Is the prevalence of peanut allergy increasing? A 5-year follow-up study in children in Montreal

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Background: Studies suggest that peanut allergy prevalence might be increasing, but these results have not yet been substantiated.
Objective: We conducted a follow-up study to determine whether peanut allergy prevalence in Montreal is increasing.
Methods: Questionnaires regarding peanut ingestion were administered to parents of children in randomly selected kindergarten through grade 3 classrooms between December 2000 and September 2002 and between October 2005 and December 2007. Respondents were stratified as (1) peanut tolerant, (2) never/rarely ingest peanut, (3) convincing history of peanut allergy, or (4) uncertain history of peanut allergy. Children in group 3 with positive skin prick test responses were considered to have peanut allergy. Children in groups 2 and 4 with positive skin prick test responses had peanut-specific IgE levels measured, and if the value was less than 15 kU/L, an oral peanut challenge was performed. Multiple imputation was used to generate prevalence estimates that incorporated respondents providing incomplete data and nonrespondents.
Results: Of 8,039 children surveyed in 2005-2007, 64.2% of parents responded. Among those providing complete data, the prevalence was 1.63% (95% CI, 1.30% to 2.02%) in 2005-2007 versus 1.50% (95% CI, 1.16% to 1.92%) in 2000-2002. After adjustment for missing data, the prevalence was 1.62% (95% credible interval, 1.31% to 1.98%) versus 1.34% (95% credible interval, 1.08% to 1.64%), respectively. The differences between the prevalences in 2005-2007 and 2000-2002 were 0.13% (95% credible interval, −0.38% to 0.63%) among those providing complete data and 0.28% (95% credible interval, −0.15% to 0.70%) after adjustment for missing data.
Conclusions: This is the first North American study to document temporal trends in peanut allergy prevalence by corroborating history with confirmatory tests. The results suggest a stable prevalence, but wide CIs preclude definitive conclusions.
(J Allergy Clin Immunol 2009;123:783-8.)

Key words: *Peanut allergy, prevalence, skin prick test, peanut-specific IgE, food challenge, epidemiology*

During the last 2 decades, the medical literature reports an increase in allergic diseases,[1] including peanut allergy. Based primarily on longitudinal studies conducted in the United States and the Isle of Wight,[2,3] it is speculated that the prevalence of peanut allergy might have doubled over 5 years. However, this apparent increase might be attributed to a failure to apply rigid and inclusive diagnostic criteria, methodological differences, overlapping CIs, and/or nonresponse bias. Between December 2000 and September 2002, we conducted the first Canadian study to estimate the prevalence of peanut allergy[4]; ours was also the first study in North America to corroborate history with confirmatory testing and the largest study worldwide to fully incorporate these techniques.[4] Although our estimate of peanut allergy prevalence of 1.5% (95% CI, 1.16% to 1.92%) in Montreal exceeds North American and most European estimates,[2-5] it cannot be concluded that the prevalence of peanut allergy is increasing. Our study did not evaluate prevalence over time, and comparisons with other studies are hampered by differences in methodologies and sampling frames, overlapping CIs, and nonresponse bias. To determine whether the prevalence of peanut allergy is increasing, we conducted a follow-up study between October 2005 and December 2007 using the identical methodology and sampling frame of our 2000-2002 study. It is only by replicating a methodology that corroborates clinical history with comprehensive diagnostic testing, sampling an identical population, ensuring an adequate sample size, and adjusting for nonresponse that we can determine whether this speculated increase is real.

METHODS

Sampling frame

We conducted a cross-sectional study, revisiting the schools participating in our original study[4] and randomly selecting kindergarten through grade 3 classrooms. The study was approved by the Institutional Review Board of the McGill University Health Centre, school boards, individual schools, and parents. Children were recruited between October 2005 and December 2007. Public schools refusing to participate in the follow-up study were replaced by other randomly selected schools.

For our 2-sample design (ie, a comparison of the prevalence between the 2000-2002 and 2005-2007 studies), we required 4,315 children in each sample to estimate the prevalence to an accuracy of at least ±0.625% with a 95% CI,

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Supported by the Canadian Institutes of Health Research (MOP-77584); the Allergy, Genes and Environment Network Centres of Excellence (AllerGen NCE); Anaphylaxis Canada; the Canadian Allergy, Asthma and Immunology Foundation; and Novartis Pharmaceuticals. Dr Clarke received a $2500 unrestricted grant from Novartis Pharmaceuticals. Drs Joseph and Clarke are National Scholars of Fonds de la recherche en santé du Québec.

Disclosure of potential conflict of interest: M. Ben-Shoshan receives grant support from AllerGen. R. S. Kagan is on the Board of Directors for the Canadian Society of Allergy and Clinical Immunology (CSACI) and receives honoraria for speaking for King Pharmaceuticals and Merck Frosst. R. Alizadehfar is on the speakers' bureau for Paladian Pharma and receives research support from Allergen. A. E. Clarke receives grant support from AllerGen; Anaphylaxis Canada; and the Canadian Allergy, Asthma and Immunology Foundation. The rest of the authors have declared that they have no conflict of interest.

Received for publication October 10, 2008; revised January 29, 2009; accepted for publication February 2, 2009.

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0091-6749/$36.00

doi:10.1016/j.jaci.2009.02.004

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

assuming the prevalence had increased from 1.5% to 3.0%. Because we expected a response rate similar to the 56% seen in our original prevalence study,[4] 7,705 children (from 6-7 randomly selected classes per 63 schools previously sampled) were required to attain the desired sample.

Criteria for diagnosis of peanut allergy

The diagnosis of peanut allergy was made (as in our original study) only if one of the following was fulfilled: (1) a child who had never or rarely ingested peanut or had an uncertain clinical history of an IgE-mediated reaction to peanut had either a positive skin prick test (SPT) response to peanut AND a serum peanut-specific IgE level of greater than 15 kU/L OR a positive SPT response to peanut AND a positive double-blind, placebo-controlled food challenge (DBPCFC) result 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 a peanut-specific IgE level of greater than 0.35 kU/L OR a positive DBPCFC result.

A convincing clinical history of an IgE-mediated reaction 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 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.[6]

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 within 10 to 15 minutes of placement.[7] In children who required a SPT to determine whether they were allergic and previous results were unavailable, the SPT was performed by our nurse in the presence of an allergist at the child's school or hospital allergy clinic by using the prick technique and glycerinated peanut extract supplied by ALK-Abelló (Hørsholm, Denmark). In this technique a drop of peanut extract was placed on the skin, and a solid-bore smallpox needle (Hollister-Stier, Spokane, Wash) was passed through it; histamine phosphate in 50% glycerin served as the positive control, and 50% glycerosaline served as the negative control. In children with a convincing or uncertain history who had a negative SPT response with commercial extract, the test was repeated with crude extract (ie, peanut butter). Although the positive predictive values of a SPT response of 3 mm or larger is only 61%,[7] it might be considered diagnostic in patients who experience a systemic anaphylactic reaction after the ingestion of an isolated food.[8]

The serum level of peanut-specific IgE was measured with the CAP System Fluoro Enzyme Immunoassay (Phadia AB Diagnostics, Uppsala, Sweden). In children who had never or rarely ingested peanut or had an uncertain history, peanut-specific IgE levels of greater than 15 kU/L were considered sufficient to diagnose peanut allergy without performing a DBPCFC.[9-12] In children with a convincing history, it is believed that a very high positive predictive value is attained at a much lower threshold of peanut-specific IgE. Hence, in children with a convincing history, peanut-specific IgE levels of greater than 0.35 kU/L were considered sufficient to diagnose peanut allergy.

The DBPCFC was conducted in the hospital under the supervision of an allergist,[13] as previously described.[4]

Determining whether a child is allergic to peanut

A questionnaire was administered to parents to determine whether the children fulfilled the criteria for peanut allergy. The questionnaire inquired about the child's frequency of ingestion of the following peanut-containing foods: peanut, peanut butter, Snickers bars, peanut Glossettes, Oh Henry bars, peanut-containing granola bars, ice cream with peanut, Crunchy Nut Corn Flakes cereal, peanut M&M's, or other peanut-containing foods. They were also queried on characteristics of the most severe reaction to peanut, diagnostic testing for peanut allergy, confirmation of peanut allergy by a physician, the presence of atopy (asthma, allergic rhinitis, eczema, hives, anaphylaxis, and other food allergies), and demographic details, including the age, sex, and ethnicity of the child.

When responses were incomplete or unclear, parents were contacted. This questionnaire enabled children to be stratified into 4 mutually exclusive groups: (1) ingests peanut-containing products regularly without problems, (2) never or rarely ingests peanut, (3) has a convincing clinical history of an IgE-mediated reaction to peanut, or (4) has an uncertain clinical history of peanut allergy.

A child was considered to ingest peanut regularly (group 1) if the child tolerated at least 1 peanut-containing food on a monthly basis or at least 2 different peanut-containing foods on at least 2 occasions each. Such a child was considered not allergic to peanut and did not warrant any further investigation. Children who never ingested peanut or ingested peanut less frequently than defined above (but had never experienced a reaction) were assigned to group 2. Children who experienced a reaction after peanut ingestion or contact were stratified into either group 3 or 4 depending on whether their signs/symptoms were compatible with a convincing history of an IgE-mediated reaction to peanut.

A SPT to peanut was required to determine whether children in groups 2, 3, or 4 were sensitized 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 underwent measurement of peanut-specific IgE levels and potentially a DBPCFC. For children in group 3, if the SPT response was positive, the child was assumed to be allergic to peanut. If the SPT response was negative, the child underwent measurement of peanut-specific IgE levels and potentially a DBPCFC. It should be noted that in the first year of our original study, peanut-specific IgE measurement was unavailable at our institution, and therefore children who never or rarely ingested peanut or had an uncertain clinical history of an IgE-mediated reaction to peanut with a positive SPT response were offered a DBPCFC to establish the diagnosis of peanut allergy.

Statistical analysis

A preliminary point estimate and 95% CI for the overall prevalence of peanut allergy was calculated by using standard binomial formulas.[14] This preliminary estimate was based on the observed proportion of children with peanut allergy of the total number who completed the questionnaire and required diagnostic testing (ie, full responders). Given that the numerator and denominator might both be affected by selection bias, selection bias–adjusted estimates of prevalence were derived by using the information provided by parents who completed the questionnaire but whose children withdrew before completion of diagnostic testing (ie, partial responders) and also from those who did not complete the questionnaire (ie, nonresponders) through a Bayesian bias correction technique called multiple imputation.[15,16] This method imputes the patient-specific probability of peanut allergy on the basis of all available information while accounting for the fact that the information is imperfect.

A logistic regression model was fit to data provided by full responders on age, sex, ethnicity, grade, characteristics of the most severe reaction to peanut, results of diagnostic testing for peanut allergy, presence of atopy, and the likelihood that they had previously declared to the school they were allergic to peanut to perform the multiple imputations for partial responders. (Schools were requested to provide nonnominal data on the sex and number of children per participating class who had declared to the school that they are allergic to peanut, and this was used to determine the probability that children had self-declared they were allergic to peanut.) This regression model was then used to predict the probability of peanut allergy for each partial responder. A similar model was created to impute the probability of peanut allergy among nonresponders, but because less information was available in this subset of children, only the covariates of sex, school grade, and likelihood of self-declaration of peanut

TABLE I. Distribution of children in kindergarten through grade 3 in Montreal and distribution of study participants (2000-2002 vs 2005-2007)

	Public schools, 2000-2002	Public schools, 2005-2007	Private schools, 2000-2002	Private schools, 2005-2007
No. of schools	327	332	95	98
No. of schools selected (% selected)	68/327 (20.8)	*	15/95 (15.8)	*
No. of schools participating (% participating)	49/68 (72.1)	49†	14/15 (93.3)	14
Total no. of children attending schools	73,944	67,256	11,559	8,843
No. of children surveyed (% surveyed)	5,997/73,944 (8.1)	6,224/67,256 (9.3)	1,771/11,559 (15.3)	1,815/8,843 (20.5)
No. of respondents (% of children surveyed responding)	3,310/5,997 (55.2)	3,919/6,224 (63.0)	1,029/1,771 (58.1)	1,242/1,815 (68.4)

Information was provided by Ministère de l'Éducation, du Loisir et du Sport, Quebec, Canada.
*Not applicable in 2005-2007 because only schools selected in the 2000-2002 were revisited.
†Eight of the public schools participating in the original study refused to participate in the second study and were replaced by other randomly selected public schools.

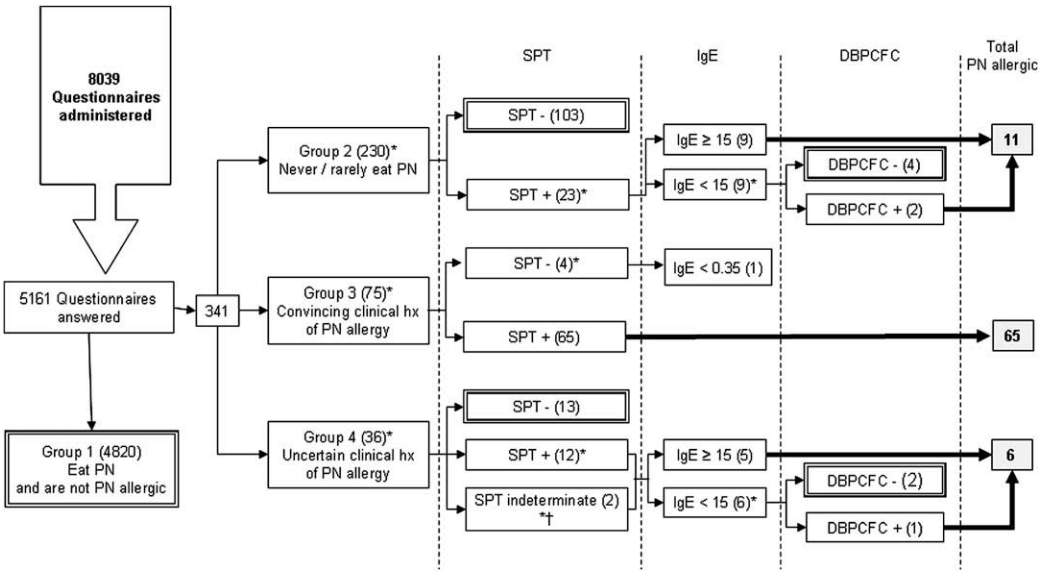

FIG 1. Evaluating the prevalence of peanut allergy. *PN,* Peanut. *The number of children eligible for SPTs, measurement of peanut-specific IgE levels, or DBPCFCs exceeds the number undergoing the tests because of parental refusal; these were classified as partial responders. †An indeterminate SPT response signifies a negative SPT response performed on a child receiving antihistamines.

allergy status were used. Throughout, the prevalence estimates calculated by using multiple imputation were reported with 95% credible intervals (CrIs; the Bayesian analogue to frequentist CIs).

In our original study schools provided more detailed data on self-declared peanut allergy. A sensitivity analysis was performed to account for the possibility that the difference in the level of data obtained influenced prevalence estimates for nonresponders. This sensitivity analysis consisted of the following: (1) re-estimating the prevalence in the original study among full responders, partial responders, and nonresponders by using data of comparable detail to those obtained in the second study and (2) adjusting the prevalence in the follow-up study among the full responders, partial responders, and nonresponders by a factor reflecting the difference between the 2000-2002 estimates by using detailed versus less detailed data on self-declaration.

RESULTS

All 14 private schools participating in the original study were revisited (Table I). Eight of the original 49 public schools refused to participate in the follow-up and were replaced by 8 other randomly selected public schools.

Parents of 5,161 (64.20%) of the 8,039 children surveyed responded (Fig 1). Among the 5,161 children, 4,820 (93.40%) tolerated peanut (group 1). Two hundred and thirty never or rarely ingested peanut (group 2), 75 had a convincing history of peanut allergy (group 3), and 36 had an uncertain history (group 4). Sixty-five of those in group 3 had a positive SPT response and thus were considered allergic to peanut. Of the 230 children in group 2, 23 had a positive SPT response, and 9 of these had a peanut-specific IgE level of greater than 15 kU/L and were considered allergic to peanut. Of the 9 with a peanut-specific IgE level of less than 15 kU/L, 2 had a positive DBPCFC result. Hence 11 children in group 2 were considered allergic to peanut. Of the 36 children in group 4, 12 had a positive SPT response and 2 had an indeterminate SPT response (because they were receiving antihistamines). Of these 14 children, 5 had a peanut-specific IgE level of 15 kU/L or greater and were considered allergic to peanut. Of the 6 children with a peanut-specific IgE level of less than 15 kU/L, 1 had a positive DBPCFC result. Thus 6 children in group 4 were considered allergic. In conclusion, 82

TABLE II. Estimates for prevalence of peanut allergy in 2000-2002 and 2005-2007

	Prevalence, 2005-2007	Prevalence, 2000-2002	Between-study difference, 2000-2002 and 2005-2007
Full responders	1.63% (95% CI, 1.30% to 2.02%)	1.50% (95% CI, 1.16% to 1.92%)	0.13% (95% CI, −0.38% to 0.63%)
Full and partial responders	2.06% (95% CrI, 1.68% to 2.51%)	1.76% (95% CrI, 1.38% to 2.21%)	0.30% (95% CrI, −0.27% to 0.87%)
Full responders, partial responders, and nonresponders	1.83% (95% CrI, 1.48% to 2.23%)	1.34% (95% CrI, 1.08% to 1.64%)	0.49% (95% CrI, 0.03% to 0.94%)
Sensitivity analysis no. 1 for full responders, partial responders, and nonresponders	1.83% (95% CrI, 1.48% to 2.23%)	1.61% (95% CrI, 1.29% to 1.98%)	0.22% (95% CrI, −0.27% to 0.71%)
Sensitivity analysis no. 2 for full responders, partial responders, and nonresponders	1.62% (95% CrI, 1.31% to 1.98%)	1.34% (95% CrI, 1.08% to 1.64%)	0.28% (95% CrI, −0.15% to 0.70%)

TABLE III. Characteristics of study participants

	Allergic to peanut, 2000-2002 (n = 64)	Not allergic to peanut, 2000-2002 (n = 4,190)	Allergic to peanut, 2005-2007 (n = 82)	Not allergic to peanut, 2005-2007 (n = 4,942)
Age (y), mean (SD); 95% CI	7.4 (1.2); 7.1 to 7.6	7.4 (1.2); 7.3 to 7.4	7.1 (1.0); 6.9 to 7.3	7.1 (1.1); 7.0 to 7.1
Male (% [95% CI])	59.4 (46.4 to 71.5)	48.6 (47.1 to 50.2)	63.0 (51.5 to 73.4)	49.2 (47.8 to 50.6)
Atopic features (% [95% CI])				
Asthma	56.3 (43.3 to 68.6)	11.0 (10.1 to 12.0)	53.1 (41.7 to 64.3)	9.6 (8.8 to 10.4)
Eczema	41.3 (29.0 to 54.4)	9.1 (8.2 to 10.0)	43.2 (32.2 to 54.7)	8.6 (7.9 to 9.5)
Other food allergies	53.1 (40.2 to 65.7)	3.3 (2.8 to 3.9)	54.3 (42.9 to 65.4)	2.8 (2.4 to 3.3)
Ethnicity* (% [95% CI])				
White	70.3 (57.6 to 81.1)	69.5 (68.0 to 70.9)	67.9 (56.4 to 78.1)	63.8 (62.4 to 65.2)
Black	6.3 (1.7 to 15.2)	7.3 (6.5 to 8.2)	6.4 (2.1 to 14.3)	6.5 (5.8 to 7.2)
Asian	10.9 (4.5 to 21.2)	6.6 (5.8 to 7.4)	9.0 (3.7 to 17.6)	8.0 (7.2 to 8.8)
Arabic	0 (0.0 to 5.6)†	7.3 (6.5 to 8.1)	2.6 (0.3 to 9.0)	7.4 (6.7 to 8.2)
Hispanic	0 (0 to 5.6)†	2.8 (2.3 to 3.3)	0 (0 to 4.6)†	2.7 (2.3 to 3.2)

*On the island of Montreal, the population is 75% white, 7.1% black, 10.1% Asian, 4.1% Arabic, and 3.1% Hispanic (Canada 2006 Census statistics, data on visible minorities).
†One-sided 97.5% CI.

participants were allergic to peanut (11 in group 2, 65 in group 3, and 6 in group 4).

Among full responders, the prevalence was 1.63% (95% CI, 1.30% to 2.02%; Table II); among full and partial responders, it was 2.06% (95% CrI, 1.68% to 2.51%); and among full responders, partial responders, and nonresponders, it was 1.83% (95% CrI, 1.48% to 2.23%). The differences between the prevalence in 2005-2007 and 2000-2002 were 0.13% (95% CI, −0.38% to 0.63%) among those providing complete data, 0.30% (95% CrI, −0.27% to 0.87%) for full and partial responders, and 0.49% (95% CrI, 0.03% to 0.94%) for full responders, partial responders, and nonresponders. The prevalence of peanut allergy among full responders in public schools was 1.62% (95% CI, 1.24% to 2.07%) and did not differ significantly from the prevalence among full responders in private schools (1.67%; 95% CI, 1.02% to 2.56%).

If the prevalence is re-estimated in the original study among the full responders, partial responders, and nonresponders by using data of comparable detail with those obtained in the second study, it increased from 1.34% to 1.61% (95% CrI, 1.29% to 1.98%), yielding a between-study difference of 0.22% (95% CrI, −0.27% to 0.71%; Table II). Alternatively, if the prevalence is adjusted in the 2005-2007 study among the full responders, partial responders, and nonresponders by a factor reflecting the difference between the 2000-2002 estimates by using detailed versus less detailed data on self-declaration, it decreases from 1.83% to 1.62% (95% CrI, 1.31% to 1.98%), yielding a between-study difference of 0.28% (95% CrI, −0.15% to 0.70%).

Because the positive predictive value of an SPT in those with a convincing history based on 2 mild symptoms might be as low as 61%,[7] we performed a sensitivity analysis assuming that these patients had a 61% probability of being allergic to peanut instead of the 100% probability assumed in our original calculations. These represented only 13.3% of the patients in group 3 in 2005-2007 and 7% of the patients in 2000-2002. Accordingly, the adjusted prevalence estimates in 2005-2007 for full responders, partial responders, and nonresponders decreased from 1.62% to 1.58% (95% CrI, 1.28% to 1.94%) and in 2000-2002 from 1.34% to 1.32% (95% CrI, 1.04% to 1.64%), yielding a between-study difference of 0.26% (95% CrI, −0.17% to 0.70%).

Given that it is reported that 15% of children who had a previous allergic reaction to peanut but who did not experience a reaction for 2 or more years might have outgrown their allergy,[17] it is possible that 2 of the 12 children receiving diagnoses of allergy in our study based on a remote reaction to peanut and a remote positive SPT response might have outgrown their allergy.

Children with peanut allergy were of similar age and ethnicity as those without peanut allergy but were substantially more atopic (Table III).

DISCUSSION

This is the first North American study to document temporal trends in peanut allergy prevalence by corroborating history with confirmatory testing. Despite American and British studies, as

well as anecdotal reports suggesting that the prevalence of peanut allergy is increasing,[2,3] we have shown that the prevalence has remained relatively stable in Montreal schoolchildren between 2000-2002 and 2005-2007. Our findings are consistent with recent reports suggesting that the prevalence of eczema and asthma might have stabilized or decreased in developed countries, which already have a relatively high prevalence of atopic conditions.[18-20] In contrast, the prevalence might be increasing in developing countries, which are undergoing rapid economic growth with subsequent decrease in family size and increase in hygiene.[19,21-23] It is postulated that the decreasing microbial burden influences the immune system by shifting toward a T_H2 cell response, which is responsible for triggering allergic disorders (ie, the hygiene hypothesis).[23,24] Although additional other environmental hypotheses are advanced to explain the anticipated increase in peanut allergy prevalence,[25] these have not been substantiated.[25,26] These hypotheses include increased consumption of vegetable oil deficient in omega-3, which might protect against atopy[27]; decreased consumption of fresh fruits and vegetables rich in antioxidants, which might also protect against atopy[25]; excess or inadequate vitamin D, which have both been shown to promote allergy[28-30]; and low-dose cutaneous sensitization to peanut.[31]

It has also been speculated that early introduction of peanut to infants and young children might promote the development of peanut allergy.[32] Hence guidelines published by the American Academy of Pediatrics in 2000 recommended peanut avoidance during pregnancy and lactation, as well as delayed introduction to the infant diet until the age of 3 years.[33] The parents of children surveyed in our initial prevalence study conducted between 2000 and 2002 would not have had any guidelines to follow because most children were born 5 to 8 years before their publication. However, it is possible that some of the parents of children in our follow-up study might have endorsed these recommendations. We did not collect data on peanut avoidance before age 3 years and hence do not know whether the percentage of parents restricting peanut during pregnancy, lactation, or infancy differed between our 2 studies. Nevertheless, it could be postulated that such a restriction might have potentially reduced the risk of peanut allergy and attenuated an increase in the prevalence of peanut allergy. However, recent studies have demonstrated that delayed introduction of peanut has no effect on peanut allergy prevalence.[26,34-36] Furthermore, recent reports suggest that early introduction of peanut to the infant diet might be associated with a decrease in peanut allergy prevalence.[37] Accordingly, guidelines published by the American Academy of Pediatrics in 2008 do not support maternal dietary restrictions during pregnancy and lactation nor do they support selective food restriction after the age of 4 months.[38]

Previous studies documenting an increase in the prevalence of peanut allergy have limitations. On the Isle of Wight in the United Kingdom, Grundy et al,[3] by evaluating the clinical history of a 1989 and a 1994 birth cohort at age 4 years, claimed that the prevalence of peanut allergy increased from 0.5% (95% CI, 0.1% to 0.8%) to 1.0% (95% CI, 0.5% to 1.7%).[39] However, wide CIs precluded definitive conclusions (odds ratio, 2.08; 95% CI, 0.79 to 5.49). Furthermore, estimates from the first study were based solely on self-report of a convincing history and a positive SPT response; children with a positive SPT response who were never exposed to peanut or with an uncertain history were not considered.

In the United States Sicherer et al[2] demonstrated, through a population-based telephone survey, that the prevalence of self-reported peanut allergy in children 18 years or younger increased from 0.4% (95% CI, 0.2% to 0.7%) in 1997[40] to 0.8% (95% CI, 0.5% to 1.2%) in 2002,[2] with the difference barely achieving statistical significance. Although these investigators replicated the methodology of their original study and adjusted for the false-positive rate of self-report, their study also excluded patients with either no previous exposure or an uncertain history, who might be found to be allergic on appropriate evaluation.

Furthermore, in both of these longitudinal studies, participation rates were lower at follow-up, potentially leading to an overestimate of prevalence in the second cohort. Parents who believe their child is allergic might be more likely to participate and hence might be overrepresented in the second cohort, contributing to an apparent increase in point prevalence. It is also possible that Grundy et al[3] and Sicherer et al[2] report an increasing prevalence because there was more environmental change occurring during the intervals they examined.[18]

Although we have attempted to overcome the limitations of previous research by corroborating self-report with confirmatory testing, replicating the methodology and sampling frames of our initial study, and recruiting an adequate sample, our study has some potential limitations. Because we have reproduced the methodology of our original study as much as possible, our assumptions regarding parental reporting and diagnostic testing should affect both studies similarly and should not influence our conclusions on temporal change in prevalence. However, our studies differed slightly in the data obtained on nonresponders. Although we did not observe an increase in prevalence between 2000-2002 and 2005-2007 when we compared 2 subsets of responders (full responders and full and partial responders), we did observe a small increase when the full responders, partial responders, and nonresponders were compared. It is possible that the less detailed data available on nonresponders in the follow-up study might explain this estimated increase in prevalence. We therefore performed sensitivity analyses to account for the potential effect of this differing level of data and showed that there was no longer a between-study difference in prevalence for the full responders, partial responders, and nonresponders. Our initial and follow-up study also differed slightly in response rates. The lower participation rate in our 2000-2002 study (ie, 56% vs 64%) might have resulted in a slight overestimation of prevalence relative to that in 2005-2007, resulting in an artificial narrowing of our estimate of the between-study difference.

Finally, it should be noted that in the first year of our original study peanut-specific IgE measurement was unavailable at our institution. Hence children in groups 2 and 4 with a positive SPT response were offered a DBPCFC. Considering that this is a much riskier procedure, parents might have been more likely to refuse, leading to a potential underestimation of peanut allergy. Despite this, we did not demonstrate a between-study difference in prevalence.

Despite our finding that the prevalence of peanut allergy has remained stable in Montreal over a 5-year period, peanut allergy is a substantial societal concern. Studies comparing temporal trends in peanut allergy prevalence between countries and over longer intervals are crucial because they might elucidate genetic and environmental processes involved in the pathogenesis of food allergy.

We thank Joanna Priestly, RN; Popi Panaritis; and the school staff, parents, and children whose participation made this study possible.

Clinical implications: Stable peanut allergy prevalence is consistent with eczema and asthma trends. A better understanding of environmental and genetic factors that might influence prevalence is crucial.

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