International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Utilizing Molecular Based Component Resolved Diagnostics in Wheat Allergy and its Potential Benefit in Management of Wheat Dependent Exercise Induced Anaphylaxis: A Case Series

Arjun Dang[1], Asim Israr Khan[1], Ritu Malani[2], Binish Jawed[1]

[1]Department of Allergy, Dr Dangs Lab LLP, New Delhi, India
[2] Department of Internal medicine, Pulmonology & Allergy, Max Healthcare, Delhi, India

Abstract

Purpose: Wheat dependent exercise induced anaphylaxis (WDEIA) is an uncommon IgE-mediated food allergy in which allergic reactions occur when wheat ingestion is followed by physical exercise or other cofactors. Omega-5 gliadin (ω5 gliadin; Tri a 19) is the wheat component most frequently associated with this condition. Diagnosis is often difficult in routine practice, and component resolved diagnostics (CRD) may assist in identifying clinically relevant sensitization.

Methods: A retrospective review was conducted of patients who underwent CRD for wheat allergy components during the study period. Clinical details were analyzed in patients with documented reactions suggestive of WDEIA.

Results: Out of 1,056 patients tested, 28 (2.65%) showed sensitization to ω5 gliadin. Patient age ranged from 11 to 81 years, with a median age of 42 years. A marked male predominance was noted (85.7%). Specific IgE levels to ω5 gliadin varied widely, ranging from 0.37 to 44.65 kUA/L. Sensitization to other wheat components was infrequent, with α-amylase trypsin inhibitor detected in four patients and wheat nsLTP in three patients.

Four representative patients are described in detail, each with well-documented clinical episodes consistent with WDEIA. These cases illustrate the heterogeneity of clinical presentation, the role of physical exercise and cofactors such as aspirin or NSAIDs, and the diagnostic value of CRD in establishing the underlying mechanism and guiding management.

Conclusion: CRD allows identification of ω5 gliadin sensitization across a broad clinical spectrum and is useful in supporting the diagnosis of suspected WDEIA in routine clinical settings. This case series highlights the potential of CRD to revolutionize the diagnosis and management of WDEIA, enabling more precise and personalized care for affected individuals.

Keywords: WDEIA, wheat-dependent exercise-induced anaphylaxis; CRD, component resolved diagnostics; Gliadin.

Introduction

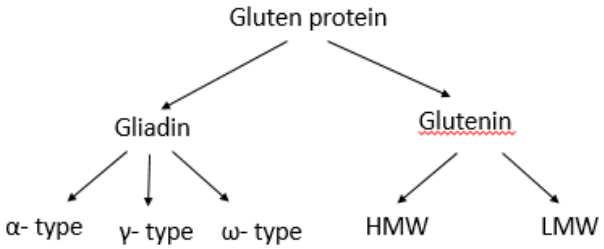
Wheat dependent exercise induced anaphylaxis (WDEIA) is a rare but potentially life-threatening food allergy that manifests when wheat ingestion is followed by physical exercise, often in the presence of

cofactors such as aspirin, other nonsteroidal anti-inflammatory drugs (NSAIDs), alcohol, stress, fatigue, or infections. This condition is characterized by an immunoglobulin E (IgE) mediated allergic response, with omega-5 gliadin (ω5 gliadin) identified as the primary allergen implicated.[1][2][,3]

The diagnosis of WDEIA relies on a multimodal evaluation that includes a detailed clinical history, serum IgE profiling, skin testing, basophil activation tests, and, when appropriate, challenge tests incorporating relevant cofactors.[4] Although strict avoidance of wheat is often advised, it may not necessarily promote long term tolerance. In fact, continued consumption of gluten containing cereals in the absence of cofactors may support tolerance development. Emergency management of WDEIA aligns with that of other IgE mediated food allergies, with adrenaline auto injectors playing a critical role in self-treatment.[5][6] Despite advancements, the mechanisms underlying WDEIA are not fully understood, necessitating further research to elucidate them.[1][3] Overall, WDEIA represents a complex interplay of allergenic proteins, cofactors, and host susceptibility, requiring individualized diagnosis and management strategies, including dietary counseling and emergency preparedness.[1][6]

Gliadin and glutenin are two major allergenic components of gluten protein. Gliadin is further classified into α, γ, and ω gliadins, while glutenins are categorized into low and high molecular weight glutenins. α and γ gliadins are primarily responsible for wheat allergy in children, whereas ω gliadins and high molecular weight glutenins are involved in wheat dependent exercise induced anaphylaxis.[1][7] (Figure 1)

Figure 1. Components of gluten



The placebo controlled oral wheat exercise challenge test is regarded as the gold standard for diagnosis.[4] However, reproducing this test poses significant challenges due to the substantial variability in the necessary amounts of food and exercise intensity. Moreover, this approach entails risks, as it can provoke severe systemic reactions in patients. In instances of acute reactions and in professions with high demands, such as those in the military, aviation, or law enforcement, the advised course of action is to follow a wheat free diet to prevent the occurrence of anaphylaxis. In milder instances, it may be suggested to refrain from wheat consumption along with the use of cofactors.[1][4]

Methods

A retrospective analysis was conducted on patient data collected between April 2023 and May 2025, focusing on 28 individuals who tested positive for specific IgE (sIgE) antibodies against ω5 gliadin. From this cohort, four representative cases were selected for detailed evaluation based on the presence of allergic symptoms in contexts consistent with wheat dependent exercise or drug induced anaphylaxis triggers.

Venous blood samples were collected using serum separation tubes (BD Vacutainer; REF#367956) under standard aseptic conditions. CRD using microarrayed allergens on the ALEX-2 by Macro Array Diagnostics was utilized to identify IgE sensitization to 294 allergens or their components, including molecular components of wheat. Two kit variants were employed depending on processing requirements:

variant 02-2001-01 for manual analysis and variant 02-5001-01 for automated testing on the MAX 9k platform.

ALEX is a multiplex immunoassay test based on Enzyme Linked Immunosorbent Assay (ELISA). Allergen extracts or molecular allergens, which are coupled to nanoparticles, are systematically deposited onto a solid phase, forming a macroscopic array. Initially, the particle bound allergens react with sIgE present in the patient's sample. After incubation, non-specific IgE is washed off. The procedure continues by adding an enzyme labeled anti-human IgE detection antibody, which forms a complex with the particle bound sIgE. Following a second washing step, a substrate is added, which is converted to an insoluble, colored precipitate by the antibody bound enzyme. Finally, the enzyme substrate reaction is stopped by adding a blocking reagent. The amount of precipitate is proportional to the concentration of sIgE in the patient sample. The lab test procedure is followed by image acquisition and analysis of results with MADx's Raptor Analysis Software. The results are reported in IgE response units (kU/L). According to the assay protocol, a sIgE concentration greater than 0.35 kUA/L was considered a positive result for sensitization. (Figure.2)

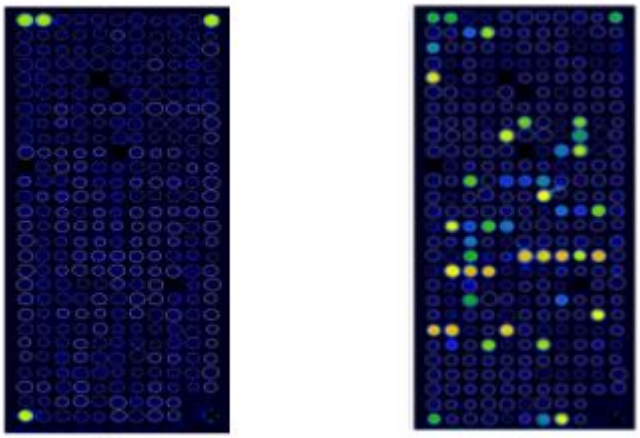


Figure.2. (a) is negative and (b) is positive CRD results, images analyzed with MADx's Raptor Analysis Software

Results

Among the total cohort of 1,056 patients tested for wheat allergy components by CRD, 28 individuals (2.65%) were positive for ω5 gliadin (Tri a 19), the principal allergen associated with WDEIA. The age of these positive patients ranged broadly from 11 to 81 years, reflecting the wide demographic affected by this condition. The median age was approximately 42 years. When stratified by age groups, the highest positivity rate was observed in the 21–40 years category, comprising 39.3% (11/28) of positive cases. This was followed by the 41–60 years group at 35.7% (10/28), those above 60 years at 17.9% (5/28)

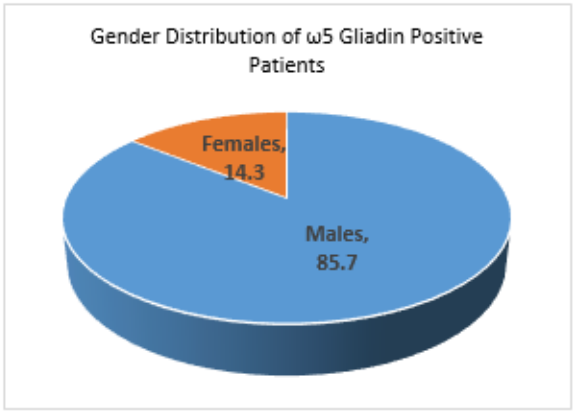


Figure 3: Gender Distribution of ω5 Gliadin Positive Patients

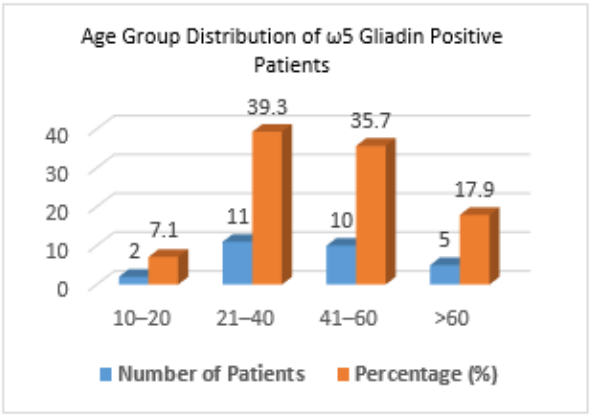


Figure 4: Age Group Distribution of ω5 Gliadin Positive Patients

and the 10–20 years group at 7.1% (2/28).(Figure 4) This distribution highlights a predominance of WDEIA sensitization in

young to middle aged adults, which aligns with the typical age range of physically active individuals who are more likely to engage in exercise triggering anaphylaxis. The cohort included 24 males (85.7%) and 4 females (14.3%), indicating a marked male predominance in sensitization. (Figure 3).

Patient no.	Allergen	Molecular Component	Protein	Specific IgE Conc. (kUA/L)
1	Wheat	Tri a 19	ω 5 Gliadin	3.39
2	Wheat	Tri a 19	ω 5 Gliadin	5.12
3	Wheat	Tri a 19	ω 5 Gliadin	2.70
4	Wheat	Tri a 19	ω 5 Gliadin	2.16
5	Wheat	Tri a 19	ω 5 Gliadin	1.46
6	Wheat	Tri a 19	ω 5 Gliadin	0.38
7	Wheat	Tri a 19	ω 5 Gliadin	12.68
8	Wheat	Tri a 19	ω 5 Gliadin	3.46
9	Wheat	Tri a 19	ω 5 Gliadin	3.03
10	Wheat	Tri a 19	ω 5 Gliadin	1.13
11	Wheat	Tri a 19	ω 5 Gliadin	9.46
12	Wheat	Tri a 19	ω 5 Gliadin	5.10

13	Wheat	Tri a 19	ω 5 Gliadin	0.55
14	Wheat	Tri a 19	ω 5 Gliadin	2.23
15	Wheat	Tri a 19	ω 5 Gliadin	1.45
16	Wheat	Tri a 19	ω 5 Gliadin	4.55
17	Wheat	Tri a 19	ω 5 Gliadin	44.65
18	Wheat	Tri a 19	ω 5 Gliadin	7.35
19	Wheat	Tri a 19	ω 5 Gliadin	4.18
20	Wheat	Tri a 19	ω 5 Gliadin	0.40
21	Wheat	Tri a 19	ω 5 Gliadin	29.02
22	Wheat	Tri a 19	ω 5 Gliadin	6.05
23	Wheat	Tri a 19	ω 5 Gliadin	1.54
24	Wheat	Tri a 19	ω 5 Gliadin	13.44
25	Wheat	Tri a 19	ω 5 Gliadin	3.32
26	Wheat	Tri a 19	ω 5 Gliadin	4.76
27	Wheat	Tri a 19	ω 5 Gliadin	0.37
28	Wheat	Tri a 19	ω 5 Gliadin	38.68
		Tri a aA_TI	α -Amylase Trypsin Inhibitor	3.65
29	Wheat	Tri a 14	nsLTP	1.42
		Tri a aA_TI	α -Amylase Trypsin Inhibitor	15.03
30	Wheat	Tri a 14	nsLTP	0.32
31	Wheat	Tri a aA_TI	α -Amylase Trypsin Inhibitor	2.52
32	Wheat	Tri a aA_TI	α -Amylase Trypsin Inhibitor	2.20
33	Wheat	Tri a 14	nsLTP	0.61

The sIgE concentrations to ω5 gliadin among positive patients ranged from 0.37 kUA/L to 44.65 kUA/L, demonstrating a wide variability in sensitization intensity (Table 1). The majority of patients exhibited moderate to high levels of sIgE, consistent with clinical reactivity. In addition to ω5 gliadin, sensitization to other wheat components was also observed, albeit at lower frequencies, with 4 patients (0.004%) positive for α-Amylase Trypsin Inhibitor (Tri a aA_TI) and 3 patients (0.003%) positive for nonspecific Lipid Transfer Protein (nsLTP, Tri a 14).

Case Descriptions:

Case 1

Patient: 37-year-old male

Clinical Presentation: On April 9, 2024, the patient consumed a meal consisting of a granola bar, milk, omelet, and buttered toast at approximately 6:30 PM. At 8:00 PM, he engaged in a session of pickleball. Within 15 minutes, he developed sudden-onset generalized itching, urticaria, facial and auricular swelling,

throat tightness, and subsequently collapsed. Emergency medical services administered intravenous adrenaline, hydrocortisone, pheniramine, and intravenous fluids. He recovered fully within 30 minutes.

Previous History: The patient had previously consumed similar foods without any adverse effects. However, in December 2023, he experienced itching after consuming beer, egg parantha, and mutton, which resolved spontaneously on rest.

Diagnostic Workup: CRD panel revealed sensitization to Wheat Tri a 19 (ω5 gliadin) with a sIgE level of 5.10 kUA/L.

Diagnosis: WDEIA.

Management: The patient was advised to strictly avoid wheat products, refrain from food intake at least 3 hours prior to exercise, and consider pre-exercise antihistamines in specific situations. The patient remained symptom-free with adherence to dietary and activity-related precautions.

Outcome: The patient had no further episodes during six months of follow-up.

Case 2

Patient: 44-year-old male

Clinical Presentation: The patient had a complex history of multiple anaphylactic episodes. On March 26, 2024, he developed a systemic allergic reaction following a bee sting, which resolved with emergency care. On May 1, 2024, he consumed a meal that included wheat-based foods and paracetamol, and developed generalized itching, facial swelling, vomiting, and subsequent collapse within one hour. He received IV adrenaline and corticosteroids. On May 15, 2024, he had another episode after eating bread and taking a combination NSAID (ibuprofen and paracetamol). Symptoms again included pruritus, urticaria, and hypotension requiring emergency treatment.

Previous History: The patient reported tolerance to similar foods and medications in the past, though he noted intermittent episodes of unexplained pruritus.

Diagnostic Workup: CRD showed sIgE reactivity to Wheat Tri a 19 (ω5 gliadin) at 0.55 kUA/L, Honey bee (API m1) at 0.35 kUA/L, Cockroach (arginine kinase) at 0.42 kUA/L, and cross-reactive carbohydrate determinants (CCD) at 0.41 kUA/L. Diagnosis: Insect venom hypersensitivity, possible NSAID/paracetamol hypersensitivity, and WDEIA.

Diagnosis: WDEIA.

Management: The patient was advised to eliminate wheat from his diet, avoid bee exposure, and NSAIDs. He was prescribed an epinephrine auto-injector and referred for further allergological evaluation of NSAID sensitivity.

Outcome: Symptoms improved with avoidance strategies. Follow-up showed no further reactions related to the identified triggers.

Case 3

Patient: 58-year-old male, commercial pilot.

Clinical Presentation: The patient presented with a CRD report indicating significant sensitization to Wheat Tri a 19 (Omega 5 gliadin). He had a history of recurrent urticaria and occasional angioedema for 10-12 years, often linked to food intake. In June 2023, he experienced an anaphylactic reaction with generalized urticaria, throat tightness, and dizziness after eating a sweet bun, requiring epinephrine administration. He had been taking aspirin regularly for cardiovascular prophylaxis for over two decades. Notably, one of the most severe episodes occurred after consuming a muffin followed by aspirin at

bedtime, resulting in widespread hives, hypotension, and transient loss of consciousness. He also suffered from perennial allergic rhinitis.

Previous History: The patient had multiple severe episodes after consuming seafood, pizza, and other foods, although no consistent single trigger was identified before CRD testing.

Diagnostic Workup: CRD demonstrated elevated sIgE to Wheat Tri a 19 (ω5 gliadin) at 5.12 kUA/L.

Diagnosis: WDEIA with aspirin as a cofactor; concurrent allergic rhinosinusitis.

Management: The patient was advised to completely avoid wheat and gluten, avoid any food intake for at least three hours before physical exertion, and discontinue aspirin, switching to an alternative antiplatelet agent. He was prescribed an epinephrine auto-injector and continued on daily antihistamines.

Outcome: With the implementation of avoidance strategies and medication adjustments, the frequency and severity of episodes reduced significantly. He reported improved tolerance to well-cooked wheat products and remains under regular follow-up due to his high-risk occupation.

Case 4

Patient: 27-year-old male, postgraduate student.

Clinical Presentation: The patient had a history of recurrent allergic episodes, some presenting as anaphylaxis. In 2020, while pacing in his hostel room during late-night exam preparations, he developed sudden onset generalized itching followed by urticaria around 3:00 AM. He does not recall what he ate prior to the episode. In 2021, he developed hives and lip swelling while jogging, having eaten a cheese sandwich 30 minutes prior. Both episodes were treated with injectable antihistamines and corticosteroids. In 2024, he experienced a severe anaphylactic event within 20 minutes of playing football, characterized by diffuse pruritus, urticaria, and collapse. Emergency services administered epinephrine, steroids, and supportive care. He recalled consuming wheat-based pasta about 90 minutes before the event.

Previous History: Upon further evaluation, the patient acknowledged previous episodes of exercise-induced itching and hives during football sessions, particularly following certain meals.

Diagnostic Workup: CRD revealed a high level of sIgE to Wheat Tri a 19 (ω5 gliadin) at 5.27 kUA/L.

Diagnosis: WDEIA.

Management: The patient was advised to avoid wheat-containing foods, refrain from eating for at least 3 hours before exercise, and was prescribed an epinephrine auto-injector.

Outcome: With adherence to dietary and activity precautions, the patient reported no further allergic episodes and has remained well through follow-up.

Discussion

WDEIA is an increasingly recognized but underdiagnosed clinical entity, particularly in populations with high wheat consumption and regular physical activity. The present case series highlights the clinical utility of molecular based CRD for the identification and management of WDEIA, with a focus on ω5 gliadin (Tri a 19) as the principle allergen. Our findings are consistent with previous studies demonstrating that ω5 gliadin is the major sensitizing component in WDEIA, with specific IgE to this protein serving as a highly sensitive and specific biomarker for diagnosis. [8] [12]

The demographic profile in our cohort, with a median age of 42 years and a striking male predominance, mirrors the epidemiological trends reported in other WDEIA studies.[2][9] The higher prevalence in young to middle aged adults may be attributed to increased physical activity and greater exposure to cofactors such as NSAIDs and alcohol, which are known to lower the threshold for anaphylaxis.[3] The male

predominance could reflect behavioral or occupational differences, as well as potential hormonal influences on immune responses.[1][10]

CRD has revolutionized the approach to food allergy by enabling precise identification of allergenic proteins at the molecular level. In the context of WDEIA, CRD offers several advantages over conventional testing based on allergen extract. First, it allows for the differentiation between sensitization to ω5 gliadin and other wheat proteins, such as α-amylase trypsin inhibitors (Tri a aA_TI) and non-specific lipid transfer proteins (nsLTP, Tri a 14), which may be implicated in classical wheat allergy or baker's asthma.[11][13][14] In our series, a minority of patients displayed sensitization to these additional components, underscoring the heterogeneity of wheat allergenicity and the importance of comprehensive molecular profiling.[1][11]

The clinical significance of ω5 gliadin sIgE is reinforced by the observed association between sensitization levels and symptomatic reactions in our cohort, in agreement with previously published data. [2] [12] Nevertheless, severe anaphylactic episodes were also noted in individuals with comparatively low sIgE concentrations, underscoring the complex and multifactorial nature of WDEIA. Known cofactors such as exercise, NSAIDs, alcohol, infections, and even psychological stress are known to modulate gastrointestinal permeability, augment mast cell responsiveness, and increase allergen bioavailability, collectively lowering the threshold for anaphylaxis in sensitized patients. [1][3][15]

Management of WDEIA is often complicated by the variable presentation and unpredictable timing of reactions, as well as the potential for life-threatening anaphylaxis. The cases presented in this series emphasize the need for an individualized approach that incorporates careful risk stratification, targeted dietary counseling, and structured patient education, particularly regarding avoidance of wheat ingestion in proximity to exercise and other recognized cofactors. The prescription of adrenaline auto injectors is essential, particularly in high risk individuals or those with a history of severe reactions. [9][16] Recent evidence suggests that some patients may tolerate wheat ingestion in the absence of cofactors, raising the possibility of tailored reintroduction protocols under specialist supervision.[9][17]

The gold standard for WDEIA diagnosis remains the placebo controlled oral wheat exercise challenge; however, this approach carries significant risk and logistical challenges.[1,4] CRD provides a safer, non-invasive alternative that can guide both diagnosis and management. The identification of ω5 gliadin sensitization enables clinicians to confidently diagnose WDEIA and implement targeted interventions, reducing the risk of unnecessary dietary restrictions and improving quality of life. [2][12]

Despite these advances, several gaps remain. The precise mechanisms by which cofactors lower the threshold for anaphylaxis are not fully elucidated, and the predictive value of sIgE levels for reaction severity requires further study.[9][16][18] Longitudinal studies are needed to assess the natural history of WDEIA and the impact of dietary and lifestyle interventions on long-term outcomes. Additionally, the role of emerging biomarkers and novel immunotherapeutic strategies warrants exploration.[1][15]

Overall, our findings reinforce the central role of ω5 gliadin in WDEIA and demonstrate the clinical utility of molecular based CRD in diagnosis and management. This approach facilitates personalized care, enhances diagnostic accuracy, and supports evidence-based recommendations for patients at risk of wheat-induced anaphylaxis.

Conclusion

WDEIA is a complex, multifactorial disorder that poses significant diagnostic and management challenges. Our case series demonstrates that CRD, particularly the detection of ω5 gliadin sIgE, offer a

sensitive and specific tool for identifying at-risk individuals. The integration of CRD into routine clinical practice enables precise diagnosis, risk stratification, and the development of individualized management plans, including dietary counseling and emergency preparedness. Continued research is needed to further elucidate the mechanisms of WDEIA, refine diagnostic algorithms, and optimize patient outcomes through targeted interventions.

Acknowledgement

We are grateful to the consultant and laboratory personnel at Dr Dangs Lab LLP for their valuable support and participation in this study.

Statement of Ethics

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Independent Ethics Committee Written informed consent was obtained from all patients whose cases are discussed in this article.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

This study was not supported by any sponsor or funder.

Author Contributions

Asim and Binish conducted data collection, analysis and prepared the first draft. Arjun, Asim, Shubnam contributed to the conception of the idea for the work, study design, and critical manuscript review for important content.

Data Availability Statement

Further enquiries can be made to the corresponding author. The data that support the findings of this study are not publicly available as they contain information that could compromise the privacy of research participants but are available in de-identified form from the corresponding author (BJ) upon reasonable request.

References

1. Scherf KA, Brockow K, Biedermann T, Koehler P, Wieser H. Wheat-dependent exercise-induced anaphylaxis. Clin Exp Allergy. 2016 Jan;46(1):10-20.
2. Matsuo H, Morita E, Tatham AS, Morimoto K, Horikawa T, Osuna H, Ikezawa Z, Kaneko S, Kohno K, Dekio S. Identification of the IgE-binding epitope in omega-5 gliadin, a major allergen in wheat-dependent exercise-induced anaphylaxis. J Biol Chem. 2004 Mar 26;279(13):12135-40.
3. Wölbing F, Fischer J, Köberle M, Kaesler S, Biedermann T. About the role and underlying mechanisms of cofactors in anaphylaxis. Allergy. 2013 Sep;68(9):1085-92.
4. Brockow K, Kneissl D, Valentini L, Zelger O, Grosber M, Kugler C, Werich M, Darsow U, Matsuo H, Morita E, Ring J. Using a gluten oral food challenge protocol to improve diagnosis of wheat-dependent exercise-induced anaphylaxis. J Allergy Clin Immunol. 2015 Apr;135(4):977-984.e4.
5. Faihs V, Kugler C, Schmalhofer V, Scherf KA, Lexhaller B, Mortz CG, Bindslev-Jensen C, Biedermann T, Brockow K. Wheat-dependent exercise-induced anaphylaxis: subtypes, diagnosis, and management. J Dtsch Dermatol Ges. 2023 Oct;21(10):1131-1135.
6. Cardona V, Ansotegui IJ, Ebisawa M, El-Gamal Y, Fernandez Rivas M, Fineman S, Geller M, Gonzalez-Estrada A, Greenberger PA, Sanchez Borges M, Senna G, Sheikh A, Tanno LK, Thong BY,

Turner PJ, Worm M. World allergy organization anaphylaxis guidance 2020. World Allergy Organ J. 2020 Oct 30;13(10):100472.
7. Kotaniemi-Syrjänen A, Palosuo K, Jartti T, Kuitunen M, Pelkonen AS, Mäkelä MJ. The prognosis of wheat hypersensitivity in children. Pediatr Allergy Immunol. 2010 Mar;21(2 Pt 2):e421-8.
8. Palosuo K, Alenius H, Varjonen E, Kalkkinen N, Reunala T. Rye gamma-70 and gamma-35 secalins and barley gamma-3 hordein cross-react with omega-5 gliadin, a major allergen in wheat-dependent, exercise-induced anaphylaxis. Clin Exp Allergy. 2001 Mar;31(3):466-73.
9. Du Z, Gao X, Li J, Li L, Liu J, Yin J. Clinical features and outcomes of patients with wheat-dependent exercise-induced anaphylaxis: a retrospective study. Allergy Asthma Clin Immunol. 2022 Jul 5;18(1):61.
10. Klein SL, Flanagan KL. Sex differences in immune responses. Nat Rev Immunol. 2016 Oct;16(10):626-38.
11. Canonica GW, Ansotegui IJ, Pawankar R, Schmid-Grendelmeier P, van Hage M, Baena-Cagnani CE, et al. A WAO - ARIA - GA²LEN consensus document on molecular-based allergy diagnostics. World Allergy Organ J. 2013 Oct 3;6(1):17.
12. Palosuo K, Varjonen E, Nurkkala J, Kalkkinen N, Harvima R, Reunala T, Alenius H. Transglutaminase-mediated cross-linking of a peptic fraction of omega-5 gliadin enhances IgE reactivity in wheat-dependent, exercise-induced anaphylaxis. J Allergy Clin Immunol. 2003 Jun;111(6):1386-92.
13. Tatham AS, Shewry PR. Allergens to wheat and related cereals. Clin Exp Allergy. 2008 Nov;38(11):1712-26.
14. Scala E, Till SJ, Asero R, Abeni D, Guerra EC, Pirrotta L, Paganelli R, Pomponi D, Giani M, De Pità O, Cecchi L. Lipid transfer protein sensitization: reactivity profiles and clinical risk assessment in an Italian cohort. Allergy. 2015 Aug;70(8):933-43.
15. Morita E, Kunie K, Matsuo H. Food-dependent exercise-induced anaphylaxis. J Dermatol Sci. 2007 Aug;47(2):109-17.
16. Srisuwatchari W, Kanchanaphoomi K, Nawiboonwong J, Thongngarm T, Sompornrattanaphan M. Food-Dependent Exercise-Induced Anaphylaxis: A Distinct Form of Food Allergy-An Updated Review of Diagnostic Approaches and Treatments. Foods. 2023 Oct 13;12(20):3768.
17. Christensen MJ, Eller E, Mortz CG, Brockow K, Bindslev-Jensen C. Clinical and serological follow-up of patients with WDEIA. Clin Transl Allergy. 2019 May 16;9:26.
18. Shin M. Food allergies and food-induced anaphylaxis: role of cofactors. Clin Exp Pediatr. 2021 Aug;64(8):393-399.