD symptoms, followed, with increasing age, by sensitization to inhalant allergens and the clinical picture of allergic rhinitis and asthma [20,41]. Inhalant and food allergens may participate in the same disease process through partially shared effector pathways [42].

A particular form of the interaction discussed is cross-reactivity, in which primary sensitization to an inhalant allergen may determine an IgE response to homologous proteins present in foods. AD links a barrier defect (including that related to filaggrin) with dominance of the Th2 response and the activity of cytokines such as IL-4 and IL-13, which may affect epidermal differentiation and promote the persistence of chronic inflammation. This, in turn, may create a positive feedback loop: exposure to environmental allergens facilitated by the impaired barrier promotes the induction and maintenance of Th2 responses, while the Th2 response may simultaneously exacerbate barrier dysfunction, increasing susceptibility to subsequent stimuli [10].

Within cross-reactivity, a key example is the relationship between house dust mite allergens and shellfish, interpreted in terms of allergenic protein homology. At the molecular level, tropomyosin has been identified as the most common allergen responsible for shellfish allergy, and its substantial homology with arthropod tropomyosins includes, inter alia, house dust mites and cockroaches [3,43]. In addition, after sublingual immunotherapy with mite allergens, improved shrimp tolerance has been reported in a patient with shrimp allergy [44].

An important aspect is also the co-occurrence and mutual amplification of allergic phenotypes. For example, in a cohort of children with asthma, it was shown that the risk allele in IL4RA increased the odds of AD, and AD significantly increased the odds of food allergy, with the effect of genotype on food allergy being partially mediated by AD [45]. Among preschool children with eczema, those who were sensitized more frequently presented with polysensitization, suggesting broader

and more multi-source activation of IgE responses in this group [2] In AD, primary impairment of the epidermal barrier facilitates allergen penetration and initiates an inflammatory response that secondarily deepens the barrier defect, increasing susceptibility to subsequent environmental exposures regardless of their source [46]. Within this feedback loop, type 2 mediators are important both as an effect of exposure and as a factor maintaining conditions conducive to further exacerbations, because IL-4 and IL-13 inhibit the expression of epidermal proteins and ceramides and reduce the production of antimicrobial peptides, thereby promoting the persistence of inflammation and susceptibility to environmental stimuli [26].

The intensity and significance of these interactions are influenced by age-, genetic-, and environment-related modulatory factors. In adolescents and adults, the role of food allergies resulting from prior pollen sensitization increases [5,7,42,46]. Genetic disturbances of epidermal structure and function facilitate the process of transcutaneous sensitization, which increases the likelihood that a patient will react to allergens present in the environment (including aeroallergens) and will develop sensitization and symptoms after food exposure [9,26,46]. In turn, the environment operates, on the one hand, through allergen exposures in the surroundings and, on the other, through factors that increase disease activity and susceptibility to exacerbations, such as exposure to tobacco smoke, climatic factors, or the familial context of atopy, among others [4,23,25,47]. Consequently, the interaction between inhalant and food allergens in AD exacerbations should be understood as the effect of overlapping shared inflammatory and barrier pathways with an individual genetic background, age-conditioned immunophenotypic characteristics, and the patient's environmental context, which also includes seemingly non-specific factors capable of amplifying allergic responses [13,14,48].

Table 1. Mechanisms linking inhalant and food allergens with atopic dermatitis exacerbation

Mechanism	Clinical significance
Epidermal barrier dysfunction	Facilitates penetration of inhalant and food allergens, increasing the risk of sensitization and chronic inflammation.
Transcutaneous sensitization	Exposure of food allergens through damaged skin promotes Th2 immune responses and allergen-specific IgE production, particularly during infancy.
IgE sensitization	Positive SPT or serum-specific IgE indicates sensitization but does not necessarily confirm clinically relevant food allergy.
Skin–gut axis	Alterations in gut microbiota may impair oral tolerance and contribute to allergic inflammation in patients with atopic dermatitis.
Environmental factors	Indoor pollutants, tobacco smoke and aeroallergens may enhance epithelial damage and amplify inflammatory responses.
Food elimination diets	Appropriate only after confirmation of clinically relevant food allergy; unnecessary elimination may increase nutritional deficiencies and the risk of IgE-mediated allergy.
Restoration of epidermal barrier	Regular emollient therapy and appropriate anti-inflammatory treatment reduce allergen penetration and support long-term disease control.

5. Diagnostic and therapeutic strategies

Establishing the contribution of allergens to AD exacerbations requires an approach that distinguishes sensitization alone from clinically relevant allergy and takes into account concomitant, non-allergic exacerbating factors [20]. A thoroughly obtained medical history and observation of the clinical pattern assume a primary role and should serve as the starting point for interpreting the results of allergy testing [21]. Tests that detect sensitization have an auxiliary value in this context, because a positive result does not determine that a given allergen triggers exacerbations. [14,49,50]. When a given factor is considered causative, confirmation of an exposure–symptom relationship is essential, and in equivocal situations controlled provocations remain decisive [50].

Therapeutic management aimed at allergen control depends on confirmation of the role of exposure. With regard to food allergy, elimination of products deemed clinically relevant remains the fundamental tool [46]. In the field of food allergy, dietary interventions should be implemented only in cases in which a clinical relationship has been reliably confirmed, because eliminations introduced on the basis of sensitization alone may not lead to improvement and are associated with the risk of nutritional consequences [50]. Similarly, for inhalant allergens, with the caveat that in the case of house dust mites effective exposure reduction is difficult due to the year-round presence of the allergen [41].

New research directions focus on objectivizing hypersensitivity and improving differentiation of disease phenotypes through biomarkers, as well as on targeted immunomodulation. The utility of eosinophils for monitoring, compared with total IgE, is being investigated as a measurable indicator of inflammatory activity [20].Increasing emphasis is also placed on a molecular approach, as cytokine-profile analyses and multi-allergen testing may allow clinically relevant sensitization to be distinguished from cross-reactivity and may support the individualization of avoidance recommendations or qualification for immunotherapy [48,51]. Fan and Mishra [52] describe the development of approaches targeting neuroimmunological pathways and indicate that biomarkers linked to the activity of inflammatory axes may support patient stratification and treatment selection. It is suggested that interventions modulating the microbiome may confer potential benefits. In this respect, probiotics are being investigated in clinical studies as potential modifiers of the course of AD [13,15,16]. IgE is discussed as a potential marker of disease phenotype and concomitant allergies, although available data underscore the need to interpret results in close reference to the clinical picture [49].

Recent advances in allergy diagnostics have also expanded the use of molecular allergology and functional diagnostic assays. Component-resolved diagnostics (CRD) enable the identification of IgE directed against individual allergenic proteins rather than whole allergen extracts, thereby improving diagnostic specificity and facilitating differentiation between primary sensitization and cross-reactivity. In selected patients with equivocal clinical findings, CRD may reduce diagnostic uncertainty and support individualized management decisions, particularly in cases involving peanut, hazelnut, and cashew allergy [51,53].

The basophil activation test (BAT) is an emerging functional assay that complements conventional allergy testing by assessing basophil activation following in vitro allergen stimulation. Unlike skin prick testing and serum-specific IgE measurements, BAT reflects the biological activity of allergen-specific IgE and may improve discrimination between asymptomatic sensitization and clinically relevant food allergy. Current EAACI recommendations suggest that BAT may be considered in selected patients with equivocal diagnostic findings, particularly in peanut and sesame allergy, although limited availability and the need for specialized laboratory facilities currently restrict its routine clinical use[34,53].

6. Summary and conclusions

Atopic dermatitis (AD) results from a feedback loop between a defective epidermal barrier and a dominant type 2 immune response. Barrier dysfunction facilitates penetration of inhalant and food allergens, yet sensitization alone does not prove clinical relevance. Standard tests (SPT, sIgE) do not always reflect symptoms. Management should be interdisciplinary: restoring barrier function, controlling inflammation (e.g., IL-4/IL-13 blockade), assessing true allergen relevance, and avoiding unnecessary elimination diets.

The relationship between atopic dermatitis and food allergy requires careful clinical interpretation, as sensitization alone should not be equated with clinically relevant allergy. Positive skin prick tests and elevated serum allergen-specific IgE identify immunological sensitization but should always be interpreted in the context of the patient's history and clinical presentation. When uncertainty persists, oral food challenge remains the reference standard for confirming food allergy. Therefore, diagnostic decisions should rely on an integrated assessment rather than on laboratory results alone [3,34].

Current evidence also emphasizes that effective management of atopic dermatitis extends beyond allergen avoidance. Restoration of epidermal barrier integrity through appropriate skin care, regular emollient therapy, and individualized anti-inflammatory treatment remains a cornerstone of disease management. At the same time, elimination diets should only be recommended when a causal relationship between food exposure and clinical symptoms has been confirmed, as unnecessary dietary restrictions may increase the risk of nutritional deficiencies and loss of oral immune tolerance [22,54]. Future studies should focus on improving diagnostic precision by integrating clinical assessment with molecular diagnostics, functional assays, and emerging biomarkers. Further research is also required to clarify the interactions between epithelial barrier dysfunction, environmental exposures, and the skin–gut axis in order to optimize preventive and therapeutic strategies for patients with atopic dermatitis [3,11,13].

In routine clinical practice, careful medical history taking and thorough physical examination remain fundamental for distinguishing clinically relevant food allergy from asymptomatic sensitization, irritant reactions, food intolerance, or disease flares unrelated to allergen exposure.

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