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Pollen-Food Allergy Syndrome: Pathogenesis, Diagnosis, Management, and Implications for Athletic Populations — A Narrative Review

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Abstract

Background. Pollen-food allergy syndrome (PFAS) is an IgE-mediated hypersensitivity reaction caused by cross-reactivity between airborne pollen allergens and homologous proteins in plant-derived foods. **Aim.** To provide a structured overview of PFAS pathogenesis, allergen components, regional epidemiology, clinical spectrum, diagnosis and management, with emphasis on its relevance for sports medicine. **Material and methods.** Narrative review of 25 full-text articles (1987–2025): position papers, large cohorts, randomised trials and the 2024 AAAAI Delphi consensus. **Results.** PFAS affects 4.7–53.8% of pollen-allergic patients; anaphylaxis is reported in 1.7–8.9%. PR-10 sensitisation drives the birch–Rosaceae pattern, profilins underlie milder polyreactive phenotypes, nsLTP and GRP are linked to severe, cofactor-dependent reactions. Sublingual immunotherapy with recombinant Mal d 1 and omalizumab are the most promising therapies; pollen immunotherapy alone is not currently endorsed for PFAS. Exercise, NSAIDs and uncontrolled rhinitis amplify severity, with direct relevance for athletes. **Conclusions.** PFAS is a heterogeneous, prevalent and clinically meaningful condition. Personalised diagnostics, cofactor avoidance and emerging biologicals will shape future management, especially for outdoor athletes exposed to lengthening pollen seasons under climate change.

Key words: pollen-food allergy syndrome; oral allergy syndrome; cross-reactivity; pan-allergens; food-dependent exercise-induced anaphylaxis; allergen immunotherapy; omalizumab; athletes.

1. Introduction

Pollen-food allergy syndrome (PFAS) is the most prevalent IgE-mediated food allergy in adolescents and adults living in temperate climates, and is increasingly recognised in children [1,2]. The first systematic description of the entity was provided by Amlot and colleagues in 1987, who reported that 33 of 80 highly atopic patients (41%) experienced food-induced oropharyngeal symptoms and proposed the term “oral allergy syndrome” (OAS) for a stereotyped sequence beginning with oral irritation that could progress to systemic features [3]. The acronym PFAS was introduced in 1995 to better reflect the underlying pathogenesis, namely cross-reactivity between inhaled pollen allergens and homologous proteins in plant-derived foods [1,4].

Conceptually, the syndrome belongs to class II food allergies, in which sensitisation occurs through the airways to labile allergens, in contrast to classical class I food allergies driven by digestion-resistant proteins ingested via the gastrointestinal tract [1,4,5]. The clinical phenotype is therefore largely determined by the molecular profile

of the patient: PR-10 and profilin sensitisation typically produces a self-limited oropharyngeal reaction, whereas non-specific lipid transfer proteins (nsLTPs) and gibberellin-regulated proteins (GRPs) — both heat- and digestion-resistant — are associated with systemic and life-threatening reactions [2,6,7,8].

Although PFAS is usually depicted as a benign condition, between 1.7% and 8.9% of patients experience anaphylaxis, and up to 3% develop systemic symptoms without any preceding oral phase [1,9]. The disease is dynamic and increasingly relevant for several reasons: pollen seasons are lengthening and intensifying under climate change, ragweed (*Ambrosia artemisiifolia*) is invading central and northern Europe, and the prevalence of seasonal allergic rhinitis is rising worldwide [2,10,11]. These trends are particularly important for athletes, who train and compete outdoors, often during peak pollen seasons, and in whom uncontrolled rhinitis or food-dependent exercise-induced anaphylaxis (FDEIA) can compromise both performance and safety [12,13].

The aim of this narrative review is to synthesise the current state of knowledge on PFAS — covering terminology, molecular basis, epidemiology, clinical spectrum, diagnosis and management — with particular reference to physically active populations. We highlight controversies, recent therapeutic advances and unresolved research questions.

2. Materials and methods

This study is a narrative literature review. The bibliographic search was conducted in PubMed/MEDLINE, Web of Science, Scopus and Google Scholar databases. The primary search strategy combined the terms “pollen food allergy syndrome”, “oral allergy syndrome”, “class II food allergy”, “cross-reactivity”, “PR-10”, “profilin”, “lipid transfer protein”, “gibberellin-regulated protein”, “food-dependent exercise-induced anaphylaxis”, “athletes” and “climate change”, with emphasis on full-text articles in English published between 1987 and 2025. Priority was given to international position papers and consensus documents (European Academy of Allergy and Clinical Immunology — EAACI 2015 [5]; American Academy of Allergy, Asthma and Immunology — AAAAI 2024 Delphi [9]; Italian Society of Pediatric Allergy and Immunology — SIAIP 2017 [14]), large cohort studies (T-Child birth cohort [15]; the Italian paediatric cohort of 1271 children with seasonal rhinoconjunctivitis [10]; the Polish single-centre study by Cudowska et al. [16]) and randomised controlled trials (Kinaciyan et al. for sublingual immunotherapy with recombinant Mal d 1 [17]; Wood et al. OUtMATCH for omalizumab [18]).

3. Results

3.1. Terminology, definitions and classification

The terminology of pollen-related plant-food allergy has evolved considerably. Amlot et al. introduced “oral allergy syndrome” in 1987 to describe a chronological progression of food-induced symptoms beginning with lip and throat irritation [3]. In 1988 Ortolani and colleagues linked OAS specifically to birch pollinosis and Rosaceae fruits [19], and in 1995 the umbrella term PFAS was coined to encompass the broader class II food-allergy phenomenon [1,4]. According to the EAACI position paper, up to 60% of food allergies in older children, adolescents and adults are linked with an inhalant allergy and are mediated by cross-reactive IgE binding to homologous epitopes in pollen and food [5].

Current usage distinguishes OAS — symptoms confined to the oral mucosa, regardless of cause — from PFAS, which refers to the IgE-mediated cross-reactive food allergy that develops on the basis of prior pollen sensitisation and may extend beyond the oropharynx [2,4]. Several syndromic sub-entities are widely recognised: birch–apple/Rosaceae syndrome, mugwort–celery–spice syndrome, ragweed–melon–banana association, plane tree–hazelnut–peach syndrome, latex–fruit syndrome and cypress–peach (GRP) syndrome [1,2,5,6,20]. The

lipid-transfer-protein (LTP) syndrome describes patients sensitised to nsLTPs (most often Pru p 3 from peach), in whom multiple botanically unrelated plant foods elicit reactions that range from oral itching to anaphylaxis, frequently in the presence of cofactors [8,11].

3.2. Molecular basis: cross-reactive pan-allergen families

Approximately 65% of plant food allergens belong to three protein superfamilies — the prolamin superfamily (which includes 2S albumins and nsLTPs), the cupin superfamily (7S and 11S seed-storage globulins) and the pathogenesis-related (PR) protein family [1,5]. Within these, four pan-allergen groups account for the vast majority of PFAS phenotypes: Bet v 1-related PR-10 proteins, profilins, nsLTPs and the more recently described GRPs [2,6,7,21].

3.2.1. PR-10 proteins (Bet v 1 homologues)

Bet v 1, the major allergen of *Betula verrucosa* (white birch), is the prototypical class II food sensitiser. Its homologues are widespread among rosaceous fruits (Mal d 1 in apple, Pru p 1 in peach, Pru av 1 in cherry, Pyr c 1 in pear), Apiaceae vegetables (Api g 1 in celery, Dau c 1 in carrot), legumes (Gly m 4 in soy, Ara h 8 in peanut) and tree nuts (Cor a 1 in hazelnut) [1,2,21]. PR-10 proteins are heat- and digestion-labile, which explains why PFAS triggered by them is usually limited to the oral mucosa and tolerable when the food is cooked [1,5]. Across European countries, the Global Allergy and Asthma European Network (GA²LEN) database showed that 6.8–57.4% of the population is sensitised to birch pollen, of whom 47.6–98.5% develop associated symptoms; among birch-allergic individuals, approximately 70% experience IgE-mediated cross-reactive food hypersensitivity [2]. In a German series of 260 patients with tree pollinosis, 92% were sensitised to Bet v 1, and in an Italian survey of 854 birch-pollinosis patients the figure ranged from 53% to 95% [2]. Aln g 1 from *Alnus glutinosa* (alder) shares more than 80% protein homology with Bet v 1, allowing PFAS to occur in regions where birch is absent but alder is widespread [2].

3.2.2. Profilins

Profilins are 12–15 kDa cytoskeletal proteins ubiquitous across all eukaryotic cells and highly conserved among plant species [21]. Bet v 2 (birch) and Phl p 12 (timothy grass) are the prototypes; food homologues include Mal d 4 (apple), Dau c 4 (carrot), Api g 4 (celery), Cuc m 2 (melon) and Citr l 2 (watermelon) [2,21]. Sensitisation to profilins occurs in 20–30% of patients with tree pollen allergy [1,2,21] and in 10–38% of those sensitised to birch, while 14–24% of patients sensitised to the major timothy allergen Phl p 1/5 are also positive for Phl p 12 [21]. Profilins are easily degraded by pepsin and are usually associated with mild oropharyngeal reactions, but anaphylactic episodes have been reported, particularly in patients with high pollen exposure or after ingestion of large amounts of profilin-rich foods such as lychee or fresh fruit smoothies [21].

3.2.3. Non-specific lipid transfer proteins (nsLTPs)

Non-specific LTPs are small, basic, 8–10 kDa proteins of the prolamin superfamily, classified within PR-14 [1,21]. They share a stable tertiary fold supported by four disulphide bridges and contain a hydrophobic cavity capable of hosting fatty-acid ligands [22]. This structural rigidity makes nsLTPs resistant to heat — Mal d 3 retains IgE reactivity even after 20 minutes at 90 °C — and to gastric digestion, which explains their association with severe and even systemic reactions [1,21]. Pru p 3 from peach is regarded as the primary sensitiser of LTP syndrome and a marker for severe reactions. Cor a 8 (hazelnut), Ara h 9 (peanut), Jug r 3 (walnut), Tri a 14 (wheat) and Mal d 3 (apple) are clinically relevant homologues [1,2,5].

Pascal et al. characterised 45 LTP-monosensitised patients in Barcelona and reported a heterogeneous clinical pattern: oral allergy syndrome in 75.6%, urticaria in 66.7%, gastrointestinal disorders in 55.6% and anaphylaxis in 75.6% of patients [8]. Cofactors — most often non-steroidal anti-inflammatory drugs (NSAIDs) or exercise — were involved in 40% of cases, and were indispensable in 32.4% of anaphylactic events. Pollinosis, particularly to plane tree (*Platanus acerifolia*) and mugwort (*Artemisia vulgaris*), was diagnosed in 75.6% of patients [8]. Mechanistically, Dubiela and colleagues demonstrated that binding of unsaturated free fatty acids — oleic, linoleic and elaidic acid — induces a conformational change in Pru p 3, exposing a dominant IgE epitope and enhancing the protein's allergenicity [22]. This fatty-acid-dependent exposure of epitopes provides a molecular rationale for the well-known protective effect of co-sensitisation to labile allergens such as PR-10 proteins, which appears to dampen LTP-driven phenotypes through unknown immunological pathways [1,10].

3.2.4 Gibberellin-regulated proteins (GRPs)

GRPs (or GASA proteins) are cysteine-rich antimicrobial peptides of approximately 6.9 kDa, identified during efforts to clarify peach allergies in patients negative for Pru p 3 [7]. The first member, peamaclein/Pru p 7, was officially registered in 2013; subsequent allergens include Pru m 7 (Japanese apricot), Cit s 7 (orange), Pun g 7 (pomegranate), Pru av 7 (cherry) and Cup s 7 (cypress pollen) [2,7]. GRPs are highly stable to heat and digestive enzymes, akin to nsLTPs [7]. Among 100 Japanese fruit-allergic patients, GRP monosensitisation was identified in 13%, and 65% of those without PR-10 profilin reactivity were GRP-sensitised; all 13 GRP-monosensitised patients experienced grade ≥2 systemic reaction, with anaphylactic shock in 31% [7]. Peach was the most frequent culprit (92.3%), followed by apricot (61.5%), orange (46.2%) and apple (30.8%). Cofactors — exercise or aspirin — enhanced symptom onset in 61.5% of patients [7]. Eyelid oedema and laryngeal tightness are characteristic features [7]. Clinical and serological cross-reactivity has been demonstrated between fruit GRPs and the cypress GRP Cup s 7 (Cypmaclein), suggesting a pollen-driven sensitisation route, especially in southern Europe and Japan [2,7].

3.2.5. Other relevant panallergens and emerging components

Beyond the four major families, several additional cross-reactive structures contribute to PFAS phenotypes. Thaumatin-like proteins (TLPs; PR-5) have been described in cypress, mugwort, olive, plane tree, birch pollen, apple, cherry, kiwifruit and grape [6,21]. Class I chitinases (Hev b 6 from *Hevea brasiliensis*) underpin the latex–fruit syndrome, which affects 21–58% of latex-allergic patients and most often involves banana, avocado, kiwifruit and chestnut [6,20]. Profilin-independent allergens have also been reported: Li and colleagues recently identified fructose-bisphosphate aldolase as a novel cross-reactive component between *Artemisia* pollen and red fruit ginseng (*Campanumoea lancifolia*), with an estimated 89–90% homology to Art si 14 and Art an 14 [23]. Cross-reactivity has also been documented between microfungi aeroallergens (such as *Alternaria alternata* and *Cladosporium herbarum*) and edible mushrooms; Gauld et al. described a 15-year-old patient who developed PFAS-like symptoms to fresh portobello and cremini mushrooms while tolerating canned mushrooms, consistent with a heat-labile cross-reactive allergen [24]. These observations support an open and dynamic concept of pan-allergen-mediated cross-reactivity in PFAS.

Table 1. Summary of major pan-allergen families implicated in pollen-food allergy syndrome.

Family	Prototype pollen allergen	Representative food homologues	Stability	Typical clinical phenotype
PR-10 (Bet v 1-like)	*Betula* Bet v 1; *Alnus* Aln g 1	Mal d 1 (apple), Pru p 1 (peach), Cor a 1 (hazelnut), Api g 1 (celery), Gly m 4 (soy)	Heat- and digestion-labile	Oral allergy; mild, self-limited; rarely anaphylaxis (e.g. Gly m 4 in soy beverage)
Profilins	Bet v 2 (birch); Phl p 12 (timothy)	Mal d 4, Pru p 4, Dau c 4, Cuc m 2, Citr l 2	Heat- and digestion-labile	Mild OAS; rarely systemic with high allergen loads (smoothies, lychee)
nsLTPs (PR-14)	Art v 3 (mugwort), Pla a 3 (plane tree)	Pru p 3 (peach), Cor a 8 (hazelnut), Ara h 9 (peanut), Tri a 14 (wheat)	Heat- and digestion-stable	LTP syndrome — multi-food allergy, anaphylaxis, cofactor dependence
GRPs (Snakin/GASA)	Cup s 7 (cypress), Cry j 7 (Japanese cedar)	Pru p 7 (peach), Pru m 7 (apricot), Cit s 7 (orange), Pun g 7 (pomegranate)	Heat- and digestion-stable	Severe systemic reactions; eyelid oedema, laryngeal tightness; cofactor-dependence in ~60%

3.3. Epidemiology and natural history

The reported prevalence of PFAS varies dramatically — from 0.8% to 53.8% — depending on geography, climate, the dominant pollen source, the population studied and the definition used [1,2,5]. In a UK survey of 3590 subjects, the average prevalence was 2% (range 0.8–4.1%) [1]. In Korean children with pollen allergy, OAS reached 42.7%; in Swedish 4–8-year-olds it was 25%, with 31% in birch-allergic versus 5% in those sensitised to other pollens [10]. In a multicentre study at nine sites in southern Europe, 167 of 815 (20.5%) patients with seasonal allergic rhinoconjunctivitis aged 10–60 years had PFAS, with prevalence ranging by centre from 7.5% to 41.4% [2]. In a Japanese single-centre series of 6824 outpatients in Fukui Prefecture, where birch pollen is rare, PFAS prevalence was 10.8%, with foods of the Cucurbitaceae and Rosaceae families predominating [2]. A Sasaki et al. nationwide survey of 41,264 elementary and 35,302 middle-school Japanese students reported PFAS in 0.99% and 2.8% respectively [2].

In children, the disease is far from rare. Mastrorilli et al. studied 1271 Italian children aged 4–18 with seasonal allergic rhinoconjunctivitis and identified PFAS in 300 (24%); five distinct molecular endotypes — profilin-PFAS, LTP-PFAS, PR-10-PFAS, multi-pan-allergen and no-pan-allergen — were defined by unsupervised cluster analysis [10]. Prevalence in paediatric series ranged from 5% in North America to 48% in a UK clinic, reflecting both methodological heterogeneity and regional differences [10]. Risk factors for PFAS in children included female sex, older age, parental atopy, overweight and exposure to environmental tobacco smoke [10].

Cudowska et al. evaluated 43 Polish children aged 2–14 years with pollen-food sensitisation: 65.1% had concomitant seasonal allergic rhinitis, 25.6% reported a temporal association between ingestion of pollen-related foods and exacerbation of nasal symptoms, 25.6% experienced OAS and 25.6% had anaphylactic reactions to foods (mostly cow's milk, peanut, egg or pear) [16]. Strikingly, 37.2% remained asymptomatic to plant foods despite confirmed sensitisation, underlining the limited predictive value of isolated IgE testing and the importance of correlating molecular results with clinical history [16].

The longitudinal T-Child cohort in Tokyo prospectively followed 521 children to the age of 13 and identified PFAS in 11.3%, of whom 72.7% were sensitised to at least one tree-, grass- or weed-derived allergen [15]. Sensitisation to Bet v 1 at age 5 conferred the strongest risk of PFAS at age 13 (adjusted odds ratio aOR 10.6; 95% confidence interval CI 2.64–42.5); at age 9, the association remained robust (aOR 9.10; 95% CI 4.71–17.6) [15]. Cry j 1 (Japanese cedar) sensitisation at age 9 also predicted PFAS (aOR 4.28; 95% CI 1.98–9.25), and the combination of wheezing, eczema and rhinitis at age 5 yielded a triple-symptom aOR of 3.47 (95% CI 1.34–8.98) [15]. These data support the integration of PFAS into the broader concept of the "allergic march". Risk factors identified by Kato et al. in their Fukui cohort included a history of bronchial asthma (1.7-fold), immediate-type food allergy (3.6-fold), drug allergy (2.2-fold) and latex hypersensitivity (2.4-fold) [2].

3.4. Clinical spectrum — from oral pruritus to systemic anaphylaxis

Most patients present with a constellation of immediate-type oropharyngeal symptoms appearing within 5–15 minutes of food ingestion: pruritus of the lips, tongue, palate, ears and throat, mild angioedema and occasionally short-lived blisters of the oral mucosa [1,5]. Spontaneous resolution within 10–30 minutes is the rule [5]. Foods classically associated with a higher risk of systemic reaction include almonds, apricots, cherries, celery, lentils, peaches, plums and tomatoes [1,4].

The Al-Shaikhly et al. AAAAI Delphi exercise estimated that anaphylaxis as a manifestation of PFAS affected 1.7% of patients in the original Ma survey, but more recent studies have reported higher figures: 8.9% in a Korean nationwide series and 5% in a large Italian cohort [9]. Modifiable factors associated with severe outcomes include proton-pump inhibitors (PPIs), NSAIDs, alcohol, fasting, exercise, uncontrolled asthma and bariatric surgery [9]. In a multicentre retrospective study of patients with systemic reactions to labile plant-food allergens, PPI use was significantly more frequent than in those with confined oropharyngeal symptoms [9]. Wolters and colleagues reported that 9 patients with PFAS who underwent gastric bypass surgery developed systemic reactions to Rosaceae fruits, tree nuts and peanuts that had previously been tolerated [9].

3.5. Diagnostic work-up

Diagnosis of PFAS combines a thorough clinical history, skin testing and serological assays, with confirmation by oral food challenge in selected cases [2,6,9]. The clinical history should establish the pattern of pollen allergy, the spectrum of triggering foods (raw versus cooked), the form of food (whole, juiced, smoothie), latency between ingestion and symptoms, severity of reactions and presence of cofactors. Documenting whether reactions are confined to the oral cavity or include systemic features is essential for risk stratification and management decisions [9].

Skin prick testing (SPT) with commercial extracts of fruits and vegetables suffers from poor sensitivity owing to allergen lability and lack of standardisation. Prick-to-prick (PtP) testing with the fresh culprit food is therefore preferred — sensitivity for celery, hazelnut and carrot using fresh extracts reached 100/80/100% versus 85/80/29% with commercial extracts in one comparative study [4]. Specific IgE testing (ImmunoCAP) provides

quantitative information but may detect clinically irrelevant sensitisation; for instance, in grass-pollen-allergic individuals 78% have positive SPT and 40.5% positive sIgE to wheat, while only 0.48% have genuine wheat allergy demonstrable by oral challenge [6].

Component-resolved diagnostics (CRD) — using purified or recombinant allergen panels (ImmunoCAP ISAC, ALEX, FABER) — has revolutionised PFAS evaluation [2,7,21]. CRD distinguishes sensitisation to specific allergen families and predicts clinical severity: positivity to PR-10 or profilin suggests milder phenotypes, whereas LTP or GRP positivity predicts systemic risk [9,21].

Double-blind placebo-controlled food challenge (DBPCFC) remains the gold standard for diagnostic confirmation, yet is logistically difficult for raw fruits because lyophilisation and matrix manipulation may destroy labile allergens [4]. In clinical practice, open or single-blind challenges are often used, and a combination of clinical history, fresh-food PtP, sIgE and CRD usually suffices [9].

3.6. Management

3.6.1. Avoidance, food processing and patient education

Avoidance of the raw culprit food remains the cornerstone of management for confirmed PFAS, while well-cooked, tinned or jam forms are usually tolerated for PR-10- and profilin-driven phenotypes [2,9]. However, the AAAAI panel cautioned that patients should not be given exhaustive avoidance lists for botanically related fruits and vegetables, as this may promote unnecessary dietary restriction and undue anxiety, with potential negative consequences for nutrition and quality of life [9]. Conversely, patients with a history of systemic reactions should strictly avoid the responsible foods [9]. Survey data show considerable variation: a US allergists' survey reported that 53% recommended complete avoidance, while approximately 40% adopted a case-by-case approach; in the UK, 80% of healthcare professionals recommended avoidance [9]. The risk of inadvertent exposure in juice bars and commercial salads was specifically highlighted [9].

The 2024 AAAAI Delphi consensus assigned its highest appropriateness score (median 9; disagreement index ≤0.132) to a series of practical statements: patients should be educated about the mechanistic basis of PFAS; reactions are mostly benign but rarely severe; lighter cooking methods such as steaming or stir-frying may be insufficient to fully denature relevant allergens; peeling and seed removal alone are generally inadequate; nut roasting at 140 °C for 40 minutes did not prevent PFAS in 5/17 birch-allergic adults; pollen allergen immunotherapy should not be considered an indication-driven treatment for PFAS [9].

3.6.2. Allergen-specific immunotherapy (AIT)

The therapeutic potential of pollen AIT for associated food allergies has been intensely studied, with conflicting results [5,14,25]. Asero et al. reported that 80% of 49 birch-sensitised patients receiving subcutaneous immunotherapy (SCIT) for 1–3 years experienced subjective improvement in apple-induced symptoms, with complete resolution in 54% [1]. Hansen et al., however, found that SLIT improved apple allergy in only 1/11 patients while inducing apple allergy in 3/11 [1]. The systematic review by Kallen et al. included 10 studies and 475 patients evaluating SCIT or SLIT with birch pollen for birch-related food allergy; only three studies were of moderate (rather than high) risk of bias [25]. Two of three moderate-risk studies showed reduced symptoms during food challenge, and one of three reported a significant 24-fold increase in eliciting dose, with 23% of treated versus 0% of control patients tolerating the highest food challenge dose [25]. The authors concluded that

current evidence is insufficient to draw firm conclusions, and the AAAAI Delphi consensus correspondingly classified PFAS as not a primary indication for pollen AIT [9,25].

A more targeted approach was tested by Kinaciyan et al. in a randomised, double-blind, placebo-controlled trial: 60 birch-pollen-allergic adults with apple allergy were assigned to daily SLIT with 25 µg of recombinant Mal d 1 (rMal d 1), recombinant Bet v 1 (rBet v 1) or placebo for 16 weeks [17]. SLIT with rMal d 1 significantly reduced the rMal d 1-induced sublingual oral symptom score ($P = 0.001$ versus placebo, $P = 0.038$ versus rBet v 1), reduced cutaneous reactivity ($P = 0.022$) and increased the rMal d 1-specific IgG4/IgE ratio ($P = 0.012$); 70% of rMal d 1-treated participants tolerated higher challenge doses, and 25% tolerated both the highest dose of rMal d 1 and a subsequent challenge with 20 g of fresh apple. By contrast, SLIT with rBet v 1 did not improve clinical reactivity to rMal d 1 [17]. No systemic adverse events were observed. These data support a hypothesis-driven shift towards food-specific molecular SLIT for selected single-component PFAS phenotypes [17]. The Italian SIAIP consensus on AIT in children reaffirmed that SCIT and SLIT are effective for allergic rhinitis, conjunctivitis and asthma, but classified food immunotherapy as still experimental, with a favourable evidence base only for peanut [14].

3.6.3. Biologicals — omalizumab and beyond

Omalizumab, a humanised anti-IgE monoclonal antibody binding to the Cε3 domain of free IgE, has long been used in severe asthma and chronic urticaria. Wood et al. reported the pivotal phase 3 OUtMATCH trial, in which 180 persons aged 1–55 years with allergies to peanut and at least two other foods (cashew, milk, egg, walnut, wheat or hazelnut) were randomised 2:1 to receive subcutaneous omalizumab or placebo for 16–20 weeks; the analysis population comprised 177 children and adolescents [18]. The primary endpoint — ingestion of at least 600 mg of peanut protein without dose-limiting symptoms — was met by 79/118 (67%) on omalizumab versus 4/59 (7%) on placebo ($P < 0.001$), and similar effects were observed for cashew (41% vs 3%), milk (66% vs 10%) and egg (67% vs 0%), all $P < 0.001$ [18]. Notably, 80% of treated participants tolerated 1044 mg of any one food, 69% of two foods and 47% of three foods [18]. The US Food and Drug Administration approved omalizumab for IgE-mediated food allergy in 2024, although Wood et al. emphasise that omalizumab does not eliminate food allergy or allow free ingestion, but reduces reactions to accidental exposures [18]. Although PFAS was not the primary target of OUtMATCH, the underlying mechanism — broad reduction in mast-cell activation through IgE neutralisation — supports a plausible benefit for PFAS, particularly for cofactor-driven or LTP-related phenotypes; dedicated trials are awaited [2,18].

3.6.4. Pharmacotherapy of acute reactions

For mild oropharyngeal symptoms, non-sedating H1-antihistamines are recommended on demand; chewable or liquid formulations may act faster [9]. Symptoms typically resolve within 30 minutes and rarely require active treatment [9]. For systemic reactions, intramuscular epinephrine via an autoinjector remains the first-line therapy; the prescription threshold is debated. Ma et al. found that 70% of US allergists would consider an epinephrine autoinjector (EAI) for PFAS, compared with only 18% in the UK [1]. In the AAAAI Delphi exercise, 82 of 122 surveyed US allergists reported case-by-case prescription, with throat involvement, facial oedema and generalised urticaria being the most common indications [9]. The 2014 US food-allergy practice parameter recommended EAI for patients with a history of laryngeal swelling or respiratory compromise [1,9]. The panel further endorsed shared decision-making for EAI in patients with PFAS limited to the oropharynx but with non-

modifiable risk factors such as gastric bypass surgery or asthma [9]. Novel non-injectable epinephrine formulations (intranasal, sublingual) under development may further lower the threshold for prescription [9].

3.7. PFAS in athletic populations

For athletes, three intersecting factors make PFAS a clinically distinct problem: (i) the high prevalence of pollen allergy among elite athletes, (ii) the role of exercise as the most consistent augmenting factor in food-allergic anaphylaxis, and (iii) the strict regulatory environment for permitted medications in competition. Katelaris et al. surveyed 214 Australian Olympic-level athletes from 12 sports prior to the Sydney 2000 spring Olympics: 56% reported symptoms consistent with allergic rhinoconjunctivitis, 41% had compatible symptoms plus a positive prick test to at least one allergen, and 29% had seasonal allergic rhinoconjunctivitis (positive history with a positive skin response to a seasonal allergen) [13]. Aquatic athletes were more likely to be allergic than non-aquatic athletes; equestrians were the least allergic, plausibly through self-selection [13]. Quality-of-life and symptom scores during the spring pollen season were significantly poorer in allergic athletes [13]. The authors emphasised the need to screen prospective Olympians for pollen allergy and tailor permitted medication, including intranasal corticosteroids, second-generation antihistamines and immunotherapy when symptoms are severe [13]. Helbling et al. previously found that 16.8% of 2961 Swiss elite athletes reported hay fever, 59% of whom required medication during the pollen season [13].

Food-dependent exercise-induced anaphylaxis (FDEIA) is the disorder most directly relevant to athletes with cross-reactive sensitisation. Feldweg classifies FDEIA as a food-allergy syndrome in which exercise — and, less consistently, NSAIDs, alcohol or hot/humid environmental conditions — facilitates allergen-induced mast-cell activation, possibly by increasing gastrointestinal allergen absorption or by lowering the activation threshold of mast cells and basophils [12]. In Western populations, the most commonly implicated foods are wheat (omega-5-gliadin in wheat-dependent exercise-induced anaphylaxis, WDEIA), other grains and tree nuts; in Asian populations, wheat and shellfish predominate [12]. A Japanese survey reported a prevalence of approximately 0.02% among junior high-school students [12]. The culprit food is usually ingested within 4 hours preceding exercise; symptoms range from pruritus, fatigue and urticaria during early exertion to angio-oedema, laryngeal oedema, bronchospasm, hypotension or collapse if exertion continues [12]. Importantly, peach Pru p 7 (GRP) FDEIA has been described in patients without Pru p 1 or Pru p 3 sensitisation, and 61.5% of Pru p 7-sensitised individuals required exercise or aspirin as a cofactor [7]. The case reported by Li et al. — anaphylaxis after ingestion of red fruit ginseng in a 35-year-old *Artemisia*-allergic woman — illustrates how rare cross-reactive components can produce severe outcomes that are easily overlooked [23].

Practical management for athletes includes: (i) avoidance of the culprit food for 4–6 hours before exercise, with most patients tolerating a 2-hour minimum; (ii) avoidance of cofactors, especially NSAIDs, alcohol and exercise on hot, humid days during the pollen season; (iii) identification of safe alternative foods to support energy intake; (iv) availability of an epinephrine autoinjector and a mobile phone in all training settings, with the carrier (athlete, teacher, coach or companion) clearly identified and trained in early recognition; and (v) explicit discussion of the “never push through” rule, which is particularly important in adolescent team sports where peer pressure may delay symptom recognition [12]. From a regulatory standpoint, second-generation H1-antihistamines, intranasal corticosteroids and pre-loaded adrenaline are permitted in competition under the World Anti-Doping Agency framework, whereas oral corticosteroids and beta-2-agonists may require therapeutic-use exemptions; team physicians should review medication lists in advance of the competitive season [13].

Table 2. Practical considerations for athletes with confirmed pollen-food allergy syndrome and/or food-dependent exercise-induced anaphylaxis.

Domain	Practical recommendation
Pre-exercise food avoidance	Avoid culprit food (often wheat, peach, tree nuts, soy beverage) for 4–6 hours before exercise; minimum 2 hours [12]. Consume cooked rather than raw forms of PR-10-driven culprits whenever possible [9].
Cofactor avoidance	Avoid NSAIDs, alcohol and intense exercise during peak pollen season; pay particular attention to hot, humid days. Beta-blockers and ACE-inhibitors may potentiate refractory anaphylaxis [9,12].
Pollen-season planning	Before outdoor competitions, screen for relevant pollen sensitisation; prepare with intranasal corticosteroids and second-generation antihistamines; consider AIT for severe seasonal symptoms [13].
Emergency preparedness	Carry an epinephrine autoinjector and a mobile phone at all training and competition sites; designate a trained companion (coach, teacher) [12].
Stop rule	"Never push through symptoms": any cutaneous, oropharyngeal or respiratory symptom during exertion mandates immediate cessation [12].
Anti-doping	Most rescue medications (epinephrine, H1-antihistamines, intranasal corticosteroids) are permitted; oral systemic corticosteroids and selected beta-2-agonists may require therapeutic-use exemption [13].

3.8. Environmental drivers — climate change, urbanisation and the changing pollen calendar

Pollen exposure is the upstream driver of class II food allergy, and both pollen quantity and pollen-season duration are changing under climate change [2,11]. Lake et al. modelled the future European burden of ragweed (*Ambrosia artemisiifolia*) sensitisation under two greenhouse-gas emission scenarios (RCP4.5 and RCP8.5) and three plant-invasion scenarios, using a process-based model on a 50 km × 50 km grid [11]. Their primary estimate suggests that the number of ragweed-sensitised individuals in Europe will more than double — from approximately 33 million at baseline to 77 million by 2041–2060 — driven 66% by climate, with the remainder attributable to ongoing plant invasion [11]. The largest proportional increases were predicted for Germany, France and Poland, where ragweed sensitisation is currently uncommon [11]. Higher pollen concentrations and longer pollen seasons are also expected to increase symptom severity [11].

In Japan, the prevalence of allergic rhinitis rose from 29.8% in 1998 to 39.4% in 2008 and 49.2% in 2019; nearly half (49.5%) of children aged 10–19 had hay fever in 2019 [2]. As PFAS prevalence tracks pollen-related sensitisation, the Kato review predicts further increases, particularly in children [2]. Plane tree allergy in urban

environments and grass-pollen polysensitisation also contribute to the dynamic landscape [6]. From a sport-medicine perspective, the practical implication is that future training and competition calendars should be planned with up-to-date pollen forecasts in mind, and that allergic athletes will require structured surveillance during increasingly long and intense pollen seasons [11,13].

4. Discussion

Pollen-food allergy syndrome is no longer a curiosity confined to allergologists. The sources synthesised in this review highlight three areas of active controversy. First, the boundary between OAS and PFAS remains terminologically contested. Whereas Faber and colleagues advocate abandoning the term OAS altogether — because oropharyngeal symptoms can occur without pollen sensitisation and PFAS can manifest beyond the oropharynx — the AAAAI Delphi panel still found broad consensus on retaining PFAS as the more accurate descriptor [6,9]. We endorse this stance; the umbrella term should remain PFAS, with OAS reserved for the symptom complex.

Second, the LTP and GRP syndromes challenge the classical class I/class II dichotomy. Pascal et al. showed that 75.6% of LTP-monosensitised patients in Catalonia experienced anaphylaxis, of whom one third required a cofactor [8]. Inomata et al. similarly reported anaphylactic shock in 31% of GRP-monosensitised Japanese fruit-allergic patients, with cofactor dependence in 62% [7]. Whereas the early descriptions of PFAS reported a benign syndrome confined to the oral mucosa, the LTP and GRP literature from the Mediterranean basin and Japan describes a systemic, cofactor-driven phenotype that arguably belongs to a different clinical category. Faber et al. argue that nsLTP allergy is no longer governed strictly by peach and is not confined to the Mediterranean basin [6]. These observations call for an integrated molecular nomenclature that complements rather than replaces the pollen-driven classification.

Third, the therapeutic landscape is in transition. Pollen AIT — long the most rational candidate to modify the natural history of PFAS — has produced inconsistent results, and the 2024 AAAAI Delphi panel did not endorse it as a primary indication for PFAS [9,25]. By contrast, food-specific molecular SLIT with rMal d 1 has demonstrated objective efficacy without systemic adverse events in a small but methodologically rigorous trial [17]. Meanwhile, omalizumab — newly approved for IgE-mediated food allergy on the basis of the OUtMATCH trial — opens a non-allergen-specific avenue for the most severe phenotypes [18]. These two strategies are not mutually exclusive; a future framework integrating CRD, molecular SLIT and biologicals appears plausible.

For sports medicine, the convergence of three trends — increasing pollen burden under climate change, increasing prevalence of allergic rhinitis and increasing recognition of cofactor-driven anaphylaxis — argues for systematic pre-season screening, individualised education and pre-competition planning [11,12,13]. The lack of dedicated trials of omalizumab or molecular SLIT in athletic populations is a clear research gap; so is the absence of validated PFAS-specific quality-of-life instruments for athletes.

This review has limitations. As a narrative synthesis, it does not include formal meta-analytic pooling or risk-of-bias scoring; readers seeking quantitative effect estimates for AIT should consult the systematic review by Kallen et al. [25]. The source articles were heterogeneous in design (case reports, cohort studies, position papers, randomised trials and systematic reviews), and several paediatric and adult cohorts had small sample sizes. Geographical bias is unavoidable: most evidence on LTP syndrome originates from southern Europe, and most on GRP syndrome from Japan, while data from Sub-Saharan Africa, Latin America and the Indian subcontinent are scarce.

5. Conclusions

PFAS is a heterogeneous, prevalent and clinically meaningful condition that integrates allergology, sports medicine, paediatrics and environmental health. Knowledge of the four pan-allergen families — PR-10 proteins, profilins, nsLTPs and GRPs — allows clinicians to predict the likely severity and the potential role of cofactors. Avoidance of the raw culprit food remains the cornerstone of management, but should be tailored to the individual patient and informed by component-resolved diagnosis. Pollen allergen immunotherapy is not currently endorsed as a primary indication for PFAS; food-specific molecular SLIT with recombinant Mal d 1 and the new role of omalizumab are the most promising therapeutic developments of the past decade. For athletes, systematic pre-season screening, careful identification of cofactors (especially exercise and NSAIDs) and individualised emergency planning are essential to prevent food-dependent exercise-induced anaphylaxis. As pollen seasons lengthen and ragweed and other invasive species spread across Europe, the relevance of PFAS to outdoor athletes can only grow. Future research should address head-to-head comparisons of biologicals, molecular SLIT and standardised AIT in PFAS, dedicated trials in athletic populations, and the underexplored role of GRP and minor pan-allergens in non-Mediterranean settings.

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