

REVIEW ARTICLE

Year : 2013 | Volume : 27 | Issue : 2 | Page : 108--114

Food allergy in bronchial asthma: Diagnostic modalities

Raj Kumar

Department of Respiratory Allergy and Applied Immunology, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

Correspondence Address:

Raj Kumar

Department of Respiratory Allergy and Applied Immunology, National Centre of Respiratory Allergy, Asthma and Immunology (NCRAAI), Vallabhbhai Patel Chest Institute, University of Delhi, Delhi - 110 007
India

Abstract

Food hypersensitivity is estimated to be approximately 2% in adults and 8% in children. The incidence of perceived adult food hypersensitivity varies (1.4-30%) largely across the different countries. Allergen sensitization in different countries may be the result of interchange of genetic factors, cultural and dietary habits and or exposure to new allergenic products early in life. The diagnosis of food allergy is difficult due to lack of definitive laboratory test. The clinical observation is very important. The diagnosis and confirmation of adverse reaction to specific food should initially involve obtaining a good history of symptoms that may indicated the presence of food related adverse reaction. The diagnosis is confirmed by double blind placebo controlled food challenge test, which is difficult and may have sever reaction. Hence this test should be done in the hospital set up where the facilities to treat the allergic reactions are present.

How to cite this article:

Kumar R. Food allergy in bronchial asthma: Diagnostic modalities. Indian J Allergy Asthma Immunol 2013;27:108-114

How to cite this URL:

Kumar R. Food allergy in bronchial asthma: Diagnostic modalities. Indian J Allergy Asthma Immunol [serial online] 2013 [cited 2014 Jan 9];27:108-114

Available from: <http://www.ijaai.in/text.asp?2013/27/2/108/124392>

Full Text

Introduction

"One man's meat is another man's poison" (Lucretius 96-55 BC). Any unusual or unexpected reaction to ingested food is regarded as adverse reaction to food. It may be due to food intolerance which is physiologic response and/or hypersensitivity (allergy), which is an adverse immunologic reaction. Food allergy (FA) manifestation involves immunoglobulin (Ig) E-mediated antigen-antibody reaction through T cell and immune complex mechanism. FA is abnormal or exaggerated immunologic response to specific food protein. It may be IgE-mediated with rapid onset of action and non-IgE-mediated which takes hours to produce symptoms.

In recent years, food allergies and intolerance have attracted considerable attentions from the food industry and governmental regulatory agencies around the world. Even among the medical community, food allergies have attracted reasonable attention. Why this increased interest? Some have argued that the prevalence of FA or at least severity of FA is

increasing. At least in USA, the next major event was the publication of two paper describing death associated with food allergic reaction. [1],[2]

Food hypersensitivity is estimated to be approximately 2% in adults and 8% in children. [1] The incidence of perceived adult food hypersensitivity varies (1.4-19.1%) largely across different countries. [2] Allergen-sensitization varying in different countries may be the result of interaction of genetic factors, cultural, and dietary habits and/or exposure to new allergenic products early in life. Studies show higher prevalence of allergy to soy in Japan; peanut allergy in the UK, France, Switzerland, and North America; and sesame allergy in Israel. [3] Legumes are an important source of immunoglobulin E (IgE)-mediated reactions in Mediterranean and Asian countries. [4],[5],[6] Allergic diseases have increased worldwide, including Asian countries. [7],[8] The food hypersensitivity reaction is summarized in [Table 1].{Table 1}

Most of the world's population lives in Asia. However, not much published data about FA is available from this part of the world. This problem may be due to large population with diverse racial, cultural, and socioeconomic condition. Asia is also unique as its range of different cultures and eating habits. The most common cause of food-induced anaphylaxis in a cohort of children from Singapore is bird's nest (27%), followed by egg and milk (11% combined). [9]

The true prevalence of FA in the general population in Asia is uncertain. Estimates from Chinese studies range from 4.98 to 16.4%. [10],[11] The latter percentage represented those diagnosed by skin prick test (SPT) and therefore reflects sensitization rather than true clinical allergy. In the late 1990s, a parent-reported questionnaire of children from Singapore estimated the prevalence of FA to be 4-5%. [12] A Korean study using a self-reported questionnaire in a huge cohort of children reported a lifetime prevalence of 10.9%. [13]

Similarly, variable FA prevalence was noted in other Asian countries: As high as 5.5% in a cohort of Japanese children and as low as 1.2-1.7% in Israeli infants as assessed by detailed questionnaire and SPTs. [3] A cross-sectional study of 656 children of 6 months-6 years of age in Thailand showed a prevalence of 6.25% in children less than 6 years based on a parent reported questionnaire survey versus the 0.45% established through SPT and food challenge. [14] This illustrates the discrepancy between reported and medically diagnosed allergy.

Recent studies in India show a considerable increase in the prevalence of bronchial asthma (8-15%). [15],[16] When compared with the earlier report of <1%. [17] Many asthma and rhinitis patients may be suffering from FA, but our knowledge about food hypersensitivity is limited to few studies carried out in the country. [18],[19],[20] These studies indicate that foods such as egg, milk, cereals, and legumes commonly induce IgE-mediated reaction in children and adult population.

Legumes are important causative agents of type I hypersensitivity in south Asia and Europe, but such studies are lacking in Indian population. A study [21] investigated blackgram sensitization in asthma and rhinitis patients and identifies IgE-binding proteins. Blackgram induces IgE-mediated reactions in 1.7% of asthma and rhinitis patients and contains eight major IgE-binding components, of which six retained IgE reactivity after roasting. Blackgram shares allergenicity with lentil and lima bean. A case [22] with definite history of allergy (asthma) was investigated using standard in vivo and in vitro tests. Double blind oral challenge test with dehusked horse gram (pulses) showed severe pulmonary obstruction. Intradermal tests with seed extracts (pulses) revealed marked positive skin reactions. Further, enzyme-linked immunosorbent assay (ELISA) with patient's serum showed significantly elevated IgE levels to various seed extracts compared to normal control. Immunoblotting with patient's serum showed four to ten IgE binding proteins with molecular weights (MWs) 45, 38, 35, 30, 28, and 11 kDa to be common in the food extracts, prepared from seeds of family Leguminosae. In another study, it was concluded that the purified 28 kDa protein is a potent major allergen that may have implication in diagnosis of blackgram allergy. [23]

Sensitization to food varies in different countries reflecting a possible interaction of genetic factors and cultural and dietary habits. Rice is a major food consumed worldwide and evaluated for IgE-mediated reactions. A study [24] was carried out to identify rice allergy in patients of rhinitis and asthma and to identify the allergenic proteins in raw and cooked rice. Of these, 20 (12.1%) patients demonstrated marked positive SPT and 13 showed significantly raised specific IgE to rice compared to normal controls. Double blind placebo controlled food challenge (DBPCFC) confirmed rice allergy in six out of 10 patients. Immunoblot with hypersensitive individual patients' sera showed 14-16, 33, 56, and 60 kDa proteins as major IgE-binding components in rice. Boiled rice retained four IgE reactive proteins of 16, 23, 33, and 53 kDa. IgE-mediated rice allergy affects 0.8% (0.42-1.58 at 95% confidence interval (CI)) of asthma and rhinitis cases. The subjects with severe SPT reactions (4 mm or above) and specific IgE, 6.9 ng/ml to rice demonstrated positive blinded food challenge with clinical symptoms.

Studies suggest the importance of serum total and specific IgE in clinical evaluation of allergic manifestations. Such studies

are lacking in Indian subcontinent, though a large population suffer from bronchial asthma. In a study, [25] the relevance of serum total and specific IgE was investigated in asthmatics with food sensitization. A total of 216 consecutive patients (mean age 31.9 years, SD 11.8) were screened by various diagnostic testing. Out of 216 patients, 172 were with elevated serum total IgE (201->800 IU/ml). Rice elicited marked positive SPT reactions in 24 (11%) asthma patients followed by blackgram 22 (10%), lentil 21 (9.7%), and citrus fruits 20 (9.2%). Serum total IgE and specific IgE showed significant correlation, $P = 0.005$ and 0.001 , respectively, with positive skin tests. Blinded food challenges (DBPCFC) with rice and blackgram confirmed food sensitization in 28-37% of cases. Hence concluded, serum total IgE of 265 IU/ml or more with marked positive SPT (4 mm or more) can serve as marker for atopy and food sensitization. Specific IgE, three times of normal controls correlates well with positive DBPCFC and offers evidence for the cases of FA.

Studies have shown an association between food sensitivity asthma and or other atopic disorders. But the knowledge about the true prevalence of food hypersensitivity in respiratory allergy patients is lacking in India. In a study, [26],[27] it was investigated the prevalence of FA in patients of asthma and rhinitis. Of 1,860 patients (mean age 30 ± 12 years) screened using standard questionnaire, 1,097 (58.9%) gave history of FA. Of 470 patients (history positives) skin prick tested, 29.3% showed positive reactivity to at least one food, with rice (6.2%) as top sensitizers followed by blackgram (5.9%), lentil (5.5%), and citrus fruits (5.3%). Patients with history and SPT positive reactions showed elevated specific IgE levels (0.98-79 IU/mL) against respective food allergens than normal controls (0.60-0.80 IU/mL). Prick to prick test with fresh foods demonstrated more positive reactions than SPT with antigen extracts ($P = 0.0001$). Of 184 open food challenges (OFC), most of the positive OFCs were obtained with rice, [20] followed by citrus fruits, [14] banana, [10] and blackgram. [6] Among blinded challenges on 45 individuals, 21 (47%) were assessed as positive. With DBPCFC as the end point, the prevalence of food sensitization was 1.1% (0.72-1.75 at 95% CI) of 1,860 patients. We concluded that the perception of food-related symptoms is common among respiratory allergy patients. Self-reports could be confirmed in 21/470 (4.4%), resulting in an overall estimated prevalence of 1.1% (0.72-1.75 at 95% CI) in respiratory allergy patients. The common offending foods are rice, blackgram, citrus fruits, and banana.

Food allergies are the single most common cause of generalized anaphylaxis seen in hospital emergency department accounting for about one-third of the cases. It is estimated that about 100 fatal cases of food induced anaphylaxis occur in USA each year. In addition to the cutaneous, respiratory, and gastrointestinal symptoms noted above; patients may have cardiovascular symptoms, including hypertension, vascular collapse, and cardiac dysrhythmia; presumably caused by massive mast cell mediator release. However, most food induced anaphylactic reactions are not associated with increase in serum tryptase.

Laboratory Tests and Diagnostic Approaches

Another cause of the confusion about FA has been the lack of definitive laboratory test to define disease. Clinical observation has been the main stay of diagnosis. The diagnosis and confirmation of adverse reactions to specific food should initially involve obtaining a history of symptoms that may indicate the presence of food-related adverse reaction.

A variety of the diagnostic approaches which are listed below may be utilized in an attempt to establish the diagnosis of food hypersensitivity.

HistoryDiet diagnosisElimination dietSkin prick testEstimation of specific IgEOpen challengeSingle blind challengeDouble blind placebo controlled food challenge.

Initial History

One must direct the questioning so that history is focused on:

The suspected foodsThe timing between the ingestion of food and on onset of symptomsThe smallest quantity of food that the patient suspects will produce symptomThe frequency and reproducibility of the reactionwhether exercise was associated with the reactionThe most recent occurrence of the putative food reaction, andDescription of the signs such as rhinitis, urticaria, eczema, nasal discharge, cough, asthma, sputum production, vomiting, diarrhea, cold, etc.

The initial history should include presence or absence of the following symptoms: Hives, choking, harshness of voice, fainting, sneezing, rhinorrhea, nasal congestion, watery itchy eye, breathlessness or wheezing, eczema, pruritic,

erythematous rash, swelling, nausea, vomiting, urticaria, abdominal pain, diarrhea, etc., If one or more of these symptoms are present, information should be obtained regarding the time relationship and reproducibility of the symptoms with regard to consumption of the suspected food and the amount consumed. At this point if there is reasonable probability that symptoms are related to food consumption, the procedure given below should be followed to confirm or reject the diagnosis of FA.

Physical Examination

If history is suggestive of FA, a physical examination should be performed. Pulmonary function of the patient should be assessed.

The physical examination should emphasize the cutaneous, respiratory, and gastrointestinal systems. In particular, skin rashes, abnormalities of nose and ears, unusual auscultatory findings, and abnormal discomfort should be noted. Main symptoms and signs include gastrointestinal complains, such as abdominal pain, nausea, vomiting, diarrhea, cramps, and gas. Cutaneous symptoms and signs have included pruritis caused by eczema, urticaria, angioedema, and nonspecific erythematous rashes that have been reproduced. Respiratory symptoms and signs have been reported in blind challenges which include sneezing, ocular infection, serious rhinorrhea, laryngeal edema, cough, bronchorrhea, and wheezing. Cardiovascular symptoms and signs include tachycardia, arrhythmia, and hypotension.

Skin Tests

SPTs with extracts of suspected food are done to diagnose IgE-mediated FA. If the reaction to the skin test material is definitively positive and there is a history of an immediate reaction to isolated food ingestion and clinical manifestation of allergic reaction (generalized urticaria/angioedema, masked laryngeal edema, significant wheezing, or hypotension), diagnosis of food-related adverse reaction can be made. At present, food skin tests with food extracts can be reliably performed by prick/puncture test. They have been demonstrated to detect antibody response in patients known to be sensitized. A positive skin test is indicated by the development of a wheal that is 3 mm or larger in diameter within 10-30 min. SPT reactions are graded based on the weal size of positive control, that is, histamine diphosphate (5 mg/ml).

Elisa/Radioallergosorbent Test (Rast)

In all clinical situations the skin tests are the preferred method for identifying sensitization to food allergens. Clinician should be aware that in vitro test results may vary among laboratories and a laboratory with adequate quality control should be chosen.

Elimination Diet

The purpose of food elimination diet is to know if the patient's symptoms will resolve if particular food is restricted from the diet. Several variations of the elimination of diet may be used, depending on patient's history. The resolutions of symptoms after avoidance of particular food item coupled with positive prick test is a strong supportive evidence of specific food hypersensitivity and should be verified by controlled food challenge. When the patient confirmed that foods cause symptoms, but no particular food is suspected, then elimination diet may be undertaken for 7-14 days. The elimination of food item should be done one by one. This procedure may take months and may still not be rewarding. In contrast, after completion of 1-2 weeks on a strict elimination diet, most patients will be able to state unequivocally that the diet has been very helpful or useless. If the patient's symptoms do not vanish with an eliminated diet it is very unlikely that inspected substance are the problem. If the elimination diet has been helpful, then the physician should proceed to either an open challenge test in selected circumstances or directly to DBPCFC.

Food Challenge Precautions

Before performing any food challenge a tough discussion must be undertaken with patient and/or guardian. The purpose of the procedure must be explained including a discussion of both beneficial and potential adverse reaction. The procedure should be done where management of anaphylaxis can be assured.

Open food challenge

In the open challenge, the patient ingests the suspected food and is observed for reaction. Both patient and observer (physician) are aware of the content of challenge.

Single blind challenge

Single blind challenge differs in that the patient is unaware of what he/she is eating. If these diagnoses are done in an office or hospital setting, environmental factors which could influence the outcomes of the challenge can be accounted for, psychological factors can be reduced, but subjective attitudes cannot be eliminated.

Double blind placebo controlled food challenge

DBPCFC is the state of art technique to confirm the adverse reaction to foods. This is the "gold standard" by which all studies of FA should be justified. It is also a practical procedure by which practicing allergist can accurately diagnose adverse food reaction with minimum preparation. DBPCFC is the only reliable method to confirm a suspected diagnosis of FA. [28] The double blind challenge with placebo control, where neither patient nor observer is aware of the contents of the challenge, can control for psychological factors of patients and physician bias.

In the developing recipes for DBPCFC some general principles are to be observed. [29] The concentration of suspected agent hidden in the food should be as high as possible without being detectable; the use of any ingredient, other than the active agent, that could provoke any undesirable reaction should be avoided: The same ingredients should be used in both the active and placebo food. Beside taste and smell, the texture of offending food, that is, granularly, fibrousness, and fluidity should also be taken into account.

List of Ingredients in Recipes for DBPCFC

The advantage of dried food cannot be overstated because of their convenience and easy storage. Such foods as grains (wheat, rice, barley, corn, soya, etc.) are available as flour or meals. Nuts and legumes may be purchased and powdered. Dried milk and usually dried egg white are not difficult to find out.

Fish may be problem to prepare; they may be dried and hidden in the vehicle. Being their strong test, masking may be incomplete. However, capsules often help to mask the taste. [Table 2],[Table 3],[Table 4],[Table 5] and [Table 6] shows recipe for DBPCFC with fresh fruit, vegetable, nuts, and milk. [30]{Table 2}{Table 3}{Table 4}{Table 5}{Table 6}

Vehicle

In DBPCFC the principle is to administer the challenge to the patient objectively to duplicate the conditions of timing and quantity noted in the history. Therefore, any vehicle used must allow for a truly blind challenge, masking smell, flavor, and texture of the food. The vehicle must be able to accommodate enough food in a small quantity so that multiple challenges during a few hours may be administered.

Capsules are preferred vehicle for most of investigators. Capsules have the advantage because a fairly large amount of food can be thoroughly hidden in a small space. For example, a whole egg, when dried, weighs about 5 g and this amount can be placed in about 10-15 capsules. Foods are packed into capsules by hand and excess food wiped from the outside of capsule. Placing all capsules in powdered sugar before wiping clean ensures a similar taste of both food and placebo capsule.

Milk shakes are a good vehicle for some food, as is ice cream flavored with some strong taste like grape flavor. At least two situations exist in which liquid challenge may be preferred to capsule challenge. The test is the "oral allergy syndrome" in which all the reported symptoms are limited to oropharynx. These patients may benefit from both liquid and capsule

challenges on separate occasions that may determine whether the reaction is due to local contact and/or systemic absorption. Initially, liquid challenge is preferred in patients suspected to being anaphylactically sensitive to food. Administration of quantities of suspected food in liquid may be more easily controlled and will not be subjected to waiting for capsular dissolution.

Placebo

Placebo portion of challenge may be arranged in several ways depending on the vehicle used. If capsule is used, a good placebo is dextrose, because of its commercial availability and ease of encapsulation. Many of vehicles mentioned can be used as a placebo without adding anything to them. It should be noted that, patient is not aware of what he is taking. One may occasionally omit the placebo entirely by giving the suspected food first and if reaction is negative or positive (impressive) may forgo the placebo. It is important that blind observer must be unaware of this choice.

Medication avoidance

Medication use to be avoided before food challenge includes antihistamines (96 h), beta-agonist (12 h), theophyllins (12 h), and cromolyn (12 h). It should be noted that withholding medication could result in confusion, between symptoms occurring caused by the food challenge versus those caused by inadequate medication. It is advisable to challenge after an overnight fasting to eliminate the possibility that previously ingested substances might alter the timing of the reaction.

Administration of the blind food challenge

A single blind food challenge may be used when the objectivity of observer can be assured. The allergist must be present during all challenges and should confirm suspected reactions. Administration of the food during challenge should be done in a graded fashion. The starting quantity should be less than that estimated by the patient to be required to produce symptoms. The dose may then be doubled at intervals specified by the history or every 15-60 min. If the reaction is said to take 45 min to occur, the challenge interval could be 1 h. The doubling dose should continue until the patient has convincing symptoms or 8-10 g of dried food or 60-100 g of wet food has been ingested as a single dose. It is obvious that by the time patient has reached to a dose of 8 g, patient may have had a total dose of as much as 15-20 g of dried food from beginning of test. If double blind challenge up to 8 g (dry) is negative then food must be introduced into diet openly. Occasionally, the open challenge may be positive in the face of a negative blind challenge.

Three possibilities may explain this observation:

The patient's threshold to response may be higher than achieved during the blind challenge, as suggested by the dose response relationship. Such possibility can be tested in blind challenge with greater quantities. The allergenicity of the food may be different because of the manner of preparation. The reaction may be due to psychological factor.

The symptom score sheet should be maintained by blind observer. If bronchoconstriction is suspected, spirometry should be done to detect early changes in airway function. The subjects should be monitored for 1-2 h after the completion of the challenge, since most major allergic symptoms would occur within this period. The challenge should be initiated in the morning having fasted overnight. A morning of blinded challenge with open challenge at lunch time does not put subject in under strain. Despite numerous claims delayed onset of food reaction caused by immunological mechanism are rare and limited, primarily to diarrhea observed in children, including gluten-sensitive enteropathy, and cow's milk and soy protein enteropathesis. [31] Delay in time of true late reaction in intestine, skin, and respiratory system caused by foods may occur, but their existence must be confirmed.

Treating of adverse reactions during challenge

Since adverse reactions, some potentially severe may occur during challenges, prompt management is essential. Appropriate personnel and equipment should be available to treat anaphylactic reactions. For any severe reaction to a food challenge, epinephrine (subcutaneously or intramuscular) is the drug of choice. Antihistamine and parental steroids can also be used. In situations in which shock is developing appropriate intravenous fluid replacement should be done promptly. If the symptoms do not promptly resolve, or if recur after treatment, gastric lavage to remove remaining food antigen should be considered.

References

1. Bruijneel-Koomen C, Ortolani C, Aas K, Bindslev-Jensen C, Bjorksten B, Moneret-Vautrin D, *et al.* Adverse reactions to foods. European Academy of Allergology and Clinical Immunology Subcommittee. *Allergy* 1995;50:623-35.

- 2 Crespo JF, Rodriguez J. Food allergy in adulthood. *Allergy* 2003;58:98-113.
- 3 Dalal I, Binson I, Reifen R, Amitai Z, Shobat T, Rahmani S, *et al.* Food allergy is a matter of geography after all: Sesame as a major cause of severe IgE-mediated food allergic reactions among infants and young children in Israel. *Allergy* 2002;57:362-5.
- 4 Ibanez Sandin D, Martinez San Ireneo M, Maranon Lizana F, Fernandez-Caldas E, Alonso Lebrero E, Laso Borrego T. Specific IgE determinations to crude and boiled lentil (*Lens culinaris*) extracts in lentil-sensitive children and controls. *Allergy* 1999;54:1209-14.
- 5 Pascual CY, Fernandez-Crespo J, Sanchez-Pasor S, Padial MA, Diaz-Pena JM, Martin-Munoz F, *et al.* Allergy to lentils in Mediterranean pediatric patients. *J Allergy Clin Immunol* 1999;103:154-8.
- 6 Sanchez-Monge R, Pascual CY, Diaz-Perales A, Fernandez-Crespo J, Martin-Esteban M, Salcedo G. Isolation and characterization of relevant allergens from boilded lentils. *J Allergy Clin Immunol* 2000;106:955-61.
- 7 Harwanegg C, Hiller R. Protein micro array in diagnosing IgE mediated diseases: Spotting allergy at a molecular level. *Expert Rev Mol Diagn* 2004;4:539-48.
- 8 Wang GW, Leung TF. Asthma epidemiology and hygiene hypothesis in Asia. *Allergy Clin Immunol Int* 2004;16:155-60.
- 9 Goh DL, Lau YN, Chew FT, Shek LP, Lee BW. Pattern of food-induced anaphylaxis in children of an Asian community. *Allergy* 1999;54:84-6.
- 10 Wang Z. An allergy prevalence survey in population of 10,144 people. *Zhonghua Liu Xing Bing Xue Za Zhi* 1990;11:100-2.
- 11 Kim JS, Ouyang F, Pongracic JA, Fang Y, Wang B, Liu X, *et al.* Dissociation between the prevalence of atopy and allergic disease in rural China among children and adults. *J Allergy Clin Immunol* 2008;122:929-35.
- 12 Lee BW, Chew FT, Goh DY. Changing prevalence of childhood allergic diseases in Singapore. In: 5th West-Pacific Allergy Symposium and 7th Korea-Japan Joint Allergy Symposium; 11-14 June, 1997. Seoul, South Korea; 1997. p. 17-22.
- 13 Lee SI, Shin MH, Lee HB, Lee JS, Son BK, Koh YY, *et al.* Prevalence of symptoms of asthma and other allergic disease in Korean children: A nationwide questionnaire survey. *J Korean Med Sci* 2001;16:155-64.
- 14 Santadusit S, Aththapaisalsarudee S, Vichyanond P. Prevalence of adverse food reactions and food allergy among Thai children. *J Med Assoc Thai* 2005;88:S27-32.
- 15 Chhabra SK, Gupta CK, Chhabra P, Rajpal S. Prevalence of bronchial asthma in school children in Delhi. *J Asthma* 1998;35:291-6.
- 16 Gaur SN, Sanjay R, Ashish R. Prevalence of bronchial asthma and allergic rhinitis among school children in Delhi. *Int Med J Thailand* 2004;20:8-13.
- 17 Vishwanathan R. Definition, incidence, etiology and natural history of asthma. *Indian J Chest Dis* 1964;6:108-24.
- 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* 2001;87:140-5.
- 19 Parihar H, Kumar L, Puri R, Kumar V. The incidence of allergic diseases and feeding patterns in children up to 2 years of age. *Indian J Paediatr* 1984;51:7-12.
- 20 Sharman J, Kumar L, Singh S. Allergenicity of common foods restricted in respiratory allergy. *Indian J Paediatr* 2000;67:713-20.
- 21 Kumar D, Kumar R, Sridhara S, Arora N, Gaur SN, Singh BP. Sensitization to blackgram in patients with bronchial asthma and rhinitis: Clinical evaluation and characterization of allergens. *Allergy* 2006;61:104-10.
- 22 Kumar R, Sridhara S, Verma J, Arora N, Singh BP. Clinico-immunologic studies on allergy to pulses - a case report. *Indian J Allergy Appl Immunol* 2000;14:15-20.
- 23 Kumari D, Arora N, Kasera R, Sridhara S, Kumar R, Singh BP. Isolation and characterization of a 28kDa major allergen from blackgram (*Phaseolus mungo*). *Immunobiology* 2012;217:895-904.
- 24 Kumar R, Srivastava P, Kumari D, Fakhr H, Sridhara S, Arora N, *et al.* Rice (*Oryza sativa*) allergy in rhinitis and asthma patients: A clinico-immunological study. *Immunobiology* 2007;212:141-7.
- 25 Kumar R, Singh BP, Srivastava P, Sridhara S, Arora N, Gaur SN. Relevance of serum IgE estimation in allergic bronchial asthma with special reference to food allergy. *Asian Pac J Allergy Immunol* 2006;24:191-9.
- 26 Kumar R, Kumari D, Srivastava P, Fakhr H, Arora N, Gaur SN, *et al.* Prevalence of food allergy and its impact on bronchial asthma and rhinitis patients. *Allergy* 2007;62 Suppl 83:167-551.
- 27 Kumar R, Kumari D, Srivastava P, Khare V, Fakhr H, Arora N, *et al.* Identification of IgE-Mediated food allergy and allergens in older children and adults with asthma and allergic rhinitis. *Indian J Chest Dis Allied Sci* 2010;52:217-24.
- 28 Huijbers GB, Colen AA, Jansen JJ, Kardinaal AF, Vlieg-Boerstra BJ, Martens BP. Masking food for food challenge: Practical aspects of masking foods for double blind placebo-controlled food challenge. *J Am Diet Assoc* 1994;94:645-9.

- 29 Noe D, Bartemucci L, Mariani N, Cantari D. Practical aspects of preparation of foods for double-blind, Placebo-Controlled food challenge. *Allergy* 1998;53 (Suppl 46):75-7.
- 30 Bock SA, Sampson HA, Atkins FM, Zeiger RS, Lehrer S, Sachs M, *et al.* Double blind placebo-controlled food challenge (DBPCFC) as an office procedure: A manual. *J Allergy Clin Immunol* 1988;82:986-97.
- 31 Kuitunen P, Visakorpi JK, Savilahti E, Pelkonen P. Malabsorption syndrome with cow's milk intolerance: Clinical findings and course in fifty-four cases. *Arch Dis Child* 1975;50:351-6.

Thursday, January 09, 2014

[Site Map](#) | [Home](#) | [Contact Us](#) | [Feedback](#) | [Copyright and Disclaimer](#)

IgE mediated

Cutaneous: Urticaria/angioedema, atopic dermatitis

Respiratory: Rhinoconjunctivitis, asthma

Gastrointestinal: Oral allergy syndrome, gastrointestinal anaphylaxis, infantile colic(subset), allergic eosinophilic gastroenteritis(subset), infantile gastroesophageal reflux(subset)

Generalized: Systemic anaphylaxis, food associated or exercise induced anaphylaxis

Non-IgE mediated

Cutaneous: Dermatitis, herpetiformis, contact dermatitis

Respiratory: Food induced pulmonary hemosiderosis

Gastrointestinal: Food induced enterocolitis syndrome, food induced proctocolitis syndrome, food induced enteropathy syndrome, gluten-sensitive enteropathy(ceeliac disease), allergic eosinophilic gastroenteritis syndrome, gastroesophageal reflux, dermatitis herpetiformis mechanism(s) unknown, migraine headache, cow milk induced intestinal blood loss

Whole Milk	100 ml	Water	10ml
Banana	120 g	Banana	120 g
Sugar	13 g (1 tbs)	Sugar	13 g (1tbs)
Curd	80 g	Water	120 g
Banana	60 g	Banana	150 g
Apple Sauce / Juice	100 g	Apple Sauce / Juice	13 g
Sugar	13 g	Sugar	Pinch amount

Active food	Weight	Placebo food	Weight
Hazelnut/peanut	25 g (2 tbs)		
Water	100 ml	Water	100 ml
Peppermint syrup	48 ml (4 tbs)	Peppermint syrup	42 ml
Rice flour	11 g	Rice flour	11 g
Cocoa powder	10 g (1 tbs)	Cocoa powder	10 g
Saffron	Pinch amount	Saffron	Pinch amount
Milled rice grains	1 tbs	Milled rice grains	2 tbs

Note: Undesirable reactions to any ingredients other than the active agent were avoided by choosing with great care ingredients for recipes

Active food	Weight	Placebo food	Weight
Fresh fruit pulp	100 g		
Sugar	13 g	Sugar	13 g
Wheat meal	11 g (1 tbs)	Wheat meal	11 g (tbs)
Peppermint syrup	12 ml (1 tbs)	Peppermint syrup	12 ml (tbs)
Coffee	50 ml	Coffee	100 ml
Saffron	Pinch amount	Saffron	Pinch amount

Note: The preparation of recipes and the method of hiding the taste, smell, color, and texture of ingredients to be used were carefully examined in our laboratory. Saffron, peppermint syrup, bitter cocoa powder, and groundnut coffee which were mask fresh fruit and nuts, thus masked the smell and flavor of the suspected food without any adverse reaction

Active food	Weight	Placebo food	Weight
Spinach or beets	70 g		
Vegetable marrow pulp	50 g	Vegetable marrow pulp	60 g
Wheat meal	22 g (2 tbs)	Wheat meal	22 g
Water	100 ml	Water	100 ml
Coffee	30 ml (2 tbs)	Coffee	100 ml
Peppermint syrup	24 ml (2 tbs)	Peppermint syrup	24 ml
Saffron	Pinch amount	Saffron	Pinch amount

Spinach and beets were squeezed after cooking vegetable marrow pulp and peel were cooked

Food	Wet weight (g)	Dry weight (g)	Weight/capsule
Rice	0.90	0.90	0.90
Wheat	0.90	0.90	0.90
Soy	2.83	0.68	0.68
Corn	8.25	0.66	0.66
Egg (dried)	2.36	0.59	0.59
Milk (defatted)	6 ml	0.61	0.61
Peanut	1.83	0.44	0.44

Vehicles for dried food: Capsules (00 white/orange, opaque gelatin, tinted with titanium oxide)