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# PATIENTS WITH POLLEN-FOOD ALLERGY ENCOUNTERS

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**Abstract.** In sensitized people, the interaction of a specific pollen allergen with a corresponding food allergen activates pollen-food allergy syndrome (PFAS). Although oral symptoms are theost prevalent signs, certain individualsmay also face extra symptoms like nasal congestion, skin reactions, breathing difficulties, or potentially fatal anaphylactic shock. The onset of pollen-food allergy syndrome (PFAS) entails mast cells in the oral mucosa that become sensitized to particular pollen antigens attached to IgE. These mast cells subsequently cross-react with food antigens, initiating a localized type I allergic response upon consuming foods with protein antigens (panallergens) that have structural resemblances to pollen antigens. The distribution of PFAS is affected by local differences in the types and amounts of pollen. Research on per- and polyfluoroalkyl substances (PFAS) is a global effort due to the prevalence of different pollen allergens like alder and grass associated with this condition. This review explores the recent discoveries regarding the pathophysiology, epidemiology, and clinical features of PFAS. We also emphasize important research voids that need to be filled to enhance patient outcomes for individuals impacted by PFAS

**Key words:** Pollen-food allergy, ethiology, pathogenesis, cross-reaction, clinical types

The characteristic of pollen-food allergy syndrome (PFAS) is sensitivity to pollen antigens, which causes swelling of the lips and oral mucosa, as well as tingling and itching shortly after consuming the trigger food. Respiratory problems, sinus irritation, and stomach troubles may occur on rare occasions. Nonetheless, severe cases rarely include systemic symptoms such as anaphylaxis, skin eruptions, vomiting, and wheezing. PFAS have become more prevalent in recent years, and clinicians frequently encounter them in their job. Additionally, oral allergy syndrome (OAS), an immediate-type allergy that relies on IgE and causes symptoms in the oral mucosa in response to a dietary antigen, includes PFAS as a subtype.

As a result, while not all PFAS symptoms are OAS symptoms, the majority are.

The Japanese Guidelines for Food Allergy 2020 divide food allergies into four clinical types: neonatal and infantile gastrointestinal allergies, special type food allergies (such as food-dependent exercise-induced anaphylaxis [FDEIA] and oral allergy syndrome [OAS]), and immediate-type food allergies (such as urticaria and anaphylaxis). 1. In people with immediate-type food allergies, the gastrointestinal tract becomes sensitized to proteins that are difficult to digest. Because the triggering and sensitizing antigens are the same, ingesting the sensitizing allergen results in allergy symptoms. The patient becomes sensitive to allergens inhaled, such as pollen antigens, in the case of PFAS, a special type of food allergy, and similar molecules in plant-based foods react with these allergens to cause the allergy.

When people who are sensitive to certain pollen allergens come into touch with the matching specific food antigens, they develop symptoms of PFAS. [2]

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These reactions depend on how similar the primary allergen components are to one another.
The pathophysiology of PFAS is often mediated by pan-allergens, which are commonly present in a broad range of foods and pollens. Plant survival depends on basic proteins known as pan-allergens; as a result of the genes encoding these proteins being conserved over time, a variety of plants have developed cross-reactivity and allergic reactions. Pathogen-related (PR) protein-10/Bet v 1-related proteins, lipid transfer proteins (LTPs), profilin, and other pan-allergens are examples of antigen families. The majority of PFAS symptoms are caused by type I allergic reactions that are exclusive to the oral mucosa. Patients experience swelling and itching of the tongue, lips, oral mucosa, and pharyngeal mucosa within an hour of consuming the trigger food. The symptoms that PFAS can cause include those related to the nose (nasal obstruction, rhinorrhea, sneezing), eyes (tearing, conjunctival hyperemia, itching), skin (facial swelling, itching, generalized rash), digestive system (abdominal pain, vomiting, diarrhea), respiratory system (hoarseness, wheezing, dyspnea), and, in rare cases, anaphylaxis. [2] Symptoms usually appear when raw food is consumed. Since soy milk, a processed soybean product, is often not heat treated for more than 30 minutes during processing, consuming it raw can result in anaphylaxis. This preserves Gly m4's antigenicity. Bean sprouts, a vegetable made from sprouted soybeans, should also be handled carefully because few cooking methods call for heat treatment for more than 30 minutes. Systemic symptoms are influenced by the allergens' stability in the associated diet and pollen. There are many different allergen components in food, and each one has a different tolerance for heat and digestion.

In a similar vein, Pur p 3 in peaches is LTP and likely to cause systemic symptoms, but Pur p 1 in peaches is PR-10 and unlikely to do so. Furthermore, GRP, which is also very stable like LTP, is Pur p 7 in peaches. 30, 32LTP is present in a number of pollens, such as mugwort (Art v 3) and ragweed (Amb a 6). Sensitization to specific pollen-derived LTPs and GRPs may cause cross-reactivity among homologous proteins of food origin and increase symptom severity.

The number of patients with allergic rhinitis is increasing worldwide. In Europe, climate change has led to an increase in the amount and duration of birch pollen dispersal, increasing the prevalence of birch pollen sensitization.[4] In Japan, allergic rhinitis prevalence was 29.8%, 39.4%, and 49.2% in 1998, 2008, and 2019, respectively, and it continues to increase, with nearly half (49.5%) of children aged 10–19 years developing hay fever in 2019. Although cedar pollen is the most predominant pollen antigen in Japan, non-cedar pollen allergies are also on the rise, contributing to an increased PFAS prevalence. In Japan, the birch's range is restricted to all of Hokkaido and the higher-elevation areas north of central Japan. Therefore, several patients with PFAS associated with birch pollinosis are found in Hokkaido. Notably, PFAS also occurs in areas where birch pollen dispersal is rarely observed. In a previous survey of 6824 outpatients with PFAS in Fukui Prefecture, where birch pollen is rarely dispersed, we found that PFAS prevalence was 10.8%, with *Cucurbitaceae* and *Rosaceae* foods. Serum antigen-specific IgE antibody positivity was significantly higher in the group of patients with birch exposure (PFAS group vs. controls, 31.7% vs. 8.6%), alder (17.7% vs. 2.0%) and timothy grass (31.7% vs. 19.9%). Furthermore, the main allergenic components of each pollen, Bet v1 (birch, 29.3% vs. 9.3%), Aln g1 (alder, 26.5% vs. 7.3%) and Phl p1 (timothy grass, 24.1% vs. 13.9%) were also significantly higher in patients with PFAS. This suggests that alder and timothy grass pollens are established causative pollens in Fukui Prefecture.[4] The alder belongs to the same family as birch and is common in deciduous forests widely distributed throughout Japan from Hokkaido to northern Kyushu. Bet v 1, the major allergen of birch pollen, and Aln g 1, the major allergen of alder pollen, have more than 80% protein homology; thus, PFAS is observed upon alder pollen sensitization even in areas without birch pollen. Cedar pollen is associated with

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tomatoes. Other pollinosis, such as alder pollinosis, frequently overlap with cedar pollinosis, and because tomatoes contain pan-allergens such Sola l 4 (PR-10) and Sola l 1 (profilin), PFAS could be produced by pollens other than cedar. [5] In a survey of over 40,000 US people, Gupta et al. found a 10.8% prevalence of food allergies. In a multicenter research conducted at nine sites in southern Europe, 167 (20.5%) of 815 individuals with seasonal allergic rhinitis aged 10 to 60 had PFAS, with prevalence ranging from 7.5% to 41.4%. Thus, PFAS incidence varies greatly due to regional variances in pollen sensitization patterns, which are determined by geographical factors such as pollen type and quantity. PFAS, a history of hypersensitivity symptoms in

A high index of suspicion is needed if complaints are caused by fruits (such as apples, melons, and kiwis), legumes (such as soybeans and bean sprouts), and vegetables (such as carrots and celery). Typically, symptoms appear only with raw foods and foods not thoroughly cooked through, within minutes after consumption, and are limited to oral lesions.[6] Although additional testing is not always required in patients with seasonal allergic rhinitis who report characteristic symptoms to typical raw food triggers, it is important to confirm sensitization to the specific pollen associated with the causative food. To definitively identify the causative food, a positive prick test result or detection of food-specific IgE in the serum is used. Regarding the prick test, commercial food extracts are not suitable as prick test antigens because the antigen is altered. The prick-to-prick test is performed by introducing a prick test needle into unprocessed food and then directly into the patient's skin.[5] If the prick test result is positive for raw fruits and vegetables but negative for cooked fruits and vegetables, PFAS can be diagnosed. The prick test provides rapid results with high sensitivity and specificity.[8] For several allergens, skin tests have been considered to have higher diagnostic sensitivity and specificity than serum-specific IgE tests.[6] However, because PFAS induces symptoms through cross-reactivity between pollen and food antigens, a negative result may be obtained if the causative antigen is present in trace amounts. The diagnosis can usually be made with a detailed clinical history, serum-specific IgE, and prick test. However, an oral challenge test may be performed if the history and diagnostic tests are inconclusive, or the patient's diet is unnecessarily restricted. Caution should be exercised in patients with a history of severe anaphylactic reactions to exceedingly small amounts of allergen exposure. PFAS is caused by cross-reactivity between pollen and food antigens and can occur in children and adults of all ages. An understanding that symptoms are mostly confined to the oral mucosa, but occasionally, respiratory, gastrointestinal, skin, or anaphylactic manifestations can be induced is important for its management. The most important criterion in diagnosing PFAS is a complete medical history; skin prick tests and serum-specific IgE are adequate diagnostics. In theory, avoiding foods that produce oral symptoms can help prevent the development of PFAS. However, because pan-allergens produce PFAS, once the disease develops, the number of foods that induce allergic reactions increases dramatically, making it difficult to eradicate the antigen. The etiology of PFAS is exacerbated by the various allergenic substances found in foods and pollen. As a result, having a good understanding of PFAS is critical for both diagnosis and counseling patients.

**References:**

1. D. Mittag, s. Vieths, l. Vogel, w.m. becker, h.p. rihs, a. Helbling, et al. Soybean allergy in patients allergic to birch pollen: clinical investigation and molecular characterization of allergens J allergy clin immunol, 113 (2004), pp. 148-154
2. P.l. amlot, d.m. kemeny, c. Zachary, p. Parkes, m.h. lessof oral allergy syndrome (oas): symptoms of ige-mediated hypersensitivity to foods Clin allergy, 17 (1987), pp. 33-42
3. G. Carlson, c. Coop pollen food allergy syndrome (pfas): a review of current available literature Ann allergy asthma immunol, 123 (2019), pp. 359-365

eISSN: 2349-5715 pISSN: 2349-5707

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4. M. Vanek-krebitz, k. Hoffmann-sommergruber, m. Laimer da camara machado, m. Susani, c. Ebner, d. Kraft, et al. Cloning and sequencing of mal d 1, the major allergen from apple (malus domestica), and its immunological relationship to bet v 1, the major birch pollen allergen Biochem biophys res commun, 214 (1995), pp. 538-551
5. M.h. shamji, r. Valenta, t. Jardetzky, v. Verhasselt, s.r. durham, p.a. würtzen,The role of allergen-specific ige, igg and iga in allergic disease Allergy, 76 (2021), pp. 3627-3641
6. T.e. velez, p.j. bryce, k.e. hulse Mast cell interactions and crosstalk in regulating allergic inflammation Curr allergy asthma rep, 18 (2018), p. 30