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Family history of food allergy, asthma, allergic rhinitis, and atopic dermatitis: genetic and environmental factors – a narrative review

Abstract

Background: Atopic diseases, including Asthma, Allergic Rhinitis, Atopic Dermatitis, and Food Allergy, are among the most common immune-mediated disorders in childhood. These conditions share complex pathogenic mechanisms that involve genetic susceptibility, IgE-mediated immune responses, epithelial barrier dysfunction, immune dysregulation, and environmental factors. The increasing prevalence of atopic diseases worldwide highlights the need better to understand the interactions between inherited risk and early-life exposures.

Objective: This narrative review summarizes current evidence on the contributions of family history, genetic, and environmental determinants to the development of Asthma, Allergic Rhinitis, Atopic Dermatitis, and Food Allergy, emphasizing their interrelationships within the Atopic March.

Methods: A comprehensive literature search was conducted in the PubMed/MEDLINE database, with no restrictions on publication date. Studies addressing family history, genetic predisposition, epigenetic regulation, microbiome composition, early-life dietary factors, and environmental exposures associated with atopic diseases were reviewed. Relevant original studies, systematic reviews, clinical guidelines, and consensus documents were considered.

Results: Evidence indicates that family history represents an important marker of susceptibility to atopic diseases, reflecting the contribution of genetic variants, epigenetic modifications, and shared environmental exposures. Early-life factors, including alterations in the microbiome, dietary patterns, mode of delivery, breastfeeding, allergen exposure, and lifestyle factors, may influence immune maturation and modify the risk of developing allergic disorders. The coexistence and sequential progression of atopic diseases support the concept of the Atopic March as a dynamic interaction between genetic predisposition and environmental influences.

Conclusion: Understanding the complex interplay among heredity, epigenetic mechanisms, and environmental factors is essential for identifying children at increased risk, improving early diagnosis, and developing personalized preventive and therapeutic strategies for atopic diseases.

Keywords: atopic diseases, asthma, allergic rhinitis, atopic dermatitis, food allergy, atopic march, family history, genetics, epigenetics, environmental factors

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Introduction

Atopic diseases, including Asthma, Allergic Rhinitis (AR), Atopic Dermatitis (AD), and Food Allergy (FA), represent a major global health burden, particularly during childhood. These disorders frequently begin early in life and share common immunological pathways involving IgE-mediated responses, epithelial barrier dysfunction, and immune dysregulation. A positive family history of allergic diseases is one of the strongest indicators of susceptibility, reflecting the combined influence of genetic inheritance, epigenetic regulation, and shared environmental exposures. However, the development and progression of atopic diseases cannot be explained solely by genetic predisposition, as early-life environmental factors, including diet, microbiome composition, lifestyle, and external exposures, may modulate immune maturation and disease expression. Understanding the complex interactions among hereditary background, environmental determinants, and the Atopic March is essential for improving risk assessment, early diagnosis, and preventive strategies. The objective of this narrative review is to summarize current evidence on the roles of family history, genetic mechanisms, and environmental factors in the development of Asthma, Allergic Rhinitis, Atopic Dermatitis, and Food Allergy, and to emphasize their relationships within the Atopic March.

Literature search strategy

A literature search was conducted in the PubMed/MEDLINE and Google Scholar databases, covering English-language publications without date restrictions, prioritizing recent studies and publications considered historically relevant to the understanding of the topic. Relevant studies were selected based on their scientific contributions to understanding the complex interactions among genetic susceptibility, epigenetic regulation, environmental exposures, and immune mechanisms underlying atopic diseases. Pertinent articles, including Original studies, systematic reviews, consensus documents, international guidelines, and observational studies that presented information related to the objective of this review, were included. The search strategy combined Medical Subject Headings (MeSH terms) and free-text keywords related to the main concepts of this review. The following terms and their combinations were used: ("Asthma"

OR "Allergic Rhinitis" OR "Atopic Dermatitis" OR "Food Allergy") AND ("family history" OR "familial aggregation" OR "genetic predisposition" OR "heredity") AND ("environmental factors" OR "microbiome" OR "epigenetics" OR "early-life exposure" OR "diet").

Overview of atopic diseases: epidemiology and clinical interrelationships

The term "allergy" refers to type 1 hypersensitivity reactions[1] and describes an adverse immune response initiated by the host upon exposure to a substance.[2] Although wide individual variations may be observed, allergic diseases tend to occur in the first decades of life and, thereby, reflect the maturation of the immune system. Overall, no clinical symptoms are demonstrable at birth, and although IgE production begins in the eleventh week of gestation, no food sensitization, as measured by cord blood serum IgE levels, is detectable. However, during the first months of life, the first IgE responses to food proteins may be observed, particularly those to the hen's egg and cow's milk. Even in entirely breastfed infants, high amounts of specific serum IgE antibodies can be detected in a hen's egg. It has been proposed that hen's egg protein exposure occurs through the mother's milk.[3]

Allergy epidemiology and atopic march

The World Health Organization (WHO) recognizes allergic diseases as a major global health concern. The global prevalence of allergic diseases is estimated at 30% to 40%, including 400 million individuals with AR, 300 million with Asthma, 200–250 million with FA, and 150 million with drug allergy.[4,5] AR and AD affect approximately 29.4% and 26.1% of adults worldwide, respectively. Recent estimates indicate that 8% of children and 11% of adults have FA.[6] Over the past two decades, the prevalence of childhood allergic diseases has also increased.[7] Collectively, allergic diseases impact millions globally and impose a significant burden on health systems and economies. Although Asthma, AR, AD, and FA are clinically distinct, evidence indicates that they are genetically related, with correlation estimates of 0.55 for Asthma and AD, 0.47 for Asthma and AR, and 0.62 for AD and AR.[8] Asthma, AR, AD, and FA typically manifest in early childhood as part of the Atopic March, a sequential progression of clinical signs and atopic diseases. This sequence generally begins with AD and FA, followed by the onset of Asthma and AR.[6,9] Indeed, FA prevalence in Western civilizations has risen in recent years and is viewed as the "second wave" of the allergic epidemic.[10]

Risk of allergic diseases

Exposures during conception, pregnancy, and early childhood are critical determinants of immune system development, with an increased risk of allergic sensitization and disease.[11] These exposures include maternal smoking during pregnancy, prematurity,[12] low birthweight,[11] maternal obesity,[13] cesarean delivery, low Apgar score,[14] lack of exclusive breastfeeding,[15] residence in industrialized cities, and parental atopy.[16] Definitely, children with healthier dietary patterns, like a higher consumption of fruits and vegetables, are less likely to develop symptoms of Asthma, AR, and AD.[17–19]

Genetics, environmental, and atopic diseases

The prevalence of atopy has increased markedly over the past four decades. Clinical disorders such as Asthma, AR, and AD frequently exhibit familial clustering, reflecting the interplay of significant genetic factors that are likely to have a greater impact on the pathogenesis of atopic diseases than environmental factors. Consequently, the rise in allergic diseases has led to a greater proportion of individuals with at least one first-degree relative affected by these conditions. Notably, the substantial increase in FA prevalence has occurred more recently, lagging behind the rise observed in other allergic diseases.[20–22]

Family history of atopy

Previously, in the 70s, 80s, and 90s, publications reported that a family history of atopy predisposes to allergic disorders[23] and that childhood Asthma affects more boys than girls.[24] Studies suggest that elevated cord serum IgE levels predict the development of atopy.[25,26] Additionally, maternal influence is generally considered more significant than paternal influence,[27,28] but the relative contributions of heredity and maternal environment remain unknown. Indeed, even back then, a search for reliable genetic markers of atopy had been inconclusive.[29]

The occurrences of atopic diseases and their associations with parental atopic diseases have varied between studies. At 10 and 13 years of age, the prevalence of AR was found to be twofold higher in the offspring of parents with allergic diseases than in those of parents without.[30] Evidence from epidemiological studies indicates that infants with an atopic family history are genetically predisposed to allergies, especially those born to actively atopic mothers.[2,31,32] Indeed, Allergies will develop in 30% to 50% of children with a single parental allergy, and 60% to 80% of children with biparental allergy.[2,33]

The four allergic diseases

Asthma, AR, AD, and FA represent a closely related group of conditions that have increased in prevalence over the past four decades. The global rise in these allergies has led to more frequent familial clustering of diagnoses. FA, in particular, has experienced a more recent surge, following earlier increases in other atopic diseases. Evidence indicates a genetic connection among these conditions, with strong associations observed between their occurrences. The recent rise in FA may further contribute to the increased prevalence of other allergic disorders.

Food allergy

Adverse food reactions can be classified into Immune-Mediated and Non-Immune-Mediated reactions. FA is an immune-mediated condition that encompasses a spectrum of adverse responses to specific food proteins. FA is broadly classified into those mediated by immunoglobulin E (IgE) ("atopy"), those without IgE involvement (non-IgE), and those with a mixed signature of IgE-dependent and independent pathway activation.[34]

Therefore, FA is a complex, multifactorial disease with both environmental and genetic risk factors thought to contribute to its pathogenesis. It elicits abnormal immunological reactions upon exposure to specific food proteins, resulting in adverse clinical reactions, most severely anaphylaxis, which can be life□threatening.[35] The onset of FA commonly occurs in infancy and early childhood, with the highest prevalence in this age group, reported to range from 8 to 17%.[36–40] The predictors of early development of allergic sensitization and clinical allergy have been identified. Epidemiologic studies have revealed an increasing prevalence of FA globally, particularly in countries with a Westernized diet.[41]

Food allergy epidemiology

Over the past decades, the pediatric prevalence of FA has been increasing worldwide and has arisen as a "Second Wave" of the allergy epidemic.[2,42–48] Really, the prevalence of pediatric FA is increasing worldwide and affects approximately 2.5% of the world's population (1% to 10%).[2,49–51] Moreover, it is a significant global public health

problem among children.[52–57] Although most food allergies arise in children, many childhood-onset food allergies resolve during school age.[58] Similarly, new-onset allergies in adulthood: with at least one food allergy.[59] Both accurate diagnosis of FA and evaluation of resolution are essential to prevent serious health issues.

Food allergy and genetics

Genetic factors contribute to the development of FA, however, interactions between risk alleles and environmental exposures are likely responsible for the rapid increase in FA prevalence. The genetic component of allergy development is well established, though risk levels vary. Children with one parent affected by allergic disease have twice the risk of developing FA, while those with two affected parents have a threefold increased risk.[60] Additionally, FA in an index child significantly predicts FA in siblings (Odds Ratio, 2.6).[61] The involvement of multiple genes underscores the complex, multifactorial nature of FA. Similar to other allergic diseases, the genetic architecture of FA appears to involve several common low-penetrance variants with variable expressivity, although the contribution of rare deleterious mutations remains unexplored. Nonetheless, genetic risk remains a significant contributor to the development of FA, however, it is unlikely to explain the recent increase in FA as observed in Asthma and AD. Thus, it remains unclear whether the same genetic polymorphisms associated with Asthma and eczema are present in patients with FA, or whether unique polymorphisms are present in FA.

Food allergy and environmental factors

FA has a hereditary component, however, such dramatic rises in FA rates, particularly in developed countries, are hypothesized to result from shifts in environmental exposures associated with a Westernized lifestyle (cesarean delivery, diet, access to antibiotics, urbanization, and industrialization).[62]

The onset of FA symptoms often occurs during infancy or toddlerhood, sometimes as early as the first few months of life. Numerous lifestyle factors, such as maternal diet during pregnancy and lactation, delivery mode, the timing of introduction to allergenic foods, farming lifestyle, animal exposure, family size, and the use of antibiotics, have all been associated with the risk of FA, highlighting pregnancy and the early postpartum period as potentially critical windows to modify FA risk. Socioeconomic variations directly affect immune development through pathogen exposure (hygiene hypothesis),[63,64] or indirectly through a leaky epithelial barrier in the skin, airways, and gut mucosa (barrier hypothesis),[65,66] and the development of allergic diseases. Accordingly, there is some evidence that heritability contributes to the increase in food allergies, but it is likely mainly due to environmental risk factors, given the short time frame. Some of the genes with evidence for association with FA have also been shown to be associated with other allergic diseases, such as AD, Asthma, and AR.

Family history and food allergy

Comparison of the prevalence of FA family studies among first-degree families of affected cases or between unaffected controls demonstrated that the disease may be familial. However, genes are not necessarily involved. Nongenetic factors, such as shared environmental factors, play a significant role in the onset of FA, including food-related health problems among lower-income children compared with higher-income groups. The most widely used indicators of FA are familial aggregation and genetic component.[61,67,68]

The link between the risk of FA in children and allergic diseases, as well as family allergic sensitization, has been extensively reported,[69–74] with estimates that the risk of FA/sensitization doubles if one parent has an allergic disease and is 3-fold higher if both parents have an allergic disease. Definitely, an association between a parental history of allergic diseases and the offspring's FA is more powerful if the mother has an allergic disease.[75]

In the Australian Health Nuts study, children who had two or more atopic family members were shown to have an increased risk of having FA with an odds ratio of 1.8.[75] The same findings were reported in a Japanese cohort, in which a history of atopy in both parents was associated with an odds ratio of 2.6.[76] Twin and family studies have shown that genetic composition may play a significant role in the development of FA.[35,61,77] In these studies, genetic differences account for about 15-35% of the observed individual differences in food-specific IgE.[61] Monozygotic twins showed higher concordance rates for sensitization to the peanut allergen than dizygotic twins, demonstrating the role of genetic influence: those with more similar genes (monozygotic twins) were more likely to share a similar phenotype.[77,78] A family history of allergy is a significant risk factor for the onset of FA in offspring. However, cohort studies detected significant associations,[75,76,79–82] and others detected no associations.[83–86]

In a population study of 1-year-olds, individuals with family members with a history of any allergic disease (high risk) had a higher prevalence of FA than those with no family history of allergic disease.[2,75] Presently, it is widely accepted that a family history of 'allergy' in general is at increased risk of FA. So, the increase in FA may be related to the rise in other allergies, as this allergy epidemic has increased the proportion of people with a family history of allergy.

The prevalence of food allergy in children below five years old appears to be higher in Western countries compared with Asian countries. However, Australian-born children of Asian parents have a higher prevalence of food allergy compared with both Asian children born in Asia and Australian-born Caucasian children, suggesting that the effect of genetic predisposition to food allergy may differ depending on early-life environmental exposures.[50,87]

The prevalence and manifestations of FA vary significantly worldwide, affecting individuals of all ages. The rising prevalence of FA has prompted investigations into its underlying mechanisms, which have identified complex interactions among genetic, dietary, and environmental factors, dietary diversity and the composition of the early-life microbiome are presumed to link these elements.[88–91]

FA is a complex disease influenced by genetic, epigenetic, and environmental factors. These interactions contribute to FA development. The main factors are dietary diversity and early-life microbiome composition.[89–91] The increase in FA may be partially attributable to the concurrent rise in other allergies, leading to a greater proportion of individuals with a family history of allergy. The observed increase in FA prevalence is likely driven primarily by environmental risk factors, although heritability also contributes, as evidenced by gene-environment interactions. Despite the short time frame, which suggests a predominant environmental influence, some evidence supports a heritable component. It remains uncertain whether the global rise in other allergic diseases has directly contributed to the increase in FA. Studies indicate that FA and other allergies are more prevalent among individuals with a first-degree relative with FA.

Asthma

Complex gene-environment interactions, with heterogeneity in clinical presentation, chronic airway inflammation, excessive mucus production, and increased airway sensitivity, characterize Asthma.

Certainly, Gene-environment interactions are described as the "modern environment" hypothesis, which suggests that urbanization, increased population density, industrialization, and air pollutants, along with genetic predispositions, are more likely to trigger respiratory conditions.[92,93] Asthma is a global health problem, as its prevalence among children in many countries continues to rise.[94] The World Health Organization describes it as the most prevalent non-communicable disease among children globally.[95] Asthma and FA frequently co-occur, sharing risk factors and underlying mechanisms. Really, children with FA are more likely to be diagnosed with Asthma.[96,97] FA, and the presence of specific IgE antibodies to at least one of the six most common allergenic foods, have been reported among asthma patients,[98,99] as they tend to have greater sensitization to food allergens than the general population.[96] In children with severe FA, a stronger correlation between symptomatic FA and Asthma is observed. With the rising prevalence of Asthma globally, the prevalence of FA and associated health risks among asthmatic children is also likely to rise.[96,100]

Allergic rhinitis

AR is a chronic inflammatory condition, mediated by immunoglobulin E (IgE), that affects the nasal mucosa. In childhood, the pathophysiology of AR is further shaped by the phenomenon of "Atopic March," in which upper airway inflammation frequently precedes or coexists with the development of Asthma and AD. Worldwide, AR impacts up to 40% of children, and up to 40% of patients with AR also present with Asthma, with symptom onset typically occurring as early as ages two to three.[101–103] A family history of Asthma, AR, AD, and FA, male sex, are important risk factors for AR.[104] AR impairs quality of life, disrupts sleep, and reduces overall productivity.[105] Patients frequently have other allergic conditions, such as allergic conjunctivitis. Thus, it is central to consider the family history of atopy, given the strong genetic predisposition to AR.[106–108]

Atopic dermatitis

AD is a chronic disorder whose pathogenesis involves T-cell-driven inflammation, epidermal dysfunction, unquestionably genetic predisposition, and environmental factors.[109] AD increases the risk of multiple comorbidities, including Asthma, AR, and FA. According to the World Health Organization (WHO), the incidence of AD in children is increasing annually.[95,110–112]

Common environmental factors that exacerbate AD symptoms include air pollutants, environmental and dietary allergens, seasonal changes, temperature fluctuations, and microbial infections.[113–115] Concerning AD specifically, common food allergens such as dairy, peanuts, eggs, and gluten have been associated with being common triggers for food-triggered AD.[116,117] Food-triggered AD can manifest in immediate or latent reactions. IgE-mediated immune responses often trigger AD symptoms that manifest immediately and result in the exacerbation of pruritus as well as flare-ups of erythematous lesions.[118] In latent reactions, exacerbation of AD symptoms is delayed for hours to days after consumption of the food trigger.[119] AD associated with RA and Asthma forms the "atopic triad". Markedly, over 85% of AD patients are infants and young children.[120]

Food allergy, asthma, allergic rhinitis and atopic dermatitis interaction

In summary, FA is strongly interconnected with other major atopic diseases and represents a model disease that demonstrates how genetic susceptibility interacts with environmental influences during early life. The increasing prevalence of FA reflects alterations in epithelial barrier function, microbiome development, dietary patterns, and immune regulation rather than genetic changes alone. Also, a meta-analysis demonstrated that FA is associated with an increased risk of Asthma and AD.[121] FA and Asthma have been shown to coexist and share similar risk factors and underlying pathologies.[96,97,122] Nonetheless, in their clinical management, the potential comorbidity with FA is often ignored. So, FA investigations should be part of routine Asthma management. Food Allergy occupies a central position within the Atopic March because it frequently appears early in life and may identify children with increased susceptibility to subsequent allergic diseases. Understanding these interactions is essential for prevention strategies and personalized management of children at risk of progressing through the Atopic March.

Perspectives in genetic approach

Recent advances in genomic technologies, particularly next-generation sequencing (NGS), whole-exome sequencing (WES), and whole-genome sequencing (WGS), have significantly enhanced the identification of rare pathogenic variants and common susceptibility loci associated with complex immune-mediated diseases. In contrast to monogenic disorders, allergic diseases result from the interplay of multiple genetic loci, epigenetic mechanisms, and environmental exposures. As a result, genomic approaches are increasingly important for identifying disease susceptibility genes, refining genotype–phenotype correlations, and supporting precision medicine. Despite these advances, the routine clinical application of these technologies in the management of allergic disease remains in development. The combined effects of multiple genetic variants, environmental factors, and epigenetic modifications shape multifactorial diseases. Accordingly, NGS-based methods are critical for detecting disease-associated variants, characterizing genetic heterogeneity, and improving genotype–phenotype correlations. Although these techniques are not yet standard for all patients with allergic diseases, they have substantially advanced the understanding of disease susceptibility, elucidated molecular pathways underlying immune dysregulation, and informed precision medicine strategies. Furthermore, recent developments in molecular genetics have facilitated the identification of genetic factors underlying complex diseases. NGS, including targeted gene panels, WES, and WGS, has enabled the detection of both rare pathogenic variants and common susceptibility loci implicated in multifactorial diseases. Future approaches will likely move toward a risk-based model, in which children are identified based on their likelihood of developing allergic diseases before clinical manifestations become established. This strategy may allow for earlier diagnosis, targeted surveillance, individualized preventive interventions, and improved long-term outcomes. [123-126]

Clinical implications

The clinical expression of atopy depends on interactions among inherited genetic susceptibility, epigenetic regulation, epithelial barrier integrity, microbiome development, and environmental exposures. Therefore, early identification of children at increased risk for allergic diseases represents an important opportunity for preventive strategies and personalized clinical management. From a practical perspective, pediatricians and specialists should recognize that:

1) Family history remains one of the most valuable clinical predictors of atopic risk.
2) The presence of one atopic disease increases the likelihood of developing other allergic conditions.
3) Early-life environmental exposures may modify disease expression.

4) Children with AD, FA, or a strong family history require careful longitudinal follow-up.

5) Prevention should focus on supporting immune tolerance rather than simply avoiding allergen exposure.

Conclusions

Asthma, Allergic Rhinitis, Atopic Dermatitis, and Food Allergy constitute an assembly of immune-mediated conditions with overlapping pathophysiological mechanisms. These diseases share genetic predisposition and IgE-mediated immune responses and are strongly influenced by environmental and epigenetic factors, often interconnected through the Atopic March. While each condition exhibits distinct clinical features, they commonly display familial clustering and a tendency to coexist. This interrelationship underscores the necessity for an integrated approach to patient management to identify comorbidities and mitigate early progression along the Atopic March. Understanding the association between family history of atopy—including Asthma, AR, AD, and FA—is essential for informing families about increased risk, identifying high-risk groups for targeted prevention, and providing indirect measures of genetic susceptibility for gene-environment interaction research. Atopic diseases are not isolated disorders but interconnected manifestations of a complex biological network in which hereditary susceptibility interacts with environmental influences. Family history provides an important clinical window into this susceptibility, while future precision-based approaches may enable early intervention to modify the trajectory of allergic disease.

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Author contributions

Study design (NCM, MAC, JASC), Bibliographic research, and acquisition of data (JASC, JTD, GNH, CDFJ), analysis and interpretation of data (NCM, JTD, MAC), drafting of the manuscript (NCM, MAC), critical revision of the manuscript (NCM, JASC). All authors have contributed significantly to this study.

Consent for publication

All authors approved the final manuscript and consented to publication.

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Conflicts of interest

The authors declare no conflicts of interest.

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