International Journal of Health Sciences and Research
Vol.14; Issue: 1; January 2024
Website: www.ijhsr.org
Review Article *ISSN: 2249-9571*

Treatment and Strategy for Food Allergies in Paediatrics

Dr. Sana Shaikh[1], Dr. Ab Samad[2], Ab Haque Bamer[3], Sunil S. Tade[4], Areeb Inamdar[5], Dr. Bijoy Panda Kumar[6], Dr. Shuklendu Deo[7]

[1]Medical Information Specialist, Department of Medical Communications, Eversana (India) PVT. Ltd, Pune,
[2]Medical Advisor Department of Medical Affairs, Celagenex Research (India) PVT. Ltd, Thane,
[3]Associate Professor Department of Pharmacology, Vivekvardhini Sevabhavi Sansthas College of Pharmacy at Post Pingali, Parbhani,
[4]Associate Professor Department of Pharmaceutics Vivekvardhini Sevabhavi Sansthas College of Pharmacy at Post Pingali, Parbhani,
[5]Department. of Biology-Biotechnology, Maulana Azad College of Arts, Science and Commerce, Dr. Rafiq Zakaria Campus, Aurangabad,
[6] Department of Pharmacy Practice, Krishna Institute of Pharmacy, Krishna Vishwa Vidyapeeth (Deemed to be University), Karad,
[7] MS Regulatory Affairs College of Professional Studies Northeastern University Boston Ma 02115,

Corresponding Author: Dr. Sana Shaikh

DOI: https://doi.org/10.52403/ijhsr.20240112

ABSTRACT

Food allergy (FA) has significantly increased in the last decade, and its diagnosis is a continuous challenge. Mild cases are often neglected or detected late, and in children, parents may not be able to accurately interpret symptoms. It is crucial to differentiate FAs from food intolerance and toxic reactions. To provide personalized management, accurate diagnosis is essential. Modern diagnostic tests, such as component diagnosis and epitope reactivity, allow for a more accurate therapeutic approach and reliable prognosis evaluation. Investigations like serum IgE, elimination diets, oral food challenges, single, blind, and double-blind tests, and skin tests are used. Anaphylaxis risk can be assessed using molecular diagnostics/component-resolved diagnosis (CRD) and a basophilic activation test (BAT). These tests allow for planned, individualized therapy based on molecular and clinical characteristics. Understanding immunological processes, diagnostics, and immunotherapies in FAs is crucial for evaluating food allergen exposure, detecting allergic responses, analyzing clinical manifestations, highlighting diagnostic options, and demonstrating appropriate therapeutic strategies.

Keywords: Food allergy, paediatric, basophilic activation test, component-resolved diagnosis

INTRODUCTION

Food allergies are immunologic reactions to food proteins, causing over 20% of diet changes in adults and children [1]. Food intolerances are allergic responses not caused by the immune system; these can occur from metabolic substances, toxins, or pharmacologically active components. Food allergies that are not mediated by IgE are often misunderstood as food intolerances [2].

PREVALANCE

Prevalence in India

The prevalence of food allergy in India is not well understood, especially among children. Current studies rely on self-reported questionnaires, which can overestimate prevalence [3]. A EuroPrevall-INCO study found 1.2% prevalence in southern India, with cow's milk and apple accounting for 0.5 and 0.5 respectively.

More information on the prevalence of food allergies in children is awaited [4].

Prevalence in Central and East Asia

Research shows that the prevalence of paediatric food allergies varies significantly between Central and East Asian nations. In Thailand, a study found a food allergy prevalence of 1.1% in children aged 4-7 years [5], while in China, it was 6.2%[6]. In South Korea, it was 12.6% in children aged 12-15 years and 11.3% in children aged 6-12 years [7]. The major components of the Asian diet, such as fish, shellfish, bird's nest, buckwheat, and royal jelly, are the most frequently reported triggers for severe allergic responses. Hospital-based studies in Singapore [8], Thailand [9], and Hong Kong [10] suggest crustacean shellfish is a significant food trigger. Royal jelly has been linked to anaphylaxis in Hong Kong and Australia [11], while peanut allergy is rarely found in Asian populations [12].

Prevalence of Food Allergy among Immigrants of Asian Origin

A study by Koplin et al. [13] found a significant link between parental birth country and food allergy in Australian children aged 11 to 15 months. The study found that peanut allergy was three times more common in infants whose parents were born in East Asia than in Australian-born children. The study found that peanut sensitization was found in 5.4% of children with both parents born in Australia, 15.9% with one parent born in East Asia, and 19.6% with both parents born in East Asia. Peanut allergy was found in 2.3% of infants born in Australia with both parents, 6.7% with one parent born in East Asia, and 7.7% with both parents born in East Asia. The study's large sample size and standardized objective food allergy testing suggest that the rise in peanut allergy incidence in Australian children born to Asian parents appears to have happened in a single generation.

A recent survey study by Panjari et al. [14] investigated the link between parental migration and the development of food allergies in Asian children and children born in Australia. The study found that in a sample of 60,000 children, nut allergy prevalence was 3.1%, with peanut and tree nut allergies at 2.7% and 1.7%, respectively. Asian children were twice as likely to be allergic to nuts, while children born in Asia who later immigrated to Australia had a lower incidence of nut allergy. The study also found that parental migration from Asia to Australia may be a major risk factor in the development of nut allergies in the next generation.

Kamdar et al. [15] conducted a large-scale study in the United States, examining food allergy patterns among Asian Indian children and white children. The study involved 114 Asian Indian children, with an average age of 3.3 years and 66.7% being male. The most common food allergy was tree nuts, reported by 59% of the children. Other common allergies included chickpea flour, capsicum, and Indian lentils. Despite the small sample size, a wide range of food allergies were recorded, including avocado, banana, beef, bulgur wheat, coconut, maize, eggplant, garlic, ginger, green peas, jalapeo peppers, kiwi, melon, rice, and tomato. 11% of parents self-reported having a food allergy.

TYPES OF FOOD ALLERGENS

Food allergies in South Asia and the Indian subcontinent have been documented, with case studies and anecdotal accounts being the most common. In 1997, Patil et al [16] described two cases of acute allergic responses to fenugreek were reported, one by inhalation and the other through topical application of fenugreek seed powder. Fenugreek is a plant used in Indian cooking and is considered a traditional cure for diabetes, appetite stimulation, and milk production in breastfeeding women [17]. Inhaling fenugreek caused rhinorrhea, wheezing, and a brief loss of consciousness, while applying fenugreek to the scalp caused numbness, facial edema, and wheezing. Both patients and non allergic

controls tested for allergies to fenugreek and chickpea, with negative results in the control group.

Food allergy responses are influenced by geographical and ethnic disparities, possibly due to different eating habits. Traditional South Asian diets include grains and legumes like pigeon pea, black gram, and mung bean. A study by Patil et al. [18] in Mumbai, India, used in vivo and in vitro testing to diagnose patients with clinical histories of chickpea allergy. The skin prick test obtained a perfect score, and the diagnostic specificity was 64%. Skin prick testing and serum specific IgE measurement could be combined to accurately detect chickpea allergy in individuals with suggestive symptoms.

A study by Babu et al. [19] found a sex preponderance of sensitization and allergy to eggplant. A cross-sectional study on 741 individuals found a sensitization prevalence of 9.2%, with cutaneous responses (urticaria and pruritus) being the most prevalent allergic reactions. Cutaneous reactions affected approximately 60% of those who responded, followed by wheezing and rhinorrhea, which affected 30% of those who reacted. These findings suggest that a combination of skin prick testing and serum specific IgE measurement could be effective in detecting food allergies.

In 2013, Kasera et al. (20) the first instance of allergy to amaranth grain was reported in India. Amaranth grains, also known as rajgira in India, are minuscule buds of the Amaranthus plant's flowers and contain numerous nutrients. One female experienced anaphylaxis after taking her first rajgira, which she had consumed several years prior. A Pub Med search revealed that lentil allergy has been recorded in several countries, including Turkey, Spain, and Italy. Mushroom allergy has also been reported in Japan, Spain, and Turkey [21]. Mustard seed has been reported in Spain [22], and cumin has been reported in the United States [23]. Some cases of legume allergies, such as fenugreek and chickpea, have been reported in Norway [24], Belgium, and Spain. The increasing prevalence and recognition of food allergies in emerging nations is an atypical phenomenon, with studies revealing an increase in more westernized regions [25]. Factors such as altered cooking habits and greater consumption of processed foods should be studied as well.

RISK FACTORS

Food allergies are more common in males in childhood, but are more common in white people in North America. Non-white populations experienced a threefold rise in food allergies over a ten-year period [26]. Food allergies are caused by complex interactions of genetic and environmental variables in childhood, including male sex, ethnicity, genetics, microbial exposure, allergy exposure, and vitamin D deficiency [27]. In Australia, 12-month-old infants with East Asian parents had a threefold greater incidence of food allergies compared to non-East Asian descent [13]. The breakdown of skin barrier integrity and delay in introducing potentially allergenic foods to infants can promote topical sensitization and evade oral tolerance. The risk of food allergy increases in newborns with earlier start and severity of eczema [28]. Sibling risk is a common clinical problem, with 66.6% of siblings being food sensitive and 13.6% clinically reactive [29]. Concerns about the safety of childhood vaccinations have emerged, as the pathway of sensitization in IgE-mediated food allergy is cutaneous, and early-life consumption of foods like peanut might be protective [30].

DIAGNOSIS

An accurate Food Allergy (FA) diagnosis is crucial for providing education and management techniques to reduce the risks of potentially fatal allergic reactions. Correctly diagnosing food tolerance encourages dietary liberation, especially in light of the paradigm shift supporting early introduction of allergenic foods to avoid food allergy [31]. Double-blind placebo-

controlled food challenges are the gold standard for FA diagnosis, but their feasibility is limited due to inherent risks and high resource requirements.

Skin prick tests (SPT) and serum-specific IgE (sIgE) are used in clinical practice because they are relatively safe and affordable to conduct. However, the usual positive results show low specificity to clinical FA, with around half of sensitized individuals being able to tolerate the food without reacting. To reduce the requirement for diagnostic food challenges, numerous studies have identified thresholds for these tests with a 95% positive predictive value (PPV) to FA reviewed [32][33]. Although SPT and sIgE thresholds with 95% PPV to FA are routinely utilized to reduce the requirement for diagnostic food challenges, a proportion of children remain in the immunologic grey area, meaning they are food-sensitized but have a PPV of less than 95%. New techniques that can effectively detect FA while eliminating the need for food challenges are definitely needed.

Allergen component-resolved diagnostics (CRD) are recommended as a more accurate method of diagnosis because it measures sIgE to specific allergen proteins. A systematic review found that sIgE to Ara h 2 had higher diagnostic accuracy than other tests [34]. A meta-analysis of 19 studies revealed that while sIgE to Ara h 1, Ara h 2, and Ara h 3 had high specificity to peanut allergy, Ara h 2 had the highest sensitivity [35].

Another promising approach to FA diagnosis based on cellular tests appears to be more sensitive and specific than traditional techniques. The basophile activation test (BAT) involves flow cytometry to assess the expression of activation markers on the surface of basophiles stimulated with food allergens and controls [36]. In a study of 104 children, BAT outperformed SPT, sIgE, and sIgE to Ara h 2 in distinguishing between peanut-allergic and peanut-sensitized tolerant children [37].

The mast cell activation test (MAT) is another promising technique that, unlike BAT, utilizes stored plasma rather than fresh whole blood. In terms of specificity, MAT and BAT performed equally well, but MAT's sensitivity was lower than BAT's. Importantly, in all situations when basophiles were non-responsive, MAT provided definite results [38].

Despite continued advances and the development of novel molecular approaches, a reliable diagnostic test to eliminate the necessity for oral food challenges remains elusive. The appropriate threshold necessitates a trade-off between false negatives and false positives, and this varies in the published literature [39].

A possible strategy to enhance food allergy diagnosis without the requirement for OFC is proposed by several studies. This process requires performing first-line tests for conventional SPT and/or sIgE using established 95% PPVs. If the findings are unclear, a second-line test of CRD, BAT, or MAT might be requested, which has been demonstrated to significantly reduce the need for OFC [39]. However, if all tests are unclear, OFC remains the gold standard and may be necessary to confirm the diagnosis.

STRATEGY AND TREATMENT OF FOOD ALLERGY

The traditional approach to food allergy (FA) treatment focuses on patient education, strict avoidance of offending foods, and early treatment of adverse reactions. However, recent research has adopted a more active strategy, including early dietary introduction of potentially allergenic foods, active testing for related allergens, active monitoring and desensitization to known food allergens, and active risk management. These approaches can enhance quality of life and decrease the development of new allergies but may increase the complexity of managing children with FA. Primary prevention is the optimal scenario for actively managing FA.

In the past two decades, infant feeding advice has been modified, with

recommendations for complementary feeding postponed until six months, with longer delays for certain allergic foods like peanuts [40]. Current guidelines from 2008 recommend exclusive breastfeeding for six months and avoidance of potentially allergenic foods until six months of age, with no recommendation for maternal allergen avoidance during pregnancy or lactation. Research shows that early exposure of an allergic meal may play an essential role in avoiding FA development with certain foods [41] [42]

The Learning Early About Peanut Allergy (LEAP) study investigated the impact of early peanut consumption on the possibility of developing peanut allergy and reported an improvement in peanut tolerance in high-risk allergy patients. The Enquiring About Tolerance (EAT) research suggests that introducing six allergenic foods into infants' diets at three months of age results in a decreased prevalence of FA at three years of age. Interventional studies are ongoing to provide clear guidelines on infant introductory feeding.

EMERGENCY TREATMENT

Emergency therapy for anaphylaxis is tailored to the individual's severity, with patients with third-degree severity receiving intramuscular adrenaline. In severe cases, adrenaline is administered if symptoms have progressed, circulatory signs occur, or respiratory signs cannot be eased with inhaled bronchodilators [43] [44]. Treatment must begin promptly and vital signs are monitored. Adrenaline is usually administered at a dosage of 0.01 mg/kg, with a maximum dose of 0.5 mg for patients over 12 years and 0.3 mg for children under 12. If symptoms persist, intravenously infusions may be used [45] [46]. Body position is crucial in anaphylaxis management, with patients placed in a lateral decubitus position to prevent aspiration syndrome. Oxygen is administered using a 10 L/min O2 mask, and intravenous rehydration and rebalancing is done with Ringer's solution or saline at intervals. In case of respiratory disorders, Basic Life Support (BLS) procedures are followed, and vital signs are regularly reviewed.

IMMUNOTHERAPY AND ADMINISTRATION APPROACHES

Food allergens immunotherapy aims to induce specific antigen immune tolerance in food allergies [47], allowing patients to consume previously triggered foods without experiencing symptoms. This is achieved by administering food allergens in progressively increasing doses [48], with three current administration approaches

ORAL IMMUNOTHERAPY (OIT)

The treatment strategy for incriminated food involves consuming small amounts and gradually increasing the dosage [49] . No exact protocols for initiation, maintenance, and duration of therapy have been developed. The OIT is successful in inducing tolerance, but further research is needed on long-term consequences. Safety is modest, but anaphylaxis risk exists. Adjuvant therapy with Omalizumab is suggested to improve safety and efficacy. The age at which OIT can be initiated is suggested for children above six years, but opinions on the minimum age vary, necessitating further research and guidelines.

SUBLINGUAL IMMUNOTHERAPY (SLIT)

SLIT is a sublingual delivery method for allergenic proteins from food, which is less effective than oral immunotherapy (OIT) in building long-term tolerance [50]. However, it is safer than OIT in terms of safety. SLIT is recommended for patients who do not tolerate OIT or for desensitization. After sublingual administration, food is administered orally, reducing the risk of severe adverse effects. The development of quantified food extract-isolated proteins could address the method's limitations in protein administration.

EPICUTANEOUS IMMUNOTHERAPY (EPIT)

It is a skin-based treatment that has shown excellent safety with no reported anaphylaxis incidents. However, it can cause erythema, pruritus, eczema, and atopic dermatitis [51]. EPIT has modest results for developing food tolerance, but its main limitation is the amount of allergen administered, indicating a need for improvement in OIT's safety.

NEW TREATMENT STRATEGIES

New pharmacological therapies for food allergies aim to manage inducing cells and decrease allergic reactions [52]. These medicines are non-allergic and function non-specifically, differentiating them from immunotherapy. Targeted biological therapy and microbiome reconstitution agents are among the therapeutic strategies.

OMALIZUMAB

Omalizumab is an anti-IgE monoclonal antibody that works by binding to the Fc region of IgE antibodies, inhibiting receptor binding on mast cells and basophils [53], preventing degranulation and allergic manifestations. It should be initiated before the OIT and continued for a few weeks after [54]. The recommendation considers the increased safety profile and effectiveness of immunotherapy. However, the administration of Omalizumab in food allergy treatment is not standardised, and more research is needed to determine the optimal time to discontinue therapy and appropriate doses. It can be administered to children over six years old.

DUPILIMUMAB

Dupilumab is a human monoclonal IgG4 antibody that inhibits IL-4 and IL-13 signaling in adult patients with severe atopic dermatitis. Approved in 2017 by the US Food and Drug Administration and the European Medicines Agency, it is currently undergoing two Phase II clinical trials in the food allergy area [55].

CONCLUSION

The lack of data on food allergies among children in India necessitates detailed clinical history and targeted allergy testing for diagnosis. Oral food challenge is the gold standard in cases of diagnostic uncertainty. Active food allergy treatment offers benefits such as tolerance development and improved quality of life. Limiting allergenic potential without unnecessary restrictions is crucial. Parents and children should be educated on early allergic reactions and avoid high-risk foods. Understanding immunological processes, diagnostics, and immunotherapeutic alternatives is essential for developing strategies for diagnosing, treating, and preventing food allergies (FAs). However, a lack of food labelling policy and the non-availability of adrenaline autoinjectors hinder effective management of FAs in children in India.

Declaration by Authors

Ethical Approval: Not Required
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Sicherer SH, Sampson HA. Food allergy. J Allergy Clin Immunol. 2010 Feb;125(2 Suppl 2):S116-125.
2. Sicherer SH, Sampson HA. Food allergy: Epidemiology, pathogenesis, diagnosis, and treatment. J Allergy Clin Immunol. 2014 Feb;133(2):291–307; quiz 308.
3. Shek LP-C, Lee BW. Food allergy in Asia. Curr Opin Allergy Clin Immunol. 2006 Jun;6(3):197–201.
4. Mahesh PA, Wong GWK, Ogorodova L, Potts J, Leung TF, Fedorova O, et al. Prevalence of food sensitization and probable food allergy among adults in India: the EuroPrevall INCO study. Allergy. 2016;71(7):1010–9.
5. Lao-araya M, Trakultivakorn M. Prevalence of food allergy among preschool children in northern Thailand. Pediatr Int Off J Jpn Pediatr Soc. 2012 Apr;54(2):238–43.

6. Chen J, Liao Y, Zhang H, Zhao H, Chen J, Li H. [Prevalence of food allergy in children under 2 years of age in three cities in China]. Zhonghua Er Ke Za Zhi Chin J Pediatr. 2012 Jan;50(1):5–9.
7. Lee SI, Shin MH, Lee HB, Lee JS, Son BK, Koh YY, et al. Prevalences of symptoms of asthma and other allergic diseases in korean children: a nationwide questionnaire survey. J Korean Med Sci. 2001 Apr;16(2):155–64.
8. Thong BYH, Cheng YK, Leong KP, Tang CY, Chng HH. Immediate food hypersensitivity among adults attending a clinical immunology/allergy centre in Singapore. Singapore Med J. 2007 Mar;48(3):236–40.
9. Jirapongsananuruk O, Bunsawansong W, Piyaphanee N, Visitsunthorn N, Thongngarm T, Vichyanond P. Features of patients with anaphylaxis admitted to a university hospital. Ann Allergy Asthma Immunol Off Publ Am Coll Allergy Asthma Immunol. 2007 Feb;98(2):157–62.
10. Smit DV, Cameron PA, Rainer TH. Anaphylaxis presentations to an emergency department in Hong Kong: incidence and predictors of biphasic reactions. J Emerg Med. 2005 May;28(4):381–8.
11. Leung R, Lam CW, Ho A, Chan JK, Choy D, Lai CK. Allergic sensitisation to common environmental allergens in adult asthmatics in Hong Kong. Hong Kong Med J Xianggang Yi Xue Za Zhi. 1997 Jun;3(2):211–7.
12. Liew WK, Chiang WC, Goh AE, Lim HH, Chay OM, Chang S, et al. Paediatric anaphylaxis in a Singaporean children cohort: changing food allergy triggers over time. Asia Pac Allergy. 2013 Jan;3(1):29–34.
13. Koplin JJ, Peters RL, Ponsonby A-L, Gurrin LC, Hill D, Tang MLK, et al. Increased risk of peanut allergy in infants of Asian-born parents compared to those of Australian-born parents. Allergy. 2014 Dec;69(12):1639–47.
14. Panjari M, Koplin JJ, Dharmage SC, Peters RL, Gurrin LC, Sawyer SM, et al. Nut allergy prevalence and differences between Asian-born children and Australian-born children of Asian descent: a state-wide survey of children at primary school entry in Victoria, Australia. Clin Exp Allergy J Br Soc Allergy Clin Immunol. 2016 Apr;46(4):602–9.
15. Conference Abstract - Asian Indian food allergy survey: Unique ethnic food allergens [Internet]. [cited 2022 Apr 10]. Available from: https://www.mdlinx.com/allergy-immunology/conference-abstract.cfm/55242/?conf_id=207968
16. Patil SP, Niphadkar PV, Bapat MM. Allergy to fenugreek (Trigonella foenum graecum). Ann Allergy Asthma Immunol Off Publ Am Coll Allergy Asthma Immunol. 1997 Mar;78(3):297–300.
17. Fenugreek [Internet]. NCCIH. [cited 2022 Apr 8]. Available from: https://www.nccih.nih.gov/health/fenugreek
18. Patil SP, Niphadkar PV, Bapat MM. Chickpea: a major food allergen in the Indian subcontinent and its clinical and immunochemical correlation. Ann Allergy Asthma Immunol Off Publ Am Coll Allergy Asthma Immunol. 2001 Aug;87(2):140–5.
19. Harish Babu BN, Mahesh PA, Venkatesh YP. A cross-sectional study on the prevalence of food allergy to eggplant (Solanum melongena L.) reveals female predominance. Clin Exp Allergy J Br Soc Allergy Clin Immunol. 2008 Nov;38(11):1795–802.
20. Kasera R, Niphadkar PV, Saran A, Mathur C, Singh AB. First case report of anaphylaxis caused by Rajgira seed flour (Amaranthus paniculatus) from India: a clinico-immunologic evaluation. Asian Pac J Allergy Immunol. 2013 Mar;31(1):79–83.
21. Tepetam FM, Dağdeviren B, Bulut İ, Karabay CY, Barış S, Aydıner Karakoç E. A patient with mushroom allergy; a new etiological agent of Kounis syndrome. Tuberk Ve Toraks. 2016 Jun;64(2):171–4.
22. Malet A, Valero A, Lluch M, Bescos M, Amat P, Serra E. Hypersensitivity to mustard seed. Allergy. 1993;48(1):62–3.
23. Boxer M, Roberts M, Grammer L. Cumin anaphylaxis: a case report. J Allergy Clin Immunol. 1997 May;99(5):722–3.
24. Namork E, Fæste CK, Stensby BA, Egaas E, Løvik M. Severe allergic reactions to food in Norway: a ten year survey of cases reported to the food allergy register. Int J Environ Res Public Health. 2011 Aug;8(8):3144–55.
25. Gupta RS, Springston EE, Warrier MR, Smith B, Kumar R, Pongracic J, et al. The prevalence, severity, and distribution of childhood food allergy in the United States. Pediatrics. 2011 Jul;128(1):e9-17.

26. Branum AM, Lukacs SL. Food allergy among children in the United States. Pediatrics. 2009 Dec;124(6):1549–55.
27. du Toit G, Tsakok T, Lack S, Lack G. Prevention of food allergy. J Allergy Clin Immunol. 2016 Apr;137(4):998–1010.
28. Fernández-Rivas M, Bolhaar S, González-Mancebo E, Asero R, van Leeuwen A, Bohle B, et al. Apple allergy across Europe: how allergen sensitization profiles determine the clinical expression of allergies to plant foods. J Allergy Clin Immunol. 2006 Aug;118(2):481–8.
29. Gupta RS, Walkner MM, Greenhawt M, Lau CH, Caruso D, Wang X, et al. Food Allergy Sensitization and Presentation in Siblings of Food Allergic Children. J Allergy Clin Immunol Pract. 2016 Oct;4(5):956–62.
30. Du Toit G, Roberts G, Sayre PH, Bahnson HT, Radulovic S, Santos AF, et al. Randomized trial of peanut consumption in infants at risk for peanut allergy. N Engl J Med. 2015 Feb 26;372(9):803–13.
31. Ierodiakonou D, Garcia-Larsen V, Logan A, Groome A, Cunha S, Chivinge J, et al. Timing of Allergenic Food Introduction to the Infant Diet and Risk of Allergic or Autoimmune Disease: A Systematic Review and Meta-analysis. JAMA. 2016 Sep 20;316(11):1181–92.
32. Peters RL, Gurrin LC, Allen KJ. The predictive value of skin prick testing for challenge-proven food allergy: a systematic review. Pediatr Allergy Immunol Off Publ Eur Soc Pediatr Allergy Immunol. 2012 Jun;23(4):347–52.
33. Calvani M, Arasi S, Bianchi A, Caimmi D, Cuomo B, Dondi A, et al. Is it possible to make a diagnosis of raw, heated, and baked egg allergy in children using cutoffs? A systematic review. Pediatr Allergy Immunol Off Publ Eur Soc Pediatr Allergy Immunol. 2015 Sep;26(6):509–21.
34. Klemans RJB, van Os-Medendorp H, Blankestijn M, Bruijnzeel-Koomen C a. FM, Knol EF, Knulst AC. Diagnostic accuracy of specific IgE to components in diagnosing peanut allergy: a systematic review. Clin Exp Allergy J Br Soc Allergy Clin Immunol. 2015 Apr;45(4):720–30.
35. Nilsson C, Berthold M, Mascialino B, Orme ME, Sjölander S, Hamilton RG. Accuracy of component-resolved diagnostics in peanut allergy: Systematic literature review and meta-analysis. Pediatr Allergy Immunol Off Publ Eur Soc Pediatr Allergy Immunol. 2020 Apr;31(3):303–14.
36. Santos AF, Shreffler WG. Road map for the clinical application of the basophil activation test in food allergy. Clin Exp Allergy J Br Soc Allergy Clin Immunol. 2017 Sep;47(9):1115–24.
37. Santos AF, Douiri A, Bécares N, Wu S-Y, Stephens A, Radulovic S, et al. Basophil activation test discriminates between allergy and tolerance in peanut-sensitized children. J Allergy Clin Immunol. 2014 Sep;134(3):645–52.
38. Santos AF, Couto-Francisco N, Bécares N, Kwok M, Bahnson HT, Lack G. A novel human mast cell activation test for peanut allergy. J Allergy Clin Immunol. 2018 Aug;142(2):689-691.e9.
39. Dang TD, Tang M, Choo S, Licciardi PV, Koplin JJ, Martin PE, et al. Increasing the accuracy of peanut allergy diagnosis by using Ara h 2. J Allergy Clin Immunol. 2012 Apr;129(4):1056–63.
40. Burks AW, Tang M, Sicherer S, Muraro A, Eigenmann PA, Ebisawa M, et al. ICON: food allergy. J Allergy Clin Immunol. 2012 Apr;129(4):906–20.
41. Katz Y, Rajuan N, Goldberg MR, Eisenberg E, Heyman E, Cohen A, et al. Early exposure to cow's milk protein is protective against IgE-mediated cow's milk protein allergy. J Allergy Clin Immunol. 2010 Jul;126(1):77-82.e1.
42. Koplin JJ, Osborne NJ, Wake M, Martin PE, Gurrin LC, Robinson MN, et al. Can early introduction of egg prevent egg allergy in infants? A population-based study. J Allergy Clin Immunol. 2010 Oct;126(4):807–13.
43. Matyas M, Hasmasanu MG, Zaharie G. Antioxidant Capacity of Preterm Neonates Assessed by Hydrogen Donor Value. Med Kaunas Lith. 2019 Oct 30;55(11):E720.
44. Meyer R, Fox AT, Chebar Lozinsky A, Michaelis LJ, Shah N. Non-IgE-mediated gastrointestinal allergies-Do they have a place in a new model of the Allergic March. Pediatr Allergy Immunol Off Publ Eur Soc Pediatr Allergy Immunol. 2019 Mar;30(2):149–58.
45. Manuyakorn W, Tanpowpong P. Cow milk protein allergy and other common food allergies and intolerances. Paediatr Int Child Health. 2019 Feb;39(1):32–40.

46. Calamelli E, Liotti L, Beghetti I, Piccinno V, Serra L, Bottau P. Component-Resolved Diagnosis in Food Allergies. Med Kaunas Lith. 2019 Aug 18;55(8):E498.
47. Fogg MI, Brown-Whitehorn TA, Pawlowski NA, Spergel JM. Atopy patch test for the diagnosis of food protein-induced enterocolitis syndrome. Pediatr Allergy Immunol Off Publ Eur Soc Pediatr Allergy Immunol. 2006 Aug;17(5):351–5.
48. Calvani M, Bianchi A, Reginelli C, Peresso M, Testa A. Oral Food Challenge. Med Kaunas Lith. 2019 Sep 27;55(10):E651.
49. Yum HY, Yang HJ, Kim KW, Song TW, Kim WK, Kim JH, et al. Oral food challenges in children. Korean J Pediatr. 2011 Jan;54(1):6–10.
50. Paranjape A, Tsai M, Mukai K, Hoh RA, Joshi SA, Chinthrajah RS, et al. Oral Immunotherapy and Basophil and Mast Cell Reactivity in Food Allergy. Front Immunol [Internet]. 2020 [cited 2022 Apr 15];11. Available from: https://www.frontiersin.org/article/10.3389/fimmu.2020.602660
51. Schoos A-MM, Bullens D, Chawes BL, Costa J, De Vlieger L, DunnGalvin A, et al. Immunological Outcomes of Allergen-Specific Immunotherapy in Food Allergy. Front Immunol [Internet]. 2020 [cited 2022 Apr 27];11. Available from: https://www.frontiersin.org/article/10.3389/fimmu.2020.568598
52. Perkin MR, Logan K, Bahnson HT, Marrs T, Radulovic S, Craven J, et al. Efficacy of the Enquiring About Tolerance (EAT) study among infants at high risk of developing food allergy. J Allergy Clin Immunol. 2019 Dec;144(6):1606-1614.e2.
53. Čelakovská J, Bukač J, Vaňková R, Salavec M, Krejsek J, Andrýs C. Allergy to walnuts and hazelnuts in atopic dermatitis patients and analysis of sensitization to molecular components. Food Agric Immunol. 2021 Jan 1;32(1):105–20.
54. Łubiech K, Twarużek M. Lactobacillus Bacteria in Breast Milk. Nutrients. 2020 Dec;12(12):3783.
55. Regeneron Pharmaceuticals. A Study to Evaluate the Efficacy and Safety of Dupilumab Monotherapy in Pediatric Patients With Peanut Allergy [Internet]. clinicaltrials.gov; 2021 Aug [cited 2022 Apr 26]. Report No.: NCT03793608. Available from: https://clinicaltrials.gov/ct2/show/NCT03793608

How to cite this article: Sana Shaikh, Ab Samad, Ab Haque Bamer, Sunil S. Tade, Areeb Inamdar, Bijoy Panda Kumar et.al. Treatment and strategy for food allergies in paediatrics. *Int J Health Sci Res.* 2024; 14(1):93-101. DOI: *https://doi.org/10.52403/ijhsr.20240112*

******