JOURNAL OF EDUCATION, HEALTH AND SPORT
eISSN 2391-8306 · Open Access · Peer-reviewed
apcz.umk.pl/JEHS · Nicolaus Copernicus University in Toruń

Cite as: PŁACHTA, Weronika, SIERADZKA, Amelia, ZDRODOWSKA, Gabriela, WYRZYKOWSKA-KUCHARSKA, Julia, JECZKAŁO, Margarita, SHEVKOPLIAS, Yuliia and LITWIN, Kamil. Oral Allergy Syndrome: A Review of Epidemiology, Pathogenesis, and Current Management Strategies. Journal of Education, Health and Sport. 2026;93:72600. https://doi.org/10.12775/JEHS.2026.93.72600

ARTICLE TIMELINE
Received: 25.05.2026 Revised: 20.06.2026
Accepted: 20.06.2026 Published: 23.06.2026

INDEXING & EVALUATION
MEiN points: 40 Unique ID: 201159
Disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

The journal has been awarded 40 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32318). Unique Journal Identifier: 201159. Scientific disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

Punkty Ministerialne z 2019 – aktualny rok 40 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).

Oral Allergy Syndrome: A Review of Epidemiology, Pathogenesis, and Current Management Strategies

Weronika Płachta[1], ORCID

https://orcid.org/0009-0003-2460-9435

E-mail: weronikaplachta888@gmail.com

[1]Medical College, The Jan Kochanowski University in Kielce, 19A IX Wieków Kielc Street, 25-516 Kielce, Poland

Amelia Sieradzka[1], ORCID

https://orcid.org/0009-0005-9588-3065

E-mail: amelia.sieradzka@gmail.com

[1]Medical College, The Jan Kochanowski University in Kielce, 19A IX Wieków Kielc Street, 25-516 Kielce, Poland

Gabriela Zdrodowska[1], ORCID

https://orcid.org/0009-0001-5222-6015

E-mail: zdrodowskagabriela@gmail.com

[1]Medical College, The Jan Kochanowski University in Kielce, 19A IX Wieków Kielc Street, 25-516 Kielce, Poland

Julia Wyrzykowska-Kucharska[1], ORCID

https://orcid.org/0009-0008-9600-3703

E-mail: juliawyrzykowska8@gmail.com

[1]Medical College, The Jan Kochanowski University in Kielce, 19A IX Wieków Kielc Street, 25-516 Kielce, Poland

Margarita Jeczkało[1], ORCID

https://orcid.org/0009-0003-1103-090X

E-mail: echkalo2712@gmail.com

[1]Medical College, The Jan Kochanowski University in Kielce, 19A IX Wieków Kielc Street, 25-516 Kielce, Poland

Yuliia Shevkoplias[2], ORCID

https://orcid.org/0009-0003-8862-3391

E-mail: Juliashev@gmail.com

[2] Group of Medical Care Facilities, 20/22 Limanowskiego Street, 63-400 Ostrów Wielkopolski, Poland

Kamil Litwin[3,4], ORCID

https://orcid.org/0009-0004-6994-2802

E-mail: kamilicjusz@gmail.com

[3] Medical College, The Jan Kochanowski University in Kielce, Emergency Medicine Facility, Health Sciences Institute, 7 Radiowa Street, 25-317 Kielce, Poland

[4] Hospital of the Ministry of the Interior and Administration, Emergency Department, Paramedic, 51 Wojska Polskiego Street, 25-375 Kielce, Poland

Corresponding Author

Weronika Płachta

E-mail: weronikaplachta888@gmail.com

Oral Allergy Syndrome: A Review of Epidemiology, Pathogenesis, and Current Management Strategies

ABSTRACT

Background:

Oral Allergy Syndrome (OAS) is an immediate allergic reaction occurring in the oral mucosa caused by food allergens, especially in individuals allergic to pollen. This reaction is caused by cross-reactivity between pollen proteins and homologous proteins located in raw vegetables, fruits and nuts. It leads to symptoms localized in the oral cavity and throat, such as itching, tingling, and mild swelling of the lips, tongue, and palate. In most cases, symptoms are mild and short-lived; however, in some individuals, they may progress to more severe reactions, including systemic symptoms or even anaphylaxis.

Aims:

The purpose of this article is to provide a broad overview of the epidemiology, etiopathogenesis, clinical symptoms, diagnostic criteria and current treatment strategies related to OAS.

Methods:

A comprehensive literature review was conducted using PubMed databases. The search included keywords such as „oral allergy syndrome", „pollen–food allergy syndrome", „cross-reactivity", „anaphylaxis" and „IgE-mediated hypersensitivity". Relevant original studies, review articles, and clinical guidelines were included.

Conclusion:

OAS is often underdiagnosed, and, at times, it can lead to fatal consequences. It is therefore crucial to raise awareness and search for new effective methods of diagnostic approaches. Identifying cross-reactive allergens and implementing preventive measures can significantly reduce the risk of OAS episodes, particularly life-threatening anaphylaxis.

Key words: food allergy, allergic reaction, Pollen-food allergy syndrome, PFAS, OAS, cross reaction, IgE-mediated hypersensitivity, anaphylaxis

INTRODUCTION

Oral allergy syndrome (OAS) was firstly described by Amlot et al. [1] in 1987 as immediate allergic reaction which concerns the oral mucosa triggered by food antigens. This specific type of allergic reaction mainly occurs in people allergic to plant pollens. Symptoms are the result of a cross-reaction between proteins present in pollens and homologously similar proteins found in some raw vegetables, fruits, nuts or seeds [2,3,4,5,6]. For an allergic reaction occurrence, prior contact with the allergen with the participation of immunoglobulin E (IgE) is necessary [2,3,5]. Characteristic symptoms include itching, burning, tingling and swelling in the mouth, lips, tongue and throat, usually appearing immediately after eating raw food. In most patients, symptoms are mild and limited to the oral cavity, but in rare cases more severe systemic reactions such as anaphylaxis may occur [1,2,3,4,5,6]. Oral allergy syndrome affects a significant percentage of the population with pollen allergy, and its prevalence is increasing with increasingly better diagnostics and greater patient awareness [2,3,4,5,6]. Understanding the pathophysiological mechanisms and the development of diagnostic and therapeutic methods are crucial for effective treatment and improving the quality of life of patients affected by this condition.

METHODS

To ensure a thorough overview, we surveyed the literature via PubMed, targeting studies related to oral allergy syndrome and pollen–food allergy syndrome. We synthesized data from original research, review papers, and current clinical guidelines to inform this review.

EPIDEMIOLOGY

Oral Allergy Syndrome (OAS), also known as pollen-food allergy syndrome, is a condition resulting from cross-reactivity between pollen allergens and certain food proteins. This syndrome predominantly affects individuals with allergic rhinitis, particularly those sensitized to specific pollens [7].

The prevalence of OAS varies depending on geographic location and dietary habits. In Europe, especially among patients allergic to birch pollen, the most common symptom-triggering foods are apples, carrots, and hazelnuts. In Japan, studies have reported OAS in 10–15% of individuals with pollen allergies. These differences highlight the influence of local pollen exposure and regional dietary patterns on the development of the syndrome [8].

Demographic factors such as age and gender also play a significant role in the epidemiology of OAS. The syndrome most frequently affects young adults with a history of allergic diseases and is more common in women – the female-to-male ratio is approximately 2:1 [9].

The severity of other allergic diseases may influence the occurrence of OAS. The syndrome is more commonly observed in individuals with asthma and those with higher levels of specific IgE against birch pollen allergens and total IgE. Risk factors for developing OAS in birch pollen-sensitized individuals include female gender, adulthood, a long history of birch pollen allergy, a family history of atopy, and lack of desensitization with birch pollen vaccine [10].

POLLEN SOURCE	FRUIT	VEGETABLES	OTHERS
BIRCH	Apple Peach Cherry Almond	Celery Carrot Potatoe	Soybean Bean sprout Peanut Hazelnut
MUGWORT	Mango Kiwi	Celery Carrot	Peanut
RAGWEED	Melon Watermelon Banana	Zucchini Cucumber	-
TIMOTHY GRASS	Melon Watermelon Kiwi Orange	Potatoe Tomatoe Celery	Peanut

Table 1. Foods reported to be associated with pollen types in OAS [11].

COMPARISON

Compared to other allergic diseases, OAS demonstrates a unique epidemiological profile in terms of age, gender, and geographic distribution. Allergic rhinitis (AR) is significantly more common – affecting up to 18% of the adult population worldwide [12]. This condition often develops in childhood or adolescence, initially more frequently in boys, while in adulthood it is more prevalent in women, which mirrors the gender pattern observed in OAS [13]. While OAS symptoms are triggered by foods in pollen-sensitized individuals, AR is primarily induced by inhalant allergens. Notably, AR is a well-documented risk factor for the development of OAS [14].

Allergic asthma also shares an atopic background with OAS, although it differs in severity and epidemiological characteristics. According to WHO data, asthma affects over 262 million people globally, with the highest prevalence in highly developed countries. Asthma often begins in early childhood, initially more common in boys, though women predominate in adulthood [15].

Atopic dermatitis (AD), another related allergic disease, usually begins in early childhood and often precedes the development of AR or asthma as part of the so-called atopic march[16]. It is estimated that AD affects approximately 20% of children and 10% of adults, with the highest prevalence in industrialized countries [17]. In contrast to OAS, which usually appears later and requires prior sensitization to pollens, AD is often the first manifestation of atopy. Both conditions are associated with genetic and environmental factors, but AD is more strongly linked to skin barrier dysfunction, often due to filaggrin gene mutations [18].

In summary, OAS holds a unique place among allergic diseases – it mainly affects adults, particularly women, and requires prior exposure to pollen allergens. By comparison, AR, asthma, and AD usually develop earlier, vary in geographic distribution, and result from a broader spectrum of genetic and environmental factors.

ETHIOPATHOGENESIS

OAS is primarily observed in patients previously sensitized to inhalant allergens. Antigen-presenting cells process the allergen and present it to naive Th cells. These Th cells then differentiate into Th2 cells, which release cytokines such as IL-4, IL-5 and IL-13. IL-4 and IL-13 promote the production of allergen-specific IgE antibodies. IL-5 contributes to the recruitment and activation of eosinophils, which intensifies local inflammation. The IgE antibodies bind to mast cells and basophils, priming them for re-exposures. Subsequent contact to the allergen causes their degranulation and histamine release which results in localized allergic symptoms [19].

Proteins involved in the pathogenesis of OAS are categorized into three major protein superfamilies: the PR-protein family, the cupin superfamily, and the prolamin superfamily. The prolamin family is further divided into 2S albumins and lipid transfer proteins (LTPs) [20].

The PR-protein family plays a significant role in the pathogenesis of OAS, with the PR-10 subfamily being particularly important. One of the major birch pollen allergens, Bet v 1, is a PR-10 protein and serves as a primary sensitizer. Its homologous proteins in foods— Mal d 1 (apple), Pru p 1 (peach), Gly m 4 (soy)—are responsible for OAS symptoms in patients with birch allergy [21]. PR proteins are thermolabile so they loose their allergenic potential when exposed to heat [22].

LTPs (Mal d 3 in apple, Pru p 3 in peach) are heat- and digestion-resistant allergens capable of inducing severe reactions [20] .These proteins are recognized as important markers of OAS severity [22].

Another group within the prolamin family—the 2S albumins—play a limited role in OAS pathogenesis, as no aeroallergen has been found to act as a primary sensitizer [20].

The cupin family includes a diverse range of proteins, however, like 2S albumins, no aeroallergens have been identified as primary sensitizers [20].

Profilins cross-react mostly between pollens (Phl p 12 in grass, Amb a 8 in ragweed) and foods (Cuc m 2 in melon, Api g 4 in celery). Like PR-10 proteins, profilins are thermolabile and mostly cause localized symptoms. Relevant profilins include Ana c 1 (pineapple), Api g 4 (celery), Ara h 5 (peanut), Dau c 4 (carrot), and Cor a 2 (hazelnut) [20].

Recent studies suggests that the gut microbiota plays a significant role in modulating food allergen cross-reactivity reactions [23]. Dysbosis has been linked to increased allergic sensitivity, while presence of beneficial microbes (Lactobacillus, Bifidobacterium) promote Treg development and IgA production. Microbial metabolites, especially short-chain fatty acids reinforce epithelial tight junctions which prevents the antigens from translocating into the bloodstream. SCFAs also promote the differentiation of regulatory T cells which helps with sustaining the immune tolerance [24].

CLINICAL ASPECTS

OAS manifests in varying ways depending on the individual. The symptoms usually appear quickly after consuming foods that trigger the reaction, often within minutes, and are characterized by itching or a burning sensation in the lips, mouth, ears, and throat. In some instances, there may be redness around the mouth (perioral erythema) and widespread hives. Extra-oral symptoms may also presents as breathing difficulties, the development of a rash, or low blood pressure. Generally, the symptoms are mild and resolve within minutes to half an hour, though in some cases, more generalized

symptoms, such as nausea, vomiting, stomach pain, diarrhea, eczema, asthma, hives, swelling of the throat, or, in rare cases, anaphylaxis [25].

Pastorello et al. proposed a classification of OAS reactions into four levels of severity: (I) mild symptoms limited to the mouth, (II) both oral and gastrointestinal symptoms, (III) oral and systemic reactions like hives, swelling, nasal issues, and asthma, and (IV) life-threatening reactions such as swelling of the larynx and shock [25].

DIAGNOSTIC PROCESS

Current diagnostic approaches for OAS involve an in-depth medical history, skin prick testing (SPT), and serological testing for specific IgE (sIgE). OAS is generally diagnosed by taking a history from the relevant patients, performing a skin prick test, and measuring serum levels of allergen-specific IgE antibodies [26]. While SPT is commonly used, its reliability is limited due to variability in extract-based reagents. The gold standard for diagnosing food allergies in OAS patients remains the double-blind, placebo-controlled food challenge (DBPCFC), though it is contraindicated in individuals with a history of severe reactions. The basophil activation test (BAT) offers a functional assessment of allergen sensitivity and severity prediction, but its complexity restricts its use to specialized investigations. Serological testing with singleplex and multiplex systems provides a safe and accessible diagnostic tool, particularly when using purified allergenic molecules rather than extracts. This molecular-based approach enhances precision medicine by identifying specific allergens, assessing cross-reactivity, and predicting potential severity, thus enabling personalized patient management [23].

TREATMENT

Treatment of OAS requires a personalized approach, as the symptoms result from cross-reaction mechanisms between pollen and food allergens and may occur with different intensity in each patient. Strategy related to the management of OAS included prophylactic measures aimed at preventing symptom development, pharmacotherapy and immunotherapy. In most cases, OAS does not require the implementation of pharmacotherapy and can be well-controlled by providing effective patient education [27].

Preventing the development of OAS is primarily associated with avoiding allergen sources. However, this approach remains controversial, as allergic reactions often result from cross-reactivity with airborne allergens. Patient education plays a vital role, focusing on proper food preparation and the influence of thermal processing on allergenic potential. For example, cooked fruits or vegetables may be well-tolerated, whereas roasted peanuts often become more allergenic [27]. Patients should also be advised to carefully examine the labels of processed foods, as these may contain hidden allergens. These challenges can lead to excessive and unnecessary avoidance of various products due to concerns about allergic reactions, which can result in nutritional deficiencies and reduced quality of life. Therefore, it is recommended to adjust the strategy to the patient's individual needs, including severity of symptoms, dietary preferences and nutritional requirements [19].

Pharmacological treatment is recommended for patients with moderate, localized symptoms to shorten the duration of discomfort and improve quality of live. H1-receptor blockers or glucocorticosteroids can help alleviate symptoms such as pruritus and swelling. New generation antihistamines are typically used during the peak season for pollen allergies (when the risk of cross-reactivity is increased) or before consuming allergy-triggering foods. Non-sedating drugs such as loratadine, cetirizine or fexofenadine are preferred in management of OAS syndrome due to their safety profiles [19,28].

It should also be noted that comorbidities, such as bronchial asthma or atopic dermatitis, may contribute to a more severe course of food allergic reactions. Proper management and control of these conditions are essential in mitigating the severity of allergic symptoms [29].

The only causative treatment in allergies is allergen-specific immunotherapy (ASIT). The aim of this therapy is to induce immunological tolerance and provide anti-inflammatory effects by modifying the inflammatory mediators involved in allergic responses. While ASIT is a well-established and effective therapy for patients with inhalant allergies, there is currently insufficient evidence to support its use in patients with OAS caused by cross-reactivity with airborne allergens in the absence of respiratory symptoms [27,30,31]. However, in recent years ASIT has gained increasing relevance in management of pollen-food allergy syndrome, especially in cases where allergy symptoms significantly impaired patients' quality of life [19,30,32,33]. It should be emphasized that ASIT carries the risk of complications, such as localized skin reactions accompanied by pain and swelling in subcutaneous administration, and, in rare cases, life-threatening anaphylactic shock [34,35,36].

ANAPHYLAXIS

A growing body of research indicates that OAS can occur across all age groups, with a substantial proportion of affected adults experiencing systemic and anaphylactic reactions [37].

The most common triggers of anaphylaxis in OAS are peanut, followed by apple, walnut, pine nut, peach, and ginseng. A significant association was observed between anaphylaxis and sensitization to alder, hazel, willow, poplar, timothy, and ragweed [38].

In the treatment of anaphylactic shock, proper assessment of symptom severity is essential to initiate treatment promptly[39]. Early administration of epinephrine improves hospitalization rates and reduces mortality. The injection site for epinephrine is the mid-thigh's anterolateral aspect. The recommended dose is 0.01 mL/kg (0.01 mg/kg), with a maximum single dose of 0.5 mL (0.5 mg) for patients aged 12 years and older and 0.3 mL (0.3 mg) for those under 12 years [40]. If symptoms persist despite multiple intramuscular adrenaline injections (approximately three doses), continuous intravenous administration should be considered [41].

CONCLUSIONS

The literature analysis clearly indicates that oral allergy syndrome (OAS) constitutes a significant challenge in contemporary allergology. The results of this review suggest that differences in the regional prevalence of OAS are primarily due to the varied occurrence of airborne allergens. The pathophysiology of OAS based on thermolabile PR-10 proteins usually limits symptoms to the oral mucosa; however, reactivity to proteins resistant to digestion and temperature carries a real risk of systemic anaphylaxis. The evolution of diagnostic methods indicates the need to move away from classical skin tests.

In summary, OAS requires an integrated approach combining molecular allergology with personalized medicine, and future research should focus on developing standardized diagnostic protocols and assessing the long-term effectiveness of modern forms of immunotherapy in reducing food hypersensitivity, in order to improve patients' quality of life and ensure their safety.

Disclosure

Supplementary Materials

Not applicable.

Author Contributions

Conceptualization: Płachta Weronika, Sieradzka Amelia, Zdrodowska Gabriela, Wyrzykowska-Kucharska Julia, Litwin Kamil, Jeczkało Margarita, Shevkopila Yulia

Methodology: Płachta Weronika, Sieradzka Amelia, Zdrodowska Gabriela, Wyrzykowska-Kucharska Julia, Litwin Kamil, Jeczkało Margarita, Shevkopila Yulia

Formal analysis: Wyrzykowska-Kucharska Julia, Litwin Kamil, Jeczkało Margarita

Investigation: Płachta Weronika, Sieradzka Amelia, Zdrodowska Gabriela

Writing-rough preparation: Zdrodowska Gabriela, Wyrzykowska-Kucharska Julia, Shevkopila Yulia

Writing-review and editing: Płachta Weronika, Sieradzka Amelia, Zdrodowska Gabriela, Wyrzykowska-Kucharska Julia, Litwin Kamil, Jeczkało Margarita, Shevkopila Yulia

Visualization: Płachta Weronika, Sieradzka Amelia, Zdrodowska Gabriela, Wyrzykowska-Kucharska Julia, Litwin Kamil, Jeczkało Margarita, Shevkopila Yulia

Supervision: Płachta Weronika, Litwin Kamil

All authors have read and agreed to the published version of the manuscript.

Funding statement:

No financial support was received.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Our work did not involve direct human subject or obtaining their consent for participation in the study.

Data Availability Statement:

Since this is a review paper, our work does not contain new data or analyses.

Conflict Of Interest:

The authors declare no conflict of interes

REFERENCES

1. Amlot P, Kemeny D, Zachary C, Parkes P, Lessof M. Oral allergy syndrome (OAS): symptoms of IgE-mediated hypersensitivity to foods. Clin Allergy. 1987;17(1):33–42. DOI: 10.1111/j.1365-2222.1987.tb02317.x

2. Kiguchi T, Yamamoto-Hanada K, Saito-Abe M, Sato M, Irahara M, Ogita H, Yoshitsune Miyagi Y, Inuzuka Y, Toyokuni K, Nishimura K, Ishikawa F, Miyaji Y, Kabashima S, Fukuie T, Narita M,Ohya Y. Pollen-food allergy syndrome and component sensitization in adolescents: A Japanese population-based study. 2021. PLoS ONE 16(4): e0249649. DOI: 10.1371/journal.pone.0249649

3. Osawa Y, Ito Y, Takahashi N, Sugimoto C, Kohno Y, Mori S, Morikawa T, Kato Y, Okamoto M, Kanno M, Takabayashi T, Fujieda S, Epidemiological study of oral allergy syndrome in birch pollen dispersal-free regions. 2020; 69(2):246-52 DOI: 10.1016/j.alit.2019.09.008

4. Shrestha P, Diaz-Menindez M, Chiarella SE, Gonzalez-Estrada A, Oral allergy syndrome due to a hidden allergen in Mojo sauce. 2020; 124(6):623-24. doi: 10.1016/j.anai.2020.03.027

5. Ota M, NishidaY , Yagi H, Sato K, Yamada S, Arakawa Y, Takizawa T, Regional differences in the prevalence of oral allergy syndrome among Japanese children: A questionnaire-based survey ;41(2):142-49. DOI: 10.12932/AP-130120-0739

6. Matsumoto M, Takenaka M, Aoyagi K, Tomita Y, Arima K, Yamauchi-Takihara K, Murota H, Factors associated with the development of oral allergy syndrome: A retrospective questionnaire survey of Japanese university students. 2021; 70(4):458-62 DOI: 10.1016/j.alit.2021.02.003

7. Poncet P, Sénéchal H, Charpin D. Update on pollen-food allergy syndrome. Expert Rev Clin Immunol. 2020;16(6):561–78. https://doi.org/10.1080/1744666X.2020.1774366

8. Kim JH, Kim SH, Park HW, Cho SH, Chang YS. Oral Allergy Syndrome in Birch Pollen-Sensitized Patients from a Korean University Hospital. J Korean Med Sci. 2018 3;33(33):e218. https://doi.org/10.3346/jkms.2018.33.e218

9. Sedaghat AR, Kuan EC, Scadding GK. Epidemiology of chronic rhinosinusitis: prevalence and risk factors. J Allergy Clin Immunol Pract. 2022;10(6):1395–1403. https://doi.org/10.1016/j.jaip.2022.01.016

10. Baseggio Conrado A, Patel N, Turner PJ. Global patterns in anaphylaxis due to specific foods: a systematic review. J Allergy Clin Immunol. 2021;148(6):1515–23.e3. https://doi.org/10.1016/j.jaci.2021.03.048

11. Yukinori K, Taiyo M, Shigeharu F. Comprehensive review of pollen-food allergy syndrome: Pathogenesis, epidemiology, and treatment approaches, Allergology International 2025, 74(1), 42-50. https://doi.org/10.1016/j.alit.2024.08.007

12. Bousquet J, M van Hage Correction to: ARIA 2016: Care pathways implementing emerging technologies for predictive medicine in rhinitis and asthma across the life cycle. Clin Transl Allergy. 2016;10:60. https://doi.org/10.1186/s13601-016-0137-4

13. Pinart M, Benet M, Annesi-Maesano I, von Berg A, Berdel D, Carlsen KCL, Carlsen KH, Bindslev-Jensen C, Eller E, Fantini MP, Lenzi J, Gehring U, Heinrich J, Hohmann C, Just J, Keil T, Kerkhof M, Kogevinas M, Koletzko S, Koppelman GH, Kull I, Lau S, Melén E, Momas I, Porta D, Postma DS, Rancière F, Smit HA, Stein RT, Tischer CG, Torrent M, Wickman M, Wijga AH, Bousquet J, Sunyer J, Basagaña X, Guerra S, Garcia-Aymerich J, Antó JM. Comorbidity of eczema, rhinitis, and asthma in IgE-sensitised and non-IgE-sensitised children in the MeDALL population-based birth cohort studies. Allergy. 2014;75(3):679–91. https://doi.org/10.1016/S2213-2600(13)70277-7

14. Caliskaner Z, Naiboglu B, Kutlu A, Kartal O, Ozturk S, Onem Y, Erkan M, Gulec M, Colak C, Sener O. Risk factors for oral allergy syndrome in patients with seasonal allergic rhinitis 2011 1;16(3):e312-6. https://doi.org/10.4317/medoral.16.e312

15. Goshua A, Sampath V, Efobi JA, Nadeau K. The Role of Climate Change in Asthma 2023;1426:25-41. https://doi.org/10.1007/978-3-031-32259-4_2

16. Suwala Z, Marchewka u, Bieńskowski B, Feja K, Łuczak E, Jachowicz K, Petryla P, Ostafil K, Rektor K, Minknerm< Allergic march: a comprehensive approach to diagnosing, treating and preventing atopic diseases. Journal of Education, Health and Sport. 2023;45(1):388-402. https://doi.org/10.12775/JEHS.2023.45.01.028

17. Nutten S. Atopic dermatitis: global epidemiology and risk factors. Ann Nutr Metab. 2015;66 Suppl 1:8-16. https://doi.org/10.1159/000370220

18. Tsakok T, Woolf R, Smith CH, Weidinger S, Flohr C. Atopic dermatitis: the skin barrier and beyond. Br J Dermatol. 2019; 180(3):464-74. https://doi.org/10.1111/bjd.16934

19. Haidar L, Bănărescu CF, Uța C, Moldovan SI, Zimbru EL, Zimbru RI, Ciurariu E, Georgescu M, Panaitescu C. Pollen–Food Allergy Syndrome: Allergens, Clinical Insights, Diagnostic and Therapeutic Challenges. *Appl Sci*. 2025;15(1):66. https://doi.org/10.3390/app15010066

20. Carlson G, Coop C. Pollen food allergy syndrome (PFAS): A review of current available literature. *Ann Allergy Asthma Immunol*. 2019;123(4):359–65. https://doi.org/10.1016/j.anai.2019.07.022

21. Kato Y, Morikawa T, Fujieda S. Comprehensive review of pollen-food allergy syndrome: Pathogenesis, epidemiology, and treatment approaches. *Allergol Int*. 2025;74(1):42–50. https://doi.org/10.1016/j.alit.2024.08.007

22. Högerle C, San Nicolo M, Gellrich D, Eder K, Gröger M. Clinical relevance of profilin sensitization concerning oral allergy syndrome in birch pollen sensitized patients. *J Asthma Allergy*. 2022;15:249–55. https://doi.org/10.2147/JAA.S348650

23. Alessandri C, Ferrara R, Bernardi ML, Zennaro D, Tuppo L, Giangrieco I, Ricciardi T, Tamburrini M, Ciardiello MA, Mari A. Molecular approach to a patient's tailored diagnosis of the oral allergy syndrome. *Clin Transl Allergy*. 2020;10:22. https://doi.org/10.1186/s13601-020-00329-8

24. Oliva CT, Musa I, Kopulos D, Ardalani F, Maskey A, Wilson A, Yang N, Li X-M. The gut microbiome and cross-reactivity of food allergens: current understanding, insights, and future directions. Frontiers in Allergy 2025;4:1503380. https://doi.org/10.3389/falgy.2024.1503380

25. Kashyap RR, Kashyap RS. Oral allergy syndrome: an update for stomatologists. J Allergy (Cairo). 2015;2015:543928. http://doi.org/10.1155/2015/543928.

26. Kamei A, Izawa K, Ando T, Kaitani A, Yamamoto R, Maehara A, Ide T, Yamada H, Kojima M, Wang H, Tokushige K, Nakano N, Shimizu T, Ogawa H, Okumura K, Kitaura J. Development of mouse model for oral allergy syndrome to identify IgE cross-reactive pollen and food allergens: ragweed pollen cross-reacts with fennel and black pepper. Front Immunol. 2022;13:945222. https://doi.org/10.3389/fimmu.2022.945222.

27. Schmidt E. Diseases of the Oral Mucosa: Study Guide and Review. Springer Nature. 2022;431-32. https://doi.org/10.1007/978-3-030-82804-2.

28. Banerji A, Long AA, Camargo C. Diphenhydramine versus nonsedating antihistamines for acute allergic reactions: a literature review. Allergy Asthma Proc. 2007;28(4):418-426. https://doi.org/10.2500/aap.2007.28.3015.

29. Rymarczyk B, Rogala B. Food allergy. Lekarz POZ. 2018;4(5):396-405. Polish.

30. Kallen EJJ, Welsing PMJ, Löwik JM, Van Ree R, Knulst AC, Le TM. The effect of subcutaneous and sublingual birch pollen immunotherapy on birch pollen-related food allergy: a systematic review. Front Allergy. 2024;5:1360073. https://doi.org/10.3389/falgy.2024.1360073.

31. Rosada T, Ukleja-Sokołowska N, Bartuzi Z. The importance of specific immunotherapy in the treatment of food cross-reactions. Alergia Astma Immunologia. 2020;25(4):180-86. Polish.

32. de Silva D, Rodríguez Del Río P, de Jong NW, Khaleva E, Singh C, Nowak-Wegrzyn A, Muraro A, Begin P, Pajno G, Fiocchi A, Sanchez A, Jones C, Nilsson C, Bindslev-Jensen C, Wong G, Sampson H, Beyer K, Marchisotto MJ, Fernandez Rivas M, Meyer R, Lau S, Nurmatov U, Roberts G, GA2LEN Food Allergy Guidelines Group. Allergen immunotherapy and/or biologicals for IgE-mediated food allergy: A systematic review and meta-analysis. Allergy. 2022;77(6):1852-62. https://doi.org/10.1111/all.1521.

33. Hamada M, Kagawa M, Tanaka I. Evaluation of subcutaneous immunotherapy with birch pollen extract for pollen-food allergy syndrome. Asia Pac Allergy. 2021;19;11(4):e39. https://doi.org/10.5415/apallergy.2021.11.e39.

34. Besh OM, Besh DI, Sorokopud OO, Kondratiuk MO, Slaba OR. ASIT therapy: advantages and adverse effects. own results and literature data. Wiad Lek. 2018;71(2):341-45.

35. James C, Bernstein DI. Allergen immunotherapy: an updated review of safety. Curr Opin Allergy Clin Immunol. 2017;17(1):55-59. https://doi.org/10.1097/ACI.0000000000000335.

36. De Filippo M, Votto M, Caminiti L, Panasiti I, Carella F, De Castro G, Landi M, Olcese R, Vernich M, Marseglia GL, Ciprandi G, Barberi S. Safety of allergen-specific immunotherapy in children. Pediatr Allergy Immunol. 2022;33:27– 30. https://doi.org /10.1111/pai.13622.

37. Skypala IJ. Can patients with oral allergy syndrome be at risk of anaphylaxis? Curr Opin Allergy Clin Immunol. 2020;20(5):459–64. https://doi.org/10.1097/ACI.0000000000000679 .

38. Kim M, Ahn Y, Yoo Y, Kim DK, Yang HJ, Park HS, Lee HJ, Kim MA, Jeong YY, Kim BS, Bae WY, Jang AS, Park Y, Koh YI, Lee J, Lim DH, Kim JH, Lee SM, Kim YM, Jun YJ, Kim HY, Kim Y, Choi JH. Clinical manifestations and risk factors of anaphylaxis in pollen-food allergy syndrome. Yonsei Med J. 2019;60(10):960–68. https://doi.org/10.3349/ymj.2019.60.10.960 .

39. Babuska-Roczniak M, Wojtanowska-Kaczka M, Racz O, Marek H, Sośnicki K, Roczniak W, Proceedings of an emergency medical team (paramedics) with a patient in anaphylaxis. Journal of Education, Health and Sport.2020;10(6):108-116 https://doi.org/10.12775/JEHS.2020.10.06.012
40. Czyż R, Leśkiewicz M, Czyż I, Anaphylaxis: the current state of knowledge. Journal of Education, Health and Sport. 2018; 8(9):605-15 http://dx.doi.org/10.5281/zenodo.1412111
41. Ebisawa M, Ito K, Fujisawa T. Japanese guidelines for food allergy 2020. Allergol Int. 2020;69(3):370–86. https://doi.org/10.1016/j.alit.2020.03.004 .