



**THE INTERPLAY BETWEEN FOOD ALLERGY AND ATOPIC DERMATITIS:
MECHANISM, DIAGNOSIS, AND MANAGEMENT**

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ABSTRACT

Atopic dermatitis (AD) and food allergy (FA) are two conditions that frequently coexist and present significant challenges in both diagnosis and management. This review aims to describe the link between atopic dermatitis and food allergy, as well as their diagnosis, management, and prevention, especially in the Asian context. The findings indicate that impairment of the skin barrier and exposure to food allergens through the skin (epicutaneous sensitization) play central roles in the development of FA among individuals with AD. From a diagnostic perspective, it is essential to distinguish between food sensitization and clinically significant food allergy in order to avoid unnecessary elimination diets. Management strategies encompass comprehensive care for AD, including skin-barrier maintenance and anti-inflammatory therapy, alongside evaluation for FA when clinically indicated, and avoidance of confirmed trigger foods. Preventive efforts, particularly in high-risk infants, show promise through early skin-barrier care and controlled introduction of food allergens. In the Indonesian and broader Asian contexts, adaptation of these strategies should consider local dietary patterns and the availability of allergy and dermatology services.

Keywords: atopic dermatitis; diagnosis; food allergy; management; skin barrier

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INTRODUCTION

Atopic dermatitis (AD) and food allergy (FA) represent two interrelated immunological disorders whose co-occurrence poses significant challenges in clinical practice. Epidemiological and mechanistic evidence increasingly supports a bidirectional relationship, wherein skin barrier dysfunction in AD patients predisposes them to sensitization to food allergens, and conversely, food allergens may exacerbate or perpetuate AD (Papapostolou et al., 2022).

Central to this interplay is the dual-allergen-exposure hypothesis, which postulates that early exposure to food allergens through a compromised skin barrier, such as that seen in AD, can lead to immune sensitization, while oral exposure may promote immune tolerance (Brough et al., 2020, 2022). In AD, defects in skin barrier proteins (e.g., filaggrin), lipid abnormalities, and microbial dysbiosis can facilitate the penetration of food antigens (Darlenski et al., 2021; Hansen-Sackey & Hartono, 2025; Kim et al., 2019). Upon entry, these allergens are taken up by dermal dendritic cells, which migrate to lymph nodes and drive a type 2 (Th2) immune polarization, leading to IgE class-switching and sensitization (Liu et al., 2024; Ogulur et al., 2025).

Moreover, there is emerging recognition of a skin gut axis in this pathology: chronic skin inflammation in AD may influence gut immune and epithelial homeostasis via systemic inflammatory mediators, microbial metabolites, and neuroimmune pathways, thereby contributing to food allergy beyond mere sensitization (Davis et al., 2024). From a diagnostic standpoint, differentiating between food sensitization and clinically meaningful food allergy is crucial, as overdiagnosis may result in inappropriate dietary restrictions and nutritional risk. Similarly,

therapeutic strategies must address both restoring barrier integrity and modulating immune responses to avoid triggering or perpetuating FA in AD patients.

Preventive approaches are gaining traction, especially in high-risk infants: early barrier repair using emollients, controlled introduction of dietary allergens, and aggressive management of eczema may collectively reduce the risk of FA development (Tham et al., 2024). Nevertheless, critical gaps remain, including optimal timing, choice of interventions, and identification of biomarkers to stratify risk. Skin tape-stripping techniques, for example, are under investigation as noninvasive means to identify early biomarkers in infants (Kim et al., 2024). This article critically reviews the underlying mechanisms of the AD–FA nexus, outlines current diagnostic paradigms, and evaluates established and emerging strategies for management and prevention. Thereby, we aim to provide a comprehensive framework to inform both clinical decision-making and future research in this evolving field.

METHOD

This article was conducted as a narrative literature review focusing on the relationship between atopic dermatitis and food allergy, encompassing mechanisms, diagnostic approaches, and management strategies. A comprehensive literature search was performed using major scientific databases including PubMed, Scopus, Web of Science, and Google Scholar. The search covered articles published between 2010 and 2025. The following keywords and their combinations were used: “atopic dermatitis”, “food allergy”, “skin barrier”, “epicutaneous sensitization”, “dual allergen exposure hypothesis”, “diagnosis”, “management”, “prevention”, “microbiome”, “infant allergy”. Boolean operators (AND, OR) were applied to refine the search.

Inclusion criteria: (1) peer-reviewed original research articles, systematic reviews, meta-analyses, and clinical guidelines; (2) publications in English; (3) studies involving human subjects and relevant experimental models; and (4) articles addressing at least one of the following: pathophysiology, diagnosis, management, or prevention of AD and FA. Exclusion criteria: (1) conference abstracts without full text; (2) case reports with limited generalizability; (3) non-peer-reviewed publications; and (4) articles not directly related to the AD–FA relationship.

All retrieved articles were initially screened based on title and abstract. Relevant full texts were then reviewed independently. Disagreements regarding eligibility were resolved through discussion and consensus. Key information extracted from selected studies included: (1) study design and population; (2) pathophysiological mechanisms; (3) diagnostic approaches and their clinical relevance; (3) management and prevention strategies; and (4) key outcomes and conclusions. Data were synthesized qualitatively and organized into thematic sections: mechanisms/pathophysiology, diagnosis, management, and prevention, allowing integrated interpretation of findings.

RESULT

Mechanisms/Pathophysiology

Skin barrier disruption

One of the central mechanisms linking atopic dermatitis (AD) to food allergy (FA) is the dysfunction of the epidermal barrier, which facilitates the penetration of allergens and promotes immune sensitization. In AD, structural abnormalities occur at multiple levels of the skin barrier:

Filaggrin deficiency and cornified envelope defects

Filaggrin, encoded by the FLG gene, is a critical protein in the terminal differentiation of keratinocytes and in the formation of the cornified cell envelope. Loss-of-function mutations in FLG compromise the integrity of the stratum corneum, increasing transepidermal water loss and rendering the skin more permeable to environmental antigens (Kubo et al., 2012). Moreover, filaggrin-derived products (e.g., natural moisturizing factors) help maintain skin hydration and

acidity; their depletion further undermines barrier function (Yang et al., 2020). In addition to filaggrin, reductions in other structural proteins such as loricrin, involucrin, and tight-junction components (e.g., claudin-1) have been observed in AD, further weakening the epidermal barrier (Hansen-Sackey & Hartono, 2025).

Lipid abnormalities in the stratum corneum

The intercellular lipid matrix of the stratum corneum, composed of ceramides, cholesterol, and free fatty acids, is substantially altered in AD. Patients often show reduced total ceramide content, shortened ceramide chain length, and an altered lipid profile, which together impair the barrier's structural cohesion (Yang et al., 2020). These lipid disturbances are exacerbated by type-2 cytokine milieu (e.g., IL-4, IL-13), which suppress enzymes involved in lipid synthesis, further degrading barrier capacity (Hansen-Sackey & Hartono, 2025). Additionally, increased skin pH in AD (often due to genetic and environmental factors) activates serine proteases, which disrupt cell–cell adhesion and degrade key barrier proteins, worsening permeability (Hansen-Sackey & Hartono, 2025).

Tight junction (TJ) disruption

Tight junctions in the granular layer of the epidermis (notably claudin-1) form an additional “liquid–liquid” barrier. Their impairment in AD contributes to increased paracellular permeability, allowing allergens to bypass the stratum corneum and reach deeper immune cells. Loss of TJ integrity also correlates with altered lipid metabolism and reduced expression of structural barrier molecules (Yang et al., 2020).

Epicutaneous sensitization

Because of the compromised skin barrier, food allergens can more easily traverse the epidermis and elicit immune responses:

Dual-allergen exposure hypothesis

According to this hypothesis, early cutaneous exposure to food proteins via a disrupted skin barrier (especially in AD) favors Th2-type immune sensitization, whereas early oral exposure promotes immune tolerance (Tham & Leung, 2019).

Immune activation in the skin

When allergens penetrate through a damaged barrier, they encounter keratinocytes and immune cells. Keratinocytes respond by releasing cytokines such as thymic stromal lymphopoietin (TSLP), IL-1, and TNF- α , which activate Langerhans cells (LCs) and other dendritic cells (Kubo et al., 2012). Activated Langerhans cells extend their dendrites beyond tight junctions to sample antigens in the epidermis and then migrate to regional lymph nodes, where they present allergenic peptides to naïve T cells (Kubo et al., 2012). Under the influence of Th2-polarizing cytokines (e.g., IL-4, IL-13), these T cells drive B-cell class switching to IgE, establishing allergen-specific sensitization (Brough et al., 2020).

Experimental evidence

Animal models support this mechanism: for instance, mice with *FLG* deficiency or tape-stripped skin that are exposed topically to peanut or ovalbumin develop systemic allergen-specific IgE responses and, upon re-exposure, show signs of allergic reaction (Tham & Leung, 2019).

Clinical and epidemiological correlates

Observational human studies also support the relevance of epicutaneous sensitization. For example, exposure to peanut oil on inflamed or eczematous skin correlates with a higher risk of peanut allergy in children (Papapostolou et al., 2022). Moreover, infants with higher transepidermal water

loss in the first days of life are more likely to develop AD, suggesting that skin barrier defects precede clinical disease (Tham & Leung, 2019).

Microbial dysbiosis

Atopic dermatitis (AD) is closely linked to imbalances in both the skin and gut microbiome. On the skin, helpful bacteria decrease while *Staphylococcus aureus* increases, releasing substances that weaken the skin barrier and intensify inflammation and itching. Similar imbalance occurs in the gut, where beneficial short-chain fatty acid-producing bacteria decline and harmful microbes become more dominant, contributing to immune dysregulation through the gut-skin axis. Early-life factors also shape microbiome development and AD risk. Vaginal delivery, breastfeeding, and growing up in rural environments support a healthier and more diverse microbiome, while antibiotics can disrupt this process. Certain probiotic-derived metabolites further help regulate inflammation, highlighting the important role of the microbiome in the development and severity of AD (Hansen-Sackey & Hartono, 2025).

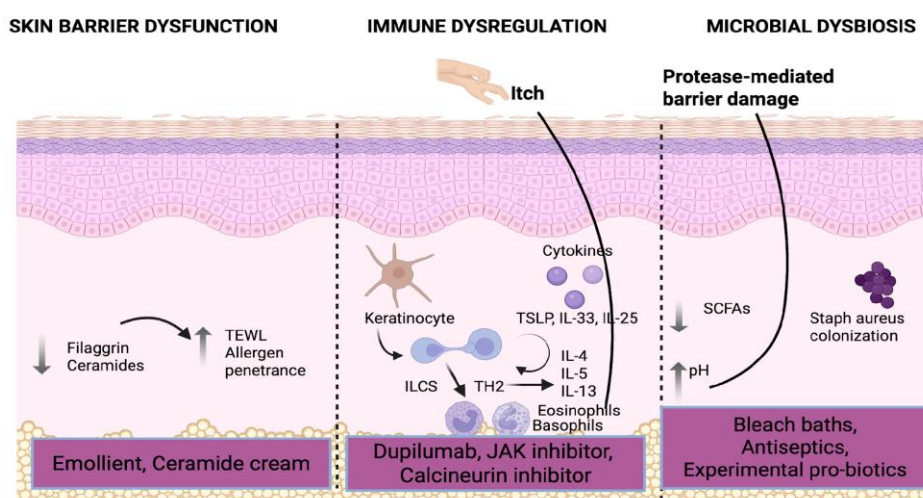


Figure 1. The pathophysiology of atopic dermatitis involves complex interactions between skin-barrier defects, immune dysregulation, and microbial imbalance, all of which contribute to chronic inflammation (Hansen-Sackey & Hartono, 2025).

DISCUSSION

Diagnosing food allergy (FA) in individuals with atopic dermatitis (AD) requires a systematic and clinically grounded approach due to the high rate of sensitization and the risk of overdiagnosis. A detailed clinical history is the essential first step, emphasizing the timing, reproducibility, and spectrum of symptoms associated with food ingestion (Sicherer & Sampson, 2014). In the context of AD, it is particularly important to differentiate immediate IgE mediated symptoms, such as urticaria, angioedema, wheezing, vomiting, or anaphylaxis, from delayed eczematous flares, which are frequently multifactorial and not necessarily driven by foods (Tsakok et al., 2016).

Diagnostic testing, including skin prick testing (SPT) and serum allergen-specific IgE (sIgE), plays an important role but should not be used as screening tools in the absence of a suggestive history (Dinardo et al., 2025; U.S. Department of Health and Human, 2011). Both SPT and sIgE exhibit high sensitivity but limited specificity, particularly in patients with moderate-to-severe AD who often present with polysensitization and elevated baseline IgE levels (Papapostolou et al., 2022). Therefore, sensitization alone, whether detected by SPT or sIgE, cannot confirm clinical allergy.

Component-resolved diagnostics (CRD) enhance diagnostic specificity by identifying IgE responses to defined allergenic proteins. Sensitization to more stable components (e.g., Ara h 2 in peanut, ovomucoid in egg) correlates more strongly with systemic allergic reactions, whereas sensitization to labile proteins may indicate milder or cross-reactive phenomena. Despite its advantages, CRD should be interpreted within clinical context and is best used for risk stratification (Calamelli et al., 2019). Despite its advantages, CRD should be interpreted within clinical context and is best used for risk stratification.

The role of atopy patch testing (APT) remains limited due to inconsistent reproducibility, heterogeneous protocols, and low predictive value. Current international guidelines do not recommend routine use of APT in diagnosing FA in AD patients (Eigenmann & Calza, 2000). Basophil activation testing (BAT), although highly specific and promising, is available only in specialized centers and is not yet standard practice (Rydzy et al., 2025).

The oral food challenge (OFC) continues to be the gold standard for definitive diagnosis. Double-blind, placebo-controlled food challenges offer the greatest accuracy, though open challenges are often adequate in clinical practice (de Weger et al., 2025). OFCs are particularly important when sensitization is detected without a clear history of reaction, or when dietary elimination may risk nutritional compromise, as commonly observed in infants and young children with AD (Foong et al., 2021). Overall, diagnostic evaluation should be guided by history, supported by targeted testing, and confirmed by OFC where appropriate. The overarching objective is to correctly identify clinically relevant FA while avoiding unnecessary dietary avoidance, which can negatively impact growth, feeding behavior, and quality of life.

Table 1.
Diagnostic tools for food allergy in patients with atopic dermatitis

Diagnostic method	Purpose	Strengths	Limitations	Clinical utility in AD
Clinical History	Evaluates symptom timing, type, reproducibility, and cofactors	Foundational step; guides testing	Subjective; caregiver recall bias; eczema flares often multifactorial	Essential for deciding whether testing is warranted
Skin Prick Test (SPT)	Detects IgE-mediated sensitization	High sensitivity; rapid; inexpensive	Low specificity, especially in AD; false positives common	Best used when history suggests immediate reactions
Serum Allergen-Specific IgE (sIgE)	Measures circulating IgE to specific foods	Standardized; unaffected by skin inflammation	Cannot confirm allergy; high IgE levels in AD reduce specificity	Useful adjunct to SPT in risk estimation
Component-Resolved Diagnostics (CRD)	Identifies IgE binding to specific allergen molecules	Higher specificity; predicts severity; distinguishes cross-reactivity	Requires expertise; higher cost; limited availability	Valuable for risk stratification (e.g., peanut, egg, tree nuts)
Atopy Patch Test (APT)	Evaluates delayed-type eczematous reactions	Designed for non-IgE pathways	Poor reproducibility; lack of standardization	Not recommended for routine diagnosis
Basophil Activation Test (BAT)	Detects functional allergic response at the cellular level	High specificity; may reduce OFC need	Specialized equipment; not widely available	Emerging option for complex cases
Oral Food Challenge (OFC)	Confirms or excludes clinical allergy	Gold standard; distinguishes sensitization from true allergy	Time-consuming; risk of anaphylaxis; requires expertise	Indispensable when history/testing are inconclusive or elimination poses risk

Management Strategies

Effective management of co-existing atopic dermatitis (AD) and food allergy (FA) requires a multi-layered, integrated approach that addresses both skin barrier integrity and immunologic

reactivity. Below, we outline current and emerging strategies grounded in clinical evidence and mechanistic insights.

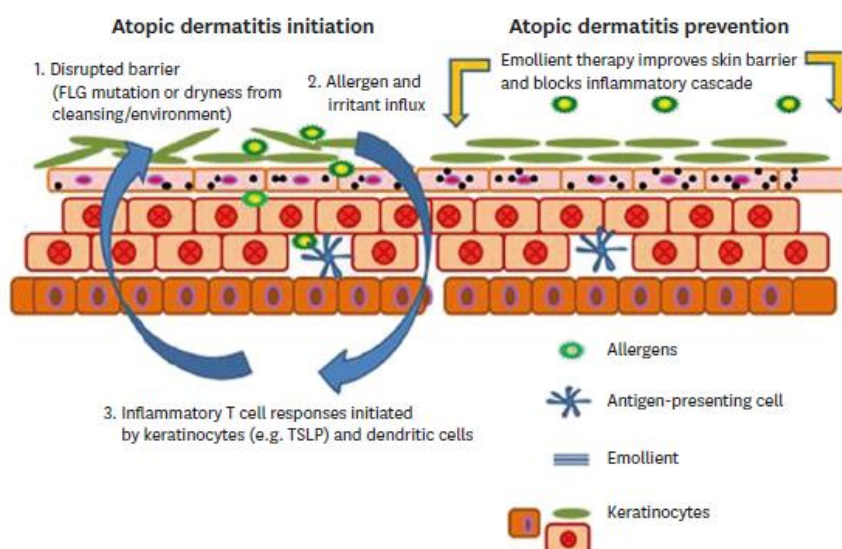


Figure 2. Prophylactic use of emollients to strengthen the skin barrier in early life has been proposed as a strategy to lower the risk of developing atopic dermatitis. This approach suggests that maintaining optimal skin hydration and barrier integrity from infancy may help prevent the onset of AD (Tham & Leung, 2019)

Skin barrier maintenance

Proactive emollient therapy

Regular application of emollients from early life aims to restore and maintain the skin barrier, thereby reducing transepidermal water loss (TEWL) and limiting epicutaneous allergen penetration (Sweeney et al., 2021)

Selection of emollient formulation

Not all moisturizers are created equal. Emerging data suggest that trilipid creams, designed to mimic the skin’s natural lipid composition (ceramides, cholesterol, free fatty acids in a 3:1:1 ratio), may be more effective for barrier repair than simpler formulations (Dębińska & Sozańska, 2023).

Challenges and heterogeneity in evidence

Clinical trials of emollient therapy for FA prevention have yielded mixed results. Meta-analyses have indicated modest protective effects, particularly when emollients are applied continuously, but the data are not uniform (Zhong et al., 2022). Factors contributing to heterogeneity include variation in the type of emollient (pH, lipid content), frequency, application area (whole-body vs. partial), and adherence (Braun et al., 2024).

Anti-inflammatory therapy

Topical corticosteroids and proactive treatment

In infants with AD, early and proactive anti-inflammatory therapy using topical corticosteroids helps not only control eczematous inflammation but may also reduce the risk of FA over time by limiting the skin’s pro-sensitization environment (Yamamoto-Hanada & Ohya, 2024). Proactive regimens (e.g., intermittent use even during quiescent phases) have been associated with reductions in IgE sensitization (Dębińska & Sozańska, 2023).

Anti-type 2 biologics and emerging agents

Though not yet standard for FA prevention, biologic therapies targeting type-2 inflammation (e.g., IL-4/IL-13 blockade) have theoretical and emerging practical relevance: by interrupting the “outside–inside–outside” vicious cycle, they may simultaneously improve barrier function and reduce sensitization risk (Beck et al., 2022).

Allergen exposure and dietary strategies

Early introduction of allergenic foods

Based on the dual-allergen-exposure hypothesis, timely dietary introduction of allergenic foods (e.g., peanut, egg) in infancy can promote oral tolerance and prevent FA, particularly in high-risk infants with or without AD (Sweeney et al., 2021).

Avoidance and elimination diets

In individuals with confirmed FA, strict avoidance of the offending food(s) remains the cornerstone of management (Cesare et al., 2025). However, in the context of AD, prolonged or unnecessary elimination should be avoided, as it may compromise nutritional status and even risk loss of tolerance (Papapostolou et al., 2022).

Preventive and risk-reduction strategies

Early, aggressive ad management

Timely and aggressive control of skin inflammation in early life (especially in infants) may prevent or reduce epicutaneous sensitization and lower the subsequent risk of FA (Tham et al., 2024).

Microbiome-targeted therapies

Novel approaches are investigating the role of probiotics, postbiotics, and other microbial therapies to modulate immune responses and restore barrier function (Ma et al., 2025). Such interventions hold promise, but further clinical trials are needed to establish efficacy and safety in the AD–FA context.

Patient and family education, monitoring, and follow-up

Comprehensive education

Education on proper skincare, correct application of emollients, avoidance of irritants, and recognition of allergic reactions is essential (Simpson et al., 2025).

Nutritional supervision

When elimination diets are necessary, collaboration with dietitians or nutritionists is crucial to ensure dietary adequacy and avoid deficiencies, especially in infants and children (guideline recommendations) (Kotchetkoff et al., 2024).

Reassessment and reintroduction

Periodic reassessment, including supervised food challenges, should be performed to evaluate for the development of tolerance and to avoid unnecessary long-term dietary restrictions (Gargano et al., 2021).

CONCLUSION

The relationship between atopic dermatitis (AD) and food allergy (FA) is driven by shared mechanisms involving skin-barrier dysfunction, type-2 immune activation, and microbial imbalance. A compromised epidermal barrier facilitates epicutaneous sensitization to food proteins, while systemic inflammation and gut–skin interactions may further promote FA development. Accurate diagnosis requires careful distinction between sensitization and true clinical allergy to prevent unnecessary dietary restrictions. Management must therefore integrate optimal AD control,

targeted evaluation for FA, and safe dietary practices. Preventive strategies, including early barrier protection, controlled allergen introduction, and microbiome-supportive interventions, show promise in reducing FA risk, particularly in high-risk infants. In Indonesia and other Asian settings, these approaches should be adapted to local dietary patterns, healthcare access, and cultural practices.

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