A population-based study on peanut, tree nut, fish, shellfish, and sesame allergy prevalence in Canada

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Background: Recent studies suggest an increased prevalence of food-induced allergy and an increased incidence of food-related anaphylaxis. However, prevalence estimates of food allergies vary considerably between studies.

Objectives: To determine the prevalence of peanut, tree nut, fish, shellfish, and sesame allergy in Canada.

Methods: Using comparable methodology to Sicherer et al in the United States in 2002, we performed a cross-Canada, random telephone survey. Food allergy was defined as perceived (based on self-report), probable (based on convincing history or self-report of physician diagnosis), or confirmed (based on history and evidence of confirmatory tests).

Results: Of 10,596 households surveyed in 2008 and 2009, 3666 responded (34.6% participation rate), of which 3613 completed the entire interview, representing 9667 individuals. The prevalence of perceived peanut allergy was 1.00% (95% CI, 0.80%-1.20%); tree nut, 1.22% (95% CI, 1.00%-1.44%); fish, 0.51% (95% CI, 0.37%-0.65%); shellfish, 1.60% (95% CI, 1.35%-1.86%); and sesame, 0.10% (95% CI, 0.04%-0.17%). The prevalence of probable allergy was 0.93% (95% CI, 0.74%-1.12%); 1.14% (95% CI, 0.92%-1.35%); 0.48% (95% CI, 0.34%-0.61%); 1.42% (95% CI, 1.18%-1.66%); and 0.09% (95% CI, 0.03%-0.15%), respectively. Because of the infrequency of confirmatory tests and the difficulty in obtaining results if performed, the prevalence of confirmed allergy was much lower.

Conclusion: This is the first nationwide Canadian study to determine the prevalence of severe food allergies. Our results indicate disparities between perceived and confirmed food allergy that might contribute to the wide range of published prevalence estimates. (J Allergy Clin Immunol 2010;125:1327-35.)

Key words: *Food allergy, peanut allergy, tree nut allergy, fish allergy, shellfish allergy, sesame allergy, perceived food allergy, probable food allergy, confirmed food allergy*

Food allergy affects up to 2.5% of the adult population and 6% to 8% of children less than 3 years of age and is associated with significant morbidity and mortality.[1,2] The incidence rate of anaphylaxis is increasing, and recent US reports suggest that it may be as high as 49.8 per 100,000 person-years.[3-8] Foods are primary inciting allergens for anaphylaxis,[8-12] and hospitalizations because of food-induced anaphylaxis are reported to have increased by 350% during the last decade.[11,13]

Peanut and tree nut account for the majority of severe reactions,[10,11,14] but fish, shellfish, and sesame are also reported to cause severe reactions, especially in Asia and parts of Europe.[12,15-20] However, there is considerable heterogeneity in the prevalence estimates of these severe food allergies, possibly because of differences in study design, methodology, or study populations. The prevalence estimates of food allergies range between 0% and 2% for peanut,[21-23] 0% and 7.3% for tree nut,[23-26] 0% and 2% for fish,[21,27,28] 0% and 10% for shellfish,[21,26,27,29,30] and 0% and 0.79% for sesame.[19,24,31,32] There have been a few population-based studies estimating the prevalence of peanut, tree nut, fish, and shellfish allergies in the United States,[23,27] but no such studies have been conducted in Canada. Recently, our research team reported that the prevalence of peanut allergy in Montreal school children had stabilized between 2002 and 2007, although it exceeded (1.63%; 95% CI, 1.30%-2.02%)] estimates from most other countries except the United Kingdom (UK).[22]

The Surveying Canadians to Assess the Prevalence of Common Food Allergies and Attitudes towards Food LAbelling and Risk (SCAAALAR) study, launched in 2008, was designed to estimate the prevalence of food allergies responsible for the majority of severe/fatal anaphylactic reactions (peanut, tree nut, fish, shellfish, and sesame) in Canada.

METHODS

Selection of study population

Households were chosen by purchasing, from Info-Direct, a random selection of telephone numbers and their accompanying addresses from the electronic white pages. (Info-Direct maintains an electronic listing of all Canadian household telephone numbers listed in the white pages and updates these records monthly). Households were limited to the 10 Canadian provinces; the territories were excluded because it was thought that there would be considerable cultural difference between individuals living in these regions and the rest of Canada. Interviews were conducted from May 2008 to March 2009.

Survey methodology

The telephone surveys were conducted by teams of similarly trained interviewers based at either McGill (Montreal, Quebec) or McMaster (Hamilton,

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Abbreviations used
IQR: Interquartile range
SPT: Skin prick test
UK: United Kingdom

Ontario) Universities, using Computer Assisted Telephone Interview software (WinCati 4.2; Sawtooth Technologies Inc, Northbrook, Ill). Respondents were eligible to participate if they were 18 years or older, were living in the household, and appeared to have no language-mental-hearing barriers. The initial age-eligible household respondent was invited to participate and asked whether any household member had an allergy to peanut, tree nut, shellfish, fish, or sesame. If any household member reported an allergy, the self-reported allergy was validated by querying the potentially allergic individual (or an appropriate surrogate if the allergic individual was not eligible or was unavailable at the time of the interview) on symptoms related to ingestion of the food and diagnosis and management of the allergy. If no food allergy was reported in the household, demographic data were obtained. In addition, data on attitudes toward food labeling for allergens and the societal risk associated with food allergy were also collected (results of the surveys on food labeling and risk perception will be described in subsequent articles).

To optimize response rates and minimize bias, a maximum of 10 attempts was made to contact households during different days and times between the hours of 9:30 AM and 9:00 PM (local time) Monday through Friday and 10:30 AM and 5:00 PM (local time) on Saturdays and Sundays. In addition, households were advised that we were conducting a survey on food allergies a few weeks in advance by a mailed information letter.[33]

The study was approved by the Institutional Review Boards of the McGill University Health Centre and McMaster University.

Questionnaire

We used a standardized questionnaire developed previously by Sicherer et al[23,27] to determine the general population prevalence of peanut, tree nut, fish, and shellfish allergy[23,27] in the United States, and modified it to incorporate questions regarding sesame allergy. In addition, in cases in which respondents reported that the allergy was diagnosed by a physician, we requested permission to obtain confirmatory information from the physician. To increase response rate among physicians, up to 3 letters were sent requesting medical information regarding the use of confirmatory tests to diagnose the food allergy.

The participant questionnaire included questions on specific types of tree nut (eg, hazelnut, pecan, and pistachio), fish (eg, tuna, cod, and salmon), and shellfish including crustaceans (eg, shrimp and lobster) and mollusks (eg, clams and squid). Individuals were queried on the history of the most severe allergic reaction (ie, whether they experienced typical IgE-mediated symptoms such as pruritus, urticaria, flushing, rhinoconjunctivitis, angioedema, throat tightness, gastrointestinal complaints, breathing difficulties, wheeze, cyanosis, or circulatory collapse), interval between exposure and symptom onset, whether medical care was sought, whether epinephrine was administered, whether diagnosed by a physician, and whether confirmatory tests (ie, skin prick tests [SPTs], measurement of serum allergen-specific IgE, and/or food challenge) were performed. Demographic data were collected including number, age, and sex of household members; education level of the household respondent; whether the household respondent was born in Canada, and country of origin of respondent if not born in Canada, and number of years living in Canada; and household income level. The questionnaire was translated into French and back-translated to English.

Definitions of food allergy

We developed 3 definitions of food allergy.

1. Perceived food allergy. This includes all cases of self-reported food allergy, regardless of history or presence of supporting confirmatory tests.
2. Probable food allergy. This refers to those self-reporting food allergy who have a convincing history of food allergy or who report a physician confirmed food allergy. A convincing clinical history of an IgE-mediated reaction to a specific food 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 120 minutes after ingestion or contact (or inhalation in the case of fish and shellfish). 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.[22,34-36]
3. Confirmed food allergy. Participants were considered to have a confirmed allergy only if one of the following was fulfilled:
   a. They had a convincing clinical history of an IgE-mediated reaction attributed to food and their physician provided confirmation of a positive SPT defined as a wheal diameter at least 3 mm larger than that elicited by the negative control within 10 to 15 minutes of placement[37] OR a serum food-specific IgE ≥0.35 kU/L OR a positive food challenge.
   b. They were never exposed to the food or had an uncertain clinical history (ie, any history other than convincing) of an IgE-mediated reaction and their physician provided confirmation of a positive SPT AND a food-specific IgE above previously published thresholds (ie, ≥15 kU/L for peanut and tree nut and ≥20 kU/L for fish[38]) OR a positive SPT AND a positive food challenge OR a positive food challenge alone. It should be noted, however, that for peanut allergy, a SPT ≥8 mm in those ≥2 years and a SPT ≥4 mm in those <2 years were considered sufficient diagnostic criteria in those never exposed or with an uncertain history. It has been reported that these thresholds are highly predictive of peanut allergy.[39,40] It should be noted that although these thresholds are widely used among allergists in different countries including Canada,[41] they are not universally accepted,[42] and there are physicians who would use a higher threshold of 13 mm.[43]

Statistical analysis

Preliminary point estimates and 95% CIs for the overall prevalence of perceived and probable food allergy were calculated, accounting for the fact that households were the primary sampling units in this survey data, rather than individuals.[44]

Given that sufficient confirmatory test data were not available for all participants, a third estimate was computed, based on the data provided, as a tentative lower bound for the prevalence of confirmed food allergy in all participants.[45,46] However, with no results of food challenges having been obtained, a proportion of true negatives among self-reported cases could not be established. Hence, the lower end of a 1-sided binomial 97.5% CI for the proportion of confirmed cases was first calculated, with a value that decreases as the number of confirmed observations gets smaller. As an example, if for a given allergy, 15 of 15 cases providing test results were confirmed, the lower end of the interval would be 78%, whereas it would only be 48% if only 5 of 5 cases were confirmed. This percentage was then multiplied by the proportion of all responders who reported a comparable history to that of confirmed cases. Pursuing the same example, if 15 cases were confirmed among patients with a convincing history, and 5 among those with an uncertain history, the prevalence estimate for confirmed allergy would be the sum of 78% of the proportion of convincing histories plus 48% of that of uncertain histories. Relevant 95% CIs were also adjusted to account for the multilevel aspect of this data.

RESULTS

Participation rate

Of 10,596 households contacted, 3666 responded (34.6% participation rate), of which 3613 completed the entire interview, representing 9667 individuals.

TABLE I. Demographic characteristics

	SCAAALAR population	Canadian population
College/university/professional degree or diploma	60.5%	32.9% (as of 2001)
High school diploma	90.7%	68.7% (as of 2001)
Born in Canada	85.6%	80.6% (as of 2006)
Immigrated to Canada in the last 10 years	1.9%	6.3% (as of 2006)
Married/cohabitation	70.3%	72.5% (as of 2006)
Dwelling owned	82.1%	68.0% (as of 2006)
Median annual household income	$70,000	$63,600 (as of 2006)
Household income under low-income cutoff*†	8.9%	14.5% (as of 2006)
Rural (based on postal code) location	15.5%	13.7% (as of 2001)
Rural‡	39.0%	32.4% (as of 2007)
Residing in Atlantic Canada	5.4%	6.9% (as of 2006)
Quebec	39.5%	23.4%
Ontario	32.6%	38.9%
Prairies	12.2%	17.5%
British Columbia	10.3%	13.2%

SCAAALAR, Surveying Canadians to Assess the Prevalence of Common Food Allergies and Attitudes towards Food LAbelling and Risk.
*Among respondents who provided income-related information, representing 61% of our household sample.
†Low income cutoffs, defined as income levels at which families or unattached individuals spend at least 70% of before tax income on food, shelter, and clothing and is determined according to family size and geographic location.
‡Residing outside Canadian metropolitan areas or in Canadian metropolitan areas with a population ≤100,000.

Compared with the general Canadian population, immigrants within the last 10 years as well as those with lower household income are underrepresented in our study population (Table I).

Prevalence estimates

The prevalence of perceived peanut allergy was 1.00% (95% CI, 0.80%-1.20%); tree nut, 1.22% (95% CI, 1.00%-1.44%); fish, 0.51% (95% CI, 0.37%-0.65%); shellfish, 1.60% (95% CI, 1.35%-1.86%); and sesame, 0.10% (95% CI, 0.04%-0.17%; Fig 1-5; Table II).

The prevalence of probable peanut allergy was 0.93% (95% CI, 0.74%-1.12%); tree nut, 1.14% (95% CI, 0.92%-1.35%); fish, 0.48% (95% CI, 0.34%-0.61%); shellfish, 1.42% (95% CI, 1.18%-1.66%); and sesame, 0.09% (95% CI, 0.03%-0.15%; Fig 1-5; Table II).

Although most participants self-reporting food allergy had testing performed (Table III), only 56.7%, 55.9%, 51.0%, 34.2%, and 70.0% of those self-reporting peanut, tree nut, fish, shellfish, and sesame allergy allowed us to contact their physician to obtain confirmatory test results. In over 50% of cases, these physicians failed to provide results, and in only 21.6%, 10.2%, 6.1%, 4.5%, and 40.0% of those self-reporting food allergy were these results sufficient to establish the diagnosis (Table III). None of the patients reported a food challenge. Confirmatory tests for peanut, tree nut, and shellfish were performed less often in adults (Table III). Based on the results obtained, the prevalence of confirmed peanut allergy was 0.61% (95% CI, 0.47%-0.74%), and the prevalence of confirmed tree nut, fish, shellfish and sesame allergy was 0.68% (95% CI, 0.54%-0.83%), 0.10% (95% CI, 0.07%-0.14%), 0.73% (95% CI, 0.59%-0.86%), and 0.03% (95% CI, 0.01%-0.06%), respectively (Fig 1-5; Table II).

Characteristics of reactions

Initial allergic reactions in children with probable peanut, tree nut, and sesame allergy occurred at a median age of 2 years (interquartile range [IQR], 1-4), 7 years (IQR, 2-12), and 2 years (IQR, 1-4), respectively (Table IV). Initial reactions in participants 18 years and older with probable peanut, tree nut, and sesame allergy occurred at a median age of 11 years (IQR, 2-30), 20 years (IQR, 10-40) and 10 years (IQR, 2-15), respectively. Initial reactions to fish and shellfish occurred in children at a median age of 4 years (IQR, 2.5-5) and 6.5 years (IQR, 4-9) and in adults, at a median age of 12 years (IQR, 5-25) and 25 years (IQR, 17-37; Table IV).

Recurrent reactions were common and occurred in 73.7%, 77.4%, 88.9%, 74.6%, and 87.5% of those with peanut, tree nut, fish, shellfish, and sesame allergy, respectively (Table IV). Among those with moderate or severe reactions (defined above),[22,34-36] to peanut, tree nut, fish, shellfish, and sesame, only 36.1%, 38.7%, 21.1%, 14.6%, and 37.5% reported receiving epinephrine treatment, respectively (Table IV).

The most prevalent tree nut, fish, and shellfish associated with allergic reactions were reported to be hazelnut, cod/salmon, and shrimp, respectively. These were also the most common foods associated with moderate/severe reactions.

DISCUSSION

We have conducted the first nationwide study on food allergy prevalence that attempts to confirm participant self-report of allergy by obtaining physician records of diagnostic testing. However, retrieving such information proved to be challenging because all participants did not undergo such testing, and of those who did, many participants or physicians refused to provide results. Hence, our prevalence estimates of confirmed allergy are very conservative, and we have therefore also provided estimates for perceived and probable allergy, which likely better approximate true prevalence. The difference between perceived and confirmed estimates certainly contributes to the wide range of published values for food allergy prevalence.[21]

Although we tried to increase the participation rate through the use of an introductory letter and by calling on different days and different times of the day, it is still relatively low. This is consistent with recently reported trends of low participation rates in telephone surveys, especially among persons with lower

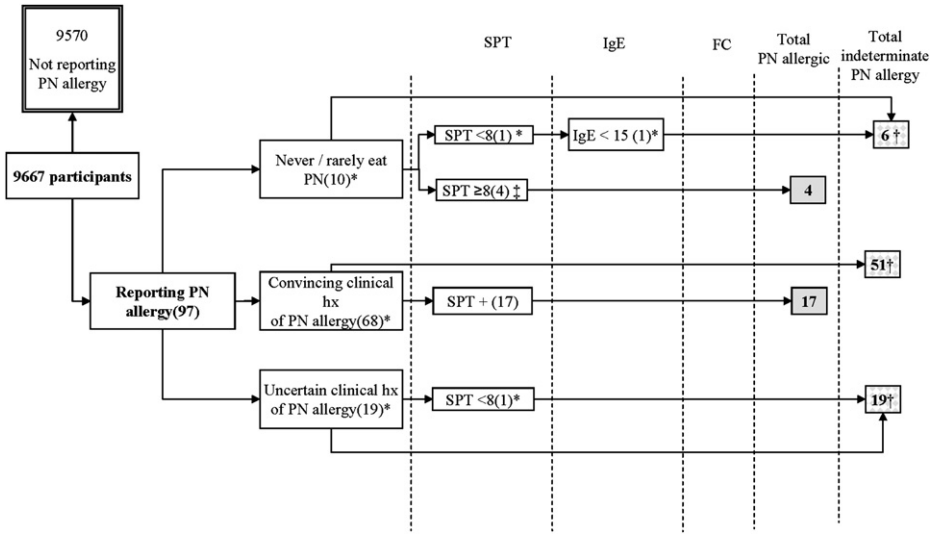

FIG 1. Algorithm for the diagnosis of confirmed peanut allergy. *The number of participants eligible for SPTs, measurement of PN-specific IgE levels, or FCs exceeds the number of available test results because participants did not have the tests done, participants refused to release medical information from the treating physician, or physicians did not provide test results. †Data provided not sufficient to establish the diagnosis of allergy. ‡For those below 2 years, the cutoff is 4 instead of 8 mm.

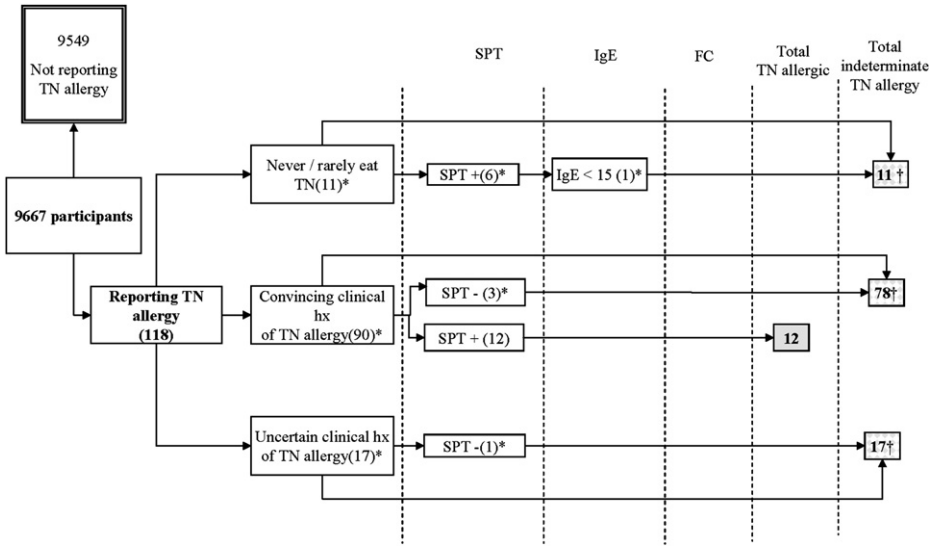

FIG 2. Algorithm for the diagnosis of confirmed tree nut allergy. *The number of participants eligible for SPTs, measurement of TN-specific IgE levels, or FCs exceeds the number of available test results because participants did not have the tests done, participants refused to release medical information from the treating physician, or physicians did not provide test results. †Data provided not sufficient to establish the diagnosis of allergy.

education.[47,48] This low participation rate is also in line with the most recent food allergy telephone survey conducted by Sicherer et al[49] in 2008 (42% participation rate). In addition, although digital telephone surveys using white pages sampling (through Infodirect) are suitable to collect information for prevalence on most common self-reported health conditions in the population including minorities,[50-52] they may result in selection bias because of exclusion of unlisted numbers,[50] persons who are primary or exclusive cell-phone users, ethnic minorities, immigrants,[53] and lower socioeconomic groups.[54] Accordingly, these latter 2 groups are relatively underrepresented in our study. Further, our low response rate may have led to a higher participation rate among those with food allergies. However, we believe that our estimates for the prevalence of perceived and probable food allergy are valid given that these estimates for peanut allergy in Canadian and Quebec children (Canada, perceived 1.77% and probable 1.68%; Quebec, 1.69% and 1.69%, respectively) are consistent with our estimates for confirmed peanut allergy in Montreal school children (1.63%), in whom the participation rate was 64.2%.[22]

Our results demonstrate that there is substantial misconception on behalf of both health care providers and patients regarding the diagnosis and management of food allergy. In our study,

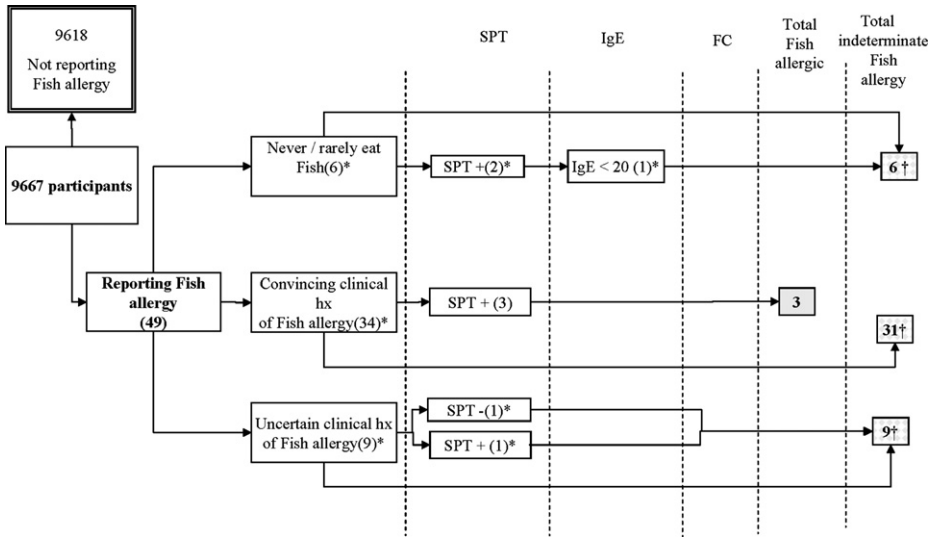

FIG 3. Algorithm for the diagnosis of confirmed fish allergy. *The number of participants eligible for SPTs, measurement of fish-specific IgE levels, or FCs exceeds the number of available test results because participants did not have the tests done, participants refused to release medical information from the treating physician, or physicians did not provide test results. †Data provided not sufficient to establish the diagnosis of allergy.

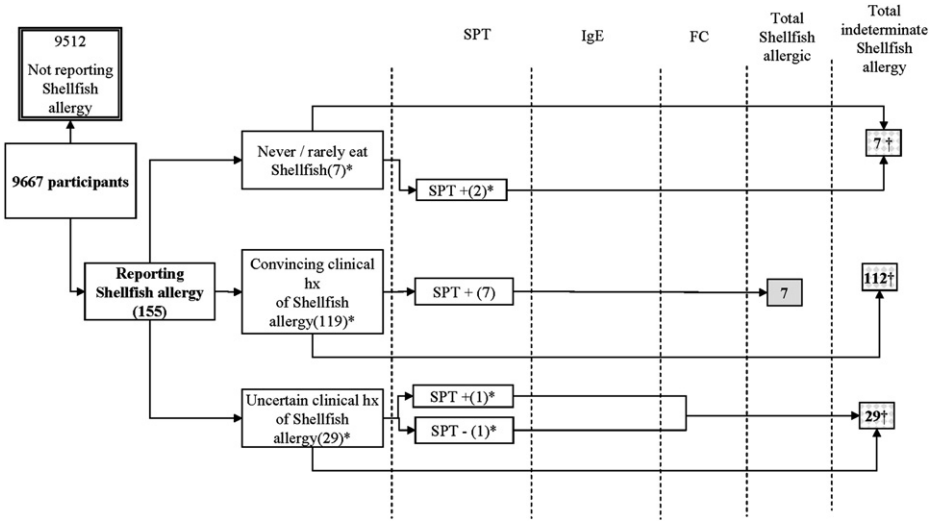

FIG 4. Algorithm for the diagnosis of confirmed shellfish allergy. *The number of participants eligible for SPTs, measurement of shellfish-specific IgE levels, or FCs exceeds the number of available test results because participants did not have the tests done, participants refused to release medical information from the treating physician, or physicians did not provide test results. †Data provided not sufficient to establish the diagnosis of allergy.

physicians underused the confirmatory tests required to establish or refute the diagnosis of food allergy, supporting our recent observation on the underuse of confirmatory tests in children never exposed to peanut or with an uncertain history.[41] Underuse of confirmatory tests was most frequent in adults reporting shellfish allergy and cannot be entirely attributed to recall bias, given that shellfish allergy usually develops in adulthood.[28] Inadequate use of confirmatory tests can have substantial consequences, with some mislabelled allergic and burdened with a lifetime of unnecessary dietary vigilance, whereas others may be falsely reassured that they are not at risk for fatal anaphylaxis. Furthermore, most of our participants with food allergy had experienced at least 1 repeat reaction, and few reactions were managed appropriately with epinephrine.

It is possible that some participants deemed to have a convincing history for tree nut or fish allergy did not actually experience an IgE-mediated reaction with the potential to develop into anaphylaxis. Tree nut allergy was the most prevalent food allergy reported in our study, and our estimates exceed most others.[27,55] It is possible that the 4.5% of participants with probable tree nut allergy who reported symptoms limited to itching/swelling of the mouth immediately after oral contact with a specific nut have a pollen-food allergy syndrome[56-60] and are less likely to experience severe anaphylactic reactions. It is also

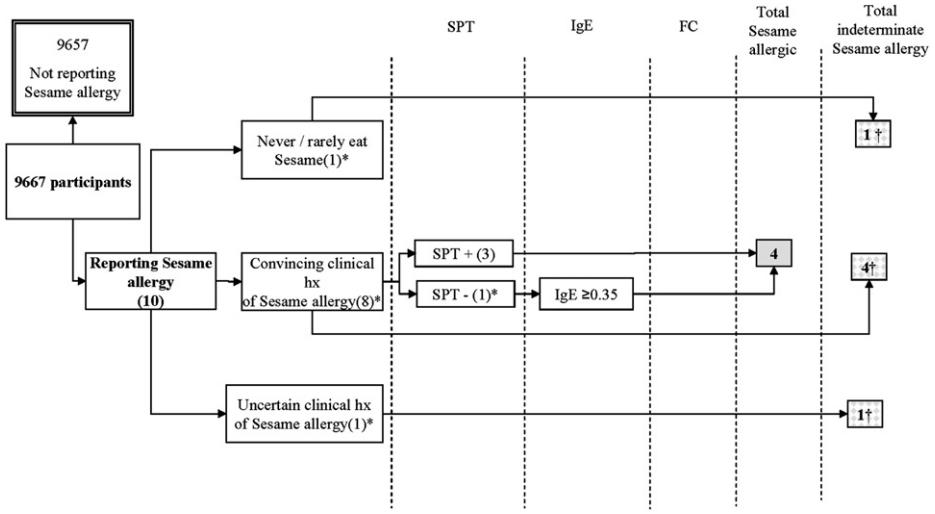

FIG 5. Algorithm for the diagnosis of confirmed sesame allergy. *The number of participants eligible for SPTs, measurement of sesame-specific IgE levels, or FCs exceeds the number of available test results because participants did not have the tests done, participants refused to release medical information from the treating physician, or physicians did not provide test results. †Data provided not sufficient to establish the diagnosis of allergy. *FC,* Food challenge; *Hx,* history; *PN,* peanut; *TN,* tree nut.

TABLE II. Prevalence estimates for perceived, probable, and confirmed food allergy

Participants	Peanut	Tree nut	Fish	Shellfish	Sesame
Children (%) (95% CI)					
Perceived	1.77 (1.21-2.33)	1.73 (1.16-2.30)	0.18 (0.00-0.36)	0.55 (0.21-0.88)	0.23 (0.03-0.43)
Probable	1.68 (1.14-2.23)	1.59 (1.04-2.14)	0.18 (0.00-0.36)	0.50 (0.18-0.82)	0.23 (0.03-0.43)
Confirmed	1.03 (0.67-1.39)	0.69 (0.40-0.97)	0	0.06 (0.01-0.10)	0.03 (0.00-0.06)
Adults (%) (95% CI)					
Perceived	0.78 (0.58-0.97)	1.07 (0.84-1.30)	0.60 (0.43-0.78)	1.91 (1.60-2.23)	0.07 (0.01-0.13)
Probable	0.71 (0.52-0.90)	1.00 (0.78-1.23)	0.56 (0.39-0.73)	1.69 (1.39-1.98)	0.05 (0.00-0.11)
Confirmed	0.26 (0.18-0.34)	0.35 (0.27-0.44)	0.12 (0.08-0.16)	0.71 (0.58-0.84)	0.01 (0.00-0.02)
Entire study population (%) (95% CI)					
Perceived	1.00 (0.80-1.20)	1.22 (1.00-1.44)	0.51 (0.37-0.65)	1.60 (1.35-1.86)	0.10 (0.04-0.17)
Probable	0.93 (0.74-1.12)	1.14 (0.92-1.35)	0.48 (0.34-0.61)	1.42 (1.18-1.66)	0.09 (0.03-0.15)
Confirmed	0.61 (0.47-0.74)	0.68 (0.54-0.83)	0.10 (0.07-0.14)	0.73 (0.59-0.86)	0.03 (0.01-0.06)

possible that patients reporting fish allergy may have had scombroid fish poisoning because of bacterial contamination of fish and production of histamine[61] or an IgE-mediated reaction to *Anisakis simplex* associated with consumption of raw fish.[62,63] However, given that all participants reporting fish allergy had either multiple reactions or a positive SPT to fish, the diagnosis of scombroid fish poisoning or *Anisakis* allergy is unlikely.

It is possible that a small percentage of children who did not experience a recent reaction had actually developed tolerance. Although we had data only on the date of the most severe reaction and not the most recent, if we assume that the most severe reaction is actually the most recent, 15% of children not having a reaction to peanut in the past 2 years,[64] 9% not experiencing a reaction to tree nut in the past year,[65] 17.2% not experiencing a reaction to fish in the past 2 years,[66] and 20% not experiencing a reaction to sesame in the past 2.3 years[67,68] might have outgrown their allergy. Thus, our probable prevalence estimates in participants with peanut, tree nut, fish, and sesame allergy would decrease to 0.88% (95% CI, 0.71%-1.09%), 1.11% (95% CI, 0.91%-1.34%), 0.47% (95% CI, 0.34%-0.63%) and 0.09% (95% CI, 0.04%-0.17%), respectively. This clearly represents a lower bound because some of the participants might have experienced a more recent but less severe reaction. Given that there are no reports on the rate of resolution of shellfish allergy, we were unable to conduct a similar sensitivity analysis.

Our estimates of the median age of the initial reaction to peanut, tree nut, and sesame in children are similar to published estimates,[68-70] but for adults, the median age exceeds that reported in most other studies. This is likely a result of recall bias—that is, adults have difficulty recalling the date of a personal remote reaction and likely report the date of a more recent one, whereas parents usually recall the date of their child's initial reaction.[71,72] The median age of the initial reaction to fish and shellfish in both children and adults is comparable to other reports, possibly because the onset of these allergies is usually at an older age.[27,73] In addition, the age of the initial introduction of a food (for which we did not collect data) may have influenced the age of the initial reaction.[31]

►

TABLE III. Number and percentage of participants with reported and sufficient confirmatory tests*

Participants	Peanut	Tree nut	Fish	Shellfish	Sesame
Children, N (%*)					
Self-report of tests†	35 (89.7)	33 (86.8)	3 (75.0)	11 (91.7)	4 (80.0)
Consent to contact MD	30 (76.9)	30 (78.9)	2 (50.0)	9 (75.0)	3 (60.0)
Results provided by MD	16 (41.0)	16 (42.1)	1 (25.0)	5 (41.7)	2 (40.0)
Results sufficient to confirm allergy	16 (41.0)	8 (21.1)	0 (0.0)	2 (16.7)	2 (40.0)
Adults, N (%*)					
Self-report of tests†	42 (72.4)	56 (70.0)	34 (75.6)	69 (48.3)	5 (100.0)
Consent to contact MD	25 (43.1)	36 (45.0)	23 (51.1)	44 (30.8)	4 (80.0)
Results provided by MD	8 (13.8)	9 (11.3)	6 (13.3)	8 (5.6)	2 (40.0)
Results sufficient to confirm allergy	5 (8.6)	4 (5.0)	3 (6.7)	5 (3.5)	2 (40.0)
Entire study population N (%*)					
Self-report of tests†	77 (79.4)	89 (75.4)	37 (75.5)	80 (51.6)	9 (90.0)
Consent to contact MD	55 (56.7)	66 (55.9)	25 (51.0)	53 (34.2)	7 (70.0)
Results provided by MD	24 (24.7)	25 (21.2)	7 (14.3)	13 (8.4)	4 (40.0)
Results sufficient to confirm allergy	21 (21.6)	12 (10.2)	3 (6.1)	7 (4.5)	4 (40.0)
Difference in reported tests percentages in children versus adults (%)	17.3 (2.4, 32.3)	16.8 (2.1, 31.6)	−0.6 (−44.8, 43.7)	43.4 (25.8, 61.1)	−20 (−55.1, 15.1)

MD, Medical doctor.
*Among those reporting food allergy.
†Including those who did not know whether tests were done.

TABLE IV. Characteristics of reactions

Participants	Peanuts	Tree nut	Fish	Shellfish	Sesame
Children					
Initial reaction median age (y) (IQR)*	2 (1-4)	7 (2-12)	4 (2.5-5)	6.5 (4-9)	2 (1-4)
Participants with probable allergy reporting at least 1 allergic reaction (N)	30	26	4	10	5
% With recurrent reactions*	56.7	58.3	50.0	44.4	80.0
% With moderate/severe reaction*	90.0	88.5	100.0	90.0	100.0
% Treated with epinephrine†	29.6	34.8	25.0	33.3	20.0
Adults					
Initial reaction median age (y) (IQR)*	11 (2-30)	20 (10-40)	12 (5-25)	25 (17-37)	10 (2-15)
Participants with probable allergy reporting at least 1 allergic reaction (N)	49	73	37	122	4
% With recurrent reactions*	84.8	84.1	93.8	77.0	100.0
% With moderate/severe reaction*	91.8	95.9	91.9	93.4	75.0
% Treated with epinephrine†	40.0	40.0	20.6	13.2	66.7
Entire study population					
Initial reaction median age (y) (IQR)*	4 (2-16)	15.5 (6-30)	8 (5-25)	25 (14-35)	3 (1.5-12.5)
Participants with probable allergy reporting at least 1 allergic reaction (N)	79	99	41	132	9
% With recurrent reactions*	73.7	77.4	88.9	74.6	87.5
% With moderate/severe reaction*	91.1	93.9	92.7	93.2	88.9
% Treated with epinephrine†	36.1	38.7	21.1	14.6	37.5

*Among participants with probable food allergy reporting at least 1 allergic reaction.
†Among participants with moderate/severe reactions as defined in the text.

Our definitions for food allergy differed slightly from those used previously by Sicherer et al[23,27] in the United States. However, to compare our results to US estimates, we have used comparable definitions. Our 2009 nationwide estimates for the perceived prevalence of peanut allergy exceeded those published by Sicherer et al[74] in 2002 by 0.27% (95% CI, 0.02%-0.52%) for all participants and by 0.88% (95% CI, 0.24%-1.52%) for children. Canadian estimates for the perceived prevalence of tree nut allergy were higher by 0.44% (95% CI, 0.18%-0.71%) for all participants and by 1.15% (95% CI, 0.55%-1.76%) for children. Canadian estimates for the prevalence of peanut and tree nut combined, based on a convincing history, exceeded US estimates by 0.31% (95% CI, 0.02%-0.60%). In contrast, our 2009 estimates for the probable prevalence of shellfish allergy were lower than US 2002 estimates by 0.69% (95% CI, 0.37%-1.01%) for all and by 0.96% (95% CI, 0.56%-1.37%) for adults. The difference between Canadian and US estimates for fish allergy was not significant (0.07%; 95% CI, –0.10% to 0.23%).[27]

The observed difference in prevalence estimates between Canada and the United States might be a result of several factors. Our study was conducted 7 years later than Sicherer's,[74] and therefore, temporal trends may contribute to an increase in true prevalence as well as enhanced awareness and an attendant increase in perceived prevalence. Several studies suggest an increase in the prevalence of peanut allergy during the last decade[23,74,75] that has recently stabilized.[22,76] The difference may also be a result of inherent differences in the 2 countries. Despite assumed similarities in Canadian and US dietary habits,

studies report differences in lifestyles, food availability, and nutrition fortification between the countries that might affect the emergence of food allergies.[77-80] Finally, some of the observed differences may be attributed to the lower response rate in our study (ie, 35% vs 67.3% in the US seafood study and 52% in the US peanut and tree nut study), which might have led to overrepresentation of those with food allergies.

Given that there are no US estimates for sesame allergy, we were able to compare our estimates only to previously published UK and Israeli estimates.[31] The prevalence of sesame allergy in Canada and Israel is similar and much lower than in the UK. This contrasts sharply with the prevalence of peanut allergy, which is similar in Canada and the UK (1.85%; 95% CI, 1.45%-2.32%) and much higher than in Israel (0.17%; 95% CI, 0.07%-0.34%).[31]

In conclusion, our results reveal significant disparities between perceived and confirmed food allergies. Guidelines regarding increased use of confirmatory tests in general and food challenges in particular should be disseminated and might contribute to a more accurate diagnosis in those never exposed or with an uncertain history. Research should be expanded to include vulnerable populations such as those of lower socioeconomic status and immigrants, and the role of environmental factors in the pathogenesis of food allergies should be explored.

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Clinical implications: Guidelines regarding increased use of confirmatory tests in general and food challenges in particular should be disseminated and might contribute to a more accurate diagnosis in those reporting food allergies.

REFERENCES

1. Sampson HA. Food allergy, part 2: diagnosis and management. J Allergy Clin Immunol 1999;103:981-9.
2. Bock SA. Prospective appraisal of complaints of adverse reactions to foods in children during the first 3 years of life. Pediatrics 1987;79:683-8.
3. Yocum MW, Khan DA. Assessment of patients who have experienced anaphylaxis: a 3 year survey. Mayo Clin Proc 1994;69:16-23.
4. Yocum MW, Butterfield JH, Klein JS, Volcheck GW, Schroeder DR, Silverstein MD. Epidemiology of anaphylaxis in Olmsted County: a population-based study. J Allergy Clin Immunol 1999;104:452-6.
5. Sheikh A, Alves B. Hospital admissions for acute anaphylaxis: time trend study. BMJ 2000;320:1441.
6. Lin RY, Anderson AS, Shah SN, Nurruzzaman F. Increasing anaphylaxis hospitalizations in the first 2 decades of life: New York State, 1990 -2006. Ann Allergy Asthma Immunol 2008;101:387-93.
7. Calvani M, Di LD, Polo A, Spinelli A, Zappala D, Zicari M. Hospitalizations for pediatric anaphylaxis. Int J Immunopathol Pharmacol 2008;21:977-83.
8. Decker WW, Campbell RL, Manivannan V, Luke A, St Sauver JL, Weaver A, et al. The etiology and incidence of anaphylaxis in Rochester, Minnesota: a report from the Rochester Epidemiology Project. J Allergy Clin Immunol 2008;122:1161-5.
9. Simon MR, Mulla ZD. A population-based epidemiologic analysis of deaths from anaphylaxis in Florida. Allergy 2008;63:1077-83.
10. Pumphrey R. Anaphylaxis: can we tell who is at risk of a fatal reaction? Curr Opin Allergy Clin Immunol 2004;4:285-90.
11. Liew WK, Williamson E, Tang ML. Anaphylaxis fatalities and admissions in Australia. J Allergy Clin Immunol 2009;123:434-42.
12. Piromrat K, Chinratanapisit S, Trathong S. Anaphylaxis in an emergency department: a 2-year study in a tertiary-care hospital. Asian Pac J Allergy Immunol 2008;26:121-8.
13. Branum AM, Lukacs SL. Food allergy among children in the United States. Pediatrics 2009;124:1549-55.
14. Sampson HA, Mendelson L, Rosen JP. Fatal and near-fatal anaphylactic reactions to food in children and adolescents. N Engl J Med 1992;327:380-4.
15. Crespo JF, Pascual C, Burks AW, Helm RM, Esteban MM. Frequency of food allergy in a pediatric population from Spain. Pediatr Allergy Immunol 1995;6:39-43.
16. Pascual CY, Reche M, Fiandor A, Valbuena T, Cuevas T, Esteban MM. Fish allergy in childhood. Pediatr Allergy Immunol 2008;19:573-9.
17. Leung TF, Yung E, Wong YS, Lam CW, Wong GW. Parent-reported adverse food reactions in Hong Kong Chinese pre-schoolers: epidemiology, clinical spectrum and risk factors. Pediatr Allergy Immunol 2009;20:339-46.
18. Gangur V, Kelly C, Navuluri L. Sesame allergy: a growing food allergy of global proportions? Ann Allergy Asthma Immunol 2005;95:4-11.
19. Dalal I, Binson I, Reifen R, Amitai Z, Shohat T, Rahmani S, et al. Food allergy is a matter of geography after all: sesame as a major cause of severe IgE-mediated food allergic reactions among infants and young children in Israel. Allergy 2002;57:362-5.
20. Derby CJ, Gowland MH, Hourihane JO. Sesame allergy in Britain: a questionnaire survey of members of the Anaphylaxis Campaign. Pediatr Allergy Immunol 2005; 16:171-5.
21. Rona RJ, Keil T, Summers C, Gislason D, Zuidmeer L, Sodergren E, et al. The prevalence of food allergy: a meta-analysis. J Allergy Clin Immunol 2007;120:638-46.
22. Ben-Shoshan M, Kagan RS, Alizadehfar R, Joseph L, Turnbull E, St Pierre Y, et al. Is the prevalence of peanut allergy increasing? a five-year follow-up study on the prevalence of peanut allergy in primary school children in Montreal. J Allergy Clin Immunol 2009;123:783-8.
23. 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.
24. Zuidmeer L, Goldhahn K, Rona RJ, Gislason D, Madsen C, Summers C, et al. The prevalence of plant food allergies: a systematic review. J Allergy Clin Immunol 2008;121:1210-8.
25. Marklund B, Ahlstedt S, Nordstrom G. Health-related quality of life among adolescents with allergy-like conditions—with emphasis on food hypersensitivity. Health Qual Life Outcomes 2004;2:65.
26. Woods R, Thien F, Raven J, Walters E, Abramson M. Prevalence of food allergies in young adults and their relationship to asthma, nasal allergies, and eczema. Ann Allergy Asthma Immunol 2002;88:183-9.
27. Sicherer SH, Munoz-Furlong A, Sampson HA. Prevalence of seafood allergy in the United States determined by a random telephone survey. J Allergy Clin Immunol 2004;114:159-65.
28. Roehr CC, Edenharter G, Reimann S, Ehlers I, Worm M, Zuberbier T, et al. Food allergy and non-allergic food hypersensitivity in children and adolescents. Clin Exp Allergy 2004;34:1534-41.
29. Lunet N, Falcao H, Sousa M, Bay N, Barros H. Self-reported food and drug allergy in Maputo, Mozambique. Public Health 2005;119:587-9.
30. Zuberbier T, Edenharter G, Worm M, Ehlers I, Reimann S, Hantke T, et al. Prevalence of adverse reactions to food in Germany—a population study. Allergy 2004; 59:338-45.
31. Du Toit G, Katz Y, Sasieni P, Mesher D, Maleki SJ, Fisher HR, et al. Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. J Allergy Clin Immunol 2008;122:984-91.
32. Venter C, Pereira B, Grundy J, Clayton CB, Arshad SH, Dean T. Prevalence of sensitization reported and objectively assessed food hypersensitivity amongst six-year-old children: a population-based study. Pediatr Allergy Immunol 2006; 17:356-63.
33. Smith W, Chey T, Jalaludin B, Salkeld G, Capon T. Increasing response rates in telephone surveys: a randomized trial. J Public Health Med 1995;17:33-8.
34. Hourihane JO, Kilburn SA, Dean P, Warner JO. Clinical characteristics of peanut allergy. Clin Exp Allergy 1997;27:634-9.
35. Brown SG. Clinical features and severity grading of anaphylaxis. J Allergy Clin Immunol 2004;114:371-6.
36. Sicherer SH, Burks AW, Sampson HA. Clinical features of acute allergic reactions to peanut and tree nuts in children. Pediatrics 1998;102:e6.
37. Eigenmann PA, Sampson HA. Interpreting skin prick tests in the evaluation of food allergy in children. Pediatr Allergy Immunol 1998;9:186-91.
38. Sampson HA. Utility of food-specific IgE concentrations in predicting symptomatic food allergy. J Allergy Clin Immunol 2001;107:891-6.
39. Sporik R, Hill DJ, Hosking CS. Specificity of allergen skin testing in predicting positive open food challenges to milk, egg and peanut in children. Clin Exp Allergy 2000;30:1540-6.
40. Hill DJ, Heine RG, Hosking CS. The diagnostic value of skin prick testing in children with food allergy. Pediatr Allergy Immunol 2004;15:435-41.
41. Ben-Shoshan M, Kagan R, Primeau MN, Alizadehfar R, Turnbull E, Harada L, et al. Establishing the diagnosis of peanut allergy in children never exposed to peanut or with an uncertain history: a cross-Canada study. Pediatr Allergy Immunol 2010; In press.

42. Pucar F, Lim H, Clarke AE. Peanut oral challenge: a retrospective study of 140 patients. Clin Exp Allergy 2001;31:40-6.
43. Kagan RS, Hayami D, Joseph L, St-Pierre Y, Clarke AE. The predictive value of a positive prick skin test to peanut in atopic, peanut-naïve children. Ann Allergy Asthma Immunol 2003;90:640-5.
44. Cochrane W. Sampling techniques, 3rd ed. New York: Wiley & Sons; 1997.
45. Kmetic A, Joseph L, Berger C, Tenenhouse A. Multiple imputation to account for missing data in a survey: estimating the prevalence of osteoporosis. Epidemiology 2002;13:437-44.
46. Rubin D. Multiple imputation for nonresponse in surveys. New York: Wiley; 1987.
47. Feveile H, Olsen O, Hogh A. A randomized trial of mailed questionnaires versus telephone interviews: response patterns in a survey. BMC Med Res Methodol 2007;7:27.
48. Rogers A, Murtaugh MA, Edwards S, Slattery ML. Contacting controls: are we working harder for similar response rates, and does it make a difference? Am J Epidemiol 2004;160:85-90.
49. Sicherer S, Munoz-Furlong A, Sampson H. Prevalence of self-reported peanut, tree nut and sesame allergy in the US determined by a random nationwide telephone survey: results from 1997, 2002 and 2008. J Allergy Clin Immunol 2010;125: AB216.
50. Dal GE, Taylor A, Wilson D. Is there a difference in health estimates between people with listed and unlisted telephone numbers? Aust N Z J Public Health 2005;29:448-56.
51. Galea S, Bucuvalas MJ. Optimizing telephone-based population sampling. Ann Epidemiol 2006;16:273-4.
52. Ngo-Metzger Q, Kaplan SH, Sorkin DH, Clarridge BR, Phillips RS. Surveying minorities with limited-English proficiency: does data collection method affect data quality among Asian Americans? Med Care 2004;42:893-900.
53. Davern M, McAlpine D, Ziegenfuss J, Beebe TJ. Are surname telephone oversamples an efficient way to better understand the health and healthcare of minority group members? Med Care 2007;45:1098-104.
54. Stang A, Moebus S, Dragano N, Beck EM, Mohlenkamp S, Schmermund A, et al. Baseline recruitment and analyses of nonresponse of the Heinz Nixdorf Recall Study: identifiability of phone numbers as the major determinant of response. Eur J Epidemiol 2005;20:489-96.
55. Sampson HA. Epidemiology of food allergy [review]. Pediatr Allergy Immunol 1996;7:42-50.
56. Sloane D, Sheffer A. Oral allergy syndrome. Allergy Asthma Proc 2001;22: 321-5.
57. Wensing M, Penninks AH, Hefle SL, Akkerdaas JH, van RR, Koppelman SJ, et al. The range of minimum provoking doses in hazelnut-allergic patients as determined by double-blind, placebo-controlled food challenges. Clin Exp Allergy 2002;32: 1757-62.
58. Ortolani C, Ballmer-Weber BK, Hansen KS, Ispano M, Wuthrich B, Bindslev-Jensen C, et al. Hazelnut allergy: a double-blind, placebo-controlled food challenge multicenter study. J Allergy Clin Immunol 2000;105:577-81.
59. Flinterman AE, Akkerdaas JH, Knulst AC, van Ree R, Pasmans SG. Hazelnut allergy: from pollen-associated mild allergy to severe anaphylactic reactions. Curr Opin Allergy Clin Immunol 2008;8:261-5.
60. Pastorello EA, Vieths S, Pravettoni V, Farioli L, Trambaioli C, Fortunato D, et al. Identification of hazelnut major allergens in sensitive patients with positive double-blind, placebo-controlled food challenge results. J Allergy Clin Immunol 2002;109: 563-70.
61. Lavon O, Lurie Y, Bentur Y. Scombroid fish poisoning in Israel, 2005-2007. Isr Med Assoc J 2008;10:789-92.
62. Choi SJ, Lee JC, Kim MJ, Hur GY, Shin SY, Park HS. The clinical characteristics of Anisakis allergy in Korea. Korean J Intern Med 2009;24:160-3.
63. Couture C, Measures L, Gagnon J, Desbiens C. Human intestinal anisakiosis due to consumption of raw salmon. Am J Surg Pathol 2003;27:1167-72.
64. Rangaraj S, Ramanathan V, Tuthill DP, Spear E, Hourihane JO, Alfaham M. General paediatricians and the case of resolving peanut allergy. Pediatr Allergy Immunol 2004;15:449-53.
65. Fleischer DM, Conover-Walker MK, Matsui EC, Wood RA. The natural history of tree nut allergy. J Allergy Clin Immunol 2005;116:1087-93.
66. Priftis KN, Mermiri D, Papadopoulou A, Papadopoulos M, Fretzayas A, Lagona E. Asthma symptoms and bronchial reactivity in school children sensitized to food allergens in infancy. J Asthma 2008;45:590-5.
67. Aaronov D, Tasher D, Levine A, Somekh E, Serour F, Dalal I. Natural history of food allergy in infants and children in Israel. Ann Allergy Asthma Immunol 2008; 101:637-40.
68. Cohen A, Goldberg M, Levy B, Leshno M, Katz Y. Sesame food allergy and sensitization in children: the natural history and long-term follow-up. Pediatr Allergy Immunol 2007;18:217-23.
69. Ewan PW. Clinical study of peanut and nut allergy in 62 consecutive patients: new features and associations. BMJ 1996;312:1074-8.
70. Fleischer DM. The natural history of peanut and tree nut allergy. Curr Allergy Asthma Rep 2007;7:175-81.
71. Moberg C, Meding B, Stenberg B, Svensson A, Lindberg M. Remembering childhood atopic dermatitis as an adult: factors that influence recollection. Br J Dermatol 2006;155:557-60.
72. Brogger J, Eagan T, Eide GE, Bakke P, Gulsvik A. Bias in retrospective studies of trends in asthma incidence. Eur Respir J 2004;23:281-6.
73. Ross MP, Ferguson M, Street D, Klontz K, Schroeder T, Luccioli S. Analysis of food-allergic and anaphylactic events in the National Electronic Injury Surveillance System. J Allergy Clin Immunol 2008;121:166-71.
74. Sicherer SH, Munoz-Furlong A, Sampson HA. Prevalence of peanut and tree nut allergy in the United States determined by means of a random digit dial telephone survey: a 5-year follow-up study. J Allergy Clin Immunol 2003;112:1203-7.
75. Grundy J, Matthews S, Bateman B, Dean T, Arshad SH. Rising prevalence of allergy to peanut in children: data from 2 sequential cohorts. J Allergy Clin Immunol 2002;110:784-9.
76. Venter C, Hasan AS, Grundy J, Pereira B, Bernie CC, Voigt K, et al. Time trends in the prevalence of peanut allergy: three cohorts of children from the same geographical location in the UK. Allergy 2010;65:103-8.
77. Csizmadi I, Kahle L, Ullman R, Dawe U, Zimmerman TP, Friedenreich CM, et al. Adaptation and evaluation of the National Cancer Institute's Diet History Questionnaire and nutrient database for Canadian populations. Public Health Nutr 2007;10:88-96.
78. Fitzpatrick KC. Regulatory issues related to functional foods and natural health products in Canada: possible implications for manufacturers of conjugated linoleic acid. Am J Clin Nutr 2004;79:1217S-20S.
79. Barr SI, Kwan S, Janelle KC. Nutrient analysis using computer programs—comparison of a Canadian and an American database. J Can Diet Assoc 1994; 55:29-32.
80. Hofer TP, Katz SJ. Healthy behaviors among women in the United States and Ontario: the effect on use of preventive care. Am J Public Health 1996;86:1755-9.