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REVIEWING POLLEN-FOOD ALLERGY SYNDROME IN DETAIL

Abstract. The cross-reaction of a particular pollen antigen with the associated food allergen in sensitized individuals results in pollen-food allergy syndrome (PFAS). Usually, only oral symptoms are present; nevertheless, on occasion, rhinitis, skin, and respiratory symptoms, as well as anaphylactic shock, might happen. In PFAS pathogenesis, mast cells attached to pollen antigen-specific IgE dispersed in the oral mucosa cross-react with the food antigen, resulting in a localized type I allergic reaction, when food containing protein antigens (pan-allergens) with high similarity to pollen antigens is consumed. Geographical factors, such as the kind and quantity of pollen in the region, influence the occurrence of PFAS. Because so many different pollen allergens, including alder and grass, are linked to the disease, PFAS is present around the world. The current state of research on the pathophysiology, epidemiology, and clinical aspects of per- and polyfluoroalkyl substances (PFAS) is reviewed in this study. We list the unmet research needs that need to be filled in order to help patients with PFAS have better outcomes.

Key words: Pollen-food allergy syndrome, antigen, symptoms, cross-reaction

The hallmark of pollen-food allergy syndrome (PFAS) is sensitivity to pollen antigens, which causes lips and mouth mucosal edema, numbness, and itching minutes after consuming the offending meal.
On rare occasions, gastrointestinal involvement, rhinitis, and respiratory symptoms may manifest. Rarely, though, systemic symptoms including anaphylaxis, skin rashes, vomiting, and wheezing accompany severe instances. The occurrence of PFAS has recently increased, and doctors routinely come into contact with it in their work. Furthermore, PFAS is a subtype of oral allergy syndrome (OAS), an immediate-type allergy that is IgE-dependent and causes symptoms to the oral mucosa in response to a food antigen. The theory behind OAS was first put forth in 1987 by Amlot et al., who noted that oral symptoms that develop shortly after consuming foods that test positive for prick tests can indicate the development of more severe symptoms.
Therefore, the majority of PFAS symptoms—though not all—are OAS symptoms.
Latex-fruit syndrome (LFS), which is caused by the cross-reactivity of latex antigens in natural rubber goods and plant food antigens such avocado, banana, chestnut, and kiwifruit, can cause allergic symptoms in those who have a latex allergy.[1]
Food allergies are categorized into four clinical types in the Japanese Guidelines for Food Allergy 2020: immediate-type food allergies (like urticaria and anaphylaxis), special type food allergies (like food-dependent exercise-induced anaphylaxis [FDEIA] and oral allergy syndrome [OAS]), and neonatal and infantile gastrointestinal allergies. 1. Sensitization to digestion-resistant proteins takes place in the gastrointestinal system in cases with immediate-type food allergies. As a result, eating the sensitizing allergen causes allergy symptoms because the triggering and sensitizing antigens are the same. However, in the case of PFAS, a unique kind of food allergy, the patient becomes sensitive to allergens inhaled, like pollen antigens, and comparable molecules in plant-based foods react with these allergens to produce the allergy. Foods like eggs, milk, wheat, and soybeans can cause an immediate-type food allergy. These frequently result in systemic symptoms like urticaria, diarrhoea, vomiting, and shock, and a lot of the allergens that cause these have heat and digesting resistance. Fruits and

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vegetables are foods that frequently contain PFAS. Furthermore, PFAS can result from grains and legumes like walnuts and soybeans that trigger immediate-type food allergies. An allergic reaction's induction is closely linked to mast cells. It takes ntigen-specific cross-reactivity for PFAS oral symptoms to manifest.

14–16 People who are sensitized to particular pollen allergens experience symptoms of PFAS when they come into contact with the corresponding specific food antigens.[2]

The degree of similarity between the main allergen components determines these reactions.

19 Pan-allergens, which are frequently found in a wide variety of foods and pollens, frequently mediate the pathogenesis of PFAS.20Basic proteins called pan-allergens are essential to plant survival; along the course of evolution, the genes encoding these proteins have been conserved across a wide range of plants, resulting in cross-reactivity and allergic reactions. Antigen families include profilin, lipid transfer proteins (LTPs), pathogen-related (PR) protein-10/Bet v 1-related proteins, and other types of pan-allergens. Oral mucosa-specific type I allergy reactions predominate in PFAS symptoms. Within an hour of eating the triggering food, patients have itching and swelling of the lips, tongue, oral mucosa, and pharyngeal mucosa. Nose (nasal obstruction, rhinorrhea, sneezing), ocular (tearing, conjunctival hyperemia, itching), skin (facial swelling, itching, generalized rash), digestive (abdominal pain, vomiting, diarrhea), respiratory (hoarseness, wheezing, dyspnea), and, in rare instances, anaphylaxis are among the symptoms that PFAS can cause.[2]

Usually, eating raw food causes symptoms to manifest. Antigenicity is decreased and symptoms are less likely to arise with heat treatment. Anaphylaxis is more common in patients with PFAS generated by LTPs and GRPs than it is in patients with PFAS caused by PR-10 family proteins andGly m4, a PR-10/Bet v 1-associated protein that is present in soybeans, is more likely to result in anaphylaxis than other PR-10/Bet v 1-associated proteins because it is comparatively more resistant to heat treatment and digestive enzymes.[3]

Anaphylaxis may occur if soy milk, a processed soybean product, is consumed raw since it is frequently not heat treated for longer than thirty minutes during processing. This leaves the antigenicity of Gly m4 intact. Similarly, as few cooking techniques require heat treatment for longer than 30 minutes, bean sprouts, a vegetable made from sprouted soybeans, should be taken with caution. The stability of allergens in the related food and pollen influences systemic symptoms. Food has a variety of allergen components, each of which has a varied heat and digestion tolerance. For instance, Mal d 3 in apples is LTP, which retains IgE reactivity even after heat treatment and is more likely to result in systemic symptoms than Mal d 1 in apples, which is PR-10, which seldom causes systemic symptoms.[3]

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

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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 tomatoes. However, other pollinosis, such as alder pollinosis, often overlap with cedar pollinosis, and because tomatoes have pan-allergens, such as Sola l 4 (PR-10) and Sola l 1 (profilin), PFAS may be caused by pollens other than cedar.[5] In the United States, Gupta et al. reported a prevalence of food allergy of 10.8% in a survey of over 40,000 U.S. adults. In a multicenter study at 9 sites in southern Europe, 167 (20.5%) of 815 patients with seasonal allergic rhinitis aged 10–60 years had PFAS, with prevalence varying significantly among centers, from 7.5% to 41.4%.Thus, PFAS prevalence varies widely owing to regional differences in pollen sensitization patterns, influenced by geographical conditions, such as pollen type and quantity. n PFAS, a history of hypersensitivity symptoms in the oral and pharyngeal mucosa upon ingestion of certain foods is essential in making a diagnosis. In several cases, PFAS is diagnosable from the clinical course. 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.[7] 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.[8] 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. Knowledge of a detailed history is the most important aspect for PFAS diagnosis, with skin prick tests and serum-specific IgE being adequate aids. In principle, to avoid developing PFAS, consumption of foods that induce oral symptoms must be avoided. However,

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since PFAS is caused by pan-allergens, once the disease develops, the number of foods that induce allergic symptoms increases drastically and antigen elimination may not be easily achievable. The various allergenic components found in foods and pollen antigens exacerbate the etiology of PFAS. Consequently, it's essential to have precise understanding of PFAS in order to diagnose and counsel patients.

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