

ORIGINAL ARTICLE

Prevalence of confirmed IgE-mediated food allergy among adolescents in Turkey

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Background: Although food allergy is an important health problem in Western countries, the prevalence varies among geographic regions. There is limited data on the prevalence of food allergy especially for adolescent age group, and the data from Turkey and Mediterranean region are even scarce.

Methods: The study is a cross-sectional survey including a questionnaire followed by phone survey with families that have reported food allergy and clinical evaluation of children having a history compatible with food allergy after phone survey. The sample number of students has been calculated 9096 of a total of 210.000 students at the second stage (6th, 7th, and 8th grades) of state elementary schools in the metropolitan counties of Ankara province with the assumption of food allergy prevalence (P) = 1% and a δ value = 0.2 ($\alpha < 0.05$, $\beta = 0.8$).

Results: Of 11,233 questionnaires distributed at 34 schools, 10,096 (89.4%) have been returned. The number of reported food allergy was 1139 (11.2%), and it was reduced to 133 (1.3%) after phone survey. After clinical evaluation by skin test, specific IgE and double-blind placebo-controlled food challenge (DBPCFC), proven IgE-mediated food allergy was determined in 15 (0.15%) children. The foods most commonly resulting food allergy were peanut 0.05% and tree nuts 0.05%.

Conclusion: The prevalence of food allergy among adolescent age group has been confirmed to be comparatively low in Turkey. Peanuts and tree nuts were determined to be the most common causes of IgE-mediated food allergy.

Food allergy (FA) is defined as an adverse health effect arising from a specific immune response occurring on exposure to a given food (1). The prevalence of FA has been increasing in some countries and turning to be an important public health concern (2–4). Although it is a major health problem, data about the true prevalence of food allergy in the general population, especially among adolescent age group are insufficient (5). This age group needs particular attention because they seem to be the leading cause of increasing healthcare attendance for food allergy, and the majority of severe allergic reactions and deaths caused by FA appear to occur among teenage population (6).

For diagnosing food allergy, relevance of the symptoms to the suspected food and positive IgE-mediated hypersensitivity is important but the gold standard in definite diagnosis is double-blind placebo-controlled food challenge (DBPCFC) test (3). The time-consuming nature and need of experienced staff make it difficult to use this test in epidemiologic studies. There are only a small number of reports conducted among unselected groups using these standardized methods. The

frequency of self-reported food allergy varies between 10% and 15% in the general population, and actual rate is considered to be 1%–3% in the literature based on this small number of studies (5, 7). Data based on confirmed diagnosis are additionally insufficient among adolescent age group. There are two reports giving a frequency of food allergy among adolescents that have used DBPCFC but only a part of the patients could be tested during these studies (8, 9).

The aim of this study is to identify the rate of FA in the adolescent age group in Turkey based on confirmed diagnosis with oral food challenge and to document corresponding risk factors and clinical characteristics.

Materials and methods

The study design

Turkey, with approximately 70 million citizens, is the second most populated country in Europe, with 40% of its population

aged less than 18 yr. Ankara is the capital city of Turkey, with a metropolitan population of around 4.4 million. Ankara is one of the top in-migrating cities in Turkey and houses a heterogeneous group of people from a wide range of socioeconomic and sociocultural groups.

The study population

Data obtained from the Turkish Ministry of National Education revealed that the total number of students attending grades 6–8 (between 11 and 15 yr of age) of schools in Ankara was 210,000 in a total of 735 schools. A minimum sample size of 9096 was considered adequate for Ankara, taking the prevalence of FA as 1.0% and setting α error at <0.05 , β at 0.8, and d as 0.2%. The minimum sample size required was calculated as 11,500 students, after adjustment for an anticipated survey completion rate of 70%. A one-step cluster sampling design was used, and of the 735 registered elementary schools in Ankara, 34 were chosen randomly; all children attending grades 6–8 of selected schools were included.

The data collection tool

The first phase of the study was conducted from March 1, 2011 through May 31, 2011. A standardized questionnaire based on international FA guidelines and previous studies was used (1, 10–12) (Data S1). This questionnaire was pilot tested on 50 children previously diagnosed with FA. The questionnaires were distributed to the students to be filled at home by their primary caregivers.

After demographic data and contact details, parents were asked whether 'any food reaction characterized by swelling of lips, tongue, eye or face, itching on the skin, skin rash, urticaria, angioedema, rhinitis, conjunctivitis, cough, shortness of breath, syncope or loss of consciousness, which occurred within 2 h after consumption of any food', were present. Parents were asked to identify the responsible food. In case of a positive response for any of the above-mentioned symptoms, the child was accepted as having 'parent-reported FA'. The presence of physician-diagnosed allergic diseases of the children and their first-degree relatives was also asked.

Phone survey

In this phase, a pediatric allergy specialist contacted the parents of the children by phone, and a detailed clinical history was obtained. Patients with a clinical history consistent with IgE-mediated FA were invited to the outpatient clinic for further investigation (phone survey group). The parental reports were considered as not consistent with IgE-mediated FA in the following situations: (i) non-allergic food reactions (food poisoning, lactose intolerance, irritable bowel syndrome), (ii) any form of rash/skin eruption common in childhood, without any reaction after subsequent consumption of the same foods later in life, (iii) dislike of food because of the way it is cooked, (iv) puberty acnes. From students with histories not suggestive of FA, a random sample of 200 were

chosen and contacted by phone for further questioning to rule out false negativity.

Clinical evaluation

In phase 3, a detailed personal history and physical examination were completed. All participants were examined by the same pediatric allergy specialist. Skin prick tests and serum-specific IgE levels were evaluated for suspected foods in all patients who accepted further evaluation in our clinic.

Skin tests were performed with the responsible foods in commercially available extracts of standard food allergen (Stallargenes SA, Antony, France), prick-to-prick test with fresh fruits and vegetables on the volar surface of the forearm. All children who reported a reaction to chocolate were skin prick tested not only with cocoa and cow's milk but also with hazelnut, almond, peanut, walnut, and egg as these may be used in marketed chocolate in the country.

Each child was also tested with standard inhalant allergens (common grass, weed, tree and cereal pollens, *Dermatophagoides farinae*, *Dermatophagoides pteronyssinus* alternaria, and cat and dog dander) (Stallargenes SA). Reactions were read after 15 min and were assessed as positive if the wheal mean diameter was 3 mm or more than the reaction of the negative control (NaCl 0.9%).

The sIgE of the suspected foods was studied by means of ImmunoCAP methodology (PHADIA, Uppsala, Sweden). IgE levels higher than 0.35 kU/l were considered positive.

Challenge protocols were developed on the basis of FA work group report and EAACI position paper (13, 14). Double-blind placebo-controlled food challenge was performed if the result (s) of both or either one of SPT and sIgE was positive. Open oral food challenge test was performed for all patients with negative SPT and sIgE for suspected food(s), followed by a DBPCFC in case of a positive reaction. Foods were freshly prepared for each child. While preparing foods, great care was taken by the dietician to ensure the right quantity (Denver Instrument SI-234, Göttingen, Germany). Double-blind placebo-controlled food challenge was conducted by a well-trained nurse and supervised by a pediatric allergist. Hazelnut, peanut, and walnut were masked in chocolate pudding and oat grains. Kiwi, tomato, and banana were masked in grape juice, pear pulp, and a vegetable juice produced by crushing fresh mint, parsley, and thyme together in a bowl. Green, yellow, and red liquid were used. Cocoa was administered in a form diluted with warm water, and fine ground corn flour, brown liquid coloring, and cocoa essence were used for placebo. All active and placebo foods were similar in appearance, color, flavor taste, odor, consistency, texture, and mouthfeel. The dose interval was 15 min. Oral food challenge symptom score sheet was used to determine the challenge reactions (13). The challenge was stopped if clinical symptoms occurred or the highest dose was reached. Individuals were observed for 120 min for immediate reactions.

Food challenge was not performed when a patient had a convincing history of anaphylaxis to the suspected food within 2 yr. Negative DBPCFCs were followed by open challenges. In the open challenge, patients received a meal size with the

portion of their age. The foods previously suspected for FA were added to the diet of the patient in case of a negative test.

Ethical issues

The survey was undertaken following the approval of the local ethics committee (Nos: HEK 10/118-36, TBK 11/20-9). A written informed consent was obtained from the parents of all children.

Statistical analysis

Statistical analyses were performed using spss, ver. 15, statistical software package (SPSS, Inc., Chicago, IL, USA) for Windows. Percentages of positive and negative answers were presented as valid percents. The Chi-squared test was used to study the significant differences in distribution across the

groups. A p-value of ≤ 0.05 was considered indicative of statistical significance.

Results

Overall, 11,233 questionnaires were distributed, 10,096 of which were retrieved, including those from 208 parents who did not wish to participate in the study (Fig. 1). Thus, the completion rate for Phase 1 was 89.9%.

Description of the study population

The mean age was 12.9 ± 0.9 yr and 49.7% were boys. The rate of lifetime parent-reported FA was 11.3% (95% CI: 10.7–11.9), and the prevalence of reporting symptoms following intake of the same food in previous year was determined as 3.6% (95% CI: 3.2–3.8).

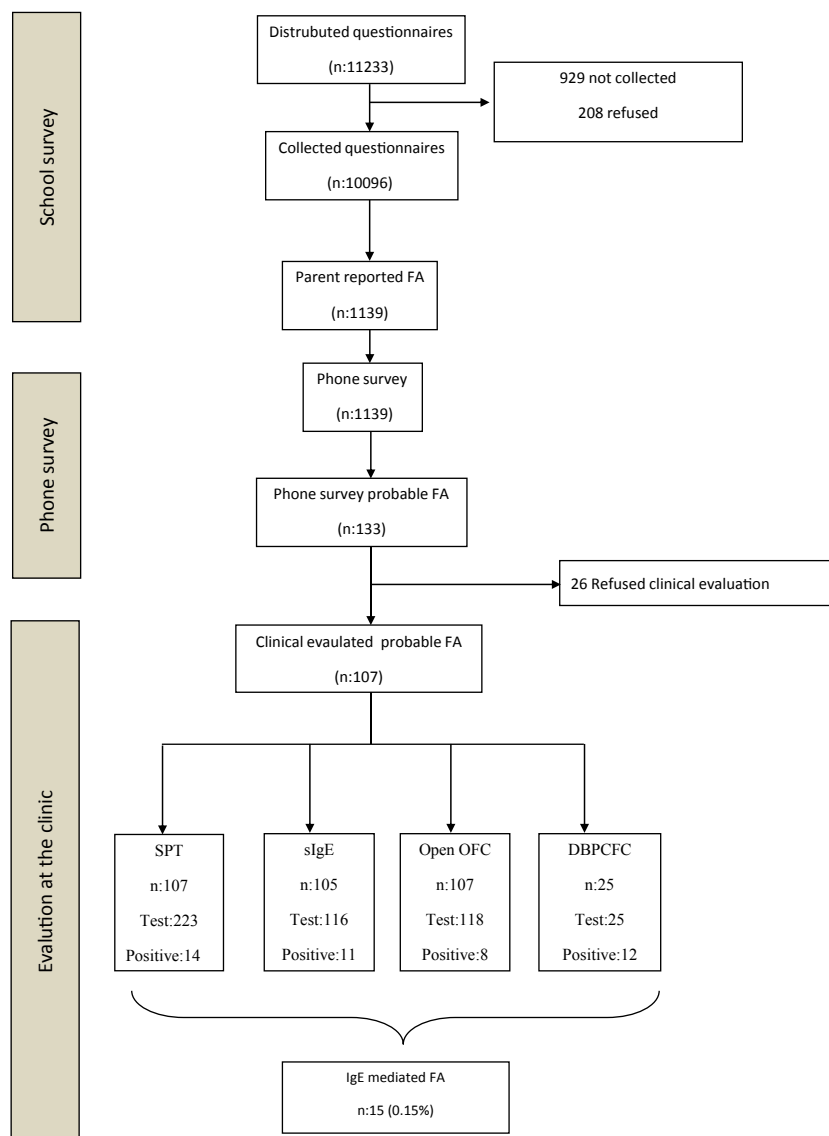


Figure 1 The study flow chart.

No significant association was detected between having a parent-reported FA and age ($p = 0.095$). However, the rate of parent-reported FA was higher among girls than in boys ($p < 0.001$). The prevalence of physician-diagnosed asthma, allergic rhinitis, drug allergy, and eczema was significantly higher in children with a suspected FA ($p < 0.001$). The most commonly cited allergic symptoms were related to cutaneous, respiratory, and gastrointestinal systems. A first-degree family history of a physician-diagnosed asthma and allergic conjunctivitis was more common in children with parent-reported food allergy ($p < 0.001$) (Table 1).

The foods most commonly reported were hen's egg (3.6%), chocolate (3.5%), tomato (1.2%), tree nuts (1.2%), and cow's milk (1.1%). Nine hundred and twenty (68%) children were reported to have reaction to one food only, while 419 (36.8%) had suspected reactions to more than one.

Phone survey

All parent-reported FA patients were contacted by phone. The frequency of clinical history consistent with IgE-mediated FA was 1.3% (with a 95%CI of 1.1–1.5). The foods most commonly excluded from the suspected FA list by means of phone survey were cow's milk and hen's egg. The phone survey on randomly selected 200 participants with negative parent-reported FA confirmed that none of them had ever showed any symptoms consistent with FA.

Results of evaluation for FA at the outpatient clinic

Of the 133 cases whose clinical histories were consistent with FA, 107 consented to participate in further clinical evaluation.

About one-third (36.4%) of these children were boys, with a median age of 12.9 yr (25%–75%, 12%–13.3%). The rates of children reporting cutaneous, digestive, respiratory, and ocular symptoms were 86.8%, 11.0%, 6.6%, and 0.7%, respectively. The reaction was reported to involve more than one organ system for 5 (4.7%) of these children. Among these 107 patients, pollen sensitization was found in 17 and house dust mite sensitization in two patients while six patients were sensitized to both allergens. There was *cladosporium* sensitization in one patient. In total, 26 (24.3%) patients among 107 had sensitization to at least one aeroallergen. Ten patients were diagnosed as allergic rhinitis, six as atopic dermatitis, and two as chronic urticaria. The median peripheral eosinophil count of the children was $100 \times 10^3/\mu\text{l}$ (min: 0, max: 1100), median eosinophil percent was 2% (min: 0.2, max: 17.3). The median serum IgE was 62 IU/ml (min: 16.4, max: 2240). Most common suspected foods were hen's egg, cow's milk, tree nuts, fruits, and spices. About one-fourth (23.3%) of the 107 children reported multiple FA.

A total of 223 skin prick test, 116 sIgE, 118 open food challenge, and 25 DBPCFC were administered to 107 patients. Skin prick test confirmed that one child had sensitization to five, another to two different foods, and the remainder had single-food sensitization (Table 2). Among the 107 patients, 16 had sensitization to at least one-food allergen. Five patients showed adverse reaction with peanut, four patients with walnut, and one patient for each of hazelnut, honey, cocoa, kiwi, tomato, and banana as a result of the applied provocations (Table 3). One patient with negative skin prick test and sIgE to peanut had wheezing and decreased FEV1 during open OFC, and thus, DBPCFC was not undertaken. Another patient who had urticaria during open sesame provocation

Table 1 Demographic and clinical characteristics of the study population

	Total (n = 10,096)	Parent-reported 'FA'			Confirmation by the phone survey		
		Positive (n = 1139)	Negative (n:8957)	p	Positive (n:133)	Negative (n:1006)	p
Age (mean $\pm$ s.d.)	12.9 $\pm$ 0.9	12.9 $\pm$ 0.9	12.9 $\pm$ 0.9		12.9 $\pm$ 0.9	12.8 $\pm$ 0.1	0.132
Male, n(%)	5019 (49.7)	468 (41.1)	4551	<0.001	44 (33.1)	4974 (49.9)	0.001
Physician-diagnosed allergic disease							
Asthma	1241 (12.3)	234 (20.5)	1007 (11.2)	<0.001	20 (15.0)	1221 (12.3)	0.347
Allergic rhinitis	1630 (16.1)	350 (30.7)	1280 (14.2)	<0.001	46 (34.6)	1584 (16.0)	<0.001
Eczema	353 (3.5)	67 (5.9)	286 (3.1)	<0.001	7 (5.3)	346 (3.5)	0.272
Drug allergy	275 (2.7)	95 (8.3)	180 (2)	<0.001	20 (15.0)	255 (2.6)	<0.001
Food allergy	356 (3.5)	356 (31.2)	0	<0.001	64 (48.5)	292 (3.0)	<0.001
Parental allergic diseases, n(%)							
Asthma	1599 (15.8)	275 (24.1)	1324 (14.7)	<0.001	38 (28.6)	1561 (15.9)	<0.001
Allergic rhinitis	2372 (23.5)	420 (36.9)	1952 (21.7)	<0.001	55 (41.4)	2317 (23.5)	<0.001
Food allergy	589 (5.8)	183 (16.1)	406 (4.5)	<0.001	32 (24.2)	557 (5.7)	<0.001
Drug allergy	942 (8.4)	191 (16.8)	751 (8.3)	<0.001	27 (20.3)	915 (9.3)	<0.001
Atopic dermatitis	989 (9.3)	155 (13.6)	834 (9.3)	<0.001	17 (12.8)	972 (9.9)	0.265
Symptoms reported by parents, n(%)							
Cutaneous	-	741 (65.1)	-		114 (85.7)		
Respiratory	-	536 (47.1)	-		8 (6.0)		
Ocular	-	344 (30.2)	-		1 (0.7)		
Cardiovascular	-	178 (15.6)	-		0		
Gastrointestinal	-	471 (41.4)	-		16 (12.0)		

Table 2 Results of clinical evaluation of patients with suspected FA

Suspected food	Number of patients	SPT Positive/ Total	splgE Positive/ Total	Open Positive/ Total	DBPCFC Positive/ Total
Egg white	36	1/36	2/36	3/34	0/5
Chocolate	15	-	-	0/15	-
Walnut	14	4/14	3/14	1/10	4/5
Hazelnut	11	1/11	1/11	2/10	1/3
Peanut	9	6/9	3/9	0/5	4/6
Blackpepper	6	0/6	1/6	0/6	-
Strawberry	6	0/6	1/6	0/6	-
Milk	5	0/5	0/5	0/5	-
Tomato	5	0/5	0/5	1/5	1/1
Redpepper	3	0/3	0/3	0/3	-
Kiwi	3	1/3	1/3	0/2	1/2
Cocoa	3	1/3	0/3	0/3	1/1
Honey	2	1/2	1/2	0/1	1/1
Banana	2	1/2	1/2	0/2	1/1
Popseed	2	0/2	0/2	0/2	-
Chicken	2	0/2	0	0/2	-
Beef	2	0/1	0/1	0/1	-
Almond	1	0/1	0/1	0/1	-
Pistachio	1	1/1	1/1	-	-
Peach	1	0/1	0/1	0/1	-
Wheat	1	0/1	0/1	0/1	-
Fish (bluefin)	1	0/1	0/1	-	-
Eggplant	1	0/1	0/1	0/1	-
Cucumber	1	0/1	0/1	0/1	-
Sesame	1	1/1	0/1	1/1	-

refused to participate in DBPCFC. OFC was not performed on a patient who has reported anaphylaxis associated with pistachio in the past 6 months. This patient was shown to have IgE-mediated FA with hazelnut, walnut, peanut, and kiwi by means of DBPCFC. Another patient reported that within one hour of ingestion of blue fin, he had urticaria and shortness of breath and was diagnosed as anaphylaxis at the emergency service. Skin prick test and splgE were negative; however, the family refused the OFC test. Of the 15 confirmed cases of FA, eight patients had pollen sensitization while none of them had house dust mite sensitization.

Of the 107 patients with a history consistent with FA completed the allergic evaluation at the outpatient clinic, and 15 (0, 15%) were confirmed to have IgE-mediated FA by means of oral provocation tests.

Discussion

Data on the frequency and nature of FA among adolescents are very limited, and review of published articles on FA to date suggests that this is the first cross-sectional study of FA on a representative sample of adolescents residing in Mediterranean region. The rate of lifetime parent-reported FA by questionnaire was 11.3%, and that as confirmed by a detailed medical history on phone was 1.3%. The rate of current IgE-mediated FA determined by OFC was 0.15% in adolescent age group in this study population.

Table 3 Characteristics of patients with proven food allergy

No	Age/ gender	Food	SPT (mm)	SplgE	OFC	Symptoms
1	12.5/M	Peanut	0	0	Open	Wheezing, decrease in FEV1
2	13.5/F	Walnut	4	0.49	DBPCFC	Pruritis
3	12/F	Honey	8	3.49	DBPCFC	Urticaria, Angioedema
4	15/M	Walnut	6	2.49	DBPCFC	Urticaria
5	12/F	Peanut	5	2.12	DBPCFC	Urticaria
6	13/F	Walnut	5	0.20	DBPCFC	Urticaria
7	14/F	Tomato	3	0	DBPCFC	Oral allergy syndrome (OAS)
8	14.5/M	Banana	5	1.01	DBPCFC	OAS
9	11.5/M	Pistachio	15	1.37	-	Anaphylaxis
		Peanut	5	1.53	DBPCFC	Cough, Itchy Mouth, Hoarseness
		Hazelnut	5	1.84	DBPCFC	Urticaria
		Kiwi	5	1.01	DBPCFC	OAS, Angioedema
10	12/F	Peanut	4	1.09	DBPCFC	Pruritis
11	14/F	Walnut	5	0.87	DBPCFC	Urticaria
12	13/F	Sesame	4	0.04	Open	Urticaria
13	11/F	Cacao	7	0	DBPCFC	Urticaria
14	11/F	Fish	0	0.09	-	Anaphylaxis
15	13/M	Peanut	4	7.30	DBPCFC	Urticaria

The rate of self-reported food allergy is considered to be 12%–13% in general (3, 5). Actually, self-reported FA varies across different countries and geographic regions as illustrated in a study conducted by European Community Respiratory Health Survey, such as 4% in Spain, 18% in Sweden, and 19% in Australia (15). Previous studies in Turkish population revealed frequency of self-reported FA as 5.7% for children and 9.5% for adults (10, 16). However, data on the frequency of adolescent age group are very limited. The rate of self-reported food allergy varies between 8.2 and 12.4% in adolescent age group (8, 17), and similar to them, it was 11.3% in our study.

The rate of 'self-perceived' adverse food reaction was reported to be seven- to 10-fold higher than determined by objective evaluations and OFCs (1, 5, 18). Accordingly, in our study, the diagnosis of IgE-mediated FA was not proven for 86% of children with suspected FA, and 96.9% of children diagnosed as FA by primary care physicians. The insufficiency of primary care physician's knowledge on FA has been reported previously (19, 20). Therefore, there is a need for the training of public and physicians about FA, and the importance of the use of objective diagnostic tests such as DBPCFC should be emphasized. The higher rate of parent-reported FA in families with allergic diseases may be related to the rising awareness of allergic symptoms.

The actual rate of FA was determined as 0.15% in our study, which was relatively low compared with the previous reports. The confirmed rate of FA in developed countries

ranges between 3 and 3.9% among children but the studies usually include children from all age groups (1, 5, 21). Among young children, the rate of FA was reported as 5%–6% in the United States (22) and the UK (23), whereas Dalal et al. found the frequency to be 1.7% in Israel (24). In fact, data on the frequency of adolescent age group are very limited. To our knowledge, there are only two studies based on representative groups including adolescents, which have used oral food challenges to make a confirmed diagnosis. The rates of confirmed food allergy were 1.0% and 5.0% in these studies (8, 9). Roehr et al. reported that 5.0% of adolescents aged between 15 and 17 yr had IgE-mediated FA (9). Although they have studied a large representative group, response rates to questionnaires and attendance to the examinations in the clinic were considerably low, and this may have affected their results. In the second study, Pereira et al. have studied 11- and 15-year-old children, and the prevalence of FA was reported as 1% in this study (8). Response rate to questionnaires was 50%, and OFC could not be performed to one-third of children in this study. Response rate to questionnaires was 90% in our study, and DBPCFC could not be performed only for three patients. The lower rate of FA in our study may be related to these differences as patients with food-related problems may have responded disproportionately to the questionnaires.

The difference in frequency of FA between countries can be explained by cultural and traditional nutritional habits, environmental factors, and possibly genetics (25). The way the foods are cooked is also known to affect their allergenic characteristics (26). Although Western-style nutritional habits have become increasingly popular, foods comprising olive oil and abundant vegetables, called Mediterranean cuisine, slowly cooked in pots are still widely consumed by the general population of Turkey (25). The consumption of early complementary feeding in early ages is asserted to be contributing to the development of tolerance (27, 28). Breastfeeding of babies within 2 yr of birth is a common practice in Turkey. However, parents turn to early complementary feeding as early as 3–4 months especially those involving probiotics, such as, yogurt and cheese (29, 30).

Most commonly suspected foods were hen's egg 3.6%, cow's milk 1.1%, and chocolate 3.5% in our study. Gelincik et al. reached results similar to ours with regard to most commonly suspected foods for FA as follows: hen's egg, chocolate, and tomato for adults (16). The commonly suspected foods for FA may vary across countries. Sandin et al. found in their study that the suspected foods varied across Sweden and Estonia (31). The above-mentioned variation across countries might be the result of nutritional and perceptual differences across cultures.

After OFC and DBPCFC, the foods that commonly cause FA were peanut (0.05%) and tree nuts (0.05%) in our study. In literature, peanuts and tree nuts were reported as the leading cause of FA in adolescents and young adults (8, 32) and

responsible for 90% of the fatalities resulting from anaphylaxis (33). Our findings are in line with an earlier study conducted in Ankara, reporting the most common cause of FA in children over 12 months as peanut and tree nuts (34). In contrary, other studies conducted among different age groups of children in Turkey reported hen's egg and tomato as the most common allergenic foods (16), whereas Orhan et al. stated that red meat, cow's milk, and cocoa were the most common food allergens in their study population (10). The latter study was carried out in Eastern Black Sea region where red meat allergy is commonly seen. This region does not receive much migration and is a place with its own special cultural characteristics. While the prevalence of FA remains comparatively low in our region, we have seen that the allergenic foods in Turkey appear to be similar to those in most European countries and the United States.

Although considered as a low frequency, a prevalence of 0.15% brings approximately a total of 25,000 students under the risk of IgE-mediated FA and anaphylaxis in the whole country. Even more strikingly, according to our results, 1.5 million students make dietary changes based on misperception of self-reported FA, which can lead to various nutritional disorders unnecessarily. This makes FA a major public health concern in our country.

The strengths of this study include the large sample size of study population for the adolescent age group and application of screening tests followed by DBPCFC. Also, the response rate was very high (89.3%). The main limitations of this study may be the risk of failure of the respondents to recall the previous adverse food reactions during the completion of the questionnaires, and we also did not ask the clinical findings of oral allergy syndrome in detail. Besides, we did not make SPT or test for specific IgE to whole sample. These factors may have caused us to miss some children with FA. This limitation was investigated and minimized by validating the negative responses of families through a further phone survey on a randomly selected 200 families, and none of them reported any FA history.

In conclusion, the rate of lifetime parent-reported FA was 11.3%, and phone survey revealed a clinical history suggestive of food allergy in 1.3%. After diagnostic evaluation including DBPCFC, the rate of current IgE-mediated FA was 0.15% in adolescents. FA among adolescent age group has been confirmed to be comparatively low in Turkey, while peanuts and tree nuts were determined to be the most common causes of IgE-mediated FA. Food allergy should be taken as a major health problem, and education of public and physicians is needed.

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References

1. Boyce JA, Assa'ad A, Burks AW, et al. Guidelines for the diagnosis and management of food allergy in the United States: summary of the NIAID-sponsored expert panel report. *J Allergy Clin Immunol* 2010; **126**: 1105–18.

2. Sicherer SH, Sampson HA. Food allergy. *J Allergy Clin Immunol* 2010; **125**: S116–25.
3. Sampson HA. Update on food allergy. *J Allergy Clin Immunol* 2004; **113**: 805–19. quiz 20.
4. Lack G. Update on risk factors for food allergy. *J Allergy Clin Immunol* 2012; **129**: 1187–97.
5. Rona RJ, Keil T, Summers C, et al. The prevalence of food allergy: a meta-analysis. *J Allergy Clin Immunol* 2007; **120**: 638–46.
6. Bock SA, Munoz-Furlong A, Sampson HA. Fatalities due to anaphylactic reactions to foods. *J Allergy Clin Immunol* 2001; **107**: 191–3.
7. Young E, Stoneham MD, Petrukevitch A, Barton J, Rona R. A population study of food intolerance. *Lancet* 1994; **343**: 1127–30.
8. Pereira B, Venter C, Grundy J, Clayton CB, Arshad SH, Dean T. Prevalence of sensitization to food allergens, reported adverse reaction to foods, food avoidance, and food hypersensitivity among teenagers. *J Allergy Clin Immunol* 2005; **116**: 884–92.
9. Roehr CC, Edenharter G, Reimann S, et al. Food allergy and non-allergic food hypersensitivity in children and adolescents. *Clin Exp Allergy* 2004; **34**: 1534–41.
10. Orhan F, Karakas T, Cakir M, Aksoy A, Baki A, Gedik Y. Prevalence of immunoglobulin E-mediated food allergy in 6-9-year-old urban schoolchildren in the eastern Black Sea region of Turkey. *Clin Exp Allergy* 2009; **39**: 1027–35.
11. Sampson HA. 9. Food allergy. *J Allergy Clin Immunol* 2003; **111**: S540–7.
12. Kanny G, Moneret-Vautrin DA, Flabbee J, Beaudouin E, Morisset M, Thevenin F. Population study of food allergy in France. *J Allergy Clin Immunol* 2001; **108**: 133–40.
13. Nowak-Wegrzyn A, Assa'ad AH, Bahna SL, Bock SA, Sicherer SH, Teuber SS. Work Group report: oral food challenge testing. *J Allergy Clin Immunol* 2009; **123**: S365–83.
14. Bindslev-Jensen C, Ballmer-Weber BK, Bengtsson U, et al. Standardization of food challenges in patients with immediate reactions to foods—position paper from the European Academy of Allergology and Clinical Immunology. *Allergy* 2004; **59**: 690–7.
15. Woods RK, Abramson M, Bailey M, Walters EH. International prevalences of reported food allergies and intolerances. Comparisons arising from the European Community Respiratory Health Survey (ECRHS) 1991–1994. *Eur J Clin Nutr* 2001; **55**: 298–304.
16. Gelincik A, Buyukozturk S, Gul H, et al. Confirmed prevalence of food allergy and non-allergic food hypersensitivity in a Mediterranean population. *Clin Exp Allergy* 2008; **38**: 1333–41.
17. Gupta RS, Springston EE, Warrier MR, et al. The prevalence, severity, and distribution of childhood food allergy in the United States. *Pediatrics* 2011; **128**: e9–17.
18. Zuidmeer L, Goldhahn K, Rona RJ, et al. The prevalence of plant food allergies: a systematic review. *J Allergy Clin Immunol* 2008; **121**: 1210–8 e4.
19. Gupta RS, Springston EE, Kim JS, et al. Food allergy knowledge, attitudes, and beliefs of primary care physicians. *Pediatrics* 2010; **125**: 126–32.
20. Erkocoglu M, Civelek E, Azkur D, et al. Knowledge and attitudes of primary care physicians regarding food allergy and anaphylaxis in Turkey. *Allergol Immunopathol (Madr)* 2012; Sep 29 [Epub ahead of print]
21. Branum AM, Lukacs SL. Food allergy among children in the United States. *Pediatrics* 2009; **124**: 1549–55.
22. Luccioli S, Ross M, Labiner-Wolfe J, Fein SB. Maternally reported food allergies and other food-related health problems in infants: characteristics and associated factors. *Pediatrics* 2008; **122**(Suppl. 2): S105–12.
23. Venter C, Pereira B, Voigt K, et al. Prevalence and cumulative incidence of food hypersensitivity in the first 3 years of life. *Allergy* 2008; **63**: 354–9.
24. Dalal I, Binson I, Reifen R, 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.
25. Lack G. Epidemiologic risks for food allergy. *J Allergy Clin Immunol* 2008; **121**: 1331–6.
26. Beyer K, Morrow E, Li XM, et al. Effects of cooking methods on peanut allergenicity. *J Allergy Clin Immunol* 2001; **107**: 1077–81.
27. Du Toit G, Katz Y, Sasieni P, et al. Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. *J Allergy Clin Immunol* 2008; **122**: 984–91.
28. Poole JA, Barriga K, Leung DY, et al. Timing of initial exposure to cereal grains and the risk of wheat allergy. *Pediatrics* 2006; **117**: 2175–82.
29. Demirtas B, Ergocmen B, Taskin L. Breastfeeding experiences of Turkish women. *J Clin Nurs* 2012; **21**: 1109–18.
30. Koksall O. Nutritional problems in Turkey during the weaning period and some solutions. *Turk J Pediatr* 1971; **13**: 59–71.
31. Sandin A, Annus T, Bjorksten B, et al. Prevalence of self-reported food allergy and IgE antibodies to food allergens in Swedish and Estonian schoolchildren. *Eur J Clin Nutr* 2005; **59**: 399–403.
32. Sampson MA, Munoz-Furlong A, Sicherer SH. Risk-taking and coping strategies of adolescents and young adults with food allergy. *J Allergy Clin Immunol* 2006; **117**: 1440–5.
33. Bock SA, Munoz-Furlong A, Sampson HA. Further fatalities caused by anaphylactic reactions to food, 2001–2006. *J Allergy Clin Immunol* 2007; **119**: 1016–8.
34. Yavuz ST, Sahiner UM, Buyuktiyaki B, et al. Phenotypes of IgE-mediated food allergy in Turkish children. *Allergy Asthma Proc* 2011; **32**: 47–55.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Data S1. Primary school survey for food allergy.

PRIMARY SCHOOL SURVEY FOR FOOD ALLERGY

Name-Surname:		Phone (home):	
Sex:	☐ Female ☐ Male	Mobile phone (Mother):	
Birthday (day/month/year):		Mobile phone (Father):	
School name:		Classroom:	

Example of how to mark questionnaire YES NO

☐ ☒

Question 1: Has your child (who brings you the questionnaire) ever had any of the complaints listed below **within 2 hours** after eating or drinking any food (milk, egg, hazelnut, peanut, walnut, strawberry, tomato, meat, etc) If yes, please check/mark all the complaints that had occurred. If you answered NO please skip to question 5.

☐ YES ☐ NO

	YES	NO
a. Skin rash, itching, urticaria	☐	☐
b. Swelling at lips, eye or other parts of the body	☐	☐
c. Cough - shortness of breath - wheezing	☐	☐
d. Nausea-vomiting-diarrea – abdominal pain	☐	☐
e. Chest pain-palpitation - syncope-loss of consciousness	☐	☐
f. Sneezing, itching, blockage or watery discharge of the nose	☐	☐
g. Redness, itching or watery of eyes	☐	☐

Question 2: After eating or drinking which food(s) did you have any allergic reaction?

☐ milk/yoghurt/cheese	☐ egg	☐ fish	☐ hazelnut	☐ peanut
☐ strawberry	☐ tomato	☐ meat	☐ chicken	☐ pistachio
☐ chocolate	☐ Others:			

Question 3: Has your child ever eaten or drunk the same food(s) in the past year?

☐ YES

☐ NO

Question 4: If your answer is yes to fourth question, has she/he had the same complaints?

☐ YES

☐ NO

Question 5: Has your child (who brings you the questionnaire) ever been diagnosed with asthma or allergic asthma or allergic bronchitis by a physician?

☐ YES

☐ NO

Question 6: Has your child (who brings you the questionnaire) ever been diagnosed with allergic rhinitis or hay fever by a physician?

☐ YES

☐ NO

Question 7: Has your child (who brings you the questionnaire) ever been diagnosed with food allergy by a physician?

☐ YES

☐ NO

Question 8: Has your child (who brings you the questionnaire) ever been diagnosed with drug allergy by a physician?

☐ YES

☐ NO

Question 9: Has your child (who brings you the questionnaire) ever been diagnosed with eczema or atopic dermatitis or atopic eczema by a physician?

☐ YES

☐ NO

Question 10: Has you or your partner/spouse ever been diagnosed with asthma or allergic asthma or allergic bronchitis by a physician?

☐ YES

☐ NO

Question 11: Has you or your partner/spouse ever been diagnosed with allergic rhinitis or hay fever by a physician?

☐ YES

☐ NO

Question 12: Has you or your partner/spouse ever been diagnosed with food allergy by a physician?

☐ YES

☐ NO

Question 13: Has you or your partner/spouse ever been diagnosed with drug allergy by a physician?

☐ YES

☐ NO

Question 14: Has you or your partner/spouse ever been diagnosed with eczema or atopic dermatitis or atopic eczema by a physician?

☐ YES

☐ NO

Question 15: Has your child's sibling/s ever been diagnosed with asthma or allergic asthma or allergic bronchitis by a physician?

☐ YES

☐ NO

☐ He/she has no sibling

Question 16: Has your child's sibling/s ever been diagnosed with allergic rhinitis or hay fever by a physician?

☐ YES

☐ NO

☐ He/she has no sibling

Question 17: Has your child's sibling/s ever been diagnosed with food allergy by a physician?

☐ YES

☐ NO

☐ He/she has no sibling

Question 18: Has your child's sibling/s ever been diagnosed with drug allergy by a physician?

☐ YES

☐ NO

☐ He/she has no sibling

Question 19: Has your child's sibling/s ever been diagnosed with eczema or atopic dermatitis or atopic eczema by a physician?

☐ YES

☐ NO

☐ He/she has no sibling