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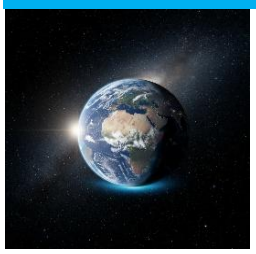

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The Impact of Nutrition and Early Dietary Introduction on the Prevalence, Severity and Management of Atopic Dermatitis in Infants. A Narrative Review

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Abstract

Background: Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease affecting up to 20% of children worldwide, characterized by epidermal barrier dysfunction and immune dysregulation. The increasing prevalence of AD has led to a discussion on efficacy of allergen avoidance versus early immune tolerance induction strategies.

Aim: This review aims to synthesize current evidence on prenatal and early-life nutritional interventions and their impact on the prevalence and severity of AD in infants.

Materials and Methods: A narrative review was conducted using a structured search of major electronic databases for studies published between January 2015 and March 2026. Eligible articles included original studies, systematic reviews, and meta-analyses addressing maternal diet, breastfeeding, complementary feeding, and micronutrient supplementation.

Results: Current evidence does not support maternal elimination diets as a preventive strategy but suggests that greater dietary diversity may reduce risk of AD. Role of breastfeeding in preventing AD remains inconclusive. Early introduction of allergenic products between 4 and 6 months promotes oral tolerance, although its impact on AD incidence is inconsistent. Vitamin D deficiency correlates with increased disease severity, and its supplementation may be beneficial. Findings regarding omega-3 fatty acids remain heterogeneous. Hydrolyzed formulas and elimination diets are not recommended for routine prevention of AD.

Conclusions: Nutritional strategies in AD are shifting toward proactive tolerance induction. Early dietary exposures play a key role in immune programming, although further research is needed to define optimal, individualized interventions.

Keywords: atopic dermatitis, nutrition, diet, infant

1. Introduction

Atopic Dermatitis (AD) is a chronic, recurrent inflammatory skin disease, typically manifesting with severe itching and eczematous lesions. It is characterized by skin barrier dysfunction and immune dysregulation, most commonly occurring in early childhood with the prevalence of up to 20% of children globally [1]. Historically, pediatric guidelines recommended delaying the introduction of allergenic foods to protect the immature immune system. However, the substantial rise in AD prevalence despite these restrictions has led researchers to the different hypotheses, highlighting the role of hygiene and the dual allergen exposure. The present model

suggests that allergic sensitization occurs through an impaired skin barrier, while oral consumption promotes immunological tolerance [2]. This review gathers and discuss the nutritional interventions that can modify the course and severity of AD based on the current literature, with strong emphasis on the ongoing shift toward proactive nutritional strategies.

Pathophysiology

Atopic dermatitis is a condition leading to the disruption of immune factors with several underlying causes including genetics, immunology, and environmental factors [1]. Most significant is genetically carried loss of function of filaggrin, a protein responsible for maintaining the integrity of skin barrier, that results in excessive penetration of pathogens, allergens and other irritants through epidermis. The immune response is mostly carried by Th2 cells, proinflammatory cytokines IL-4, IL-31 and IL-13 [3]. Impaired skin is also at a great risk of being colonized by pathological microbiome, which contributes to recurrent dermatitis and increased symptoms severity, including itching. Consequently, a self-perpetuating cycle of scratching and further skin damage develops. Additionally, hyperstimulation of immune system triggers other conditions commonly related to AD, such as asthma, food allergy, rhinitis [4].

Epidemiology

Atopic dermatitis is recognized as the most prevalent chronic inflammatory dermatosis in the pediatric population, affecting approximately 20% of children worldwide [1,3]. In Poland, epidemiological data indicate that the condition affects 3.9% of the population, with a notable prevalence of 5.3% in the 6–7 age group and 4.3% among adolescents. Research highlights a significant correlation between disease incidence and urbanization, higher socioeconomic status, and smaller family sizes [5]. Nearly 60% of cases are diagnosed within the first year of life and 85% before the age of five [6].

Clinical Manifestation

Atopic dermatitis is characterized by chronic course with periodical exacerbations and remissions. While typical symptoms are eczematous lesions and intense pruritus, the distribution differs according to the ages of the child. In infants it is expressed as acute eczematous lesions and papules, located on the face, cheeks, scalp, flexor surfaces of the limbs

and trunk, but excluding the nappy area. Other symptoms in this age group would be night-time waking and restlessness [1-3]. In older children the morphology shifts toward subacute eczematous plaques with prominent lichenification, primarily affecting the flexural folds, such as the antecubital and popliteal fossae, neck, wrists and back of the hands. In adolescents clinical image becomes similar to the adults, dominated by chronic lesions with lichenification, located on the flexural surfaces of the limbs, hands and face. The specific symptom across all age groups is intense, persistent pruritus, which leads to sleep disturbances in approximately 60% of cases. These symptoms not only cause physical discomfort but also significantly impair the psychosocial development and quality of life for both the child and their caregivers [1,6].

Atopic dermatitis in substantial proportion of infants can be the first manifestation of the atopic march, phenomenon that can be followed by development of other allergic conditions such as food allergies, asthma and rhinitis. Impaired skin barrier leads to non-physiological exposure to allergens which interferes with the development of immune tolerance and promotes sensitization [4]. Therefore, it is assumed that early treatment of atopic dermatitis and improving the skin barrier, may reduce the risk of developing other atopic diseases by limiting abnormal exposure to the allergens. These observations highlight the importance of optimizing the prevention and management of atopic dermatitis in the pediatric population to reduce the risk of sensitization and the occurrence of comorbidities in later life. Different but equally important to mention is the burden of chronic symptoms of atopic dermatitis, that can also contribute to a higher incidence of mental health disorders, including anxiety, depression, and attention-deficit/hyperactivity-disorder [1,4].

Diagnostics

Diagnosis of atopic dermatitis in children remains primarily clinical, as there are currently no definitive laboratory biomarkers for the disease. In clinical practice, the Hanifin and Rajka criteria continue to be the gold standard. The classification requires the fulfillment of at least three major criteria and three out of twenty-three minor criteria. Major criteria include pruritus, typical morphology and distribution, chronic or relapsing course, and personal or family history of atopy [2,7].

Treatment and Pediatric Guidelines

The therapeutic strategy for pediatric atopic dermatitis follows a multifaceted, stepped approach. The foundation of management across all severity levels is intensive emollient therapy, which

aims to restore the compromised epidermal barrier, reduce transepidermal water loss, and decrease the frequency of flares. According to Polish pediatric guidelines, emollients should be applied at least twice daily in generous amounts (between 200 and 500 g/week in older children; 250 g/week according to EuroGuiDerm guidelines) [8,9]. For active inflammatory lesions, topical corticosteroids remain the first-line pharmacological intervention. In the pediatric population, low-to-medium potency steroids are preferred to minimize the risk of systemic absorption and local side effects, such as skin atrophy. To maintain long-term remission, especially in sensitive areas like the face, neck, and intertriginous folds, topical calcineurin inhibitors, specifically pimecrolimus and tacrolimus are highly recommended due to their superior safety profile regarding skin thinning. A key element of modern Polish recommendations is the implementation of proactive therapy, which involves the application of topical calcineurin inhibitors or topical corticosteroids twice weekly to previously affected skin sites even after clinical clearance, significantly reducing the risk of relapse. In cases of moderate to severe atopic dermatitis that are refractory to topical treatments, the guidelines suggest advanced interventions, including Wet Wrap Therapy, which enhances the penetration of topical agents and provides a physical barrier against scratching. Systemic treatment options for children include cyclosporine A, methotrexate, or azathioprine, although these require rigorous monitoring for toxicity. Notably, the recent introduction of biological therapies, such as the IL-4/IL-13 receptor antagonist dupilumab, has transformed the management of severe pediatric atopic dermatitis, offering a targeted approach with a favorable safety profile for patients as young as 6 months [7-9]. Furthermore, patient education and the identification of individual triggers remain integral components of the holistic management plan.

2. Materials and methods

This narrative review was conducted through a structured search of major electronic databases, including PubMed, Scopus, and the Cochrane Library, for literature published between January 2015 and March 2026. The search strategy utilized combinations of keywords: "atopic dermatitis", "infant nutrition", "early allergen introduction", and "maternal diet”. Selection criteria included original research articles, meta-analyses, and systematic reviews published in English. A total of 37 key sources were selected for in-depth analysis. The analysis focused on the consistency of evidence regarding prenatal and postnatal nutritional interventions and their clinical impact on disease severity. Due to the character of this study ethical committee approval was not required.

3. Results

3.1. Maternal Nutrition During Pregnancy and Lactation

The concept of the “first 1,000 days,” encompassing the period from conception to a child’s second birthday, describes a critical developmental window during which environmental exposures, including nutrition, may shape immune system development and susceptibility to allergic diseases [10]. Maternal diet during pregnancy and lactation has therefore been increasingly investigated as a potential modifiable determinant of AD risk in offspring.

Earlier recommendations suggested that maternal avoidance of allergenic foods such as cow’s milk, eggs, or nuts during pregnancy or breastfeeding might reduce the risk of allergic disease in children. However, current evidence does not support this approach. Novel studies indicate that maternal elimination diets do not prevent AD and may adversely affect maternal nutritional status [11]. Moreover, the avoidance of allergenic foods may reduce the diversity of antigens transferred to the fetus or infant, potentially limiting opportunities for early immune tolerance induction [10].

In contrast, several cohort studies suggest that greater maternal dietary diversity may be associated with a lower risk of AD in offspring [12,13]. Diets characterized by higher intake of fruits, vegetables, and fermented dairy products appear particularly relevant. The Healthy Start cohort developed a maternal diet quality index and reported that higher consumption of vegetables and yogurt was associated with a reduced risk of AD in children (OR 0.77; 95% CI: 0.69–0.86) [12]. These findings support the hypothesis that maternal dietary patterns may influence fetal immune programming through the provision of micronutrients, bioactive compounds, and microbial components capable of modulating inflammatory pathways. Potential mechanisms include epigenetic modulation of immune-related genes during fetal development. Nutrients, such as folate and vitamin B12, as well as specific fatty acids, may influence DNA methylation patterns affecting genes associated with regulatory T-cell (Treg) differentiation and immune tolerance. Such epigenetic modifications may contribute to the regulation of Th2-skewed responses that characterize early allergic disease [10,14,15].

Among individual dietary components, omega-3 long-chain polyunsaturated fatty acids have been extensively studied. A systematic review by Garcia-Larsen et al. reported that maternal fish oil supplementation during pregnancy was associated with a reduced risk of eczema and egg allergy in offspring [16]. Data from the COPSAC2010 cohort demonstrated that the association between maternal fish oil supplementation and AD risk differed according to maternal polymorphisms in the cyclooxygenase-1 (COX-1) gene. Specifically, supplementation reduced AD risk in offspring of mothers carrying the TT genotype of the SNV rs1330344 variant (HR 0.70; 95% CI: 0.50–0.98), whereas no protective effect was observed in mothers with the CC genotype [17]. These findings highlight the potential relevance of gene–nutrient interactions and the emerging role of precision nutrition approaches in allergy prevention.

Further evidence suggests that maternal diet may interact with infant genetic susceptibility. Venter et al. indicates that maternal restrictive diets (e.g., avoiding nuts or dairy) during pregnancy do not prevent AD and may even be counterproductive by limiting the transfer of diverse antigens through breast milk [18].

Vitamin D supplementation during pregnancy has also been implicated in immune development. Data on low maternal serum concentrations of 25-hydroxyvitamin D and its association with an increased risk of AD in offspring remain inconsistent in current observational studies [3,10,15]. Evidence from the MAVIDOS randomized controlled trial demonstrated that supplementation with 1000 IU of cholecalciferol daily from mid-pregnancy reduced the incidence of eczema in infants at 12 months (OR 0.55; 95% CI: 0.32–0.97). Interestingly, the protective effect appeared more pronounced among infants who were breastfed for at least one month, suggesting potential interactions between maternal vitamin D status and immunomodulatory components of breast milk [19].

3.2. Breastfeeding and Presumptive Prevention

Breastfeeding is widely recognized as the gold standard for infant nutrition and is recommended by the World Health Organization and the American Academy of Pediatrics as the exclusive source of nutrition during the first six months of life. Human milk provides numerous immunomodulatory components, including secretory IgA, lactoferrin, cytokines, and human

milk oligosaccharides (HMOs), which contribute to immune maturation and the development of mucosal tolerance in early life [20].

Despite these biological properties, evidence supporting breastfeeding as an effective strategy for the primary prevention of AD remains inconsistent. A systematic review by Garcia et al. concluded that breastfeeding promotion as a strategy to reduce AD incidence is characterized by low certainty of evidence according to the GRADE framework [16]. Similarly, the meta-analysis by Lodge et al. reported no conclusive evidence that breastfeeding specifically protects against the development of atopic diseases across all populations, despite its well-established overall health benefits [21]. On the other hand, some cohort studies have suggested that prolonged exclusive breastfeeding beyond six months in the absence of early allergen exposure, may be associated with a higher risk of AD, possibly due to delayed induction of oral tolerance [22]. However, in infants with a family history of atopy, exclusive breastfeeding for at least four months has been associated with a reduction in AD incidence and severity [16,20]. Current guidelines therefore recommend continuing breastfeeding during the introduction of complementary foods, as the immunological components of breast milk may facilitate the development of tolerance to newly introduced dietary antigens [8]. Ongoing research focuses on how HMOs may influence the infant's skin barrier and immune maturation.

3.3. Complementary Feeding and Early Allergen Exposure

Over the past decade, recommendations regarding complementary feeding have shifted substantially from allergen avoidance toward early controlled exposure. Earlier guidelines suggested delaying the introduction of allergenic foods, however this approach coincided with a rising prevalence of food allergy and atopic diseases, prompting reconsideration of these recommendations [23].

A major turning point was the LEAP (Learning Early About Peanut Allergy) trial, which demonstrated that early peanut introduction between 4 and 11 months of age in infants at high risk for allergy reduced the prevalence of peanut allergy by more than 80% compared with strict avoidance. These findings provided strong evidence for the concept of a “window of tolerance,” a developmental period during which the infant’s gut-associated lymphoid tissue is highly receptive to the induction of oral immune tolerance [24].

Subsequent studies have explored whether similar strategies may influence the development of atopic diseases. The EAT (Enquiring About Tolerance) and the PreventADALL trials examined whether early exposure to foods such as egg, milk, wheat, fish, sesame, and peanut could reduce the risk of allergic diseases [25,26]. Although these studies did not consistently demonstrate a significant reduction in the incidence of AD in the general population, they provided evidence that early dietary exposure can promote immune tolerance and may be particularly beneficial in infants with increased atopic risk. Additional evidence suggests that dietary diversity during the first year of life may further support immune maturation. Roduit et al. reported that the introduction of a wider range of foods in infancy was inversely associated with the risk of AD and asthma, suggesting that early antigen exposure may facilitate tolerance development [27].

These observations are supported by the dual-allergen exposure hypothesis by Lack et al, which proposes that sensitization to food allergens may occur through impaired skin barriers in infants with eczema, whereas early oral exposure promotes immune tolerance. In this mechanism delayed dietary introduction may inadvertently favor sensitization through the skin [28,29]. Current consensus is that introducing complementary foods, including common allergens, between 4 and 6 months is crucial for inducing oral tolerance [30].

3.4. Role of Vitamin D and Omega Fatty Acids in AD

Vitamin D acts as a key regulator of both innate and adaptive immune responses. It is known to regulate genes involved in epidermal barrier integrity, including those related to filaggrin synthesis, and to stimulate the production of antimicrobial peptides such as cathelicidin (LL-37). Enhanced expression of these peptides may improve cutaneous defense against Staphylococcus aureus, a common pathogen associated with disease exacerbations [3,31].

Reduced serum concentrations of 25-hydroxyvitamin D are frequently observed in infants and children with AD and have been associated with increased disease severity and a higher susceptibility to skin infections [31]. Evidence from randomized controlled trials suggests that vitamin D supplementation may improve clinical outcomes in AD. A recent meta-analysis by Nielsen et al. reported a statistically significant reduction in disease severity following supplementation (standardized mean difference = -0.41; $p < 0.01$), particularly in individuals with baseline vitamin D deficiency. These effects appear more pronounced during periods of

reduced ultraviolet exposure, further supporting the role of vitamin D in immune and skin barrier regulation [3].

Omega-3 polyunsaturated fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have also been investigated for their potential anti-inflammatory effects in AD. Modern Western diets are typically characterized by a high omega-6 to omega-3 ratio, which may promote the synthesis of pro-inflammatory mediators such as leukotrienes B4. Supplementation with omega-3 fatty acids may shift this balance toward the production of specialized pro-resolving mediators, thereby contributing to the reduction of cutaneous inflammation [15,16,32].

Evidence from interventional studies suggests that omega-3 supplementation may provide clinically meaningful benefits in some patients with AD. In a randomized, triple-blind, placebo-controlled trial involving children with moderate-to-severe AD, daily supplementation with a preparation containing 2 g of fish oil (600 mg EPA and 400 mg DHA), together with gamma-linolenic acid (GLA) and vitamin D, resulted in significant clinical improvement after four months of treatment. The median SCORAD index decreased from 42 to 25 ($p < 0.001$) in the intervention group, whereas no significant change was observed in the placebo group. Importantly, supplementation was also associated with a marked reduction in the use of topical corticosteroids, which declined from 30 mg to 10 mg per month ($p < 0.001$), while corticosteroid use increased in the placebo group during the same period. In addition to objective disease severity, improvements were reported in itch intensity and sleep quality, indicating a broader impact on patient well-being [32].

3.5. Hydrolysates and Elimination Diets

The role of hydrolyzed infant formulas in the prevention of AD has been extensively debated. For many years, partially hydrolyzed (pHF) and extensively hydrolyzed formulas (eHF) were recommended for infants at high risk of allergic disease who could not be breastfed. This recommendation assumed that enzymatic hydrolysis of cow's milk proteins into smaller peptides would reduce allergenicity and prevent IgE-mediated sensitization [33].

However, more recent evidence has questioned this perspective. A large systematic review and meta-analysis published by Boyle et al. (2016) found no consistent evidence that hydrolyzed

formulas reduce the risk of allergic diseases, including AD, compared with standard cow's milk formulas [34]**.** These conclusions were supported by subsequent analyses and the 2018 Cochrane review, which similarly reported insufficient evidence to recommend hydrolyzed formulas for routine allergy prevention [35]. As a result, current international guidelines from organizations such as the EAACI no longer support the routine use of pHF or eHF solely for the prevention of atopic disease in infants without confirmed cow's milk protein allergy [30].

Interestingly, some studies suggest that specific hydrolyzed formulas may still confer benefit in selected populations. The Allergy Reduction Trial (A.R.T.) reported that a particular whey-based hydrolyzed formula reduced the incidence of AD at six months in infants at high atopic risk compared with standard formula feeding (RR 0.54, 95% CI 0.32–0.92). The favorable outcome was even more considerable in children with family history of AD (76% risk reduction; RR 0.24, 95% CI 0.07-0.78) [36]. These findings suggest that the preventive effects of hydrolyzed formulas may depend on the specific hydrolysis process and resulting peptide profile, although such benefits have not been consistently observed across products. Consequently, routine use of hydrolyzed formulas for AD prevention is not currently recommended [8].

Another important consideration is the increasing prevalence of unwarranted elimination diets in infants with AD. Although food allergies are present in approximately 30% of children with severe AD, the two conditions are not synonymous. Furthermore, elimination diets rarely result in a complete remission of skin lesions, due to the AD mechanism, which involves skin barrier defects regardless of dietary interventions. Empirical elimination of common allergens without clear clinical indication may lead to nutritional deficiencies, impaired growth, and psychosocial burden for both children and their caregivers [37]. Moreover, prolonged avoidance of previously tolerated foods may contribute to loss of oral tolerance, potentially increasing the risk of IgE-mediated allergic reactions upon re-exposure, including anaphylaxis. For this reason, current guidelines recommend that the initiation of an elimination diet should be based exclusively on a confirmed reaction, as determined by an oral food challenge, that results in the exacerbation of skin lesions or the manifestation of immediate symptoms [8,30,37].

4. Discussion

Recent findings in the literature are largely consistent with the EuroGuiDerm 2022 guidelines, while being progressively supported by emerging data on early-life nutrition and its role in immune modulation [8].

In consideration of maternal diet, there is a notable concordance between guidelines and current literature. EuroGuiDerm explicitly discourages maternal elimination diets, a position supported by recent cohort and mechanistic studies demonstrating that dietary diversity, rather than allergen avoidance, is associated with reduced risk of AD in offspring [8]. Moreover, emerging evidence on gene–nutrient interactions and epigenetic programming further strengthens this paradigm, suggesting a shift toward precision nutrition approaches in the future [3,17].

A similar ambiguity is observed in the role of breastfeeding in guidelines and recent studies. While the evidence suggests that it is beneficial for overall health, its protective effect against AD is inconsistent. Current evidence aligns with the position of EuroGuiDerm, indicating that breastfeeding should not be regarded as a standalone preventive strategy for AD [8]. Rather, it should be considered as part of a broader approach that includes both complementary feeding and immune exposure.

In the context of early allergen introduction, both EuroGuiDerm and recent landmark trials such as LEAP, EAT support the introduction of complementary foods, including allergenic products, between four and six months of age. The concept of a "window of tolerance" and the dual-allergen exposure hypothesis have been integrated into clinical practice and guidelines, effectively marking the conclusion of the historical "avoidance era" [8,24,25].

More subtle differences have been observed in micronutrient supplementation. While EuroGuiDerm guidelines recommend identifying and correcting vitamin D deficiency, without universal supplementation for AD management, recent meta-analyses and mechanistic studies suggest a potentially broader role for vitamin D as an adjunctive therapeutic agent, particularly in patients with moderate to severe or refractory AD [3,8,31]. A similar pattern is observed for omega-3 fatty acids, where clinical trials indicate possible benefits in reducing disease severity and treatment burden, although heterogeneity of results currently limits definitive guideline recommendations [15,16,32].

Finally, both the guidelines and recent literature concur in discouraging the routine use of hydrolyzed formulas and empirical elimination diets. Studies indicate that the preventive efficacy of hydrolyzed formulas is negligible in the absence of a confirmed cow’s milk protein allergy [30,37]. Concurrently, increasing recognition of the risks associated with unnecessary

dietary restrictions, including malnutrition, impaired growth, and loss of oral tolerance, has served to strengthen a more cautious and evidence-based approach to dietary interventions.

Overall, the synthesis of recent data with EuroGuiDerm 2022 guidelines suggests that the management of AD is progressing towards a more integrated and individualized model, combining established dermatological strategies with targeted nutritional interventions aimed at supporting immune tolerance and skin barrier function, two key elements in AD pathomechanism.

5. Conclusions

The management of Atopic Dermatitis in infants has shifted from reactive avoidance to proactive nutritional modulation. Early exposure to allergens and adequate intake of Vitamin D and Omega-3 appear to play an important role in modern prevention. While the EuroGuiDerm 2022 guidelines provide an excellent foundation, emerging research suggests that the role of micronutrient supplementation is likely undervalued and warrants further inclusion in future clinical protocols.

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