# Prevalence of peanut allergy in primary-school children in Montreal, Canada

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**Background: Peanut allergy is receiving increasing attention. Only one study has estimated the prevalence in North America, but it did not corroborate history with diagnostic testing.**
**Objective: We estimated the prevalence of peanut allergy in Montreal by administering questionnaires regarding peanut ingestion to children in kindergarten through grade 3 in randomly selected schools.**
**Methods: Respondents were stratified as follows: (1) peanut tolerant, (2) never-rarely ingest peanut, (3) convincing history of peanut allergy, and (4) uncertain history of peanut allergy. Groups 2, 3, and 4 underwent peanut skin prick tests (SPTs), and if the responses were positive in groups 2 or 4, measurement of peanut-specific IgE were undertaken. Children in group 3 with a positive SPT response were considered allergic to peanut without further testing. Children in groups 2 and 4 with peanut-specific IgE levels of less than 15 kU/L underwent oral peanut challenges.**
**Results: Of the 7768 children surveyed, 4339 responded, 94.6% in group 1. The prevalence of peanut allergy was 1.50% (95% CI, 1.16%-1.92%). When multiple imputation was used to incorporate data on those responding to the questionnaire but withdrawing before testing, the estimated prevalence increased to 1.76% (95% CI, 1.38%-2.21%). When data regarding the peanut allergy status of nonresponders (as declared to the school before the study) were also incorporated, the estimated prevalence was 1.34% (95% CI, 1.08%-1.64%).**
**Conclusion: Our prevalence study is the first in North America to corroborate history with confirmatory testing and the largest worldwide to incorporate these techniques. We have shown that, even with conservative assumptions, prevalence exceeds 1.0%. (J Allergy Clin Immunol 2003;112:1223-8.)**

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Peanut allergy is receiving increasing public attention. It accounts for the majority of severe food-related allergic reactions, tends to present early, usually lasts for life, and can be provoked by trace quantities of peanut in highly sensitized individuals.[1-10] Seventy percent to 80% of children allergic to peanut have their first reaction between 14 and 24 months of age, usually at the time of their first exposure.[3,4,9] Currently, there are no curative therapies, and management is limited to avoidance and symptomatic treatment of reactions with epinephrine. Although various immunomodulatory approaches to decrease peanut sensitivity hold potential, they remain investigational.[11,12] Up to 75% of known individuals with peanut allergy experience reactions caused by inadvertent exposure, reflecting the difficulty of complete peanut avoidance.[6,13,14]

Despite current opinion that the prevalence of peanut allergy might be increasing,[15,16] much of the evidence is inconclusive,[17,18] and further research with standardized methodologies is needed. Five population-based studies have estimated the prevalence of peanut allergy,[5,19-22] but only 2, conducted on the Isle of Wight, United Kingdom, corroborated history with diagnostic testing. These latter 2 studies reported an increase in peanut allergy prevalence from 0.5% to 1.5% between 1994 and 2000.[5,22] A single American and 2 European surveys conducted between 1995 and 1998 estimated the prevalence of peanut allergy to be between 0.5% and 1.0%.[19-21]

Our study estimated the prevalence of peanut allergy by surveying a randomly selected sample of Montreal schoolchildren aged 5 to 9 years and confirming peanut allergy with skin prick tests (SPTs), peanut-specific IgE measurement, and double-blind, placebo-controlled food challenges (DBPCFCs) with peanut.

## METHODS

### Sampling frame

We conducted a cross-sectional study involving a simple random selection of kindergarten through grade 3 classrooms in the public and private schools of Montreal, Quebec, Canada. All children in each selected classroom were invited to participate. The study was approved by the Institutional Review Board of the McGill University Health Centre, as well as by all participating school boards, individual schools, and all parents, who provided written informed consent. Children were recruited between December 2000 and September 2002.

*Abbreviations used*
DBPCFC: Double-blind, placebo-controlled food challenge
SPT: Skin prick test

We sought to accrue 3500 children, which would enable us to estimate the prevalence of peanut allergy to within at least ±0.4% with a 95% CI, assuming the prevalence was 1.5% or less. Given an anticipated response rate of 50%, approximately 7000 children (from 4 to 5 randomly selected classes per randomly selected school) would be required to attain the desired sample.

## Criteria for diagnosis of peanut allergy

The diagnosis of peanut allergy was made only if one of the following conditions was fulfilled:

1. a child who had never or rarely ingested peanuts or had an uncertain clinical history of an IgE-mediated reaction to peanut had either a positive SPT response to peanut AND a peanut-specific IgE level of 15 kU/L or greater OR a positive SPT response to peanut AND a positive DBPCFC response with peanut, or
2. a child who had a convincing clinical history of an IgE-mediated reaction to peanut had a positive SPT response OR peanut-specific IgE.

A convincing clinical history of an IgE-mediated reaction to peanut was defined as a minimum of 2 mild signs-symptoms or 1 moderate or 1 severe sign-symptom that was likely IgE mediated and occurred within 60 minutes after peanut ingestion or contact. Reactions were considered mild if they involved only pruritus, urticaria, flushing, or rhinoconjunctivitis; moderate if they involved angioedema, throat tightness, gastrointestinal complaints, or breathing difficulties (other than wheeze); and severe if they involved wheeze, cyanosis, or circulatory collapse.[9]

A SPT response to peanut was defined as positive when the greatest diameter of the wheal was at least 3 mm larger than that elicited by the negative control (saline) within 12 to 15 minutes of placement.[6] The SPT was performed by using a standard technique.[23] Lots of glycerinated extract from the same manufacturer were used throughout the study. When the SPT response was negative and the clinical history was convincing or uncertain, the SPT was repeated with crude extract.[24] The sensitivity and negative predictive value of the peanut SPT are 95% or greater, and the specificity and positive predictive value do not exceed 50%, except in cases of a convincing history, in which the positive predictive value is much higher.[23-28] In children in whom a SPT was required and previous results were unavailable, the SPT was performed by our study nurse in the presence of an allergist (RSK or AEC) at the child's school or hospital allergy clinic.

The serum level of peanut-specific IgE was measured with the CAP system Fluoroenzymeimmunoassay (Pharmacia Diagnostics, Uppsala, Sweden). Because the literature suggests that patients with a peanut-specific IgE level of 15 kU/L or greater have a greater than 95% likelihood of having an allergic reaction to peanut,[7,16,29,30] we assumed that children with a peanut-specific IgE level of 15 kU/L or greater were allergic to peanut without performing a DBPCFC.

The DBPCFC, considered the gold standard for the diagnosis of food allergy, was conducted in the hospital under the supervision of an allergist (RSK) according to previously published protocols.[31] Peanut flakes served as the source of peanut, and cracker crumbs served as the placebo. The peanut and placebo were disguised with either applesauce or grape jelly, depending on the child's preference. Challenges started with 10 mg of either peanut or placebo, and if tolerated, the dose was increased to 25 mg, 50 mg, 100 mg, 250 mg, 500 mg, 1 g, and 2.5 g every 15 to 30 minutes. The challenge result was considered positive if at least 2 of the mild manifestations described previously as characterizing a convincing clinical history or at least 1 of the moderate or severe manifestations occurred. If 2.5 g of peanut was tolerated, 14 g was administered in an open challenge.

## Determining whether a child is allergic to peanut

To determine whether children fulfilled the criteria for peanut allergy, a questionnaire was first administered to their parents-guardians that enabled the children to be stratified into 4 mutually exclusive groups (Fig 1): (1) ingests peanut-containing substances regularly without problems, (2) never or rarely ingest peanuts, (3) has a convincing clinical history of peanut allergy, and (4) has an uncertain clinical history of peanut allergy.

The questionnaire inquired about the child's frequency of ingestion of peanut-containing foods, characteristics of the most severe reaction to these foods, diagnostic testing performed for peanut allergy, the presence of other allergies, and other demographic details. When the parental response was incomplete or unclear, the parent was contacted by telephone. If the child ingested at least 1 peanut-containing food on a monthly basis or at least 2 different peanut-containing foods on at least 1 occasion each without any adverse event, the child was considered not allergic to peanut (group 1). However, if the child did not fulfill these criteria, the child was assigned to either group 2, 3, or 4.

A SPT to peanut was required to determine whether children in groups 2, 3, or 4 were allergic to peanut. For children in groups 2 and 4, if the SPT response was negative, the child was assumed not to be allergic to peanut. If the SPT response was positive, the child was invited to undergo peanut-specific IgE measurement and potentially a DBPCFC, depending on the results. For children in group 3, if the SPT response was positive, the child was assumed to be allergic to peanut. If the peanut SPT response was negative, the child was invited to undergo a peanut-specific IgE measurement and potentially a DBPCFC.

In the initial phases of the study, measurement of serum peanut-specific IgE was not available at our facility, and hence there were some children who progressed directly from a positive SPT response to a DBPCFC.

Parents were notified of the results of their children's testing. In circumstances in which parents refused the testing necessary to determine definitely whether their child was allergic to peanut, they were notified of the implications of not knowing the peanut allergy status of their child and were encouraged to consult with an allergist not participating in this study. When results suggested that the child might be allergic to peanut, avoidance of peanuts was advised, autoinjectable epinephrine was prescribed, and parents were instructed on its use.

## Statistical analysis

The point estimate and associated variance for the overall prevalence of peanut allergy were calculated by using standard formulas,[32] and 95% CIs were constructed. A preliminary point estimate and CI was based on the observed fraction of participants with peanut allergy of the total number of participants who completed the questionnaire and the necessary testing, as indicated in the study algorithm (ie, full responders). Given that the numerator and denominator might both be affected by selection bias, selection bias–adjusted estimates and CIs were also derived by using the information gathered from children who completed the questionnaire but withdrew before completion of the necessary diagnostic testing (ie, partial responders) and also from those who did not complete the initial questionnaire (ie, nonresponders) through a Bayesian bias correction technique called *multiple imputation*.[33,34]

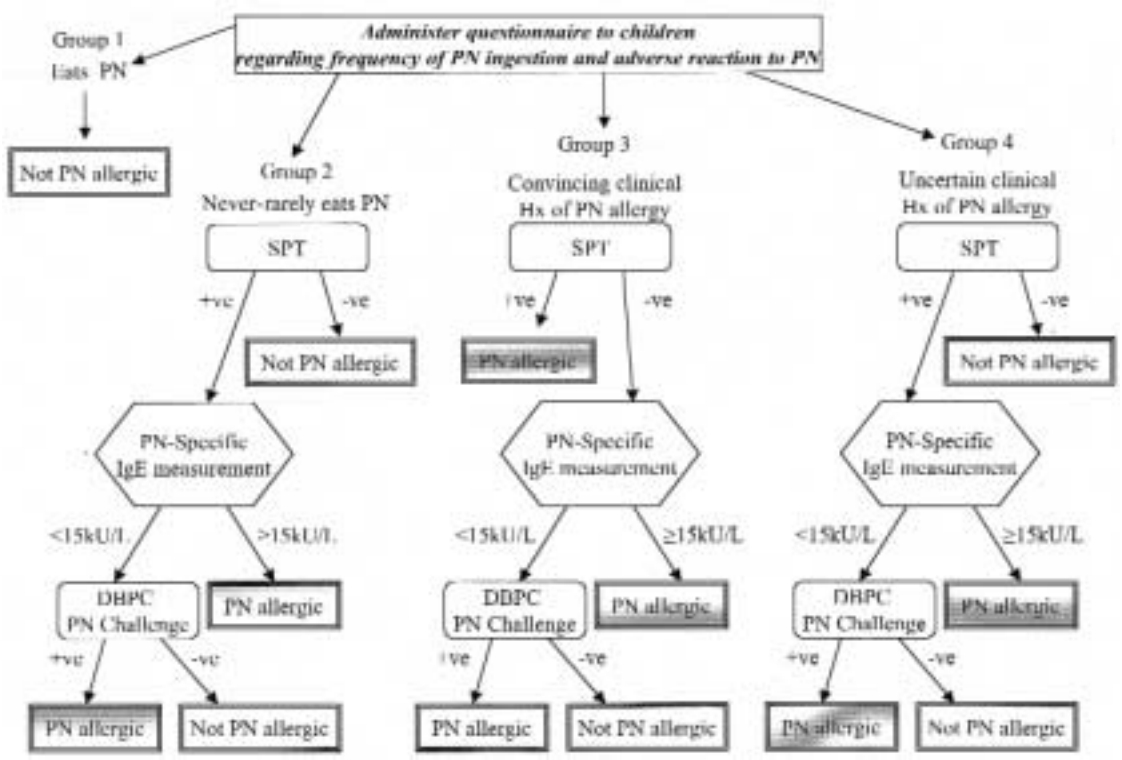


**FIG 1.** Study design for evaluating the prevalence of peanut allergy. *Hx,* History; *PN,* peanut.

These methods impute the patient-specific probability of peanut allergy on the basis of all available information, while accounting for the fact that the information is imperfect.

Logistic regression was used with the covariates of age, sex, ethnicity, grade, presence-absence of atopic features (as defined by presence-absence of asthma, allergic rhinitis, eczema, hives, anaphylaxis, other food allergies, or other allergies), characteristics of most severe reaction to peanut (if applicable), results of any diagnostic testing for peanut allergy (if performed), and whether the children had previously declared to the school they were allergic to peanut to predict the probability that the partial responders had peanut allergy.

Logistic regression was used with the covariates of school grade, sex, and self-declared peanut allergy status to predict the probability that the nonresponders were allergic to peanut. Schools were aware of which children were nonresponders and provided nonnominal data on their sex and self-declared peanut allergy status.

The above analyses assume that the information available on nonresponders is sufficient to accurately predict the prevalence of peanut allergy (ie, ignorable nonresponse).[33] A sensitivity analysis was therefore performed that was similar to the method of Kmetic et al[34] to account for the possibility that these variables do not completely adjust for selection bias (ie, nonignorable nonresponse) by increasing and decreasing the estimated prevalence of peanut allergy in the nonresponders by 25%.

## RESULTS

Seventy-six percent of the schools selected participated, 72.1% of the 68 public schools and 93.3% of the 15 private schools (Table I). Within these schools, 7768 children were surveyed, and 4339 responded.

**TABLE I.** Distribution of children in kindergarten through grade 3 in Montreal (1999-2000)* and distribution of study participants

| | Public schools | Private schools |
|---|---|---|
| No. of schools | 327 | 95 |
| No. of schools selected (% selected) | 68 (20.8) | 15 (15.8) |
| No. of schools surveyed (% of selected schools agreeing to be surveyed) | 49 (72.1) | 14 (93.3) |
| No. of children enrolled | 73,944 | 11,559 |
| No. of children surveyed (% surveyed) | 5997 (8.1) | 1771 (15.3) |
| No. of respondents (% of children surveyed responding) | 3310 (55.2) | 1029 (58.1) |

Because the prevalence estimate for peanut allergy did not differ between children in public and private schools, a weighted prevalence estimate was not needed to reflect the differential response rate between the public and private schools.
*Information provided by the Ministère de l'Education, Direction de la gestion des systèmes de collecte, Quebec, Canada.

Among the 4339 children who provided information on the initial questionnaire, 4105 tolerated peanuts (Fig 2). One hundred fifty-four had never or rarely ingested peanut (group 2), 57 had a convincing clinical history of peanut allergy (group 3), and 23 had an uncertain clinical history (group 4). Of those having a SPT, 16 in group 2, 54 in group 3, and 7 in group 4 had positive responses. In group 3, the 54 children with a positive SPT response were

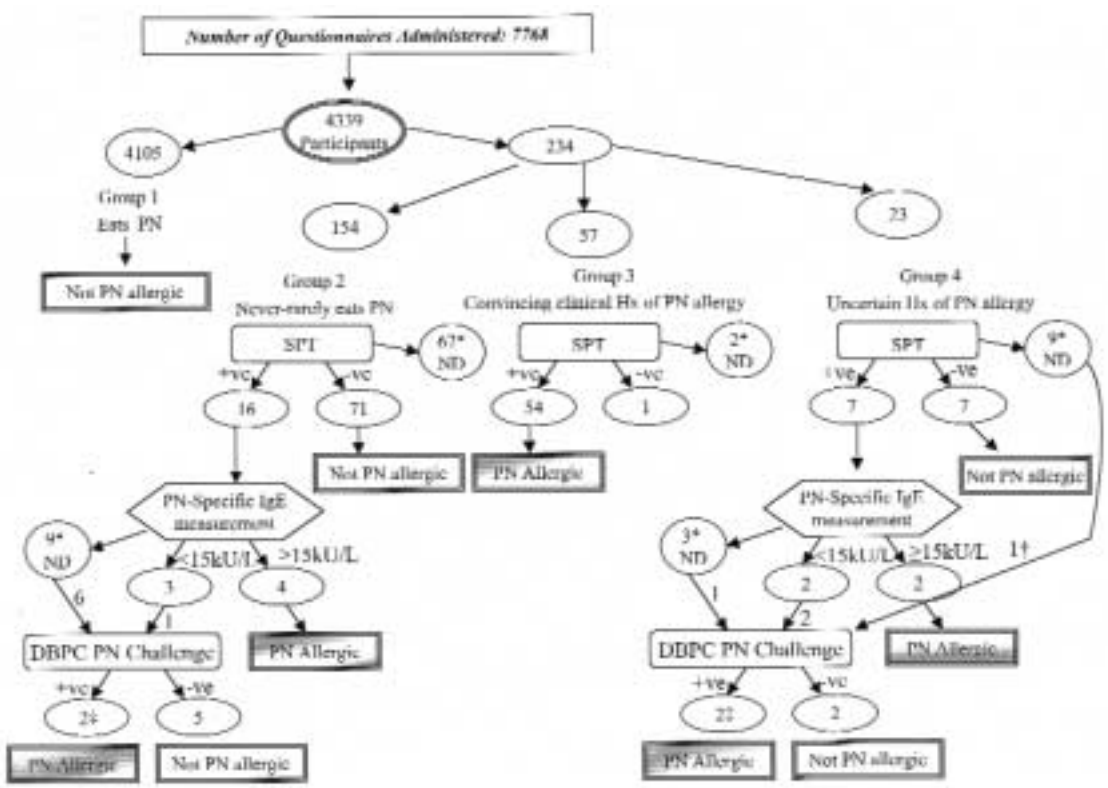


**FIG 2.** Evaluating the prevalence of peanut allergy. *Hx,* History; *ND,* not done or results not available; *PN,* peanut. *The number of children eligible for SPT or measurement of peanut-specific IgE exceeds the number actually undergoing the procedure because in some cases parents refused. †This child refused the SPT, and with parental approval, a challenge was performed, the result of which was negative. ‡One of these 2 children with a positive result on double-blind, placebo-controlled peanut challenge had a peanut-specific IgE level of less than 15 kU/L. Note: Among the 156 SPTs described in Fig 2, the results of 77 were positive, and the results of 79 were negative. Among the tests with positive results, 15 were performed by the study personnel, and 62 were obtained from the medical records. Among the tests with negative results, 70 were performed by the study personnel, and 9 were obtained from the medical records.



assumed to be allergic to peanut without further testing. In groups 2 and 4, 16 and 7 children, respectively, were eligible for measurement of peanut-specific IgE. Of children who had peanut-specific IgE measured in groups 2 and 4, 4 of 7 and 2 of 4 in the respective groups had peanut-specific IgE levels of 15 kU/L or greater and were assumed to be allergic to peanut. Of children in groups 2 and 4 who were eligible for DBPCFC, 1 of 3 and 2 of 2 consented, respectively. Additionally, 6 children from group 2 who were enrolled in the study before measurement of peanut-specific IgE and 2 children from group 4 also underwent challenges. Two of 7 challenge results in group 2 and 2 of 4 challenge results in group 4 were positive.

We report the prevalence of peanut allergy for 3 subsets of participants: (1) full responders, (2) full responders and partial responders, and (3) full responders, partial responders, and nonresponders. Among full responders, 64 were allergic to peanut, yielding a prevalence of 1.50% (95% CI, 1.16%-1.92%). In 60 of these patients, we either performed an SPT or obtained SPT results from their treating allergist; in 4 patients parents provided a verbal report. Among the subset of full and partial responders, the estimated prevalence was 1.76% (95% CI, 1.38%-2.21%). Among full responders, partial responders, and nonresponders, the estimated prevalence was 1.34% (95% CI, 1.08%-1.64%). A sensitivity analysis in which the prevalence estimate among the nonresponders was increased and decreased by 25% yielded an estimated prevalence of 1.43% (95% CI, 1.16%-1.74%) and 1.25% (95% CI, 1.00%-1.54%), respectively.

The mean age of participants was 7.4 (SD, 1.2) years (Table II). The ethnicity of the participants was reasonably representative of Montreal. When the children with peanut allergy were compared with those who were not allergic to peanut, they did not differ in terms of age or ethnicity, but there was significantly more atopy in the children with peanut allergy.

## DISCUSSION

Our prevalence study is the first in North America to corroborate history with confirmatory testing and the

**TABLE II.** Characteristics of study participants

| | Allergic to peanut (n = 64) | Not allergic to peanut (n = 4190) |
|---|---|---|
| Age, y: mean (SD) | 7.4 (1.2) | 7.4 (1.2) |
| 95% CI | (7.1-7.6) | (7.3-7.4) |
| % Male (95% CI) | 59.4 (46.4-71.5) | 48.6 (47.1-50.2) |
| Atopic features, % (95% CI) | | |
| Asthma | 56.3 (43.3-68.6) | 11.0 (10.1-12.0) |
| Eczema | 41.3 (29.0-54.4) | 9.1 (8.2-10.0) |
| Other food allergies | 87.5 (76.8-94.4) | 3.4 (2.8-4.0) |
| Ethnicity,* % (95% CI) | | |
| White | 70.3 (57.6-81.1) | 69.5 (68.0-70.9) |
| Black | 6.3 (1.7-15.2) | 7.3 (6.5-8.2) |
| Asian | 10.9 (4.5-21.2) | 6.6 (5.8-7.4) |
| Arabic | 0 (0-5.6)† | 7.3 (6.5-8.1) |
| Hispanic | 0 (0-5.6)† | 2.8 (2.3-3.3) |

*On the Island of Montreal, the population is 81.3% white, 5.7% black, 7.0% Asian, 3.4% Arabic, and 2.3% Hispanic (1996 Statistics Canada, data on visible minorities).
†One-sided 97.5% CI.

largest study worldwide to incorporate these techniques. Our design ensured that peanut-sensitized children were not considered allergic unless there was a supportive clinical history, peanut-specific IgE, or a positive DBPCFC result. We believe our results can be generalized because the study was conducted in a large, multiethnic urban area, with peanut consumption likely similar to that in other North American regions.

Potential limitations of our study include the participation rates and certain assumptions regarding the testing and questionnaire. Although our participation rates are modest, they are comparable with those reported in other prevalence studies.[19,22] We adjusted both for the possibilities that nonresponders might be less likely to be allergic and partial responders might be more likely to be allergic. After adjustment, in the first scenario the prevalence decreased to 1.34%, which still exceeds most previous estimates, and in the latter scenario the prevalence estimate increased to 1.76%.

It is possible that our assumption that peanut-specific IgE levels of 15 kU/L or greater are indicative of peanut allergy in children naive to peanut might have led to an overestimate of prevalence because this threshold has not been demonstrated for these children.[29] Although it is true that the probability of the 6 children in groups 2 and 4 who were considered to have peanut allergy on the basis of a peanut-specific IgE level of 15 kU/L or greater is less than 100%, the literature supports that it exceeds 0%; hence it would be incorrect to remove them completely from the prevalence numerator. We can, however, adjust the prevalence estimate to reflect that their probability of peanut allergy is less than 100%. The literature indicates that the probability of peanut allergy for the 2 children with an uncertain clinical history and a peanut-specific IgE level of 15 kU/L or greater is at least 95%. There is no literature on the positive predictive value of the peanut-specific IgE level in peanut-naive children. However, our work has shown that peanut-naive children with a positive SPT response have a 50% likelihood of having peanut allergy[35]; hence, we can assume that those with a positive SPT response and an increased peanut-specific IgE level would have an even greater likelihood of having peanut allergy. By using these assumptions, the prevalence estimate changes from 1.50% to 1.48% (95% CI, 1.14%-1.89%).

It is also possible that some children who were categorized as being peanut tolerant on the basis of their response that they ate at least 2 peanut-containing products on 1 occasion were mislabelled. Their ingestion might have been remote, and they might have had a subsequent peanut allergy. However, this would make our estimates conservative. Furthermore, because most children with peanut allergy have symptoms on their first known exposure to peanut and we surveyed only school-age children who would likely have already been exposed to peanut,[3,4,9] this is unlikely to result in a clinically meaningful change in the estimated prevalence. Finally, given that resolution of peanut allergy has recently been reported in up to 20% of cases,[7,13,36-38] 3 of the 16 children who we considered to have peanut allergy on the basis of a remote reaction to peanut and a remote positive SPT response might have actually lost their sensitivity.

Our study shows that the prevalence of peanut allergy is higher than what has previously been reported in the only other North American study[19] and in 3 European surveys.[5,20,21] It is possible that our study suggests that the increasing peanut allergy prevalence reported on the Isle of Wight[22] is also occurring in North America. However, this is speculative because our study does not track prevalence over time; furthermore, comparisons between studies are hampered by different methodologies for assessing allergy, different regions surveyed, overlapping CIs, and the possibility of nonresponse bias. Although we made every effort to adjust for bias, other studies with relatively low response rates did not provide adjusted estimates.[19,22] Numerous theories have been advanced to explain this perceived increase in peanut allergy prevalence. These include increased consumption of peanut

products,[16] sensitization through breast milk by maternal ingestion,[39] application of skin products that contain peanut oil,[40] early introduction of peanut-containing foods to young children, and the general trend toward increasing atopic diseases.[41,42]

Through the most rigorously conducted study on peanut allergy prevalence in North America, we have shown that even with the most conservative assumptions, prevalence exceeds 1.0%. Considering the potential severity of peanut allergy, the frequency of accidental peanut exposure, and the current perception of increasing prevalence, efforts to educate the public must be expanded, while efforts to evaluate the prevalence over time must be pursued.

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