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AN INVESTIGATIVE CASE STUDY ON ALLERGEN SPECTRUM AMONG PATIENTS IN WESTERN ROMANIA

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Abstract. *Immunoglobulin E (IgE)-mediated allergy is the most common form of hypersensitivity disorder, affecting approximately 30% of the global population. In atopic individuals, exposure to even trace amounts of allergens can trigger the production of IgE antibodies. Seasonal allergies and asthma represent a significant global health concern. The allergy panels consisted of various allergens, categorized as follows: respiratory, food, and mixed (containing both food and respiratory allergens). Food allergy is defined as an adverse immunological response to a dietary protein. Respiratory allergic disorders, including allergic rhinitis and allergic asthma, constitute major public health concerns, with a rising global prevalence. These conditions typically emerge in the spring, coinciding with the airborne dispersion of plant pollen. Among the inhalant allergens tested, pollen emerged as the most frequent trigger, indicating its prominent role in respiratory allergic responses. These findings may serve as a valuable starting point for evaluating the regional burden of allergic diseases and their major impact on public health. To gain a more comprehensive understanding of sensitization patterns and contributing environmental factors, further large scale, population based studies are warranted.The present study aims to conduct a case analysis focusing on the spectrum of allergens identified in patients from the western region of Romania.*

Keywords*:IgE-mediated allergy, food allergy, respiratory allergy, monosensitization, polisensitization*

INTRODUCTION

An allergen is defined as a protein or glycoprotein capable of binding to immunoglobulin E (IgE) (ALVAREZ-CUESTA ET AL., 2006). Exposure to trace amounts of an allergen may induce the production of IgE antibodies in atopic individuals. This phenomenon is known as allergic sensitization and typically occurs during early childhood (SHAMJI ET AL., 2021). The global prevalence of allergic diseases varies, affecting up to 30–40% of the population, with a marked increase observed among children and young adults (PAWANKAR, 2012).

IgE-mediated allergy is the most common form of hypersensitivity disorder, impacting approximately 30% of the population. Although IgE antibodies represent the least abundant immunoglobulin class in human serum, they possess the capacity to elicit strong inflammatory immune responses across various tissues and organs (ZELLWEGER & EGGEL, 2016). Repeated and subsequent exposures to the sensitizing allergen lead to increased levels of allergen-specific IgE (SHAMJI ET AL., 2021). Recent studies suggest that, in addition to the classical mechanisms of immediate hypersensitivity, complex immunological pathways are also involved, including specific T lymphocyte subtypes and pro-inflammatory cytokines (AKDIS, 2021).

The diagnosis of allergic conditions requires a thorough assessment of the patient's medical history alongside specific diagnostic tests. Once a diagnosis of food allergy is confirmed, complete elimination of the identified allergen is generally recommended. However, for certain allergies—such as those to cow's milk or eggs—consumption may be tolerated if the foods are heat-processed (NOWAK-WEGRZYN ET AL., 2016; WASERMAN ET AL., 2018). Over time, diagnostic test accuracy and reproducibility have significantly improved, enhancing both specificity and sensitivity (OZCAN ET AL., 2008).

The measurement of allergen-specific IgE antibodies can be conducted using either single-reactive systems (singleplex) or predefined panels encompassing multiple molecules tested simultaneously (multiplex) (LEE ET AL., 2009). Table 1 presents the key components involved in IgE antibody detection, as described by ANSOTEGUI ET AL. (2020).

Table 1

Testing process components

Components	Description
Reaction situs	Carbohydrate-based allergosorbents (e.g., agarose) may be used in testing procedures. A major innovation in this field is the use of a hydrophilic polymer encapsulated with covalently bound allergen
Allergen containing reageant	This represents the most complex component of the test, significantly impacting preparation, quality control, and validation processes
Allergen nature	Allergens may consist of crude extracts or single molecules obtained through recombinant DNA technology or biochemical purification.
Human sample	Both serum and plasma samples are suitable for testing.
Human anti-IgE detection reagents	Typically, polyclonal antibodies of animal origin (e.g., rabbit, sheep, or horse) are used.
Antibody markers and detection methods	The sensitivity of the assay is enhanced by the use of specific substrates.
Calibrating system	The definition of IgE antibody levels and the generation of a calibration curve are essential for quantitative analysis.
Buffer solution	The buffer system serves to normalize pH and provides an appropriate protein matrix.
Control samples	Positive and negative controls are required to validate the assay.
Data processing software	Finally, proper data analysis and interpretation ensure accurate diagnostic outcomes.

Seasonal allergies and asthma represent a significant global health concern. Data suggest that the prevalence of asthma—including forms triggered by pollen, mold, and other allergenic substances—is on an upward trajectory (BOUSQUET ET AL., 2017).

Food allergy is defined as an adverse immune response to a dietary protein. Reactions following food ingestion are associated with a wide array of signs and symptoms, which can affect any system in the body (KUZMINSKI ET AL., 2020). Among adults, approximately 10% suffer from food allergies, while the sensitization rate among children ranges from 3% to 10% (SANTOS ET AL., 2023). Although any food can potentially trigger an allergic reaction, certain food categories contain proteins with higher antigenic potential (LEHRER ET AL., 2002).

Cross-reactivity reflects phylogenetic relationships between organisms and is based on immunological recognition. Two allergens are considered cross-reactive when a single antibody or T lymphocyte is capable of reacting with both (CIOBANU & IANOVICI, 2024). Oral allergy syndrome, or pollen-food allergy syndrome, is a hypersensitivity reaction to specific foods that occurs as a result of previous exposure to inhalant plant allergens. The term “oral allergy syndrome” was first introduced by AMLOT ET AL. in 1987.

The present paper aims to conduct a case study focused on the spectrum of allergens identified in patients from the western region of Romania.

MATERIAL AND METHODS

Raw data was collected between January 4, 2021, and October 23, 2021. The primary material used was human serum, obtained by centrifuging whole blood at 4,000 revolutions per minute for 10 minutes. To obtain the raw data, an instrument employing Western blotting was used to detect IgE antibodies in the serum. The first step involved the preparation of solutions in five separate, sterilized glass containers, previously rinsed with distilled water. Following solution preparation, a work protocol was created using proprietary software integrated into the system connected to the analytical instrument. This protocol included the input of each serum code, the type of panel to be analyzed, and the corresponding panel code.

The allergy panels consisted of various allergens, categorized as follows: respiratory, food, and mixed (containing both food and respiratory allergens).

The studies presented in the speciality literature described a series of major importance allergens that can produce systemic reactions in patients. The following food allergens are known for causing hypersensitivity reactions: egg white (MINE, 2007), egg yolk (MINE & KOVACS-NOLAN, 2004), casein (RESTANI ET AL., 2009), wheat flour (MATSUO ET AL., 2004), gluten (BATTAIS F ET AL., 2005), strawberries (ANINOWSKI ET AL., 2020), rice (RICCARDO ET AL., 2007), soy (WANG ET AL., 2023), sesame seeds (ADATIA ET AL., 2017), peanuts (SICHERER & SAMPSON, 2007), cow's milk (ZEPEDA-ORTEGA ET AL., 2021), hazelnuts (CALLAMELI ET AL., 2021), pistachios (COSTA ET AL., 2019), apple (SIEKIERZYNSKA ET AL., 2021), UHT cow's milk (GEISELHART ET AL., 2021), tomato (FOETISCH ET AL., 2001), sunflower seeds (UKLEJA-SOKOŁOWSKA ET AL., 2016), pumpkin seeds (PATEL & BAHNA, 2016), cocoa (LOPES ET AL., 2019), green beans (PASTORELLO ET AL., 2010), banana (GROB ET AL., 2002), kiwi (FERNÁNDEZ-RIVAS, 2015), mulberries (PAPIA ET AL., 2020), beef and lamb (WILSON & PLATTS-MILLS, 2018), fig (CAIAFFA ET AL., 2003), orange (AHRAZEM ET AL., 2005), carrot (SCHIAPPOLI ET AL., 2002), potato (ANSARI & MU, 2018), peach (INOMATA ET AL., 2014), cherry (FUCHS ET AL., 2006), onion (ARMENTIA ET AL., 2020), olives (ESTEVE ET AL., 2012), mixed fish (SHARP & LOPATA, 2014), seafood (DAVIS ET AL., 2020), and chicken meat (HEMMER ET AL., 2016).

For the respiratory allergies, studies also suggest that the following allergens should be included: birch pollen (RAITH & SWOBODA, 2023), oak and cypress pollen (D'AMATO ET AL., 2007), olive pollen (CARNÉS ET AL., 2002), plane tree pollen (RODRÍGUEZ-RAJO ET AL., 2006), ash tree pollen (GASSNER ET AL., 2018), timothy grass (DE AMICI ET AL., 2010), ryegrass (RAWLS ET AL., 2020), wheat (CIANFERONI, 2016), meadow grass (HRABINA ET AL., 2008), cultivated rye (ŻUKIEWICZ-SOBCZAK et al., 2013), plantain (GADERMAIER ET AL., 2014), mugwort (TANG ET AL., 2015), wall pellitory (CIPRANDI ET AL., 2018), *Ambrosia elatior* (FLORINCESCU-GHEORGHE ET AL., 2019), cat dander and hair, dog hair (DÁVILA ET AL., 2018), rabbit dander (CHOI ET AL., 2007), *Aspergillus fumigatus* (SINGH ET AL., 2014), mold (TWAROCH ET AL., 2015), cockroach (POMES ET AL., 2017), *Dermatophagoides pteronyssinus* (VIDAL ET AL., 2016), *Dermatophagoides farinae* (AN ET AL., 2013), and flour mite (SUESIRISAWAD ET AL., 2015).

Both the food and respiratory allergens previously mentioned were analyzed in this study. They were contained in 3 different allergy panels, as following: food allergy panel, inhalatory panel and mixed panel. The mixed panel, including both food and respiratory allergens, contains: celery (BALLMER-WEBER ET AL., 2000), soy, peanuts, peas (BINDSLEV-JENSEN ET AL., 2004), wheat flour, shrimp (PASCAL ET AL., 2015), codfish (POULSEN ET AL., 2021), egg white, hazelnuts, kiwi, cow's milk, apple, olive pollen, birch pollen, plantain, cypress (CHARPIN ET AL., 2013), wall pellitory, oak, timothy grass, common ragweed, ash,

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mugwort, cat dander, dog hair, *Aspergillus fumigatus*, mold, *Dermatophagoides pteronyssinus*, and *Dermatophagoides farinae*.

Each serum sample was numbered from 1 to 152, and patient gender and age were recorded. The protocol was then uploaded to the system, which interfaced with secondary software for component verification, integrity check, and protocol execution. Tests were considered valid if the control band changed color to black upon substrate exposure. After completion of the protocol, the strips were scanned and results were obtained, indicating the IgE antibody titers and corresponding clinical class.

RESULTS AND DISCUSSIONS

The data was entered and processed using Microsoft Excel. In the first stage, the 152 patients were compiled into a centralized table. The table includes each patient's age, gender, the type of panel analyzed, each allergen tested, and the clinical classification based on IgE titers. A legend was then created to define the six possible antibody classes, the titer range for each, and its clinical interpretation. This legend is presented in Table 2. Of the 152 patients, 117 opted for the respiratory panel, 29 for the mixed panel, and 16 for the food panel.

Table 2

Legend

Class	Value	Interpretation
Class 0	<0.35 kU/l	No specific antibodies detected
Class 1	0.36-0.70 kU/l	Very low specific antibodies titer detected. Mainly without clinical sympomts
Class 2	0.71-3.50 kU/l	Low titer of specific antibodies – Clinical symptoms possible, particularly as values approach the upper limit of the range.
Class 3	3.51-17.50 kU/l	Moderately increased titer of specific antibodies – Clinical symptoms usually present.
Class 4	17.51-50.00 kU/l	Elevated titer of specific antibodies – Clinical symptoms almost always present.
Class 5	50.00-100 kU/l	Very high titer of specific antibodies.
Class 6	>100 kU/l	Extremely high titer of specific antibodies.

In the following stage, the number of values corresponding to each class was centralized for each allergen of the three panels, along with the total number of positive and negative patients. For the inhalatory panel, pollen-type allergens were separated from other types.

Table 3

Pollen allergens from the inhalatory panel

	Birch	Oak	Olive	Plane tree	Cypress	Ash	Timothy grass	Ryegrass	Wheat	Meadow gras	Cultivated rye	Plantain	Mugwort	*Parietaria officinalis*	*Ambrosia elatior*
Class 0	107	101	99	101	109	91	62	83	78	61	65	100	87	109	18
Class 1	4	4	4	5	4	10	6	4	11	4	13	5	9	3	11
Class 2	2	5	7	4	1	4	16	11	11	14	11	4	9	2	11
Class 3	1	5	4	5	3	8	14	13	9	10	9	6	4	2	22
Class 4	2	1	3	1	0	3	5	3	3	12	12	2	5	0	31
Class 5	0	1	0	1	0	1	10	3	5	15	6	0	3	1	24

Class 6	1	0	0	0	0	0	4	0	0	1	1	0	0	0	0
Total positive patients	10	16	18	16	8	26	55	34	39	56	52	17	30	8	99
Total negative patients	107	101	99	101	109	91	62	83	78	61	65	100	87	109	18

Table 3 presents the pollen-type allergens from the inhalatory panel. The highest number of positive values was recorded for *Ambrosia elatior* pollen, with 99 positive cases out of a total of 107. Of these, 31 patients fell into Class 4, 24 into Class 5, 22 into Class 3, and 11 patients each into Classes 1 and 2. No patients were classified in Class 6, and 18 were negative, being placed in Class 0. The next pollen type with a high number of sensitization reactions was *Poa pratensis*, with 56 positive cases: 15 in Class 5, 14 in Class 2, 12 in Class 4, 10 in Class 3, 4 in Class 1, and 1 in Class 6. A similarly high number of cases was found for *Phleum pratense* pollen, with 55 positive cases, the highest number being in Class 2. The most intense reactivity was recorded for *Ambrosia elatior*, with 31 patients in Class 5. Isolated sensitization cases were observed *for Cupressus sempervirens*, *Parietaria officinalis*, and *Betula pendula*.

Table 4

Inhalatory Panel Allergens

	Cat dander and hair	Dog hair	Rabbit dander	*Aspergillus fumigatus*	Mold	Cockroach	*D. pteronyssinus*	*D. farinae*	Flour mite
Values of Class 0	93	113	117	115	104	109	87	84	105
Values of Class 1	2	0	0	1	4	5	2	4	5
Values of Class 2	6	0	0	0	3	0	5	4	5
Values of Class 3	7	0	0	1	5	1	8	7	1
Values of Class 4	8	3	0	0	1	1	8	7	1
Values of Class 5	1	1	0	0	0	1	7	8	0
Values of Class 6	0	0	0	0	0	0	0	3	0
Total positive patients	24	4	0	2	13	8	30	33	12
Total negative patients	93	113	117	115	104	109	87	84	105

Table 4 displays the non-pollen inhalatory allergens. A high reactivity was recorded for *Dermatophagoides farinae*, with 33 positive cases: 3 in Class 6, 8 in Class 5, 7 each in Classes 3 and 4, and 4 in Class 1. Isolated cases were observed for allergens such as dog dander and *Aspergillus fumigatus*.

Table 5

Food allergens from the mixed panel

	Celery	Soy	Peanuts	Peas	Wheat flour	Shrimp	Codfish	Cow milk	Egg white	Hazelnuts	Kiwi	Apple
Values of Class 0	20	25	23	25	23	26	29	27	24	24	28	25
Values of Class 1	6	2	4	3	5	1	0	2	2	4	1	1
Values of Class 2	3	2	1	1	1	2	0	0	1	0	0	2
Values of Class 3	0	0	0	0	0	0	0	0	0	1	0	1
Values of Class 4	0	0	0	0	0	0	0	0	2	0	0	0
Values of Class 5	0	0	1	0	0	0	0	0	0	0	0	0
Values of Class 6	0	0	0	0	0	0	0	0	0	0	0	0
Total positive patients	9	4	6	4	6	3	0	2	5	5	1	4
Total negative patients	20	25	23	25	23	26	29	27	24	24	28	25

Table 5presents the food allergens included in the mixed panel. Except for codfish, which did not present any positive values, isolated sensitization cases were observed across all other food allergens.

Table 6

Respiratory allergens from the Mixed Panel

	Birch	Olive	Oak	Ash	Cypress	Timothy grass	Platain	*Ambrosia*	Wormwood	*P. officinalis*	Cat dander and hair	Dog hair	*A.fumigatus*	*A. alternata*	*D. pterynossinus*	*D.farinae*
Values of Class 0	23	19	22	21	25	16	23	11	20	22	24	25	28	27	25	21
Values of Class 1	1	6	3	2	2	1	1	4	2	2	2	1	0	1	1	1
Values of Class 2	3	1	4	4	2	5	4	4	2	4	0	1	0	1	1	4
Values of Class 3	0	1	0	1	0	3	1	3	1	1	1	0	1	0	1	0
Values of Class 4	1	1	0	1	0	4	0	5	2	0	2	0	0	0	0	2
Values of Class 5	1	1	0	0	0	0	0	1	2	0	0	2	0	0	1	1
Values of Class 6	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
Total positive patients	6	10	7	8	4	13	6	18	9	7	5	4	1	2	4	8
Total negative patients	23	19	22	21	25	16	23	11	20	22	24	25	28	27	25	21

Table 6 refers to aeroallergens from the mixed panel. As in the case of the respiratory panel, high reactivity was observed for Ambrosia, with 18 positive cases out of 29: 1 in Class 6, 1 in Class 5, 5 in Class 4, 3 in Class 3, 4 in Class 2, and 4 in Class 1. The next most reactive allergen was *Phleum pratense*, with 13 positive cases, most of which fell into Class 2.

Table 7

Selection of allergens with positive values contained in the Food Panel

	Egg white	*UHT Cow milk*	*Sesame seeds*	*Peanuts*	Apple	Carrot	Peach	Cherry	Seafood	Chicken meat
Values of Class 0	13	13	15	14	15	15	15	15	14	15
Values of Class 1	1	2	1	2	0	0	1	1	0	1
Values of Class 2	2	1	0	0	1	1	0	0	2	0
Values of Class 3	0	0	0	0	0	0	0	0	0	0
Values of Class 4	0	0	0	0	0	0	0	0	0	0
Values of Class 5	0	0	0	0	0	0	0	0	0	0
Values of Class 6	0	0	0	0	0	0	0	0	0	0
Total positive patients	3	3	1	2	1	1	1	1	2	1
Total negative patients	13	13	15	14	15	15	15	15	14	15

Table 7includes a selection of food allergens with positive values from the corresponding panel. A low level of reactivity was noted among food allergens, with a maximum of 3 positive patients for egg white and UHT cow's milk, all in Classes 1 and 2.

Out of the 152 patients analyzed, most were in the 31–40 age group, followed by the 1–10 and 21–30 groups. In terms of gender, 85 were male and 67 female. Sensitization was slightly more prevalent in women (95.50%) than in men (89.40%). Among sensitized patients, polysensitization was predominant in both genders—77.60% in males and 73.43% in females—while monosensitization accounted for 25.30% in males and 26.56% in females. Overall, 75.70% of all sensitized individuals were polysensitized. These findings are consistent with literature indicating a higher prevalence of polysensitization among patients with allergic diseases, reported in 50–80% of moderate-to-severe respiratory allergy cases (VALENTA ET AL., 2012). The predominance of *Ambrosia artemisiifolia* as a seasonal allergen, especially in Central and Eastern Europe, and the major role of house dust mites as perennial indoor allergens are well established (BUTERS ET AL., 2018; CASSET ET AL., 2023; SCHÜLKE & SCHÜLKE, 2022). These results reinforce the relevance of the current study's findings and contribute to a more comprehensive understanding of the allergen sensitization landscape in the western region of Romania.

CONCLUSIONS

The results of this study revealed a higher sensitization rate among female patients compared to males. Furthermore, polysensitization was more common than monosensitization, highlighting the complex nature of allergen exposure. Among the inhalant allergens tested, pollen emerged as the most frequent trigger, indicating its prominent role in respiratory allergic responses. These findings may serve as a valuable starting point for evaluating the regional burden of allergic diseases and their impact on public health. To gain a more comprehensive understanding of sensitization patterns and contributing environmental factors, further large-scale, population-based studies are warranted.

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BIBLIOGRAPHY

1.ADATIA, A. ET AL. (2017). Sesame allergy: current perspectives. Journal of Asthma and Allergy, 10, 141–151.
2.AHRAZEM, O. ET AL. (2005). Lipid transfer proteins and allergy to oranges. International Archives of Allergy and Immunology, 137(3), 201–210.
3.AKDIS, C. A. (2021), Mechanisms of allergen-specific immunotherapy and novel ways for vaccine development, The Journal of Allergy and Clinical Immunology, 147(6): 1667–1678.
4.ALVAREZ-CUESTA, E., BOUSQUET, J., CANONICA, G. W., DURHAM, S. R., MALLING, H. J., VALOVIRTA, E. (2006), Standards for practical allergen-specific immunotherapy, Allergy, 61(82): 1–20.
5.AMLOT, P. L., KEMENY, D. M., ZACHARY, C., et al. (1987), Oral allergy syndrome (OAS): symptoms of IgE mediated hypersensitivity to foods, Clinical Allergy, 17(1): 33–42.
6.AN, S., CHEN, L., LONG, C., LIU, X ET AL. (2013). Dermatophagoides farinae allergens diversity identification by proteomics. Molecular & Cellular Proteomics, 12(7), 1818–1828.
7.ANINOWSKI, M. ET AL. (2020). Evaluation of the potential allergenicity of strawberries in response to different farming practices. Metabolites, 10(3), 102.
8.ANSARI, I. T., & MU, T. (2018). A murine model of wheat versus potato allergy: Patatin and 53kDa protein are the potential allergen from potato. Molecular Immunology, 101, 284–293.
9.ANSOTEGUI, I. J., et al. (2020), IgE allergy diagnostics and other relevant tests in allergy, a World Allergy Organization position paper, The World Allergy Organization Journal, 13(2): 100080.
10.ARMENTIA, A. ET AL. (2020). Allergic hypersensitivity to garlic and onion in children and adults. Allergologia et Immunopathologia, 48(3), 232–236.
11.ASERO, R. ET AL. (2007). Rice: Another potential cause of food allergy in patients sensitized to lipid transfer protein. Int Arch Allergy Immunol, 143(1), 69–74.
12.BALLMER-WEBER, B. K. ET AL. (2000). Celery allergy confirmed. J Allergy Clin Immunol, 106(2), 373–378.
13.BALLMER-WEBER, B. K., et al. (2007), Clinical characteristics of soybean allergy in Europe: a double-blind, placebo-controlled food challenge study, Journal of Allergy and Clinical Immunology, 119(6): 1489–1496.
14.BINDSLEV-JENSEN, C. ET AL. (2004). Standardization of food challenges. Allergy, 59(7), 690–697.
15.BOUSQUET, J., et al. (2017), Allergic rhinitis and its impact on asthma (ARIA) 2016 revision, Journal of Allergy and Clinical Immunology, 140(4): 950–958.
16.BUTERS, J. T. M., ANTUNES, C., GALVEIAS, A., ET AL. (2018), Pollen and spore monitoring in the world, International Journal of Environmental Research and Public Health, 15(7): 1439.
17.CAIAFFA, M. F. ET AL. (2003). Fig and mulberry cross-allergy. Annals of Allergy, Asthma & Immunology, 91(5), 493–495.
18.CALAMELLI, E. ET AL. (2021). Hazelnut allergy. Medicina, 57(1), 67.
19.CARNES, J. ET AL. (2002). Olive pollen allergy. J Investig Allergol Clin Immunol, 12(2), 83–91.
20.CASSET, A., GALON, C., PONS, F., ET AL. (2023), Advances in house dust mite allergens and implications for diagnosis and treatment, *Molecular Immunology*, 161: 92–100.
21.CHARPIN, D., CALLEJA, M., PICHOT, C., PENEL, V., HUGUES, B., & PONCET, P. (2013). Cypress pollen allergy. Revue des Maladies Respiratoires, 30(10), 868–878.
22.CHOI, J. H., KIM, H. M., & PARK, H. S. (2007). Allergic asthma and rhinitis caused by household rabbit exposure: identification of serum-specific IgE and its allergens. Journal of Korean Medical Science, 22(5), 820.
23.CIANFERONI, A. (2016). Wheat allergy: diagnosis and management. Journal of Asthma and Allergy, 13–25.
24.CIOBANU, D.-G., IANOVICI, N. (2024), Overview of allergies with focus on causative agents, cross-reactivity, diagnostic methods and immunotherapy, Research Journal of Agricultural Science, 56(2): 31–39, Romania.

http://doi.org/10.59463/RJAS.2025.2.13
25.CIPRANDI, G., PUCCINELLI, P., INCORVAIA, C., & MASIERI, S. (2018). Parietaria allergy: an intriguing challenge for the allergist. Medicina, 54(6), 106.
26.COSTA, J. ET AL. (2019). Pistachio nut allergy: An updated overview. Critical Reviews in Food Science and Nutrition, 59(4), 546–562.
27.COSTA, J. ET AL. (2019). Pistachio nut allergy: An updated overview. Critical Reviews in Food Science and Nutrition, 59(4), 546–562.
28.D'AMATO, G. ET AL. (2007). Allergenic pollen and pollen allergy in Europe. Allergy, 62(9), 976–990.
29.D'AMATO, G. ET AL. (2007). Thunderstorm-asthma and pollen allergy. Allergy, 62(1), 11–16.
30.DÁVILA, I., ET AL. (2018). Consensus document on dog and cat allergy. Allergy, 73(6), 1206–1222.
31.DAVIS, C. M., GUPTA, R. S., AKTAS, O. N., DIAZ, V., KAMATH, S. D., & LOPATA, A. L. (2020). Clinical management of seafood allergy. The Journal of Allergy and Clinical Immunology: In Practice, 8(1), 37–44.
32.DE AMICI, M., ALESINA, R., MORATTI, R., & CIPRANDI, G. (2010). Component-resolved diagnosis for phleum allergy: the role of recombinants. Journal of Asthma, 47(7), 750–753.
33.ESTEVE, C. ET AL. (2012). Analysis of olive allergens. Talanta, 92, 1–14.
34.FERNÁNDEZ-RIVAS, M. (2015). Fruit And Vegetable Allergy. Chem Immunol Allergy, 101, 162–170.
FLORINCESCU-GHEORGHE, N. A., POPESCU, F., ALEXANDRU, D. O., & POPESCU, F. D. (2019). The prevalence of allergic rhinitis to Ambrosia elatior in Oltenia area and the association with allergic conjunctivitis or asthma. Current Health Sciences Journal, 45(1), 66.
35.FOETISCH, K. ET AL. (2001). Tomato (Lycopersicon esculentum) allergens in pollen-allergic patients. European Food Research and Technology, 213, 259–266.
36.FUCHS, H. C. ET AL. (2006). Natural and recombinant molecules of the cherry allergen Pru av 2 show diverse structural and B cell characteristics but similar T cell reactivity. Clinical & Experimental Allergy, 36(3), 359–368.
37.GADERMAIER, G., ET AL(2014). Plantago lanceolata: an important trigger of summer pollinosis with limited IgE cross-reactivity. Journal of Allergy and Clinical Immunology, 134(2), 472–475.
38.GASSNER, M., SCHMID-GRENDELMEIER, P., & CLOT, B. (2019). Ash pollen allergy and aerobiology. Allergo Journal International, 28, 289–298.
39.GEISELHART, S. ET AL. (2021). Cow's milk processing—friend or foe in food allergy? Foods, 10(3), 572.
40.GROB, M. ET AL. (2002). Heterogeneity of banana allergy: Characterization of allergens in banana-allergic patients. Annals of Allergy, Asthma & Immunology, 89(5), 513–516.
41.HEMMER, W., KLUG, C., & SWOBODA, I. (2016). Update on the bird-egg syndrome and genuine poultry meat allergy. Allergo Journal International, 25, 68–75.
42.HRABINA, M., JAIN, K., & GOUYON, B. (2008). Cross-reactivity between pollen allergens from common Pooideae grasses and cultivated cereals. Clinical & Experimental Allergy Reviews, 8(1), 18–20.
43.INOMATA, N. ET AL. (2014). Identification of peamaclein as a marker allergen related to systemic reactions in peach allergy. Annals of Allergy, Asthma & Immunology, 112(2), 175–177.
44.KUŹMIŃSKI, A., PRZYBYSZEWSKI, M., GRACZYK, M., ŻBIKOWSKA-GOTZ, M., SOKOŁOWSKA-UKLEJA, N., TOMASZEWSKA, A., BARTUZI, Z. (2020), Selected allergic diseases of the gastrointestinal tract, Przegląd Gastroenterologiczny, 15(3): 194–199.
45.LEE, S., LIM, H. S., PARK, J., KIM, H. S. (2009), A new automated multiple allergen simultaneous test-chemiluminescent assay (MAST-CLA) using an AP720S analyzer, Clinica Chimica Acta, 402(1–2): 182–188.
46.LEHRER, S. B., AYUSO, R., REESE, G. (2002), Current understanding of food allergens, Annals of the New York Academy of Sciences, 964(1): 69–85.
47.LOPES, J. P. ET AL. (2019). Not so sweet: True chocolate and cocoa allergy. The Journal of Allergy and Clinical Immunology: In Practice, 7(8), 2868–2871.

48.MATSUO, H. ET AL. (2004). IgE-binding epitope in omega-5 gliadin. Journal of Biological Chemistry, 279(13), 12135–12140.

49.MATSUO, H. ET AL. (2004). IgE-binding epitope in omega-5 gliadin. J Biol Chem, 279(13), 12135–12140.

50.MINE, Y. (2007). Egg proteins and peptides in human health--chemistry, bioactivity and production. Current Pharmaceutical Design, 13(9), 875–884.

51.MINE, Y.; KOVACS-NOLAN, J. (2004). New insights in the egg as a functional food. Journal of Functional Foods, 1(1), 4–12.

52.NOWAK-WEGRZYN, A., et al. (2016), Food protein-induced enterocolitis syndrome, The Journal of Allergy and Clinical Immunology: In Practice, 4(1): 24–31.

53.OZCAN, E., et al. (2008), Primary immune deficiencies with aberrant IgE production, Journal of Allergy and Clinical Immunology, 122(6): 1054–1062.

54.PAPIA, F. ET AL. (2020). Allergic reactions to genus Morus plants: A review. Clinical and Molecular Allergy, 18, 1–5.

55.PASCAL, M., GRISHINA, G., YANG, A. C., SÁNCHEZ-GARCÍA, S., LIN, J., TOWLE, D., ... & AYUSO, R. (2015). Molecular diagnosis of shrimp allergy: efficiency of several allergens to predict clinical reactivity. The Journal of Allergy and Clinical Immunology: In Practice, 3(4), 521–529.

56.PASTORELLO, E. A. ET AL. (2010). Green bean (Phaseolus vulgaris): A new source of IgE-binding lipid transfer protein. Journal of Agricultural and Food Chemistry, 58(7), 4513–4516.

57.PATEL, A., & BAHNA, S. L. (2016). Hypersensitivities to sesame and other common edible seeds. Allergy, 71(10), 1405–1413.

58.PAWANKAR, R. (2012), The unmet global health need of severe and complex allergies: meeting the challenge, The World Allergy Organization Journal, 5(2): 20–21.

59.POULSEN, L. K., HANSEN, T. K., NØRGAARD, A., VESTERGAARD, H., STAHL SKOV, P., & BINDSLEV-JENSEN, C. (2001). Allergens from fish and egg. Allergy, 56, 39–42.

60.RAITH, M.; SWOBODA, I. (2023). Birch pollen—The unpleasant herald of spring. Frontiers in Allergy, 4, 1181675.

61.RAWLS, M., THIELE, J., ADAMS, D. E., STEACY, L. M., & ELLIS, A. K. (2020). Clinical symptoms and biomarkers of Bermuda grass–induced allergic rhinitis using the nasal allergen challenge model. Annals of Allergy, Asthma & Immunology, 124(6), 608–615.

62.RESTANI, P. et al. (2009). Molecular aspects of milk allergens. Analytical and Bioanalytical Chemistry, 395(1), 47–56.

63.RODRIGUEZ-RAJO, F. J. et al. (2006). Plane tree pollen in NW Spain. Aerobiologia, 22(2), 93–100.

64.SANTOS, A. F., RIGGIONI, C., AGACHE, I., et al. (2023), EAACI guidelines on the diagnosis of IgE-mediated food allergy, Allergy, 78: 3057–3076.

65.SCHAFFNER, U., et al. (2020), Biological weed control to relieve millions from Ambrosia allergies in Europe, Nature Communications, 11(1745): 1–7.

66.SCHIAPPOLI, M. ET AL. (2002). Anaphylaxis due to carrot as hidden food allergen. Allergologia et Immunopathologia, 30(4), 243–244.

67.SCHÜLKE, S., & SCHÜLKE, N. (2022), House dust mite allergens: Role in allergic diseases and relevance for diagnosis and therapy, *Allergo Journal International*, 31(2): 46–54.

68.SHARP, M. F., & LOPATA, A. L. (2014). Fish allergy: In review. Clinical Reviews in Allergy & Immunology, 46, 258–271.

69.SICHERER, S. H.; SAMPSON, H. A. (2007). Peanut allergy: emerging concepts. J Allergy Clin Immunol, 120(3), 491–503.

70.SIDLAUSKAS, G., BERNOTAS, S. (2003), Some factors affecting seed yield of spring oilseed rape (*Brassica napus L.*), Agronomy Research, 1(2): 229–243, Lithuania.

71.SIEKIERZYNSKA, A. ET AL. (2021). Apple allergy: Causes and factors influencing fruits allergenic properties–Review. Clinical and Translational Allergy, 11(4), e12032.

72.SINGH, B., SINGH, S., R ASIF, A., OELLERICH, M., & L SHARMA, G. (2014). Allergic aspergillosis and the antigens of Aspergillus fumigatus. Current Protein and Peptide Science, 15(5), 403–423.

73.SUESIRISAWAD, S., MALAINUAL, N., TUNGTRONGCHITR, A., CHATCHATEE, P., SURATANNON, N., & NGAMPHAIBOON, J. (2015). Dust mite infestation in cooking flour: experimental observations and practical recommendations. Asian Pacific Journal of Allergy and Immunology, 33(2), 123–128.

74.TANG, R., SUN, J. L., YIN, J., & LI, Z. (2015). Artemisia allergy research in China. BioMed Research International, 2015(1), 179426.

75.TWAROCH, T. E., CURIN, M., VALENTA, R., & SWOBODA, I. (2015). Mold allergens in respiratory allergy: from structure to therapy. Allergy, Asthma & Immunology Research, 7(3), 205–220.

76.UKLEJA-SOKOLOWSKA, N. ET AL. (2016). Sunflower seed allergy. International Journal of Immunopathology and Pharmacology, 29(3), 498–503.

77.VALENTA, R., HOFER, G., ROESCH, A., ET AL. (2012), The recombinant allergen-based concept of component-resolved diagnostics and immunotherapy (CRD and CRIT), *Clinical and Experimental Allergy*, 42(3): 385–397.

78.VIDAL, C., LOJO, S., JUANGORENA, M., & GONZALEZ-QUINTELA, A. (2016). Association between asthma and sensitization to allergens of Dermatophagoides pteronyssinus. Journal of Investigational Allergology & Clinical Immunology, 26(5), 304–309.

79.WANG, J. et al. (2023). Soybean allergy. Crit Rev Food Sci Nutr, 63(23), 6182–6195.

80.WILSON, J. M., & PLATTS-MILLS, T. A. (2018). Meat allergy and allergens. Molecular Immunology, 100, 107–112.

80.ZELLWEGER, F., EGGEL, A. (2016), IgE-associated allergic disorders: recent advances in etiology, diagnosis, and treatment, Allergy, 71: 1652–1661.

81.ZEPEDA-ORTEGA, B. ET AL. (2021). Strategies and future opportunities for the prevention, diagnosis, and management of cow milk allergy. Frontiers in Immunology, 12, 608372.

82.ŻUKIEWICZ-SOBCZAK, W. A., CHOLEWA, G., KRASOWSKA, E., CHMIELEWSKA-BADORA, J., ZWOLIŃSKI, J., & SOBCZAK, P. (2013). Rye grains and the soil derived from under the organic and conventional rye crops as a potential source of biological agents causing respiratory diseases in farmers. Advances in Dermatology and Allergology/Postępy Dermatologii i Alergologii, 30(6), 373–380.