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DIAGNOSIS AND MANAGEMENT OF EXERCISE-INDUCED ANAPHYLAXIS: A LITERATURE REVIEW

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ABSTRACT

Background: Exercise-induced anaphylaxis (EIA) is a rare, potentially life-threatening allergic reaction triggered by physical activity, with food-dependent (FDEIA) and food-independent forms. Understanding its pathophysiology, triggers, and co-factors is essential for accurate diagnosis and prevention.
Aim: To summarize current evidence on the epidemiology, pathophysiology, clinical presentation, diagnosis, and management of EIA.
Methodology: A literature review was conducted using PubMed and Google Scholar.
Results: EIA accounts for 2.3-5% of anaphylaxis cases. It is mediated by IgE-triggered mast cell degranulation, with exercise altering gastrointestinal permeability, osmolarity, and pH, thereby lowering the threshold for allergic reactions. Common triggers include wheat, shellfish, and nuts, with co-factors such as NSAIDs, alcohol, and environmental extremes. Diagnosis is based on detailed history, targeted allergy testing, and, when needed, supervised exercise challenges. Management involves immediate epinephrine administration for acute episodes and long-term prevention through trigger avoidance, co-factor management, and patient education.
Conclusions: EIA is an under-recognized but preventable cause of severe allergic reactions. Early recognition, appropriate diagnostic work-up, and a personalized management plan allow most patients to continue safe physical activity.

KEYWORDS

Exercise-Induced Anaphylaxis, Food-Dependent Exercise-Induced Anaphylaxis, Physical Allergy, Mast Cell Degranulation, Anaphylaxis Management, Allergy Diagnosis, Epinephrine, Exercise-Related Allergy, Ige-Mediated Hypersensitivity

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1. Introduction

Exercise-Induced Anaphylaxis (EIA) is an uncommon form of physical allergy in which strenuous physical activity provokes an anaphylactic reaction. The condition was first described in 1979 by Maulitz et al., who reported exercise precipitating an anaphylactic reaction after shellfish ingestion [1]. EIA is now defined as an IgE-mediated hypersensitivity reaction precipitated by physical activity [2]. By definition, affected individuals are asymptomatic at rest, with exercise acting as the key catalyst that initiates the allergic cascade.

Although EIA accounts for only 2.3-5% of anaphylaxis cases in clinical series [3], it can occur at any age and in both sexes, with onset most often in adolescence or early adulthood. Atopic individuals appear to be at higher risk [4]. Fatal cases are uncommon, likely because most patients learn to recognize symptoms early and stop exercising, however, undiagnosed cases may carry significant danger. There are two recognized forms of EIA: First food-independent EIA, in which anaphylaxis is induced by exercise alone (though other cofactors may modulate it), and second, Food-dependent exercise-induced anaphylaxis (FDEIA), in which ingestion of a specific food allergen plus exercise in combination is required to provoke a reaction [5], [6]. In FDEIA, the patient can usually tolerate the food and exercise separately without anaphylaxis. It is the concurrence of both, often within a few hours of each other, that triggers the event [2]. Approximately 30-50% of reported EIA cases are food-dependent [7], [8], [9], making FDEIA a substantial subset of this syndrome. Classic EIA does not involve a specific identified allergen. In these cases, exercise alone appears to trigger anaphylaxis. However, recent studies suggest that even in so-called "food-independent" EIA, other subtle cofactors, such as medications, environmental conditions, or physiological stress, may still play a significant role in lowering the threshold for a reaction [10].

Diagnosis can be challenging, as patients may exercise safely under some conditions yet unpredictably develop anaphylaxis at other times. A clear understanding of EIA subtypes, triggers, and risk factors is essential for effective diagnosis, prevention, and management. Our review summarizes current evidence on EIA, including clinical presentation, diagnostic approaches, and treatment managment for safe exercise participation in affected individuals.

2. Methodology

We conducted a narrative literature review in PubMed and Google Scholar using combinations of “exercise-induced anaphylaxis,” “food-dependent exercise-induced anaphylaxis/FDEIA,” “cofactors,” “NSAIDs,”, “diagnosis,” and “management.” We included human studies and reviews addressing epidemiology, pathophysiology, presentation, diagnosis, triggers, co-factors, or management of EIA/FDEIA. Non-human or non-relevant papers were excluded.

3. Pathophysiology

The biochemical and cellular mechanisms underlying EIA are complex and not yet fully known [11]. Fundamentally, EIA is an IgE-mediated process centered on mast cells: during an attack, mast cell degranulation leads to release of histamine, leukotrienes, platelet-activating factor (PAF), prostaglandins and other mediators that produce multisystem allergic symptoms (cutaneous, respiratory, cardiovascular) [12]. What makes EIA unique is the exercise itself. Several hypotheses explain how exercise promotes anaphylaxis.

Physical activity alters gastrointestinal (GI) physiology. During exercise, there is a diversion of blood flow away from the splanchnic circulation and a sympathetic nervous system surge, which can increase intestinal permeability and reduce gastric acid secretion [12]. These changes allow for more efficient absorption of larger, incompletely digested food molecules from the gut. In FDEIA, this means if an athlete has recently eaten an allergenic food, exercise makes it more likely that intact allergens enter the circulation and trigger IgE-bearing mast cells [11], [13]. During exercise, especially in the presence of co-factors like nonsteroidal anti-inflammatory drugs (NSAIDs) or alcohol, the stomach produces less acid and intestinal permeability rises. This allows more structurally intact food allergens to cross the gut barrier into circulation. The allergens bind to IgE on mast cells, triggering cross-linking of FcεRI receptors and mast cell degranulation, releasing mediators (histamine, PAF, leukotrienes, prostaglandins) that cause the anaphylactic symptoms.

The exercise decrease in gastric acidity means allergens aren’t broken down as thoroughly in the stomach [12]. More intact allergenic proteins reach the intestines and bloodstream, potentiating the allergic response. Patients with wheat-dependent EIA (WDEIA) may absorb more 5 gliadin (a major wheat allergen) during exercise than at rest, helping explain why they tolerate wheat at rest but not pre-exercise [14].

Exercise leads to a rise in plasma osmolarity due to sweating and fluid shifts and acidosis from lactic acid production. These physiologic changes have been postulated to directly prompt mediator release.

Increased serum histamine during exercise has been showed in EIA patients [15]. That supports that exercise per se can induce mast cell/basophil degranulation. One theory is that changes in pH or osmolarity during strenuous activity may alter mast cell stability or IgE-antigen interactions, effectively "priming" an allergic response. Additionally, the redistribution of blood flow (with more blood to skeletal muscle and skin, and less to the gut) might bring circulating allergens into contact with a greater number of mast cells in the musculature and skin, compounding the reaction [16].

Besides food, other cofactors commonly amplify EIA reactions. NSAIDs (like aspirin or ibuprofen) are well-known to exacerbate or even enable EIA. These drugs may inhibit prostaglandin metabolism and increase gut permeability, both boosting allergic potential [17], [18]. Alcohol is another cofactor that can magnify an exercise-allergic reaction, possibly via similar permeability and metabolic effects [19]. Environmental conditions such as high ambient temperature and humidity or, conversely, very cold weather, as well as menstrual phase in females or concurrent mild illness, have all been reported to lower the threshold for EIA in susceptible individuals [11]. In one notable report, a patient's FDEIA reactions occurred only on hot, humid days and not in cooler environments, underscoring how climate stress can contribute [20]. These cofactors by themselves usually do not cause anaphylaxis, but in combination with exercise and often food, they create perfect conditions.

4. Symptoms and differential diagnoses

An athlete in the midst of an exercise-induced anaphylactic episode will manifest signs and symptoms that overlap with general anaphylaxis criteria, often beginning with cutaneous and respiratory features. Typically, patients notice flushing, pruritus, and urticaria (hives**)** early in the reaction. These may start on the chest or face and generalize quickly [2]. Some will also have a sensation of warmth, itchiness of the palms/soles or scalp, and perhaps gastrointestinal cramping as prodromal symptoms [8]. Without prompt cessation of exercise and intervention, the reaction can progress: swelling of face, lips, eyelids, or extremities are common, as is upper airway edema leading to throat tightness or difficulty swallowing. Patients often experience wheezing, cough, and chest tightness, leading to bronchospasm and laryngeal edema. This can be misinterpreted as an exercise-induced asthma attack at first, but the accompanying skin changes usually point toward anaphylaxis. As the reaction intensifies, vascular symptoms ensue: hypotension, tachycardia, lightheadedness, and even syncope or collapse from shock. In severe episodes, loss of consciousness can occur [2].

Differential diagnoses to consider include: exercise-induced bronchoconstriction (asthma), which causes wheezing and shortness of breath with exercise but typically lacks hives or hypotension. Cholinergic urticaria, a form of heat-induced hives that are smaller (2-4 mm) and transient, without systemic signs [21]. Vasovagal syncope, which can cause collapse during exercise but usually has bradycardia rather than tachycardia and no rash and, in rare cases, exercise-induced collapse from heat stroke or cardiac arrhythmia could superficially resemble anaphylaxis but would present differently (core temperature >40°C in heat stroke, arrhythmia on ECG). The presence of skin manifestations (flushing, hives, angioedema) in EIA is a key distinguishing feature, as over 80-90% of anaphylaxis cases have cutaneous symptoms [22].

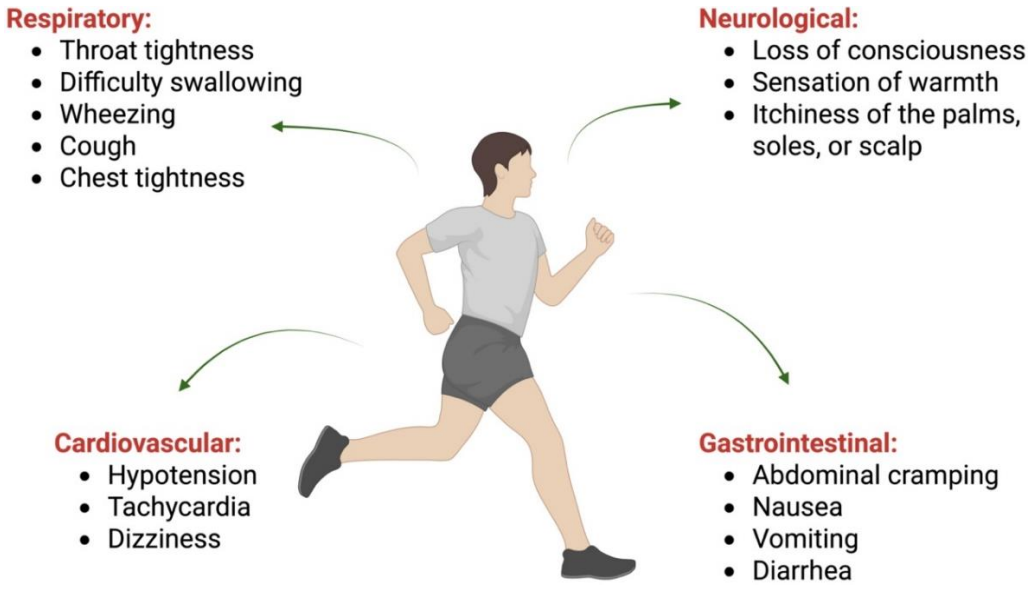

Fig. 1*. Common symptoms of Exercise-Induced Anaphylaxis (EIA).*

5. Diagnostic approach

Diagnosing EIA can be challenging, as there is no single laboratory test that definitively confirms it. The approach is therefore multifaceted, combining patient history, selective tests, and sometimes exercise provocation under controlled conditions.

5.1 Patient history and physical examination

A detailed patient history and physical examination are the most critical first steps. The clinician should ask about the specific circumstances of each anaphylactic episode, including:

1) **Exercise type and duration:** What kind of exercise were you doing and for how long?
2) **Timing of food intake:** What foods were eaten before exercise, and at what interval?
3) **Cofactors:** Were any medications (especially NSAIDs) or alcohol taken beforehand? What were the environmental conditions (temperature, humidity, pollen count)?
4) **Symptom progression:** Were there any early symptoms like itching or hives, or did the episode start with a sudden collapse? How were the episodes managed?

It's also important to note any past medical history of atopy (asthma, eczema, allergic rhinitis) or prior food allergies, as these may predispose a person to EIA. A physical exam between episodes is often normal, but some allergic individuals may have chronic hives or dermatographism (skin writing). The presence of brownish lesions that urticate (swell and itch) upon stroking should prompt an evaluation for a mast cell disorder.

5.2 Allergy testing for suspected triggers

If FDEIA is suspected allergy testing is crucial. Skin prick testing or serum-specific IgE testing for suspected foods should be performed. Common culprits include wheat, peanuts, tree nuts, and shellfish, so testing should be guided by the patient's diet history. A positive test indicates sensitization and supports an FDEIA diagnosis. However, a negative test to a strongly suspected food may require further evaluation, such as an oral food challenge with and without exercise, if it can be done safely. Testing for common environmental allergens is also reasonable, as atopy can be a predisposing factor for EIA. While there is no direct allergy test for NSAIDs or other drugs, a history of symptom exacerbation from these co-factors is highly informative [10], [23], [24].

5.3 Laboratory evaluation

Routine lab tests are generally not useful for diagnosing EIA. However, obtaining a serum tryptase level soon after an episode can be helpful. Tryptase is a mast cell enzyme that typically peaks 1-2 hours after the onset of an anaphylactic reaction. An elevated tryptase level during an exercise-induced event can confirm that an anaphylactic mechanism was involved. A normal tryptase, however, does not rule out EIA, as the sample may have been drawn too late, or the reaction may have been milder. It's also important to measure a baseline serum tryptase on a separate day when the patient has not had recent anaphylaxis. A significantly elevated baseline tryptase could indicate a mast cell disorder like mastocytosisor hereditary alpha-tryptasemia, which can increase the risk of anaphylaxis from various triggers [25], [26].

5.4 Exercise testing

In ambiguous cases, a controlled exercise challenge test can be performed in a medical setting to try and reproduce symptoms. This involves having the patient perform graded exercise, usually on a treadmill or stationary bike under supervision, after abstaining from food and medications for a specific period. Emergency medications and monitoring must be readily available. For FDEIA, the challenge might be done twice, once with exercise alone and again on a separate day with exercise after eating the suspected food. Development of symptoms or objective changes like a blood pressure drop or an increase in tryptase can confirm the diagnosis. However, these tests have a significant false-negative rate, as the condition is highly variable. A patient may have a negative test one day but still experience EIA in real life. For this reason, a negative challenge does not rule out EIA [27], [28], [29].

The diagnosis of EIA relies heavily on a clinician's ability to combine information from the patient's history with tests to confirm suspected allergies or exclude other conditions. Once a diagnosis is made or strongly suspected, the focus shifts to educating the patient and creating a management plan for future episodes.

6. Management

Managing EIA requires a two pronged approach: immediate treatment of acute episodes and long-term strategies to prevent future reactions. Patient education is essential, as is preparing for the possibility of a life-threatening event.

6.1 Acute episode managemen

An acute EIA episode must be treated as a medical emergency. The very first step is for the patient to immediately stop exercising and sit or lie down.

If the patient carries an epinephrine autoinjector, it should be administered at the first sign of significant symptoms, such as widespread hives, difficulty breathing, or dizziness. Epinephrine is the primary and most effective treatment; delays in its use are associated with worse outcomes. After administering epinephrine, the patient should call for emergency medical assistance, even if they feel better [30]. While waiting for help, the patient should be placed in a supine position (lying on their back) with their legs elevated to help maintain blood pressure. Other supportive measures include supplemental oxygen and monitoring vital signs. While medications like antihistamines and beta-agonist inhalers may help with some symptoms, they are secondary to epinephrine and are not a substitute for it. It's important to know that a biphasic reaction, where symptoms return hours later, is a possibility, which is why a medical observation period is always recommended [31].

6.2 Long-term prevention and lifestyle modifications

Long-term management focuses on avoiding known triggers and modifying a patient's exercise habits.

6.2.1 Exercise and diet

A main recommendation is to avoid eating for 4 to 6 hours before exercise, especially if a specific food trigger is known. If a patient with FDEIA ingests their known trigger, they should strictly avoid vigorous exercise for that period. For those without a specific food trigger, exercising on an empty stomach or after a very light, non-allergenic snack is the safest option. It is also important to lower the intensity of exercise and avoid exercising in extreme weather conditions, such as high heat and humidity or very cold temperature [32].

6.2.2 Medication and environmental factors

Patients with EIA should also avoid aspirin and other NSAIDs on days they plan to exercise, as these are common co-factors that can increase the risk of an attack. Acetaminophen is a safer alternative for pain relief. Similarly, alcohol consumption around the time of exercise is discouraged [33]. Environmental factors can also play a role. Patients should try to avoid exercising in extreme temperatures or during periods of high pollen count if they have hay fever. It's also advisable to avoid exercise when feeling unwell or feverish [34], [35], [36].

6.2.3 Social and personal safety

Patients with EIA should never exercise alone. They should always be with a partner or in a group where someone is aware of their condition and can administer epinephrine if needed. Wearing a medical alert bracelet or carrying a medical card can also be life-saving by informing bystanders and first responders of the condition.

6.2.4 Pharmacologic prophylaxis

While not a replacement for trigger avoidance and carrying epinephrine, some medications can be used to help prevent episodes or reduce their severity. Regular use of second-generation antihistamines, like cetirizine or loratadine, can help reduce hives and may prevent some mild episodes. They can be taken daily or 1 to 2 hours before exercise as a preemptive measure [37], [38], [39]. Omalizumab in severe cases that don't respond to other measures, omalizumab, a biologic medication that targets IgE, may be used. It has been shown to successfully prevent severe reactions in some patients with refractory EIA, but it is expensive and typically reserved for very specific cases under specialist care [40]. Mast cell stabilizers like cromolyn [37], [41], leukotriene modifiers [38], and other less common agents have been explored, but their effectiveness in preventing EIA is inconsistent or not well-established.

6.2.5 Patient education

Every patient diagnosed with EIA should be educated on how to use their epinephrine auto-injector and have a clear emergency action plan. They should know to stop exercising at the first sign of symptoms, and if symptoms do not improve quickly, to use the auto-injector and call for emergency help. Patients should be taught that using epinephrine early is always better than waiting for symptoms to worsen. Regular follow-up with an allergist is beneficial to refine the management plan, and stay updated on new therapies. With the right precautions and a solid understanding of their condition, people with EIA can often continue to lead active, healthy lives.

7. Conclusions

EIA is a rare but potentially life-threatening allergic reaction triggered by physical activity. Its diagnosis can be challenging, relying on a detailed patient history and, when appropriate, targeted allergy testing or a supervised exercise tests. Effective management requires a dual approach: immediate treatment with epinephrine during an episode and long-term prevention through avoidance of triggering factors. With appropriate education, an emergency action plan, and consistent use of an epinephrine injector, most individuals with EIA can safely maintain an active lifestyle.

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Conceptualization, E.S.; methodology, E.S, and K.M.; check, L.S.; formal analysis, L.S., E.S.; investigation, E.S.; resources, E.S., and K.M.; writing - rough preparation, E.S., and K.M.; writing - review and editing, L.S..; visualization, L.S.; supervision, L.S.; project administration, L.S., K.M., and E.S.

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REFERENCES

1. Maulitz, R., Pratt, D., & Schocket, A. (1979). Exercise-induced anaphylactic reaction to shellfish. *Journal of Allergy and Clinical Immunology, 63*(6), 433–434. https://doi.org/10.1016/0091-6749(79)90218-5
2. Minty, B. (2017). Food-dependent exercise-induced anaphylaxis. *Canadian Family Physician / Médecin de famille canadien, 63*(1), 42–43. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5257219
3. Geller, M. (2020). Clinical management of exercise-induced anaphylaxis and cholinergic urticaria. *Journal of Allergy and Clinical Immunology: In Practice, 8*(7), 2209–2214. https://doi.org/10.1016/j.jaip.2020.01.025
4. Ansley, L., Bonini, M., Delgado, L., Del Giacco, S. R., Du Toit, G., Khaitov, M., ... & Carlsen, K. H. (2015). Pathophysiological mechanisms of exercise-induced anaphylaxis: An EAACI position statement. *Allergy, 70*(10), 1212–1221. https://doi.org/10.1111/all.12677
5. Feldweg, A. M. (2017). Food-dependent, exercise-induced anaphylaxis: Diagnosis and management in the outpatient setting. *Journal of Allergy and Clinical Immunology: In Practice, 5*(2), 283–288. https://doi.org/10.1016/j.jaip.2016.11.022
6. Giannetti, M. P. (2018). Exercise-induced anaphylaxis: Literature review and recent updates. *Current Allergy and Asthma Reports, 18*(12), 72. https://doi.org/10.1007/s11882-018-0830-6
7. Fiedler, E.-M., Zuberbier, T., & Worm, M. (2002). A combination of wheat flour, ethanol, and food additives inducing FDEIA. *Allergy, 57*(11), 1090–1091. https://doi.org/10.1034/j.1398-9995.2002.23836_12.x
8. Povesi Dascola, C., & Caffarelli, C. (2012). Exercise-induced anaphylaxis: A clinical view. *Italian Journal of Pediatrics, 38*(1), 43. https://doi.org/10.1186/1824-7288-38-43
9. Castells, M. C., Horan, R. F., & Sheffer, A. L. (2003). Exercise-induced anaphylaxis. *Current Allergy and Asthma Reports, 3*(1), 15–21. https://doi.org/10.1007/s11882-003-0005-x
10. Carlisle, A., & Lieberman, J. A. (2024). Getting in shape: Updates in exercise anaphylaxis. *Current Allergy and Asthma Reports, 24*(11), 631–638. https://doi.org/10.1007/s11882-024-01176-4
11. Barg, W., Medrala, W., & Wolanczyk-Medrala, A. (2011). Exercise-induced anaphylaxis: An update on diagnosis and treatment. *Current Allergy and Asthma Reports, 11*(1), 45–51. https://doi.org/10.1007/s11882-010-0150-y
12. Srisuwatchari, W., Kanchanaphoomi, K., Nawiboonwong, J., Thongngarm, T., & Sompornrattanaphan, M. (2023). Food-dependent exercise-induced anaphylaxis: A distinct form of food allergy—An updated review of diagnostic approaches and treatments. *Foods, 12*(20), 3768. https://doi.org/10.3390/foods12203768
13. Asaumi, T., Yanagida, N., Sato, S., Shukuya, A., Nishino, M., & Ebisawa, M. (2016). Provocation tests for the diagnosis of food-dependent exercise-induced anaphylaxis. *Pediatric Allergy and Immunology, 27*(1), 44–49. https://doi.org/10.1111/pai.12489
14. Yano, H., Kato, Y., & Matsuda, T. (2002). Acute exercise induces gastrointestinal leakage of allergen in lysozyme-sensitized mice. *European Journal of Applied Physiology, 87*(4–5), 358–364. https://doi.org/10.1007/s00421-002-0653-x

15. Lindinger, M. I., & Grudzien, S. P. (2003). Exercise-induced changes in plasma composition increase erythrocyte Na+, K+-ATPase, but not Na+–K+–2Cl− cotransporter activity to stimulate net and unidirectional K+ transport in humans. *Journal of Physiology, 553*(3), 987–997. https://doi.org/10.1113/jphysiol.2003.052860
16. Nguyen, S. M. T., Rupprecht, C. P., Haque, A., Pattanaik, D., Yusin, J., & Krishnaswamy, G. (2021). Mechanisms governing anaphylaxis: Inflammatory cells, mediators, endothelial gap junctions and beyond. *International Journal of Molecular Sciences, 22*(15), 7785. https://doi.org/10.3390/ijms22157785
17. Niggemann, B., & Beyer, K. (2014). Factors augmenting allergic reactions. *Allergy, 69*(12), 1582–1587. https://doi.org/10.1111/all.12532
18. Motomura, C., Nakamura, K., Kudo, K., Takahashi, T., & Matsuo, H. (2017). Aspirin is an enhancing factor for food-dependent exercise-induced anaphylaxis in children. *Clinical and Experimental Allergy, 47*(11), 1497–1500. https://doi.org/10.1111/cea.13026
19. Versluis, A., Van Os-Medendorp, H., Kruizinga, A. G., Blom, W. M., Houben, G. F., & Knulst, A. C. (2016). Cofactors in allergic reactions to food: Physical exercise and alcohol are the most important. *Immunity, Inflammation and Disease, 4*(4), 392–400. https://doi.org/10.1002/iid3.120
20. Jo, E.-J., Kim, M. Y., Choi, Y. S., Lee, S. Y., Kim, Y. S., & Park, H. S. (2012). Food-dependent exercise-induced anaphylaxis occurred only in a warm but not in a cold environment. *Asia Pacific Allergy, 2*(2), 161–164. https://doi.org/10.5415/apallergy.2012.2.2.161
21. Casale, T. B., Keahey, T. M., & Kaliner, M. (1986). Exercise-induced anaphylactic syndromes: Insights into diagnostic and pathophysiologic features. *JAMA, 255*(15), 2049–2053. https://pubmed.ncbi.nlm.nih.gov/3514973
22. Volcheck, G. W., & Li, J. T. C. (1997). Exercise-induced urticaria and anaphylaxis. *Mayo Clinic Proceedings, 72*(2), 140–147. https://doi.org/10.4065/72.2.140
23. Kleiman, J., & Ben-Shoshan, M. (2014). Food-dependent exercise-induced anaphylaxis with negative allergy testing. *BMJ Case Reports, 2014*, bcr2013202057. https://doi.org/10.1136/bcr-2013-202057
24. Mohamed, S., Thalappil, S., & Mohamed Ali, R. (2024). A case report of food-dependent exercise-induced anaphylaxis (FDEIA) treated with omalizumab. *Frontiers in Allergy, 5,* 1472320. https://doi.org/10.3389/falgy.2024.1472320
25. Beck, S. C., Wilding, T., Buka, R. J., Baretto, R. L., Huissoon, A. P., & Krishna, M. T. (2019). Biomarkers in human anaphylaxis: A critical appraisal of current evidence and perspectives. *Frontiers in Immunology, 10,* 494. https://doi.org/10.3389/fimmu.2019.00494
26. Beyens, M., Toscano, A., Ebo, D., Gülen, T., & Sabato, V. (2023). Diagnostic significance of tryptase for suspected mast cell disorders. *Diagnostics, 13*(24), 3662. https://doi.org/10.3390/diagnostics13243662
27. Romano, A., Di Fonso, M., Giuffreda, F., Palma, R., & Artesani, M. C. (1995). Diagnostic work-up for food-dependent, exercise-induced anaphylaxis. *Allergy, 50*(10), 817–824. https://doi.org/10.1111/j.1398-9995.1995.tb05055.x
28. Dohi, M., Suko, M., Sugiyama, H., Yamashita, T., Kato, M., & Nakagawa, T. (1991). Food-dependent, exercise-induced anaphylaxis: A study on 11 Japanese cases. *Journal of Allergy and Clinical Immunology, 87*(1), 34–40. https://doi.org/10.1016/0091-6749(91)90210-F
29. Fiocchi, A., Mirri, G. P., Santini, I., Bernardo, L., Ottoboni, F., & Riva, E. (1997). Exercise-induced anaphylaxis after food contaminant ingestion in double-blinded, placebo-controlled, food-exercise challenge. *Journal of Allergy and Clinical Immunology, 100*(3), 424–425. https://doi.org/10.1016/S0091-6749(97)70258-6
30. Dribin, T. E., Waserman, S., & Turner, P. J. (2023). Who needs epinephrine? Anaphylaxis, autoinjectors, and parachutes. *Journal of Allergy and Clinical Immunology: In Practice, 11*(4), 1036–1046. https://doi.org/10.1016/j.jaip.2023.02.002
31. Hearrell, M., & Anagnostou, A. (2020). Diagnosis and management of anaphylaxis. *Journal of Food Allergy, 2*(1), 64–68. https://doi.org/10.2500/jfa.2020.2.200001
32. Soyer, O., & Sekerel, B. (2008). Food-dependent exercise-induced anaphylaxis or exercise-induced anaphylaxis? *Allergologia et Immunopathologia, 36*(4), 242–243. https://doi.org/10.1157/13127050
33. Christensen, M. J., Eller, E., Mortz, C. G., Brockow, K., & Bindslev-Jensen, C. (2019). Wheat-dependent cofactor-augmented anaphylaxis: A prospective study of exercise, aspirin, and alcohol efficacy as cofactors. *Journal of Allergy and Clinical Immunology: In Practice, 7*(1), 114–121. https://doi.org/10.1016/j.jaip.2018.06.018
34. Kulthanan, K., Tuchinda, P., Chularojanamontri, L., & Srisawat, C. (2022). Food-dependent exercise-induced wheals, angioedema, and anaphylaxis: A systematic review. *Journal of Allergy and Clinical Immunology: In Practice, 10*(9), 2280–2296. https://doi.org/10.1016/j.jaip.2022.06.008
35. Pravettoni, V., & Incorvaia, C. (2016). Diagnosis of exercise-induced anaphylaxis: Current insights. *Journal of Asthma and Allergy, 9,* 191–198. https://doi.org/10.2147/JAA.S109105
36. Morita, E., Kunie, K., & Matsuo, H. (2007). Food-dependent exercise-induced anaphylaxis. *Journal of Dermatological Science, 47*(2), 109–117. https://doi.org/10.1016/j.jdermsci.2007.03.004
37. Benhamou, A. H., Vanini, G., Lantin, J. P., & Eigenmann, P. A. (2007). Antihistamine and sodium cromoglycate medication for food cold water exercise-induced anaphylaxis. *Allergy, 62*(12), 1471–1472. https://doi.org/10.1111/j.1398-9995.2007.01484.x

38. Peroni, D. G., Piacentini, G. L., Piazza, M., Cametti, E., & Boner, A. L. (2010). Combined cetirizine–montelukast preventive treatment for food-dependent exercise-induced anaphylaxis. *Annals of Allergy, Asthma & Immunology, 104*(3), 272–273. https://doi.org/10.1016/j.anai.2009.12.002
39. Choi, J. H., Lee, H. B., Ahn, I. S., Park, C. W., & Lee, C. H. (2009). Wheat-dependent, exercise-induced anaphylaxis: A successful case of prevention with ketotifen. *Annals of Dermatology, 21*(2), 203. https://doi.org/10.5021/ad.2009.21.2.203
40. Gon, Y., Maruoka, S., & Mizumura, K. (2022). Omalizumab and IgE in the control of severe allergic asthma. *Frontiers in Pharmacology, 13,* 839011. https://doi.org/10.3389/fphar.2022.839011
41. Sugimura, T., Matsuo, H., Kaneko, S., Yokozeki, H., & Morita, E. (2009). Effect of oral sodium cromoglycate in two children with food-dependent exercise-induced anaphylaxis (FDEIA). *Clinical Pediatrics, 48*(9), 945–950. https://doi.org/10.1177/0009922809337528