ORIGINAL ARTICLE

Self-reported hypersensitivity and allergy to foods amongst Mexican adolescents: Prevalence and associated factors

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Abstract
Background: The prevalence of food allergy is on the rise on a global scale.
Objective: To determine the prevalence of food hypersensitivity (FHS) and probable food allergy (PFA), as well as the foods and factors associated with these occurrences.
Methods: A cross-sectional study was carried out among 1992 adolescents (aged 15–18 years). Each adolescent answered a structured questionnaire. A multivariate analysis was used to identify the association between the variables.
Results: The prevalence of FHS was 10.6% (the most commonly associated foods were shrimp, cow's milk and avocado) and the PFA was 7.8% (shrimp, cow's milk and pecan). The prevalences of oral allergy syndrome, food-associated urticaria and systemic reaction were 4.9%, 3.6% and 1.5%, respectively. The following factors were associated with FHS: personal history of asthma (OR 1.63; 95% CI: 1.11–2.41), allergic rhinitis (OR 2.60; 95% CI: 1.75–3.87), atopic dermatitis (OR 2.07; 95% CI: 1.25–3.43), maternal history of asthma (OR 1.80; 95% CI: 1.02–3.16), atopic dermatitis (OR 6.11; 95% CI: 2.45–15.29), and female sex (OR 1.89; 95% CI: 1.38–2.59). PFA was associated with a personal history of asthma (OR 1.65; 95% CI: 1.06–2.56), allergic rhinitis (OR 2.46; 95% CI: 1.56–3.88), atopic dermatitis (OR 2.02; 95% CI: 1.15–3.54), paternal allergic rhinitis (OR 2.52; 95% CI: 1.15–5.51), maternal atopic dermatitis (OR 7.46; 95% CI: 2.93–19.00), and female sex (OR 1.89; 95% CI: 1.31–2.72).

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Conclusion: The adverse reactions associated with foods among late adolescents are a frequent occurrence, and the most commonly associated factor is atopy.

Introduction

The prevalence of food allergy is increasing worldwide,[1] and can have an important effect on the quality of life, as food allergy is likely to provoke systemic reactions.[2]

Globally, the prevalence of self-reported hypersensitivity to food in children and adults has been estimated at 3–35%.[3] In Latin America, there is a lack of population-based studies dedicated to establishing the prevalence of adverse food reactions. Nevertheless, one of the first of such studies occurred in Colombia, where the prevalence of food allergies in children aged 9–16 was 12.6%.[4] Additional studies showed that preschool-aged Brazilian children had a prevalence of 23.5%,[5] Chile reported a prevalence of 5.5% for school-aged children,[6] and the prevalence in Mexico was 10%.[7] Among early teens in Australia, the prevalence was 5.5%.[8]

Some of the foods that were most commonly associated with food hypersensitivity (FHS) and probable food allergy (PFA) were peanut, tree nuts, cow's milk and shellfish, among others.[4-10] However, these can vary based on the geographic region, food availability, eating habits, and cultural customs.

While the factors associated with food allergy may vary, the most consistent factor is genetics.[4,9,10] Furthermore, women exhibit these symptoms more frequently than men do.[11-13]

To our knowledge, FHS and PFA in late adolescence (the final stage of childhood) have not been previously studied. Our objectives consisted of determining the prevalence of FHS and PFA, as well as their clinical characteristics, the foods that are involved, and the factors associated with them.

Methods

Design

In our cross-sectional study, we analyzed data that correspond to 1992 adolescents, aged 15–18 years. These students were enrolled in high schools within Guadalajara, Mexico, one of the nine cities that constitute the metropolitan area of the same name. The city of Guadalajara includes 1.5 million out of the approximate five million inhabitants throughout the entire metropolitan area; its large population is representative of the diverse socioeconomic strata in the region.

Sample

The samples were collected from April to June of 2016 through probabilistic sampling that was stratified and then clustered. To do so, we divided the city of Guadalajara into seven strata, and from each of these, we randomly selected at least one school (cluster); each stratum contributed a sub-sample of students through the proportional selection technique, by multiplying the complete sample size by the proportion of students that were enrolled in each stratum. To obtain a representative sample for each selected school, each school grade was considered a new stratum, and from each of these, we randomly selected a group. Lastly, we randomly selected students to participate in the study.

Questionnaire

The data were obtained through a structured questionnaire that was previously used and validated within the Mexican population.[10] Said questionnaire includes questions regarding age, sex, personal history of atopic and non-atopic diseases of each participant, and the family history of allergic diseases of each participant's parents.

Definitions

We utilized the following question to identify self-reported FHS: *Did you experience an allergic reaction after eating foods or drinking something?*[10] This study included all cases in which the student responded affirmatively, regardless of a prior history of allergy testing.[14] To define PFA, we adopted the definition proposed by Sicherer et al.; a probable allergic reaction was considered positive if the organs and the manifested symptoms were typical allergic reactions (urticaria and angioedema, difficulty breathing, wheezing, swelling of the throat, vomiting and diarrhea) and if these occurred within two hours of having ingested the allergy-causing food.[15] This definition allows us to detect up to 93% of the subjects that have specific IgE antibodies that react to the implied foods.[16] If a participant had two or more organs that were affected, we considered this to be a systemic reaction.

Statistical analyses

To estimate the prevalence of FHS, we divided the frequency of affirmative answers to the question, *Do you suffer from allergic reactions after ingesting food or drinking something?*, by the total number of people who completed the questionnaire. The same procedure was utilized to estimate the prevalence of PFA, except in this case, we utilized the previously mentioned definition. We employed the chi-square test or Fisher's exact test, as deemed necessary, in order to compare the categorical variables. We estimated the confidence intervals at 95% (95% CI) for the proportions.

The data were analyzed by using the IBM SPSS Statistics version 20.0 (IBM Co., Armonk, NY, USA) program for Windows.

To identify the association between variables, we utilized two multivariate model groups in which the dependent variables were FHS and PFA, respectively. In both groups, the independent covariates were personal, paternal or maternal history of asthma, allergic rhinitis, atopic dermatitis, and the female sex. The strength of the association between variables was evaluated by estimating the odds ratio (OR) and the 95% CI through adjusted binary logistic regression with the use of the forward conditional method. The value of $p < 0.05$ was considered to be statistically significant.

Ethics

The authors certify that this research was done under full compliance with all government policies and the Helsinki Declaration (2013). The Committee of Research Ethics at the Hospital Civil de Guadalajara approved this study. Prior to distributing our questionnaire, we explained our objective to each school district and their teachers and obtained their consent in order to be able to survey their students. If they accepted, we sought the same permission from the students, and when students agreed to participate, they were given detailed information about our study and were asked to give their verbal consent to participate.

Results

Overall, we analyzed data that corresponded to 963 men (47.0%) and 1056 women (53.0%). With regards to the personal history of allergic diseases among adolescents, we registered asthma at 12.7%, allergic rhinitis at 9.0% and atopic dermatitis at 5.2%. Where their family history was concerned, we found that the paternal history was as follows: asthma 2.6%, allergic rhinitis 2.1% and atopic dermatitis 0.7%. Maternal history corresponded to 4.5%, 3.4% and 1.1%, respectively.

The self-reported prevalence of FHS was 10.6% (95% CI: 9.4–12.1%), whereas the prevalence of PFA was 7.8% (95% CI: 6.7–9.0%). Once again, these types of reactions were predominant among women (10.1% of females vs. 5.1% of males, $p < 0.001$).

The most common self-reported symptoms for FHS were cutaneous (hives and reddening of the skin), followed by respiratory ailments (oppression of the pharynx and difficulty breathing) and lastly intestinal discomfort (abdominal pain and heartburn) (Table 1). In PFA, cutaneous symptoms were predominant (hives and reddening of the skin), followed by respiratory ailments (oppression of the pharynx and difficulty breathing) and intestinal discomfort (abdominal pain and heartburn).

The foods that were most commonly associated with self-reported FHS were shrimp, cow's milk, avocado, pecan, and

Table 1 Frequency of self-reported clinical manifestations associated to the ingestion of foods amongst 1992 late adolescents.

	Food hypersensitivity $n = 212$			Probable food allergy $n = 155$	
	n	%		n	%
Cutaneous			*Cutaneous*		
Hives	71	33.5	Reddening of skin	70	45.2
Reddening of skin	70	33.0	Itchiness of lips	59	38.1
Itchiness of lips	59	27.8	Hives	59	38.1
Edema of the mouth	51	24.1	Edema of the mouth	51	32.9
Itchiness of the body	44	20.8	Itchiness of the body	40	25.8
Skin rashes	42	19.8	Facial edema	37	23.9
Facial edema	37	17.5	Skin rashes	36	23.2
Perspiration	13	6.1	Perspiration	10	6.5
Intestinal			*Intestinal*		
Colic	45	21.2	Heartburn	26	16.8
Heartburn	41	19.3	Colic	25	16.1
Diarrhea	36	17.0	Vomiting	24	15.5
Vomiting	35	16.5	Diarrhea	21	13.5
Abdominal distention	27	12.7	Abdominal distention	21	13.5
Flatulence	26	12.3	Constipation	15	9.7
Constipation	25	11.8	Flatulence	12	7.7
Respiratory			*Respiratory*		
Pharynx oppression	62	29.2	Pharynx oppression	62	40.0
Difficulty breathing	51	24.1	Difficulty breathing	49	31.6
Sneezing	49	23.1	Nasal congestion	37	23.9
Nasal congestion	39	18.4	Sneezing	37	23.9
Runny nose	37	17.5	Runny nose	35	22.6
Coughing	35	16.5	Coughing	34	21.9
Wheezing	20	9.4	Wheezing	20	12.9

Table 2 Foods most frequently associated with food hypersensitivity and probable food allergy amongst 1992 late adolescents.

	n	%
Food hypersensitivity		
Shrimp	53	2.7
Cow's milk	37	1.9
Avocado	19	1.0
Pecan	20	1.0
Cow's milk cheese	18	0.9
Pork sausage	18	0.9
Almond	17	0.9
Banana	16	0.8
Oyster	15	0.8
Peach	13	0.7
Apple	14	0.7
Clam	14	0.7
Fish	13	0.7
Probable food allergy		
Shrimp	46	2.3
Cow's milk	20	1.0
Pecan	18	0.9
Avocado	17	0.9
Almond	16	0.8
Pork sausage	15	0.8
Peach	13	0.7
Apple	13	0.7

cow's milk cheese; for PHF, they were shrimp, cow's milk, pecan, avocado and almond (Table 2).

The prevalence of diseases that are likely regulated by IgE and the main ailments that were associated with them were oral allergy syndrome 4.9% (95% CI: 4.1–6.0%), where shellfish and fish (primarily shrimp) were the most frequent triggering agents (Table 3). The prevalence of urticaria was 3.6% (95% CI: 2.9–4.5%), and once again, shrimp was the main culprit. The frequency of systemic symptoms was 1.5% (95% CI: 1.0–2.1%), and in this case, both shrimp and peach were the primary factors.

The factors that were associated with FHS are shown in Table 4. We adjusted Model 1 to the personal history of allergic disease and sex; as a result, asthma (OR 1.75, $p = 0.004$), allergic rhinitis (OR 2.67, $p < 0.001$), atopic dermatitis (OR 2.39, $p < 0.001$) and female sex (OR 1.88, $p < 0.001$) were associated with FHS. Models 2 and 3 show that a paternal history of allergic rhinitis (OR 3.03, $p = 0.002$) and atopic dermatitis (OR 4.69, $p < 0.001$) was associated with FHS, as was a maternal history of asthma (OR 2.02, $p = 0.012$), allergic rhinitis (OR 2.08, $p = 0.019$) and atopic dermatitis (OR 7.92, $p < 0.001$). The final model, which was based on the previously identified covariates, shows that a personal history of asthma, allergic rhinitis and atopic dermatitis, along with a maternal history of asthma or atopic dermatitis and female sex, remained associated with FHS.

Table 5 displays the various models that were designed to identify the associated factors in the PFAs. Model 1 once again shows that the personal history of asthma, allergic rhinitis, atopic dermatitis and female sex were associated with PFA.

In Models 2 and 3, the paternal and maternal history of allergic rhinitis and atopic dermatitis, along with the female sex, were associated with PFA in the surveyed individuals. In the final model, a history of asthma, allergic rhinitis, and atopic dermatitis among adolescents, as well as paternal allergic rhinitis, maternal atopic dermatitis, and the female sex, remained as significant, independently associated factors with PFA.

Table 3 Primary foods associated with probable allergy symptoms that are regulated by IgE.

Oral allergy syndrome			Urticaria			Systemic symptoms		
n = 98	n	%	n = 72	n	%	n = 29	n	%
Food categories			*Food categories*			*Food categories*		
Shellfish & fish	37	37.8	Shellfish & fish	24	33.3	Fruit	10	34.5
Fruit	35	35.7	Fruit	15	20.8	Shellfish & fish	7	24.1
Tree nuts	17	17.3	Tree nuts	12	16.7	Tree nuts	5	17.2
Dairy	13	13.3	Dairy	9	12.5	Vegetables	4	13.8
Vegetables	10	10.2	Vegetables	7	9.7	Dairy	1	3.4
Individual foods			*Individual foods*			*Individual foods*		
Shrimp	36	36.7	Shrimp	18	25.0	Shrimp	8	27.6
Avocado	13	13.3	Cow's milk	9	12.5	Peach	5	17.2
Cow's milk	13	13.3	Almond	9	12.5	Brazilian nut	5	17.2
Peach	11	11.2	Walnut	8	11.1	Avocado	4	13.8
Apple	11	11.2	Avocado	7	9.7	Almond	4	13.8
Almond	11	11.2	Clam	7	9.7	Guava	3	10.3
Walnut	11	11.2	Oyster	7	9.7	Mango	3	10.3
Banana	10	10.2	Peach	6	8.3	Apple	3	10.3
Clam	10	10.2	Cow's milk cheese	6	8.3	Banana	3	10.3
Fish	9	9.2	Peanut	6	8.3	Lobster	3	10.3

Table 4 Multivariate model of factors associated with food hypersensitivity.

	OR	95% CI	p Value
Model 1			
Personal history of:			
Asthma	1.75	1.19–2.56	0.004
Allergic rhinitis	2.67	1.81–3.95	<0.001
Atopic dermatitis	2.39	1.47–3.89	<0.001
Sex (female)	1.88	1.37–2.57	<0.001
Model 2			
Paternal history of:			
Asthma	–	–	0.158
Allergic rhinitis	3.03	1.49–6.19	0.002
Atopic dermatitis	4.69	1.53–14.3	<0.001
Sex (female)	2.02	1.49–2.75	<0.001
Model 3			
Maternal history of:			
Asthma	2.02	1.17–3.51	0.012
Allergic rhinitis	2.08	1.13–3.85	0.019
Atopic dermatitis	7.92	3.30–19.02	<0.001
Sex (female)	2.00	1.46–2.72	<0.001
Final model			
Personal history of:			
Asthma	1.63	1.11–2.41	0.013
Allergic rhinitis	2.60	1.75–3.87	<0.001
Atopic dermatitis	2.07	1.25–3.43	0.005
Paternal history of:			
Allergic rhinitis	–	–	0.189
Atopic dermatitis	–	–	0.450
Maternal history of:			
Asthma	1.80	1.02–3.16	<0.001
Allergic rhinitis	–	–	0.369
Atopic dermatitis	6.11	2.45–15.29	<0.001
Sex (female)	1.89	1.38–2.59	<0.001

OR: Odds ratios obtained through logistical regression using forward conditional method.
ORs were not calculated for variables excluded from the model.
All covariates were introduced as categorical variables (with and without the characteristic of interest).

Discussion

Studies aimed at estimating the prevalence of FHS and PFA in Latin America, primarily among adolescents, are scarce. This is one of the first studies to analyze this matter in our region. We observed that the prevalence of FHS was slightly over 10% and that PFA was at 8%, and in both cases, shrimp and cow's milk were the most common triggering agents. Interestingly, in addition to shrimp, peaches were associated with systemic reactions. Moreover, we were able to identify that a personal and a family history of allergic disease, especially on the mother's side, as well as female sex, were the main factors associated with both FHS and PFA.

In Melbourne, Australia, a recent cross-sectional study that included adolescents aged 10–14 showed that the prevalence of food allergy was 5.5% (determined with the use of a questionnaire), However, when skin-prick tests or oral challenge test were applied, there was a slight decrease to 4.5%.[8] An additional study in Japan analyzed the prevalence of food allergies among female adolescents with the use of a questionnaire and reported a frequency of 8.2%.[17] In Latin America, a cross-sectional questionnaire-based study proved that the prevalence of FHS among individuals aged 9–16 was 12.6%.[4] In our country, there are limited efforts focused on researching the adverse reactions of food ingestion, and there are none that focus on adolescents. However, there was a study of young university students that identified adverse reactions to foods by utilizing the questionnaire that we have employed for this study. The university-based study recorded the prevalence of FHS at 30.1%. Interestingly, the frequency of PFA was quite different, at only 6%. An additional study analyzed the prevalence of FHS in young adults with the use of a questionnaire; their reported prevalence was 17%.[9] Notably, this makes our study the first to report the prevalence of food allergies among late adolescents in Mexico.

Table 5   Multivariate model of factors associated with probable food allergy.

	OR	95% CI	p Value
Model 1			
Personal history of:			
Asthma	1.73	1.12–2.66	0.012
Allergic rhinitis	2.78	1.81–4.28	<0.001
Atopic dermatitis	2.43	1.43–4.14	0.001
Sex (female)	1.87	1.30–2.69	0.001
Model 2			
Paternal history of:			
Asthma	–	–	0.108
Allergic rhinitis	4.43	2.16–9.10	<0.001
Atopic dermatitis	6.85	2.23–21.00	0.001
Sex (female)	2.03	1.42–2.89	<0.001
Model 3			
Maternal history of:			
Asthma	–	–	0.097
Allergic rhinitis	2.43	1.26–4.71	0.008
Atopic dermatitis	9.75	4.02–23.63	<0.001
Sex (female)	2.02	1.41–2.89	<0.001
Final model			
Personal history of:			
Asthma	1.65	1.06–2.56	0.025
Allergic rhinitis	2.46	1.56–3.88	<0.001
Atopic dermatitis	2.02	1.15–3.54	0.014
Paternal history of:			
Allergic rhinitis	2.52	1.15–5.51	0.021
Atopic dermatitis	–	–	0.214
Maternal history of:			
Allergic rhinitis	–	–	0.442
Atopic dermatitis	7.46	2.93–19.00	<0.001
Sex (female)	1.89	1.31–2.72	0.001

OR: Odds ratios obtained through logistical regression using forward conditional method.
ORs were not calculated for variables excluded from the model.
All covariates were introduced as categorical variables (with and without the characteristic of interest).

For both FHS and PFA, the most commonly associated adverse foods were shrimp and cow's milk. It seems that this could be a constant among the general population of our country, as there have been two prior studies in two different geographic regions, in which these foods were the most frequently reported triggering agents.[9,10] In a study carried out among early adolescents, the most commonly associated foods were peanuts and tree nuts.[8] In a study that included children, adolescents and adults from various European countries, hazelnuts and apples were the foods that caused the most hypersensitivity; interestingly, neither shrimp nor cow's milk were significant factors.[18]

Something similar was observed in Canada, where the most commonly associated foods among children and adults were peanuts and tree nuts.[19] A Mexican diet, which is primarily based on corn tortillas, beans and the low consumption of refined grains, usually does not include the regular consumption of shrimp and cow's milk. Consequently, we suggest that this diet is a risk factor and increases the probability of developing an allergy to these foods within our population, since in comparison to other populations, our time of exposure to such foods is considerably shorter.[20] Foods such as peanuts, which in other parts of the world are a major agent of allergic reaction,[2,8,9,21] are not a major culprit in our population, although the pecan is.

Although there were differences in age groups, the prevalence of OAS, urticaria and systemic reactions associated with foods, these were similar to the findings among adults in our city.[9] In a population of late adolescents, OAS was the most common clinical entity, and notably, it was linked to the ingestion of shellfish (mainly shrimp). However, it is likely that we are only observing the natural history of the disease among these patients, in which the persistence of a stimulated allergen begins with oral symptoms, followed by urticaria and the triggering of anaphylaxis. Up to 1.5% of late adolescent symptoms were compatible with anaphylaxis after ingesting foods, and shrimp and peaches were among the foods most related to this event. Previous epidemiological studies have shown similar findings.[4,18,22–24] Notably, none of these late adolescents had been informed by a

physician that these symptoms were characteristics of an anaphylactic reaction.

In our study, various factors were significantly associated with FHS and FPA. One such factor was a personal history of an allergic disease. Notably, previous studies have had similar results to our own.[4,9,10] Moreover, a maternal history of atopic dermatitis acts as a conducive agent, which we had previously observed among university students in our country,[10] although we have yet to find an explanation for this occurrence.

Lastly, the predominance of FHS and FPA among women, which is reported in our study, is similar to that observed for asthma, allergic rhinitis, or atopic dermatitis, among others.[11–13] It seems that within our adolescent population, these reactions are triggered during the final stage of adolescence and not during adulthood. Some of the factors that might help explain the prevalence of adverse reactions to foods among women are: hormonal (primarily estrogens), psychological sensitivity, the use of medications (such as antacids), food preferences, and the processing of information, among others.[25,26] While various hypotheses have emerged to explain the causes of food allergies, some of the most reoccurring are the hypothesis of fatty diets and allergies, the antioxidant hypothesis, the vitamin D hypothesis, the hygiene hypothesis, the exposure to food allergens, and recently the dual exposure to allergen hypothesis.[27,28]

An important limitation of studies based on self-reported symptoms that are compatible with adverse reactions and are associated with food ingestion is that they tend to overestimate or underestimate the prevalence, because most of these occurrences lack objective tests (clinical history, specific identification of IgE, or oral challenge tests). In this study, as we only included adolescents that were from an industrialized city, the generalization of our results is limited when considering rural regions, or places that do not have access to the same types of foods. Two additional limitations of our study involve the lack of data related to the triggers of allergic rhinitis symptoms in students and the inability of the questionnaire to identify a student with symptoms related to the consumption of foods containing baked or cooked milk. On the other hand, given that this is the first epidemiological study of its kind based on probabilistic sampling, our results can be extrapolated among the population of late adolescents in our city.

In conclusion, the adverse reactions associated with foods among late adolescents in Guadalajara, Mexico are a common occurrence. The prevalence of FHS was calculated at 10.6%, while FPA was estimated at 7.8%. Shrimp and cow's milk were the primary allergens in this age group. Notably, the prevalence of compatible symptoms with a systemic reaction linked to a single food was calculated at 1.5%. The most important risk factors associated with FHS and FPA were a prior personal history, a relative with an allergic disease, and the female sex.

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Conflict of interest

The authors have no conflict of interest to declare.

References

1. Verrill L, Bruns R, Luccioli S. Prevalence of self-reported food allergy in U.S. adults: 2001, 2006, and 2010. Allergy Asthma Proc. 2015;36:458–67.
2. Antolín-Amérigo D, Manso L, Caminati M, et al. Quality of life in patients with food allergy. Clin Mol Allergy. 2016;14:1–10.
3. Rona RJ, Keil T, Summers C, Gislason D, et al. The prevalence of food allergy: a meta-analysis. J Allergy Clin Immunol. 2007;120:638–46.
4. Marrugo J, Hernández L, Villalba V. Prevalence of self-reported food allergy in Cartagena (Colombia) population. Allergol Immunopathol (Madrid). 2008;36:320–4.
5. Gonçalves LC, Guimarães TC, Silva RM, et al. Prevalence of food allergy in infants and pre-schoolers in Brazil. Allergol Immunopathol (Madrid). 2016;44:497–503.
6. Hoyos-Bachiloglu R, Ivanovic-Zuvic D, Álvarez J, et al. Prevalence of parent-reported immediate hypersensitivity food allergy in Chilean school-aged children. Allergol Immunopathol (Madrid). 2014;42:527–32.
7. Ontiveros N, Valdez-Meza EE, Vergara-Jiménez MJ, Canizalez-Román A, Borzutzky A, Cabrera-Chávez F. Parent-reported prevalence of food allergy in Mexican schoolchildren: a population-based study. Allergol Immunopathol (Madrid). 2016;44:563–70.
8. Sasaki M, Koplin JJ, Dharmage SC, et al. Prevalence of clinic-defined food allergy in early adolescence: the Schoolnuts study. J Allergy Clin Immunol. 2018;141, 391–398.e4.
9. Bedolla-Barajas M, Bedolla-Pulido TR, Camacho-Peña AS, González-García E, Morales-Romero J. Food hypersensitivity in mexican adults at 18 to 50 years of age: a questionnaire survey. Allergy Asthma Immunol Res. 2014;6:511–6.
10. Puente-Fernández C, Maya-Hernández RL, Flores-Merino MV, Romero-Figueroa M, del S, Bedolla-Barajas M, et al. Self-reported prevalence and risk factors associated with food hypersensitivity in Mexican young adults. Ann Allergy Asthma Immunol. 2016;116:523–7.
11. Akcay A, Tamay Z, Ergin A, Guler N. Prevalence and risk factors of atopic eczema in Turkish adolescents. Pediatr Dermatol. 2014;31:319–25.
12. Ziyab AH. Prevalence and risk factors of asthma, rhinitis, and eczema and their multimorbidity among young adults in kuwait: a cross-sectional study. Biomed Res Int. 2017:1–10, 2184193.
13. Chu LM, Pahwa P. Prevalence and associated factors for self-reported asthma in a Canadian population: the Canadian Community Health Survey, 2014. J Asthma. 2018;55:26–34.
14. Ben-Shoshan M, Harrington DW, Soller L, et al. A population-based study on peanut, tree nut, fish, shellfish, and sesame allergy prevalence in Canada. J Allergy Clin Immunol. 2010;125:1327–35.
15. Sicherer SH, Munoz-Furlong A, Burks AW, Sampson HA. Prevalence of peanut and tree nut allergy in the US determined by a random digit dial telephone survey. J Allergy Clin Immunol. 1999;103:559–62.
16. Sicherer SH, Burks AW, Sampson HA. Clinical features of acute allergic reactions to peanut and tree nuts in children. Pediatrics. 1998;102:1–6.
17. Fujimori A, Yamashita T, Kubota M, Saito H, Takamatsu N, Nambu M. Comparison of the prevalence and characteristics of food hypersensitivity among adolescent and older women. Asia Pac J Clin Nutr. 2016;25:858–62.
18. Eriksson NE, Möller C, Werner S, Magnusson J, Bengtsson U, Zolubas M. Self-reported food hypersensitivity in Sweden, Denmark, Estonia, Lithuania, and Russia. J Investig Allergol Clin Immunol. 2004;14:70–9.
19. Soller L, Ben-Shoshan M, Harrington DW, et al. Overall prevalence of self-reported food allergy in Canada. J Allergy Clin Immunol. 2012;130:986–8.

20. Bedolla-Barajas M, Bedolla-Pulido TR, Macriz-Romero N, Morales-Romero J, Robles-Figueroa M. Prevalence of peanut, tree nut, sesame, and seafood allergy in Mexican adults. Rev Invest Clin. 2015;67:379–86.

21. Nwaru BI, Hickstein L, Panesar SS, et al., EAACI Food Allergy and Anaphylaxis Guidelines Group. Prevalence of common food allergies in Europe: a systematic review and meta-analysis. Allergy. 2014;69:992–1007.

22. Osterballe M, Hansen TK, Mortz CG, Høst A, Bindslev-Jensen C. The prevalence of food hypersensitivity in an unselected population of children and adults. Pediatr Allergy Immunol. 2005;16:567–73.

23. 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.

24. McWilliam VL, Koplin JJ, Field MJ, et al. Self-reported adverse food reactions and anaphylaxis in the SchoolNuts study: a population-based study of adolescents. J Allergy Clin Immunol. 2018;141:982–90.

25. Afify SM, Pali-Schöll I. Adverse reactions to food: the female dominance – a secondary publication and update. World Allergy Organ J. 2017;10:1–8.

26. Ridolo E, Incorvaia C, Martignago I, Caminati M, Canonica GW, Senna G. Sex in respiratory and skin allergies. Clin Rev Allergy Immunol. 2018:1–11.

27. Lack G. Epidemiologic risks for food allergy. J Allergy Clin Immunol. 2008;121:1331–6.

28. Sicherer SH, Sampson HA. Food allergy: a review and update on epidemiology, pathogenesis, diagnosis, prevention, and management. J Allergy Clin Immunol. 2018;141:41–58.