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Peanut Allergy in Mexican Children: What Is the Effect of Age at First Consumption?

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ABSTRACT

Studies suggest that children who start solid foods early are at risk for developing food allergies. Herein, we evaluated the effects of the introduction of peanuts to the diets of children on emerging peanut allergies.

Children with allergic rhinitis and asthma were enrolled in the present study and evaluated in four stages. In the first stage, a clinical history was completed for all participants. In the second stage, skin tests were conducted to detect the sensitization to peanuts. In the third stage, the parents were interviewed about the peanut-eating habits of their children. In the fourth stage, children with a convincing history of allergy or a positive peanut skin test result were subjected to an open oral food challenge (OOFC).

Three hundred children in four groups were included, 58.2% of the subjects were male, and the mean age was 7.3±3.9 years. The median age of first exposure to peanuts in patients with peanut allergies was greater than that in children without peanut allergies (2 years versus 1 year; p=0.009). The multivariate analysis, including only those children subjected to the OOFC, revealed that the consumption of peanuts after the age of ≥2 years is a risk factor for developing a peanut allergy (odds ratio=8.0, 95% confidence interval 1.3-50.0, p=0.026).

The results of the present study showed that the late introduction of peanuts to children increases the risk of developing a peanut allergy.

Keywords: Child; Food hypersensitivity; Peanut hypersensitivity; Risk factors; Skin tests

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INTRODUCTION

Food allergy is one of the main manifestations of allergic disease in children, and peanuts rank among the major products associated with allergic diseases.

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Various studies worldwide have shown increases in the prevalence of peanut allergies,[1-3] and explanations for this trend remain unknown. Although genetic and environmental factors[4] play a relevant role, other elements, including the characteristics of the gut microbiota with respect to the hygiene hypothesis,[5,6] the consumption of peanuts during pregnancy and breast-feeding,[7,8] the introduction of peanuts to the diet,[9] and the exposure to products containing peanut derivatives,[10] might also affect this phenomenon.

Recent evidence has emerged, indicating that the late introduction of solids to the diets of infants could increase the risk of developing allergies.[11,12] Foods such as peanuts, wheat, oats, oatmeal, eggs, potatoes, fish, rice, and barley (among others) have been significantly associated with an increased risk of allergy sensitization or the development of allergic disease.[9,11-14]

The objectives of the present study were to determine the prevalence of a convincing history of sensitization and examine the development of peanut allergies in children with allergic disease and to evaluate the role of peanut introduction to the diets of children in the risk of developing a peanut allergy.

METHODS AND PATIENTS

Study Design and Population

We designed a comparative cross-sectional study in a sample of children with allergic disease who were recruited from September 2011 to September 2012. Children younger than 16 years old examined for the first time in the allergy clinic with a history compatible with asthma, allergic rhinitis, or atopic dermatitis and at least one positive skin prick test (SPT) for one of the tested allergens were included in the present study. Subjects with dermographism or clinical suspicion of immunodeficiency were not included.

Sample Selection

The sample was selected in four stages as summarized in Figure 1.

Stage 1

Each of the participants underwent a complete allergy clinical history, and subjects compatible with a diagnosis of allergic rhinitis, asthma, or atopic dermatitis were initially selected.

Stage II

The participants were subjected to SPT to detect sensitization to aeroallergens from the region, among which peanut extract was included. In the event that any of the skin test results were positive, the parents of the children were invited to participate in the present study, at which time the parents provided written informed consent.

Stage III

The parents of the participating children filled out a structured questionnaire provided during an interview in which the interviewer asked questions related to family history of allergic disease, peanut consumption habits, and symptoms associated with peanut consumption. Subjects responding affirmatively to the question “Has your child had any kind of discomfort, reaction, or symptom less than 2 hours after eating peanuts or a peanut product?” or who had positive SPT results to the peanut allergen test proceeded to the fourth stage.

Stage IV

The children selected in the previous stage were subjected to an open oral food challenge (OOFC), and the parents provided written informed consent. Children suspected of having anaphylaxis did not participate in the OOFC.

Measurements

Skin Prick Test

In addition to the routinely used allergens, to determine sensitization to peanuts, including a commercial extract (Hollister-Stier Laboratories, Spokane, WA, USA), all allergens were administered using a puncture technique. Histamine and glycerin served as positive and negative controls, respectively.

The allergens were administered to the volar part of the forearm, and a calibrated lancet (ALK-Abelló, Hørsholm, Denmark) was used for the puncture; the results were interpreted 15 minutes after the application, and a positive test result was indicated as a papule size at least 3 mm larger than that of the negative control.[15]

Open Oral Food Challenge Test

The challenges were performed under close medical supervision. As a precaution, equipment necessary to treat an anaphylactic reaction was on hand. The

maximum quantity of peanuts used for the OOFC was two tablespoons of peanut cream (approximately 8 g of peanut protein), divided into four portions administered at 15-minute intervals. The results of the test were interpreted in accordance with[16] (a) a clear and objective clinical reaction: the challenge was stopped; (b) a questionable reaction: after 15 minutes, the dose of peanuts was increased; and (c) no clinical reaction: the total dose was administered. In addition, the parents received instructions to report by telephone any type of reaction that might arise in the 24 hours after the OOFC.

Definitions

Atopic state was defined as the presence of at least one positive result to an SPT for the tested allergens.

To identify self-reported peanut allergies, we used a standardized question previously used in several epidemiological studies:[1,17,18] "Has your child had any kind of discomfort, reaction, or symptom less than 2 hours after eating peanuts or a peanut product?" A convincing history of peanut allergy was indicated as an affirmative answer to the previous question and the involvement of organs and symptoms typical of allergic reaction (skin: urticaria and angioedema; respiratory system: difficulty breathing; gastrointestinal system: vomiting and diarrhea), triggered less than 2 hours after consuming peanuts.

Sensitization to peanuts was defined as the presence of a positive result to a skin test for the corresponding extract, and peanut allergy was defined as a positive result to the OOFC test.

Statistical Analyses

We determined the prevalence of a convincing history of peanut allergy, sensitization and allergy to peanuts, and associated clinical characteristics. For the quantitative variables, we determined the median, mean, and standard deviation. To compare proportions, we used the chi-squared test and Fisher's exact test as necessary. The quantitative variables for independent groups were compared using Student's *t* test or the Mann-Whitney U rank test according to distribution. The association between peanut allergy and age of the first exposure to peanuts was evaluated using an odds ratio (OR). We calculated the 95% confidence interval (95% CI) and OR. The risk factors were adjusted for potential confounding factors (age and sex) using logistic regression. Statistical significance was indicated as a *p* value of ≤0.05. For data processing, we used version 20.0 of IBM SPSS software (IBM Corporation, Armonk, NY, USA).

Ethics

The parents of the participating children provided written informed consent, and when necessary, the parents also provided consent for the OOFC. This study was reviewed and approved through the local ethics (Number 116/11) and research committees.

RESULTS

Three hundred and four children were included in the present study, of which 58.2% were male, and the mean age was 7.3±3.9 years. The children were exposed to peanuts for the first time at a median age of 2 years (Table 1); all of the participants had eaten peanuts at least once in their life, and the median current consumption was 4 days per month.

The prevalence of self-reported peanut allergy was 5.9% (95% CI 3.2%-8.5%) at the age of first consumption of peanuts (Table 2); a similar value (5.3%, 95% CI 2.8%-7.8%) was reported for the age of 6 to 7 years. In general, the symptoms most commonly associated with eating peanuts were coughing, runny nose, and itching nose. Only 2% (6 out of 304) (95% CI 0.4%-3.6%) of the children presented a convincing history of peanut allergy.

According to the results of the SPT, the prevalence of sensitization to peanuts was 7.2% (21 out of 304) (95% CI 4.3%-10.1%).

The clinical characteristics of the 24 children warranting an OOFC are presented in Table 3. No

Table 1. Peanut consumption patterns in children with allergic diseases

	n=304
Age at Timing of the first exposure, median (P_{25} - P_{75}), years	2 (1-2)
Current food consumption, n (%)	
Peanuts	304 (100)
Candy with peanuts	302 (99.3)
Peanut butter	119 (39.1)
Other	39 (12.8)
Current frequency of consumption, median (P_{25} - P_{75}), days/month	4 (0-8)

P_{25} - P_{75} = percentiles 25 and 75, respectively.

Table 2. Prevalence of auto-reported peanut allergies and clinical manifestations

Prevalence	n=304
Timing of consumption, (%)	
First time	18 (5.9)
Current	16 (5.3)
Type of manifestation, (%)	n=16
Coughing	10 (62.5)
Rhinorrhea	3 (18.8)
Nasal itching	3 (18.8)
Urticaria	2 (12.5)
Colic	2 (12.5)
Wheezing	2 (12.5)
Diarrhea	1 (6.2)
Fever	1 (6.2)

OOFC was suspended on account of an anaphylactic reaction. The mean age of this group was 7.5±4.2 years, and 17 cases were male (70.8%). Asthma without rhinitis and rhinitis without asthma were observed in three and six cases, respectively; no child had atopic dermatitis. In total, the results of 10 OOFC tests were considered positive, with a peanut allergy prevalence of 3.3% (10 out of 304) (95% CI 1.3%-5.3%); all cases had positive skin test results, and only two cases had a convincing history of peanut allergy. The most common symptoms after open oral challenge were nasal itching and rhinorrhea.

As shown in Table 4, children with a convincing history of peanut allergy had a tendency to eat peanuts more frequently, but statistical significance was not reached (p=0.395). Children exhibiting sensitization to

Table 3. Clinical characteristics in the children with open oral food challenge

No.	Sex	Age	Atopic disease			Convincing history of allergy	Skin prick test to peanut	OOFC	
			BA	AR	AD				
1	M	9	No	Yes	No	-	+	-	
2	M	4	Yes	Yes	No	-	+	-	
3	M	3	Yes	Yes	No	-	+	-	
4	M	6	Yes	Yes	No	-	+	-	
5	M	6	Yes	No	No	-	+	-	
6	F	8	No	Yes	No	-	+	-	
7	M	10	No	Yes	No	-	+	-	
8	M	14	Yes	Yes	No	-	+	-	
9	M	14	Yes	Yes	No	-	+	-	
10	F	5	Yes	Yes	No	-	+	-	
11	M	4	Yes	Yes	No	+	-	-	
12	F	4	Yes	Yes	No	+	-	-	
13	F	5	Yes	Yes	No	+	+	-	
14	F	1	Yes	No	No	+	-	-	
15	M	2	Yes	Yes	No	-	+	+	Nasal itching, rhinorrhea
16	M	9	Yes	No	No	-	+	+	Pharyngeal itching, coughing
17	F	5	No	Yes	No	-	+	+	Coughing, itchy skin
18	M	12	No	Yes	No	-	+	+	Nasal itching, rhinorrhea, coughing
19	M	15	Yes	Yes	No	-	+	+	Nasal itching
20	M	10	Yes	Yes	No	-	+	+	Pharyngeal and nasal itching
21	M	5	Yes	Yes	No	-	+	+	Rash
22	M	11	Yes	Yes	No	-	+	+	Nasal itching, rhinorrhea
23	F	5	No	Yes	No	-	+	+	Rhinorrhea, Nasal itching, rash
24	M	14	Si	Yes	No	+	+	+	Nasal itching

OOFC: Open oral food challenge

M: Male; F: Female; BA: Bronchial asthma; AR: Allergic rhinitis; AD: Atopic dermatitis

Table 4. Associated factors with a convincing history, sensitization and allergy to peanuts

	Convincing history		*p*	Sensitization		*p*	Allergy		*p*
	Yes n=6	No n=298		Yes n=22	No n=282		Yes n=10	No n=14	
Age (years), mean ± SD	5.5±4.4	7.3±3.9	.267	8.1±3.9	7.2±4.0	.361	8.8±4.4	6.2±4.0	.121
Male sex, n (%)	2 (33.3)	175 (58.7)	.240	17 (77.3)	160 (56.7)	.059	8 (77.3)	11 (61.1)	.294
Family history, n (%)									
Asthmatic mother	1 (16.7)	48 (16.1)	.999	4 (18.2)	45 (16.0)	.765	1 (10.0)	4 (28.6)	.358
Asthmatic father	0 (0)	37 (12.4)	.999	2 (9.1)	35 (12.4)	.999	1 (10.0)	1 (7.1)	.999
Allergic mother	1 (16.7)	65 (21.8)	.999	7 (31.8)	59 (20.8)	.253	2 (20.0)	6 (42.9)	.388
Allergic father	0 (0)	27 (9.1)	.999	2 (9.1)	25 (8.9)	.999	1 (10.0)	1 (7.1)	.999
Any allergic family	0 (0)	73 (24.5)	.342	8 (36.4)	65 (23.0)	.159	3 (30.0)	5 (35.7)	.999
Positive skin prick test, n (%)									
Indoor	5 (83.3)	258 (86.6)	.584	17 (77.3)	246 (87.2)	.188	8 (80.0)	10 (71.4)	.999
Trees	4 (66.7)	187 (62.8)	.999	14 (63.6)	177 (62.8)	.935	6 (60.0)	9 (64.3)	.999
Grasses	3 (50.0)	90 (30.2)	.375	9 (40.9)	84 (29.6)	.276	5 (50.0)	6 (42.9)	.729
Weeds	2 (33.3)	104 (34.9)	.999	8 (36.4)	98 (34.8)	.879	5 (50.0)	4 (28.6)	.403
Mold	1 (16.7)	22 (7.4)	.379	3 (13.6)	20 (7.1)	.225	1 (10.0)	2 (14.3)	.999
Milk	0 (0)	24 (8.1)	.999	0 (0)	24 (8.5)	.236	0 (0)	(0)	-----
First exposition, age (years), median	2	2	.957	2.0	0.92	.925	2	1	.009
Frequency of consumption, median days/ month	8	4	.395	4	12	.196	4	6	.709
Type of allergic disease, n (%)									
AR + BA	4 (66.7)	123 (41.3)	.240	14 (63.6)	113 (40.1)	.031	6 (60.0)	9 (64.3)	.999
AR	1 (16.7)	88 (29.5)	.675	6 (27.3)	83 (29.4)	.830	3 (30.0)	3 (21.4)	.665
BA	1 (16.7)	77 (25.8)	.999	2 (9.1)	76 (27.0)	.077	1 (10.0)	2 (14.3)	.999
AD	0 (0)	5 (1.7)	.999	0 (0)	5 (1.8)	.999	0 (0)	0 (0)	-----
AR + BA + AD	0 (0)	5 (1.7)	.999	0 (0)	5 (1.8)	.999	0 (0)	0 (0)	-----

BA: Bronchial asthma; AR: Allergic rhinitis; AD: Atopic dermatitis

peanuts showed fewer propensities to eat these foods (p=0.196), and sensitization was more common among children who suffered from asthma accompanied by allergic rhinitis (p=0.031).

The median age of first exposure to peanuts in patients with a positive result to the OOFC test for peanuts was greater than that in children without a peanut allergy, and this difference was statistically significant (p=0.009).

The multivariate analysis (Table 5), which included participants subjected to an OOFC test, revealed that children who consumed peanuts at the age of ≥ 2 years had a greater risk of developing a peanut allergy (OR=8.0, 95% CI 1.3-50.0).

Table 5. Risk of developing peanut allergy*

	Unadjusted risk			Adjusted risk **		
	OR	95% CI	*p* value	OR	95% CI	*p* value
History of peanut consumption for the first time ≥ 2 years old	11.8	1.4-98.0	0.022	8.0	1.3-50.0	0.026
History of breastfeeding	2.4	0.28-21.4	0.418	-	-	0.153
Actual age (years)	1.1	0.9-1.4	0.381	-	-	0.161
Sex (female)	0.39	0.04-3.8	0.424	-	-	0.180

* Peanut allergy: Positive result with open oral food challenge

** Adjustment variables by breastfeeding history, actual age, sex.

OR: Odds ratio (obtained with logistic regression)

95% CI: 95% confidence interval

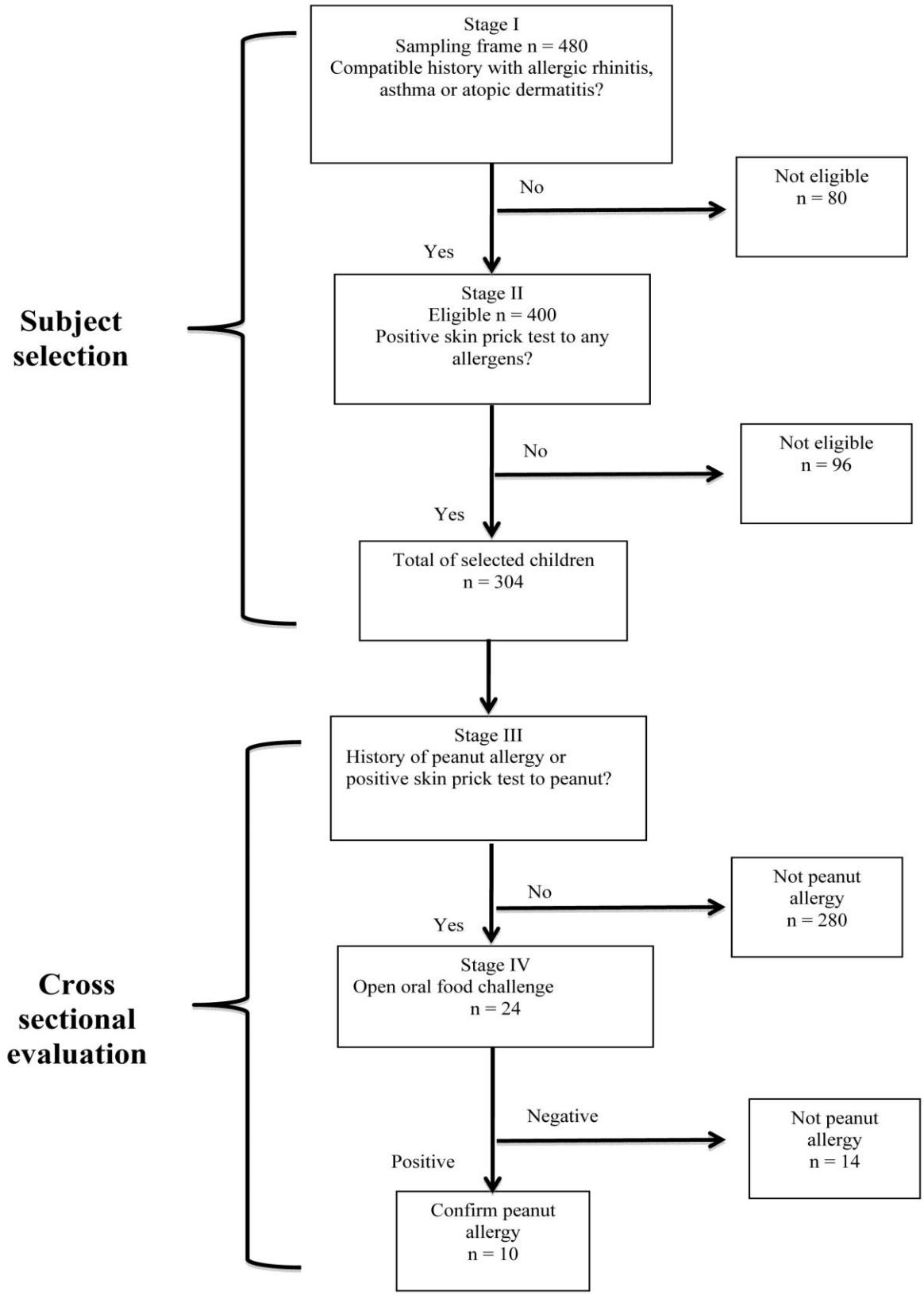

Figure 1. Flowchart of selection and evaluation of the study subjects

DISCUSSION

Mexico is an emerging country in which foods such as peanuts constitute an alternative source of protein. The consumption of peanuts is recommended in government programs as part of adequate nutrition for school-aged children. Nonetheless, until recently, little was known about the best age to introduce peanuts into the diets of children. The results of the present study showed that in children with allergic disease, the prevalence of peanut allergy was 3.3%. In addition, the results suggested that the late introduction of peanuts to the diets of children is a major risk factor associated with the development of a peanut allergy.

The prevalence of peanut allergy has increased in diverse regions of the world, potentially reflecting the definition, study populations, and methodologies used[19] but not an actual increase. Nonetheless, in the US, a telephone survey conducted during three different time periods showed that the rates of prevalence were 0.4% in 1997, 0.8% in 2003, and 1.4% in 2008.[1] Similarly, in a study of three cohorts of children in the UK,[2] the prevalence of sensitization to peanuts increased from 1.3% to 3.3% in the 1989 and 1994-1996 cohorts (p=0.003) and decreased to 2.0% in the 2001-2002 cohort, although this decrease was not statistically significant (p=0.145). In the same study, clinical allergy to peanuts also showed a significant increase from 0.5% to 1.4% (p=0.023), with a subsequent decrease to 1.2% (p=0.850). Findings from a study of a cohort of children provided estimates of the prevalence of peanut allergy according to several criteria, and these estimates were consistent with those from previous studies in the US, concluding that peanut allergy is an increasingly frequent condition.[3]

In the present study, we analyzed the role of the introduction of peanuts to the diet of children and observed that late consumption (age of≥2 years) significantly increased allergy risk (OR 8.0, 95% CI 1.3-50.0). Du Toit and colleagues obtained similar findings,[9] reporting that the prevalence of peanut allergy in Jewish children living in the UK was 10 times greater than that of children in Israel (OR 9.8, 95% CI 3.1-30.5); because peanuts are introduced to Israeli children early, these individuals consume peanuts in larger quantities and with greater frequencies than do most children in the UK.

However, the issue of the introduction of solid foods and allergy risk is not exclusive to peanuts. Indeed, the introduction of wheat prior to the age of 6 months has been associated with the risk of developing a wheat allergy (OR 3.8, 95% CI 1.18-12.28).[11] In contrast, the late introduction of oats, eggs, potatoes, and fish was significantly associated with sensitization to allergens.[12]

Thus, the time of introducing solids to the diet of children not only reduces the risk of sensitization or allergy specific to that food but also reduces the risk of developing an allergic disease. A previous study reported that the consumption of fish at least two times a week during the first year of life was associated with a reduced risk of allergic disease (OR 0.76, 95% CI 0.61-0.94) and allergic sensitization (OR 0.76, 95% CI 0.58-1.0) at the age of 4 years;[13] similarly, the consumption of wheat, rice, oats, oatmeal, fish, and eggs at a slightly earlier age reduced the risk of asthma, allergic rhinitis, and atopic sensitization.[14] The mechanisms by which foods modulate the immune response at the intestinal level have yet to be investigated and remain elusive.

Given that a considerable proportion of parents indicated that their children had some type of discomfort upon the first consumption of peanuts (5.9%), it is possible that routes other than the oral route favor sensitization. Among these, the following routes have been described: cross-reactivity with another type of allergen, early exposure (*in utero*, through breast-feeding, the type of milk formula used, and the manner of weaning), the dietary habits of the family or culture, and contamination during the preparation of other foods or perhaps through the skin.[20]

Moreover, the results of the present study also showed that the prevalence of sensitization to peanuts was 7.2% (95% CI 4.3%-10.1%). These children were subjected to an OOFC, and the results for 10 subjects were considered positive, generating an overall prevalence of 3.3% peanut allergy. The sensitization prevalence observed in the present study was consistent with the values reported in children in Egypt (7%),[21] Germany (10.9%),[22] and Mexico (5.0%).[23]

Limitations

One of the main limitations of the present study was a potential memory bias, as parents with children allergic to peanuts would remember with greater precision the time of introducing peanuts to the diets of these children. In addition, we cannot ascertain the

precise amount of peanuts or peanut products consumed, as we did not initially consider having the parents maintain a log of the food consumption for the children. Moreover, patients with atopic dermatitis, in whom the prevalence of food allergies is much greater, were not included in the present study. It is appropriate to clarify that the majority of these children were receiving regular medical attention from dermatologists. Furthermore, the population of children included in the present study was obtained from a university hospital, which serves the general public.

To the best of our knowledge, this study is the first to estimate the prevalence of peanut allergy in Mexican children. In addition, the results of the present study provide evidence that the early introduction of solid foods to the diets of children reduces the risk of developing allergies.

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The population of children in the present study was obtained from a university hospital, which serves the general public.

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