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Review

From Infancy to Adolescence: The Developmental Trajectory of Food Allergy and Its Relationship with Eosinophilic Esophagitis–Mechanisms, Epidemiology, and Emerging Therapies

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Abstract

Food allergy (FA) and eosinophilic esophagitis (EoE) represent two of the most rapidly increasing allergen-driven conditions in pediatric medicine. Both diseases share key immunological features, including Th2 polarization and epithelial barrier dysfunction. Over the past two decades, compelling epidemiological and mechanistic evidence has established EoE as a late-manifesting component of the allergic march—the well-recognized sequential progression of atopic disease in childhood, which typically begins with atopic dermatitis, followed by IgE-mediated food allergy, allergic rhinitis, and asthma. Children with IgE-mediated food allergy carry a substantially elevated risk of developing EoE, and shared genetic susceptibility loci—including CAPN14, TSLP, and filaggrin (FLG)—underscore common pathogenic pathways. We conducted a narrative review of the literature by systematically searching PubMed/MEDLINE, EMBASE, and the Cochrane Library using the terms "eosinophilic esophagitis," "food allergy," "atopic march," "IgE-mediated allergy," and "pediatric" in combination; articles published from 2000 to March 2026 were considered, with priority given to systematic reviews, meta-analyses, randomized controlled trials, and guideline documents. This narrative review comprehensively examines the epidemiology, pathomechanisms, clinical presentation, diagnostic approach, and therapeutic landscape for pediatric FA and EoE, with particular emphasis on their immunological intersections and the evolving evidence positioning EoE within atopic disease trajectories. We highlight approval of dupilumab for children as young as 1 year with EoE—representing a paradigm shift toward biologic therapy for atopic multimorbidity—and discuss the pipeline of emerging agents including cendakimab, lirentelimab, and anti-IL-5 strategies. Identification of shared pathogenic mechanisms offers promising avenues for unified prevention, early diagnosis, and precision therapeutic approaches for children with multiple atopic diseases.

Keywords: eosinophilic esophagitis; food allergy; atopic march; pediatric allergy; Th2 inflammation; esophageal barrier; dupilumab; allergic march; TSLP; CAPN14; elimination diet; biologics

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1. Introduction

The global prevalence of allergic diseases has reached epidemic proportions over recent decades, with particularly striking increases among pediatric populations in industrialized nations. FA affects an estimated 6–8% of children worldwide, while EoE—once considered exceptionally rare—now carries a global pooled prevalence of approximately 40 cases per 100,000 inhabitants, with higher rates documented in North America [1,2].

The classical paradigm of the allergic march—or atopic march—describes the stereotyped sequential development of atopic conditions beginning with atopic dermatitis (AD) in infancy, progressing to IgE-mediated food allergy, and subsequently manifesting as allergic rhinitis (AR) and asthma in older children [3,4]. While the linearity of this model has been refined by longitudinal cohort data revealing more heterogeneous trajectories, the concept remains clinically valid as a framework for understanding shared pathogenic mechanisms across atopic diseases [5,6]. Crucially, recent large-scale epidemiological studies have established EoE as a late-manifesting component of the allergic march, carrying the strongest association with pre-existing IgE-mediated FA of all atopic conditions [7,8].

The immunological links between FA and EoE are extensive and bidirectional. Both diseases feature Th2-skewed inflammation, impaired mucosal barrier function, and alarmin-mediated innate immune activation [9]. Shared genetic susceptibility, including variants in CAPN14, TSLP, and the epithelial barrier genes FLG and DSG1 predispose to both [10,11]. From a clinical standpoint, up to 90% of EoE patients carry at least one concurrent atopic diagnosis, and children with IgE-mediated FA exhibit a nine-fold elevated risk of subsequently developing EoE [7,12].

Despite this substantial overlap, FA and EoE remain distinct diseases with divergent mechanisms of allergen-specific immunity, distinct clinical phenotypes, and different therapeutic approaches. IgE-mediated FA is characterized by rapid-onset systemic reactions mediated by mast cell and basophil degranulation, whereas EoE is an eosinophil-driven chronic inflammatory disease of the esophageal mucosa largely independent of IgE-mediated mechanisms [13,14]. In IgE-mediated FA, initial allergen sensitization—most commonly via the disrupted skin barrier or the gastrointestinal mucosa—drives B-cell class switching to IgE under the influence of IL-4 and IL-13. Allergen-specific IgE binds to high-affinity FcεRI receptors on mast cells and basophils; upon re-exposure, cross-linking of receptor-bound IgE triggers rapid degranulation and release mediators such as histamine, prostaglandins, and leukotrienes, producing the immediate hypersensitivity reactions that define this disease.

The major etiological contributors to EoE extend beyond food allergens and genetic susceptibility to include sensitization to aeroallergens (particularly grass, tree, and weed pollens that exhibit seasonal modulation of esophageal eosinophilia), as well as early-life environmental factors such as antibiotic exposure, cesarean delivery, reduced microbial diversity, and formula feeding. Understanding both the shared and divergent features of these conditions has profound implications for clinical management, particularly given that therapies targeting shared Th2 pathways—most notably dupilumab—can now address multiple coexisting atopic conditions simultaneously.

This narrative review aims to provide a comprehensive, clinically oriented synthesis of the current evidence on childhood FA and EoE, focusing on their epidemiological trends, distinct pathomechanisms but also similarities, evolving diagnostic criteria, dietary and pharmacological management strategies, and the rapidly expanding biologic therapeutic pipeline. Special attention is given to the positioning of EoE within the allergic march and the clinical implications of treating these conditions within an integrated atopic disease framework.

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2. Methods

This narrative review was conducted according to a pre-specified search strategy to minimize selection bias. PubMed/MEDLINE, EMBASE, and the Cochrane Library were searched from January 2000 to March 2026 using the following MeSH terms and free-text keywords: "eosinophilic esophagitis," "food allergy," "IgE-mediated allergy," "atopic march," "allergic march," "Th2 inflammation," "dupilumab," "biologic therapy," "oral immunotherapy," "elimination diet," and "pediatric allergy," applied individually and in Boolean combination. Reference lists of included articles were hand-searched for additional relevant publications. Inclusion criteria were: (1) original research articles, systematic reviews, meta-analyses, or clinical practice guidelines; (2) human studies; (3) focus on pediatric or mixed pediatric/adult populations with FA, EoE, or both; (4) publication in a peer-reviewed journal in English, German, or with available English abstract. Article selection and data extraction were performed independently by two authors with discrepancies resolved by senior author (JdL.). No formal quality assessment tool was applied given the narrative format of the synthesis; however, level of evidence was considered when weighting individual findings.

2.1. Epidemiology

2.1.1. Food Allergy in Childhood

IgE-mediated FA is now recognized as one of the most common chronic conditions of childhood. Point prevalence estimates range from 3% to 8% in Westernized countries, with the highest rates reported in infants and preschool-age children [15,16]. The United States prevalence is estimated at approximately 8% of children, representing a significant increase from the approximately 3.5% documented in the 1990s [17]. Epidemiological data from Europe, Australia, and East Asia reflect similar upward trends, though with regional variation in the distribution of various allergens [18].

The most common food allergens responsible for pediatric FA include cow's milk, hen's egg, peanut, wheat, soy, tree nuts, fish, and shellfish—collectively the 'top eight' allergens mandated for labeling in many regulatory frameworks. Cow's milk allergy affects 2–3% of infants, typically presenting within the first year of life, and is frequently self-resolving by early childhood; peanut allergy, conversely, persists in approximately 80% of affected individuals [19,20]. The marked increase in peanut allergy prevalence—estimated to have tripled over a 15-year period in the United States—has been a particular focus of epidemiological investigation, with converging evidence implicating delayed oral introduction and cutaneous sensitization as key contributing mechanisms [21].

Atopic dermatitis serves as the most significant risk factor for developing IgE-mediated FA in infancy: approximately one-third of children with moderate-to-severe AD will develop clinically significant food allergies, predominantly to egg, milk, and peanut [22]. The dual-allergen exposure hypothesis, as described by Lack et al., proposes that epicutaneous sensitization through disrupted skin barrier promotes allergic sensitization, while early oral exposure promotes tolerance, and this framework has fundamentally reshaped primary prevention strategies [21].

2.1.2. Eosinophilic Esophagitis

EoE was first recognized as a distinct clinical and pathological entity in the early 1990s, and its epidemiology has undergone remarkable transformation since systematic population-based studies began. A landmark 2023 systematic review and meta-analysis by Hahn et al., incorporating 40 studies and over 288 million participants, reported a global pooled incidence of 5.31 cases per 100,000 inhabitant-years (95% CI, 3.98–6.63) and a global pooled prevalence of 40.04 cases per 100,000 inhabitant-years (95% CI, 31.10–48.98) [1].

Strikingly, the pooled prevalence increased nearly nine-fold from the period 1976–2001 (8.18 cases per 100,000) to 2017–2022 (74.42 cases per 100,000), an increase that substantially exceeds what can be attributed to heightened awareness or increased endoscopy utilization alone [1].

In the pediatric population specifically (as presented in Table 1), incidence rates of 5.1 cases per 100,000 person-years and prevalence rates of 19.1–34 cases per 100,000 have been documented in North American and European cohorts [23,24]. The most recent meta-analysis data suggest ongoing increases, with pooled global incidence rising to 8.43 cases per 100,000 person-years in studies conducted post-2018 [25]. EoE is consistently more prevalent in high-income countries, among males (relative risk approximately 3.4-fold compared to females), and in individuals of White/Caucasian ethnicity, although recent large-scale EHR-based analyses from the Children's Hospital of Philadelphia have demonstrated that EoE is more prevalent among non-White children than previously recognized [1,26].

Table 1. Estimated prevalence of IgE-mediated food allergy and eosinophilic esophagitis in children and adults across major Western countries/regions [27–39].

Country/Region	FA Prevalence Children (%)	FA Prevalence Adults (%)	EoE Prevalence (per 100,000)	Notes
United States	~8	~4–5	~57–74 (adults); 19–34 (children)	Highest EoE rates globally; peanut predominant FA trigger
Australia	~9–10	~3–4	~45–55 (mixed age)	Highest infant FA rates;
United Kingdom	~5–8	~2–4	~25–40 (mixed age)	Increasing EoE recognition; LEAP trial origin
Germany/DACH	~4–6	~2–3	~20–35 (mixed age)	Milk and wheat predominant EoE triggers;
Canada	~7	~3–4	~40–50 (mixed age)	Similar profile to US;

FA: food allergy; EoE: eosinophilic esophagitis; DACH: Germany, Austria, Switzerland. Prevalence figures are approximate point estimates derived from population-based studies and meta-analyses; ranges reflect methodological variation across studies. EoE prevalence shown as cases per 100,000 inhabitants. All data should be interpreted in the context of differing diagnostic criteria and ascertainment methods across countries (see Section 9, Limitations. FA prevalence figures vary substantially according to ascertainment method (self-report > food-specific IgE > skin prick test > oral food challenge); where direct national meta-analyses are unavailable, regional pooled estimates are shown.

The temporal onset of EoE within the allergic march was precisely characterized in a virtual birth cohort study of over 218,000 children using CER2 Consortium data, demonstrating that EoE typically manifests at approximately 35 months of age—after AD (~4 months), IgE-mediated FA (~13 months), and AR (~26 months)—thus constituting the temporal culmination of the pediatric allergic march [7]. This finding has important clinical implications, suggesting that early atopic disease should prompt heightened vigilance for EoE development.

2.1.3. Comorbidity and Atopic Disease Trajectories

The co-occurrence of FA and EoE is well-documented (summarized in Table 2). Studies utilizing large subspecialty care networks have found that patients with IgE-mediated food allergy carry a substantially elevated risk of EoE compared to the general pediatric population [12,14]. Conversely, analyses of EoE registries consistently demonstrate that 40–60% of EoE patients have coexisting food allergy, 30–50% have asthma, and 40–60% have allergic rhinitis [40]. Mohammad et al. reported that among 449 EoE patients, the prevalence rates of AR, asthma, and AD were 61.9%, 39%, and 46.1%, respectively, with over 21% of patients carrying all three atopic diagnoses [41].

Table 2. Summary of atopic march conditions, their typical onset, key pathomechanisms, treatment approaches, and position within atopic disease trajectories.

Condition	Typical Onset	Key Pathomechanisms	Primary Treatment	Part of Atopic Disease Trajectories
Atopic Dermatitis	3–6 months	Epicutaneous sensitization, skin barrier dysfunction (FLG mutations)	Emollients, topical steroids, dupilumab	Yes—precedes other atopic conditions
IgE-Mediated Food Allergy	12–24 months	Th2 skewing, IgE class switching (IL-4/IL-13), gut sensitization	Avoidance, epinephrine, OIT	Yes—links to asthma, AR, EoE
Allergic Rhinitis	24–48 months	Aeroallergen sensitization, mast cell/basophil activation	Antihistamines, INCS, AIT	Yes—bidirectional with asthma
Asthma	24–48 months	Airway remodeling, ILC2 activation, aeroallergen/viral triggers	ICS, LABAs, biologics	Yes—closely linked to AD and AR
Eosinophilic Esophagitis	36–60 months	Esophageal barrier defect, Th2/eosinophilic inflammation, CAPN14, TSLP	Dietary elimination, topical steroids, PPI, dupilumab	Yes—late "atopic march" member

AD: atopic dermatitis; AR: allergic rhinitis; OIT: oral immunotherapy; ICS: inhaled corticosteroids; LABA: long-acting beta-agonist; AIT: allergen immunotherapy; INCS: intranasal corticosteroids; PPI: proton pump inhibitor; Onset ages approximate from large cohort data.

The bidirectional nature of the FA-EoE relationship is supported by evidence that: (1) children with IgE-mediated FA exhibit a nine-fold increased risk of developing EoE; (2) atopic dermatitis, IgE-FA, and asthma each independently and cumulatively increase EoE incidence; and (3) the foods most commonly triggering EoE (milk, wheat, egg, soy) substantially overlap with major IgE-mediated food allergens [7,12]. Furthermore, personal history of AD, IgE-FA, and asthma appears to have a cumulative dose–response relationship with EoE risk, with patients carrying all three preceding diagnoses facing the highest probability of subsequent EoE development [7].

3. Shared and Distinct Pathomechanisms

3.1. Epithelial Barrier Dysfunction

A unifying conceptual framework for both FA and EoE is the central role of epithelial barrier dysfunction in driving allergen sensitization and subsequent type 2 inflammation. In atopic dermatitis—the archetypal 'gateway' condition of the allergic disease trajectories—loss-of-function mutations in filaggrin (FLG) impair the stratum corneum and facilitate epicutaneous allergen uptake, triggering Th2-skewed sensitization [42]. The 'outside-in' hypothesis positions impaired skin barrier as the initiating event that promotes sensitization to ingested allergens through the skin rather than the gut, ultimately undermining oral tolerance mechanisms [21].

In EoE, analogous barrier disruption occurs at the esophageal epithelium. The esophageal mucosa of EoE patients demonstrates significantly reduced expression of cell-to-cell adhesion proteins including desmoglein-1 (DSG1), claudin-1, and filaggrin variants, leading to increased epithelial permeability and abnormal antigen exposure [43,44]. The gene CAPN14—encoding calpain 14, a protease involved in esophageal epithelial cell differentiation—has emerged as a replicated EoE susceptibility locus in genome-wide association studies, with its expression regulated by IL-13 in a feedback mechanism that

amplifies esophageal barrier disruption [10,45]. Serine protease inhibitors SPINK5 and SPINK7 represent additional EoE susceptibility genes involved in barrier maintenance [10].

3.2. Th2 Inflammation and Alarmin Signaling

Both FA and EoE are driven fundamentally by type 2 helper T cell (Th2) polarization, characterized by the overproduction of cytokines IL-4, IL-5, IL-9, and IL-13 [9,46].

Barrier disruption—whether cutaneous or esophageal—triggers the release of epithelial-derived alarmins, principally thymic stromal lymphopoietin (TSLP), IL-33, and IL-25. These innate cytokines activate immature dendritic cells (DCs), mast cells, basophils, and group 2 innate lymphoid cells (ILC2s), collectively orchestrating the downstream Th2 inflammatory cascade [47].

TSLP merits particular attention given its central mechanistic role in both conditions. In EoE, esophageal TSLP expression is markedly elevated and connects innate epithelial damage to adaptive Th2-type immune responses [48]. TSLP promotes the differentiation of allergen-specific Th2 cells, drives IgE class switching in B cells via IL-4, and enhances eosinophil survival through IL-5 signaling. The potential of anti-TSLP biologics (tezepelumab) to interrupt this pathway upstream in EoE is being actively investigated [49]. In IgE-mediated FA, TSLP drives allergic sensitization through analogous mechanisms, and TSLP gene polymorphisms are associated with elevated serum IgE and food sensitization [50].

The specific effector mechanisms diverge downstream. In IgE-mediated FA, cross-linking of food allergen-specific IgE on mast cells and basophils triggers rapid degranulation, releasing histamine, prostaglandins, and leukotrienes, producing the immediate-phase response that underlies anaphylaxis [51]. IgE production itself requires not only IL-4 but also IL-13 produced by T follicular helper 13 (Tfh13) cells promoting anaphylaxis-inducing IgE [1]. EoE, in contrast, is predominantly a non-IgE-mediated disease: esophageal eosinophilia occurs largely independently of B cells and circulating IgE [13]. Instead, IL-5—the principal eosinophil growth factor—drives eosinophil recruitment, survival, and activation in the esophageal lamina propria, while IL-13 suppresses esophageal barrier gene expression (FLG, DSG1) and promotes fibrotic remodeling via TGF-β and periostin (POSTN) pathways [45,52].

3.3. The Esophageal Microenvironment and Fibrostenotic Progression

A distinctive feature of EoE that differentiates it mechanistically from IgE-mediated FA is the chronic inflammation leading into fibrostenotic remodeling of the esophageal wall that characterizes long-term untreated or undertreated disease. Activated eosinophils release major basic protein (MBP), eosinophil peroxidase (EPO), and eosinophil-derived neurotoxin (EDN), causing direct epithelial cytotoxicity [53]. Simultaneously, TGF-β1—produced by eosinophils, mast cells, and Th2 cells—drives subepithelial fibrosis through activation of esophageal fibroblasts and smooth muscle cells, ultimately producing the characteristic esophageal rings (trachealization), furrows, strictures, and small-caliber esophagus observed endoscopically [54].

Natural history studies demonstrate a compelling relationship between diagnostic delay and fibrostenotic complications. A retrospective Swiss cohort found that only 17% of patients with fewer than 2 years of symptoms at diagnosis had esophageal strictures, compared with 71% in those with more than 20 years of symptoms—with the odds of fibrostenotic disease doubling for each additional decade of untreated inflammation [55]. These data underscore the urgency of early diagnosis and effective treatment in pediatric EoE, particularly given the high prevalence of diagnostic delay averaging 2–5 years in many cohorts.

3.4. Genetic Architecture and Shared Susceptibility

Genome-wide association studies (GWAS) and candidate gene analyses have delineated overlapping genetic susceptibility for FA and EoE. One EoE susceptibility locus resides within the CAPN14 gene, which is expressed predominantly in the esophageal epithelium and is upregulated by IL-13 in a way that perpetuates barrier disruption [10]. Additional EoE susceptibility loci identified through GWAS meta-analyses include TSLP/WDR36 (chromosome 5q22.1), CAPN14 (2p23), LRCH4/LRCH3, and variants within the eotaxin-3/CCL26 locus on chromosome 7q11 [56].

FLG variants confer risk for both AD and IgE-mediated FA through their effects on cutaneous barrier integrity. Although FLG variants have a less dominant role in EoE, filaggrin-related esophageal barrier dysfunction has been documented in EoE histology [57]. The HLA-DQ region appears to confer risk for EoE in a manner that parallels celiac disease, potentially reflecting shared mechanisms of antigen presentation for dietary proteins. Importantly, the genetic architecture of EoE shows partial but incomplete overlap with FA and AD, consistent with EoE being a related but distinct disease entity positioned at the intersection of food-driven and chronic eosinophilic inflammation.

3.5. Environmental Drivers and the Hygiene Hypothesis

The dramatic increase in EoE and FA prevalence in high-income industrialized nations implicates environmental factors operating on a background of genetic susceptibility. Proposed environmental contributors include: (1) early-life dysbiosis secondary to antibiotic use, cesarean delivery, and reduced microbial diversity; (2) the hygiene hypothesis, wherein decreased childhood microbial burden impairs regulatory immune development; (3) declining Helicobacter pylori prevalence, which may previously have conferred mucosal protection against esophageal eosinophilia; (4) increasing gastroesophageal reflux disease (GERD) disrupting esophageal barrier integrity; (5) dietary changes including increased consumption of ultra-processed foods (UPF) with some mechanisms discussed, like advanced glycation end products (AGEs), that directly damage mucosal barrier integrity [9,58,59].

Epidemiologically, the timing of UPF introduction into diets of children in Western countries parallels the emergence and rise in EoE. Cesarean delivery, associated with altered neonatal gut colonization, has been reported at higher rates in pediatric EoE patients with early-onset disease, supporting a role for microbial programming in EoE susceptibility [60].

4. Clinical Presentation and Natural History

4.1. Pediatric Food Allergy Phenotypes

IgE-mediated FA in children encompasses a spectrum of immediate hypersensitivity reactions, ranging from mild urticaria and angioedema to life-threatening anaphylaxis. The clinical phenotype depends upon the age of the child, the specific allergen, and the route and dose of exposure. In infants, cow's milk allergy typically presents with cutaneous reactions, vomiting, and feeding difficulties; in older children, anaphylaxis to peanut, tree nut, or sesame is the primary concern [61]. Anaphylaxis in childhood carries a case fatality rate of approximately 0.5–1%, and risk is heightened by coexisting asthma, delayed epinephrine administration, and unrecognized or underprescribed epinephrine auto-injectors [62].

Non-IgE-mediated and mixed FA phenotypes—including food protein-induced allergic proctocolitis (FPIAP), food protein-induced enterocolitis syndrome (FPIES), and food protein-induced enteropathy—present predominantly in infants and young children with gastrointestinal symptoms including bloody stools, profuse vomiting, and diarrhea. These cell-mediated conditions have distinct natural histories and generally resolve by 2–5 years of age [63]. The co-existence of IgE-mediated and non-IgE-mediated food sensi-

tivities within the same child is not uncommon and can complicate diagnostic workup and dietary management.

4.2. EoE Across Pediatric Age Groups

EoE presents with markedly different clinical phenotypes across pediatric age groups, reflecting evolving esophageal anatomy, physiology, and the child's ability to verbalize symptoms. In infants and toddlers (0–3 years), EoE most commonly manifests as feeding difficulties, food refusal, failure to thrive, and symptoms resembling gastroesophageal reflux disease (GERD) [64]. Young children (4–9 years) typically present with vomiting, abdominal pain, and regurgitation. Adolescents more closely resemble the adult phenotype, with dysphagia, food impaction, and the chest-pain-with-eating syndrome that characterizes adult disease [65]. In adults, the clinical presentation is dominated by solid-food dysphagia and episodic food impaction requiring emergency endoscopy, with heartburn and chest pain as frequent accompanying symptoms; the inflammatory endoscopic features (edema, furrows, exudates) are often less prominent than the fibrostenotic sequelae (rings, strictures, small-caliber esophagus) reflecting years of undertreated disease.

By contrast, pediatric patients rarely present with fixed strictures at diagnosis; the predominant endoscopic findings in children are mucosal edema and linear furrows, and symptom burden is typically less specific and more age-dependent than in adults. tools developed for adult practice (e.g., patient-reported dysphagia scores) require pediatric adaptation, and symptom-based screening in young children must rely on proxy (caregiver) reporting given limited self-report capacity.

A prospective analysis of over 200,000 pediatric electronic health records from the CER2 Consortium found EoE to be a chronic, progressive condition in childhood, with symptom burden and esophageal remodeling accumulating over time [7]. Importantly, EoE is recognized as a fibrostenotic, progressive disease at the phenotypic level: inflammatory EoE in childhood may progress to mixed-inflammatory/fibrostenotic phenotypes in adolescence and adulthood if left undertreated [54]. This long-term natural history underscores the clinical imperative of early intervention.

A critical contemporary insight is the recognition that EoE encompasses multiple molecular endotypes that may have clinical relevance for treatment selection. The EoE tissue transcriptome has been characterized through cross-sectional and longitudinal studies, identifying endotypes with distinct inflammatory gene expression profiles (EoE1–EoE4 endotypes), some associated with preferential response to dietary elimination versus corticosteroids versus biologics [66]. Although endotype-guided treatment selection remains aspirational rather than established practice, this molecular stratification framework represents an important advance toward precision medicine in EoE.

5. Diagnostic Approaches

5.1. Diagnosis of Food Allergy

The diagnosis of IgE-mediated FA requires clinical history consistent with allergen-triggered reactions, supportive objective testing, and—in ambiguous cases—oral food challenge (OFC), which remains the gold standard [67]. Skin prick testing (SPT) and measurement of serum allergen-specific IgE (sIgE) provide useful supporting data but carry significant limitations in positive predictive value, particularly in the context of sensitization without clinical reactivity. Component-resolved diagnostics (CRD)—utilizing recombinant allergen components such as Ara h 2 for peanut allergy—substantially improve diagnostic accuracy by identifying sensitization to major cross-reactive versus stable allergens and are increasingly integrated into clinical practice [68].

The basophil activation test (BAT) has emerged as a diagnostic tool, particularly for difficult-to-challenge patients and when assessing risk of severe reactions. BAT assesses CD63 and CD203c expression on basophils following allergen stimulation, with sensitivity and specificity often superior to SPT and sIgE for specific allergens including peanut and cow's milk [69]. However, due to lack of standardization, comparability and methodological inconsistencies in between laboratories, BAT is currently not recommended as a routine test. The diagnostic challenge for EoE screening in the context of food allergy is discussed below.

5.2. Diagnosis of EoE

EoE diagnosis requires the integration of clinical presentation, endoscopic findings, and histopathological confirmation. Current consensus diagnostic criteria—updated in the 2018 AGREE conference guidelines and the 2024 ESPGHAN/NASPGHAN pediatric guidelines—mandate: (1) symptoms of esophageal dysfunction; (2) esophageal biopsies demonstrating a peak eosinophil count of $\geq$15 eosinophils per high-power field (eos/hpf) in one or more specimens; and (3) exclusion of alternative causes of esophageal eosinophilia [2,70].

Characteristic endoscopic features of EoE include mucosal edema with loss of vascular pattern, linear furrows, white exudates/plaques (eosinophil microabscesses), concentric rings (trachealization/fixed rings), and strictures. The EoE Endoscopic Reference Score (EREFS) quantifies these features and is now standard in clinical trials and recommended in clinical practice [71]. Given the patchy nature of EoE, biopsies from at least two esophageal levels (proximal and distal) are mandatory; obtaining $\geq$4–6 biopsy specimens dramatically increases diagnostic yield [2].

The role of allergy testing in EoE diagnosis warrants particular emphasis. The 2024 ESPGHAN guidelines carry a strong recommendation against using available allergy tests (SPT, sIgE, atopy patch testing) to predict dietary triggers of EoE, given their poor positive predictive value for identifying EoE-triggering foods [2]. However, IgE-based allergy testing may retain utility in identifying foods that could trigger acute allergic reactions—particularly IgE-mediated anaphylaxis—during food reintroduction protocols following elimination diets. This nuanced position reflects the mechanistic dissociation between IgE-mediated sensitization and EoE pathogenesis.

Non-endoscopic diagnostic tools are under development to reduce the burden of repeated endoscopies for disease monitoring. The Esophageal String Test (EST), cytosponge and blood biomarkers including eotaxin-3, ECP [72] and periostin are under evaluation as non-invasive or minimally invasive monitoring tools, but have not reached clinical applicability [73,74].

6. Management

6.1. Food Allergy Management and Oral Immunotherapy

Standard management of IgE-mediated FA has traditionally centered on strict allergen avoidance, patient/caregiver education, and provision of epinephrine auto-injectors for anaphylaxis rescue. While this approach mitigates acute risk, it does not alter the natural history of allergy and is associated with reduced quality of life, social restrictions, food anxiety, and nutritional compromise [75]. This reality has driven the development and adoption of allergen immunotherapy strategies aimed at inducing desensitization or sustained unresponsiveness.

Oral immunotherapy (OIT) is currently the most extensively studied and clinically implemented immunotherapy approach, with peanut OIT (Palforzia) available since 2020 for individuals aged 4 years and older. OIT involves gradual dose escalation of the culprit

food protein under medical supervision, aiming to raise the reaction threshold. Phase 3 trials for peanut, milk, and egg OIT have demonstrated significant desensitization in 50–80% of participants, with key limitations including high rates of adverse events, variable achievement of sustained unresponsiveness, and importantly, an association with de novo EoE in a subset of treated individuals [1,76].

The occurrence of EoE as a complication of OIT—documented in approximately 2.7–5% of peanut OIT participants in meta-analyses—represents a critical and increasingly recognized intersection between FA and EoE management [77]. OIT-associated EoE typically resolves upon discontinuation of therapy and recurs upon rechallenge, suggesting a direct causal relationship. The mechanism remains incompletely understood but may involve sustained esophageal antigen exposure activating Th2-polarized esophageal immune responses. Clinicians initiating OIT must therefore counsel families about EoE risk and maintain vigilance for dysphagia, vomiting, or new esophageal symptoms throughout the protocol.

Epicutaneous immunotherapy (EPIT) and sublingual immunotherapy (SLIT) are alternative routes under investigation, with EPIT's lower antigen dose potentially conferring a lower risk of EoE than OIT.

A further paradigm shift in IgE-mediated FA management has been the regulatory approval of omalizumab (Xolair; Novartis/Genentech), a recombinant humanized monoclonal anti-IgE antibody that binds free circulating IgE and downregulates FcεRI expression on mast cells and basophils, thereby attenuating immediate hypersensitivity responses to allergen challenge. In February 2024, the FDA approved omalizumab for the reduction in allergic reactions—including anaphylaxis—to one or more foods in adults and children aged $\geq$ 1 year with IgE-mediated FA, representing the first approved pharmacotherapy for this indication in the United States. This approval was based on the Phase 3 OUtMATCH trial (NCT03881696), a randomized, double-blind, placebo-controlled study in participants aged 1–55 years with peanut allergy plus at least two additional food allergies: following 16–20 weeks of weight- and IgE-adjusted subcutaneous omalizumab, 67% of treated participants tolerated a single 600 mg peanut protein dose (approximately 2.5 peanuts) compared with 7% in the placebo group, with parallel efficacy demonstrated for milk, egg, and cashew. Omalizumab does not induce sustained unresponsiveness and is therefore best positioned as a threshold-raising agent rather than a curative treatment; it is most appropriate for patients at high risk of severe accidental exposure reactions, those unable to undertake or who have failed OIT, and potentially as an adjunct to facilitate safer OIT escalation.

6.2. Dietary Therapy for EoE

Dietary elimination therapy remains a mainstay of EoE treatment, exploiting the disease's food-antigen-driven pathogenesis to achieve histologic remission without systemic medication [78]. Several approaches of varying stringency and efficacy have been evaluated in pediatric and adult cohorts (overview in Table 3).

The elemental formula diet—involving complete replacement of dietary protein with amino acid-based formula—achieves histologic remission in approximately 90–98% of EoE patients, establishing dietary antigens as essential disease drivers [78,79]. However, the significant impact on quality of life, palatability, social functioning, and nutritional status renders this approach primarily suitable for infants and young children or as a short-term diagnostic intervention. The empiric 6-food elimination diet (6-FED)—removing milk, wheat, egg, soy, nuts, and seafood—achieves remission in approximately 72% of children and is a well-established effective approach, though it limits available foods, reduces quality of life and requires sequential reintroduction with endoscopic monitoring to identify individual triggers [80].

Table 3. Overview of dietary elimination strategies for EoE in children, including efficacy and clinical considerations.

Strategy	Histologic Remission Rate	Efficacy	Clinical Considerations
Elemental Formula Diet	~90–98%	Highly effective	Requires exclusive elemental formula; poor palatability; mainly for young children
6-Food Elimination Diet (6-FED)	~72%	High efficacy	Eliminates milk, wheat, egg, soy, nuts, seafood; requires multiple endoscopies for reintroduction; nutritional monitoring essential
4-Food Elimination Diet (4-FED)	~54%	Moderate-high	Eliminates milk, wheat, egg, soy; simpler than 6-FED; fewer endoscopies needed; first-line dietary approach in many centers
2-Food Elimination Diet (2-FED)	~40–43%	Moderate	Eliminates milk and wheat; easiest to implement; suitable as stepwise first approach; lower remission rate
Allergy Test-Directed Diet	~45%	Variable	Based on SPT/atopy patch testing; limited by poor predictive value; not recommended as sole trigger identification method per ESPGHAN 2024

EoE: eosinophilic esophagitis; SPT: skin prick test; 6-FED: 6-food elimination diet; 4-FED: 4-food elimination diet; 2-FED: 2-food elimination diet; ESPGHAN: European Society for Paediatric Gastroenterology, Hepatology and Nutrition.

Recognition that milk is the single most common EoE trigger (implicated in approximately 50–60% of EoE cases) and wheat is the second most common (30–40%) has led to the development of stepwise, less restrictive elimination strategies. The 4-food elimination diet (4-FED, removing milk, wheat, egg, and soy) achieves remission in approximately 54% of patients, while the 2-food elimination diet (2-FED, removing milk and wheat) achieves approximately 40–43% remission—representing a pragmatic first-step approach that minimizes dietary restriction while still achieving meaningful remission rates in a substantial proportion [81,82]. A stepwise approach beginning with the 2-FED before escalating to 4-FED or 6-FED is increasingly recommended in pediatric guidelines to minimize the dietary burden of initial intervention.

6.3. Pharmacological Therapy for EoE

6.3.1. Proton Pump Inhibitors

Proton pump inhibitors (PPIs) were initially employed as a diagnostic maneuver to exclude GERD-induced esophageal eosinophilia. However, prospective trials have established that PPIs achieve histologic remission ($\geq$15 eos/hpf threshold) in approximately 33–50% of EoE patients through mechanisms extending beyond acid suppression, including direct anti-eosinophilic and anti-inflammatory effects mediated through STAT6 signaling pathway inhibition [83]. Current guidelines position PPIs as a prominent treatment option in pediatric EoE, particularly in patients with overlapping GERD or with predominantly inflammatory endoscopic phenotype [2,70].

6.3.2. Topical Corticosteroids

Swallowed topical corticosteroids—most commonly budesonide oral suspension or melting tablet and fluticasone propionate from a metered-dose inhaler swallowed rather than inhaled—have been cornerstones of EoE pharmacotherapy for two decades. Meta-analyses demonstrate histologic remission rates of 60–70% and superior symptom improvement compared to placebo [84,85]. Concerns regarding systemic absorption and adrenal suppression with long-term use necessitate appropriate monitoring in pediatric patients,

though contemporary formulations are designed to minimize systemic bioavailability. Approval is still required for adequate topical steroid medication in many countries, and especially for smaller children.

6.4. Biologic Therapies: A Paradigm Shift

The most significant therapeutic advance in EoE management in recent years has been the clinical development and regulatory approval of biologic agents. These therapies directly inhibit the cytokine pathways driving EoE pathogenesis, offering the potential for disease-modifying effects beyond symptomatic control and—crucially—addressing multiple coexisting atopic conditions simultaneously (presented in Table 4).

Table 4. Biologic therapies for eosinophilic esophagitis: Approved and in clinical development.

Agent	Target/Mechanism	Regulatory Status	Key Efficacy Data	Clinical Notes
Dupilumab (Dupixent)	IL-4Rα (blocks IL-4 and IL-13)	FDA/EMA approved (≥1 yr, ≥15 kg)	~66% histologic remission (EoE KIDS; pediatric Phase 3)	First approved biologic for EoE; also treats coexisting AD, asthma, CRSwNP
Cendakimab	IL-13 (receptor antagonist)	Phase 3 (HEROES trial)	Histologic improvement; reduced fibrostenotic markers (reduced vimentin+)	Oral administration; anti-fibrotic potential under investigation
Mepolizumab	IL-5	Phase 2 completed; Phase 3 ongoing	Reduced eosinophilia; modest symptom improvement	Approved for eosinophilic asthma; being repurposed for EoE
Benralizumab	IL-5Rα (eosinophil depletion)	Phase 2/3 ongoing	Significant eosinophil reduction in esophagus	Rapid eosinophil depletion; attractive for fibrostenotic EoE
Lirentelimab (AK002)	Siglec-8 (eosinophils and mast cells)	Phase 2/3 (KRYPTOS)	Histologic remission in adults and adolescents	Dual mast cell/eosinophil target; potential for mast cell-dominant endotype
Tezepelumab	TSLP (upstream Th2 alarmin)	Phase 2 ongoing	Early data promising	Targets upstream alarmin; may address root esophageal barrier dysfunction

IL: interleukin; FDA: U.S. Food and Drug Administration; EMA: European Medicines Agency; TSLP: thymic stromal lymphopoietin; AD: atopic dermatitis; CRSwNP: chronic rhinosinusitis with nasal polyps.

Dupilumab (Dupixent; Sanofi/Regeneron), a fully human monoclonal antibody targeting the shared IL-4 receptor alpha (IL-4Rα) subunit, thereby blocking both IL-4 and IL-13 signaling, received FDA approval for adults and children ≥ 12 years with EoE in May 2022. In January 2024, the approval was extended to children aged 1 to 11 years and weighing at least 15 kg, based on data from the Phase 3 EoE KIDS trial (NCT04394351) [86,87]. In the pivotal EoE KIDS trial, 66% of children receiving the higher weight-based dose of dupilumab achieved histological disease remission (defined as peak eosinophil count ≤ 6 eos/hpf) at 16 weeks, compared with only 3% in the placebo group. More than 53% sustained remission through 52 weeks, and 97% of enrolled patients had at least one coexisting type 2 inflammatory disease at baseline—highlighting dupilumab's value in children with atopic multimorbidity [87]. Its simultaneous efficacy across AD, asthma, chronic rhinosinusitis with nasal polyps (CRSwNP), and EoE provides the first 'umbrella' biologic strategy for children with multiple atopic diseases, addressing the systemic Th2 inflammatory state

rather than targeting individual organ manifestations in isolation. Prospective data from Geba et al. demonstrated that dupilumab treatment of AD was associated with significantly reduced incidence of new or worsened allergic events across 17 allergy categories in a large meta-analysis of clinical trial data—the first evidence that the allergic disease trajectories itself may be pharmacologically modified [88].

6.5. Pipeline Agents

Beyond dupilumab, several additional biologics are in advanced clinical development for EoE. Cendakimab, an oral IL-13 receptor antagonist, has demonstrated promise in the Phase 2/3 trial, achieving histologic improvement and—of particular mechanistic interest—evidence of reduced esophageal epithelial-to-mesenchymal transition, suggesting potential anti-fibrotic properties [49]. Anti-IL-5 agents including mepolizumab (IV and SC) and benralizumab (anti-IL-5Rα) produce consistent esophageal eosinophil reduction in Phase 2 trials, though symptomatic improvement has been rather modest, potentially reflecting the more complex nature of EoE pathogenesis [89]. Lirentelimab, targeting Siglec-8 on both eosinophils and mast cells, represents a novel mechanistic approach addressing dual effector cell populations; Phase 2/3 trial data in adults and adolescents demonstrated significant histologic remission [90]. Tezepelumab (anti-TSLP)—approved for severe asthma—is being evaluated in EoE, targeting the upstream alarmin [49]. The topical steroid APT-1011, a fluticasone propionate orally disintegrating tablet, showed superior treatment effects for histologic, endoscopic responses, and for reduction in dysphagia when applied 3 mg once daily at bedtime [91].

6.6. Esophageal Dilation

For patients with fibrostenotic EoE and symptomatic esophageal stricture or small-caliber esophagus, esophageal dilation represents an important palliative procedure that relieves dysphagia by mechanically disrupting mucosal eosinophilic strictures [92]. Dilation does not address the underlying inflammatory disease and should always be performed in conjunction with anti-inflammatory treatment. Modern techniques have demonstrated safety in pediatric patients when performed by experienced endoscopists, with perforation rates of <0.5% in contemporary series.

6.7. Practical Implications for Clinical Management

The mechanistic and epidemiological overlap between FA and EoE has several direct and actionable implications for clinical practice. First, every child presenting with confirmed IgE-mediated FA should be flagged as being at elevated risk for EoE; clinicians and caregivers should be counseled to remain vigilant for evolving esophageal symptoms—in particular feeding difficulties and vomiting in young children, and dysphagia in older children—and a low threshold for diagnostic endoscopy is warranted in this population. Second, children diagnosed with EoE should undergo structured assessment for coexisting atopic diseases (AD, asthma, allergic rhinitis, IgE-mediated FA), given that a substantial share carry at least one concurrent atopic diagnosis and that multimorbidity may influence both therapeutic choice and monitoring strategy. Third, children initiating OIT should be informed of the risk of EoE as an adverse effect; with immediate suspension of the allergen dosing regimen if EoE is confirmed. Fourth, dupilumab now offers a single biologic solution capable of achieving disease control across EoE, AD, asthma, and FA simultaneously, making it a good choice biologic in children with severe and therapy refractory atopic multimorbidity. Treatment decisions should therefore be coordinated across allergy, pediatric gastroenterology, and dermatology rather than managed in separate specialty silos. Fifth, dietary interventions for EoE—particularly milk and wheat elimination—require

structured nutritional counseling to prevent energy and micronutrient deficiencies; dietitian involvement should be standard in all centers managing pediatric EoE.

7. Quality of Life and Psychosocial Impact

Both pediatric FA and EoE exert profound and multidimensional impacts on quality of life (QoL) that extend beyond physical symptoms to encompass social, educational, nutritional, and psychological domains. Children with FA experience heightened food anxiety, social isolation at mealtimes, and disrupted participation in peer activities, with parents reporting substantially elevated levels of caregiver burden, anxiety, and hypervigilance [93]. The economic costs of pediatric FA are substantial, with direct healthcare costs and lost parental productivity adding to the disease burden.

In EoE, the chronic nature of the disease, the restrictiveness of elimination diets, and the requirement for repeated endoscopic procedures collectively impair health-related QoL. Children with EoE demonstrate altered eating behaviors—including small bites, prolonged eating duration, excessive fluid intake during meals, and food avoidance—that increasingly restrict dietary variety and social participation as they age [94]. Esophageal remodeling has been shown to correlate directly with maladaptive eating behaviors in pediatric EoE, suggesting that physical disease progression translates directly into psychosocial harm [95]. Validated pediatric QoL instruments specific to EoE, including the Pediatric EoE Symptom Score v2.0 (PEESS v2.0) and the Pediatric Quality of Life Inventory (PedsQL), provide important clinical trial endpoints, but still struggle to be integrated into routine clinical monitoring.

8. Prevention Strategies

The confluence of mechanistic and epidemiological evidence has generated actionable preventive insights for both FA and EoE, though established prevention strategies remain primarily focused on FA rather than EoE specifically.

For IgE-mediated FA, the paradigm shift from allergen avoidance to early allergen introduction has been the most impactful preventive development of the past decade. The LEAP (Learning Early About Peanut) trial demonstrated unequivocally that early oral introduction of peanut between 4 and 11 months of age in high-risk infants (with severe AD or egg allergy) reduces the risk of peanut allergy by approximately 80% at 5 years of age [96]. Subsequent guidelines in Australia, the United Kingdom, and the United States now uniformly recommend early introduction of allergenic foods for all infants, with specific protocols for high-risk infants. Skin barrier interventions—daily emollient application and proactive treatment of AD—represent a complementary prevention strategy targeting the epicutaneous sensitization route, although definitive evidence from the BEEP trial was not conclusive [97].

For EoE, no primary prevention strategy has been established in clinical practice, though several candidate interventions are supported by mechanistic and epidemiological data. These include: (1) avoiding unnecessary antibiotic prescriptions in early childhood given their association with gut and esophageal dysbiosis; (2) breastfeeding promotion, given the protective associations observed in cohort studies; (3) appropriate timing of allergenic food introduction, which may simultaneously reduce the risk of FA and, by extension, EoE risk associated with preceding FA; and (4) early aggressive treatment of AD to reduce epicutaneous sensitization that may drive subsequent EoE risk [9].

9. Future Directions and Research Gaps

Despite substantial progress, significant knowledge gaps and unmet clinical needs persist in the overlapping field of pediatric FA and EoE, representing priorities for future research.

In the realm of pathogenesis, fundamental questions remain regarding the mechanisms of allergen-specific immune responses in EoE, the relative contributions of IgE-dependent versus IgE-independent pathways, and the role of the esophageal and gut microbiome in modulating EoE susceptibility and disease activity. Emerging evidence implicates esophageal microbial dysbiosis—particularly alterations in the Streptococcus and Haemophilus genera—in EoE pathogenesis, though causality remains to be established [98]. Elucidating the mechanistic basis of EoE endotypes will be critical for advancing precision medicine approaches, particularly as the biologic pipeline expands with agents targeting distinct cytokine pathways.

Non-invasive and minimally invasive biomarker development represents an urgent clinical research priority. The requirement for repeated endoscopy with sedation—particularly burdensome in young children—represents a significant barrier to optimal disease monitoring and constitutes a disproportionate burden on patients and healthcare systems. Validation of blood biomarkers (eotaxin-3, periostin, IL-13), or other test-based assessment for routine clinical monitoring would transform EoE disease management by enabling more frequent, less invasive assessment of disease activity [73,74].

The long-term efficacy, safety, and optimal sequencing of biologic therapies in children require prospective study. Critical questions include the optimal timepoint of initiation as well as duration of dupilumab therapy, the adequate dosage for induction and maintenance, whether long-term biologic treatment prevents fibrostenotic complications, and how to manage patients in whom biologics achieve histologic but incomplete symptom remission. The potential for combination strategies—biologic therapy concurrent with dietary modification—has barely been explored. Moreover, given the high co-prevalence of EoE and FA in pediatric OIT programs, standardized monitoring protocols for OIT-associated EoE require development and implementation.

From a public health and prevention perspective, large-scale prospective birth cohort studies incorporating multiomics data—genomics, epigenomics, microbiome, metabolomics—are needed to identify the precise environmental exposures and biological pathways responsible for the epidemiological rise in FA and EoE.

Understanding whether and how the allergic march can be interrupted or attenuated by early intervention—through coordinated allergen introduction, emollient therapy, microbiome modulation, or biologic therapy—represents one of the most important questions in contemporary pediatric allergology.

Limitations of the Current Evidence Base

The existing literature on the FA–EoE relationship is subject to several important methodological limitations that warrant explicit acknowledgment. First, substantial study heterogeneity exists across the included evidence base: differences in FA and EoE diagnostic criteria (e.g., varying eosinophil thresholds per high-power field, patient age ranges, allergen panels used for FA diagnosis, and outcome definitions preclude direct quantitative synthesis across many studies and reduce the reliability of pooled prevalence and incidence estimates. Second, diagnostic bias represents a pervasive concern in EoE epidemiology: EoE is by definition an endoscopy- and biopsy-dependent diagnosis, so its apparent prevalence is inherently dependent on endoscopy access, clinical awareness, and biopsy practice patterns. The marked rise in EoE prevalence over the past two decades likely reflects in part, increased detection rates rather than a purely biological increase; disentangling these

contributions remains methodologically challenging. Third, geographic and population differences significantly constrain the generalizability of existing data. The overwhelming majority of prevalence and clinical studies originate from North America and Western Europe; data from Eastern Europe, Asia, Africa, Latin America, and the Middle East remain sparse. Ethnic and socioeconomic gradients in EoE prevalence—including the emerging recognition of underdiagnosis in non-White pediatric populations—are inadequately characterized. The distribution of food allergens triggering EoE varies geographically (e.g., seafood-dominant triggers in Asian cohorts versus dairy/wheat in Western cohorts), limiting the transferability of elimination diet protocols developed in North American populations to other health systems. Fourth, the longitudinal data needed to definitively establish causal directionality in the FA–EoE relationship—rather than co-occurrence or shared susceptibility—are limited; prospective birth cohort studies with systematic endoscopic surveillance are largely absent.

10. Conclusions

Food allergy and eosinophilic esophagitis represent two of the most clinically significant and rapidly increasing atopic diseases of childhood, united by shared immunological mechanisms, genetic susceptibility, and epidemiological trajectories. The substantially elevated EoE risk in children with IgE-mediated FA and the observed susceptibility of concomitant type 2 inflammatory diseases with possible intraindividual sequential development underscore the need for integrated, multidisciplinary care of atopic children that spans allergy, gastroenterology, and dermatology.

The shared Th2 inflammatory pathways—characterized by TSLP/alarmin-driven ILC2 and dendritic cell activation, IL-4/IL-13-mediated Th2 differentiation, and downstream eosinophil and mast cell effector responses—position biologics targeting these common pathways as particularly attractive therapeutic approaches for children with atopic multimorbidity.

Effective prevention of pediatric FA, through early allergen introduction and management of the primary atopic sensitizer—AD—remains the most impactful intervention strategy for reducing the downstream burden of EoE and the allergic march more broadly. Future research must prioritize non-invasive monitoring tools, risk and therapeutic stratification and prevention strategies targeting both conditions simultaneously, and long-term real-world outcomes of the expanding biologic therapeutic armamentarium.

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Abbreviations

The following abbreviations are used in this manuscript:

AD	Atopic dermatitis
AGE	Advanced glycation end product
AR	Allergic rhinitis
BAT	Basophil activation test
CAPN14	Calpain-14
CRD	Component-resolved diagnostics
CRSwNP	Chronic rhinosinusitis with nasal polyps
DSG1	Desmoglein-1
ECP	Eosinophil cationic protein
EoE	Eosinophilic esophagitis
EPIT	Epicutaneous immunotherapy
EREFS	EoE Endoscopic Reference Score
FA	Food allergy
FLG	Filaggrin
GERD	Gastroesophageal reflux disease
ICS	Inhaled corticosteroid
IgE	Immunoglobulin E
ILC2	Group 2 innate lymphoid cell
IL	Interleukin
OFC	Oral food challenge
OIT	Oral immunotherapy
PPI	Proton pump inhibitor
sIgE	Serum specific IgE
SPT	Skin prick testing
Th2	Type 2 T helper
TSLP	Thymic stromal lymphopoietin
UPF	Ultra-processed food

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