

Improved screening for peanut allergy by the combined use of skin prick tests and specific IgE assays

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Background: The diagnosis of peanut allergy must be based on reliable, safe criteria. Double-blind, placebo-controlled food challenges (DBPCFCs) are the gold standard but are costly and dangerous because they can trigger severe reactions. **Objective:** The aim of this study was to develop a new strategy for diagnosing peanut allergy while reducing the need for DBPCFCs.

Methods: We studied 363 children referred for an evaluation of suspected food hypersensitivity. They all benefited from the same diagnostic strategy, which included, in order, clinical history, a skin prick test (SPT), and a specific IgE assay. DBPCFCs were performed on all the children by personnel who were unaware of the results of the other tests. To assess the performance characteristics of the SPT (comparing commercial and raw peanut extracts) and the specific IgE assay, we compared the results with those provided by the DBPCFCs. For SPTs and specific IgE assays, we sought to determine the cutoff values required to exclude false-positive and false-negative results.

Results: According to DBPCFC results, 177 children were allergic to peanut, and 186 were not. The performance characteristics of the SPTs were superior with the raw extract because the negative predictive value was 100% (95% confidence interval [CI], 97.5-100). If the skin reaction with the raw extract was less than 3 mm, we could be quite certain that the child was not allergic. On the other hand, if the SPT resulted in a wheal diameter of larger than 3 mm, we could only be 74% certain that the children were allergic. Furthermore, if the SPT resulted in a wheal diameter of 16 mm or larger, we could be quite certain that the child was allergic because the positive predictive value was 100% (95% CI, 86.8-100). Specific IgE concentrations of 57 kU_A/L or greater were associated with a positive predictive value of 100% (95% CI, 87.2-100). The combined use of the tests resulting in a positive diagnosis if the SPT result was 16 mm or larger or specific IgE concentration was 57 kU_A/L or greater and in a negative diagnosis if the SPT result was less than 3 mm and the specific IgE concentration was less than 57 kU_A/L allowed us to classify subjects with almost complete certainty as being allergic or not because the predictive values were 100%.

Conclusion: Commercial extracts could not be used to reliably predict tolerance of peanut. Peanut DBPCFCs can be avoided

when SPTs with raw extracts resulted in wheals with a largest diameter of less than 3 mm and a specific IgE concentration of less than 57 kU_A/L and also when wheal diameters were 16 mm or larger or specific IgE values were 57 kU_A/L or greater. Otherwise, DBPCFCs were indispensable for the unequivocal diagnosis of peanut allergy. (*J Allergy Clin Immunol* 2002;109:1027-33.)

Key words: Children; double-blind, placebo-controlled food challenge; peanut allergy; skin prick test; specific IgE; performance characteristic

The prevalence of peanut allergy in the general population is estimated at 1%.¹⁻³ This food allergy is of special importance because it is almost impossible to avoid consuming foods containing peanuts. Symptoms can be extremely severe, with one person in 2 having an anaphylactic reaction after the ingestion of foods containing peanuts.⁴⁻⁸ Because peanuts are involved in most cases of food-induced anaphylactic shock,^{4,6-10} it is vital to establish a reliable diagnosis of peanut allergy. Previous studies have looked at skin prick tests (SPTs), specific IgE assays, and oral food challenges.¹¹⁻¹⁴ The oral food challenge remains the gold standard but is dangerous because it can trigger severe reactions and must be administered in a specialized facility capable of dealing with anaphylactic shock. This points to the need to develop less dangerous and less costly diagnostic strategies that reduce the need to administer oral food challenges and that provide a reliable diagnosis of peanut allergy.

In a study involving 20 children with peanut allergy, Sampson and Ho¹⁵ reported that peanut-specific IgE concentrations greater than or equal to 15 kU_A/L (Cap System, Pharmacia) had a positive predictive value (PPV) of greater than 95%. At this cutoff level, sensitivity and specificity were 73% and 92%, respectively. At a cutoff level of 0.35 kU_A/L, corresponding to the lower limit of detection for specific IgEs with the standard assay kit, the PPV in this study cohort was 78%, whereas in a population with an extrapolated prevalence of peanut allergy of 10%, the PPV was 15%. An IgE concentration exceeding 15 kU_A/L would be sufficient to establish a diagnosis of peanut allergy and would obviate the need for an oral food challenge. Sampson,¹⁶ in a recent prospective study with 68 children, validated this previously established diagnosis decision point. However, the threshold value of 15 kU_A/L (CAP System, Pharmacia) was determined by using a population of children with atopic dermatitis and did not correspond to results we previously published.¹⁷ The discrepancy might depend on the clinical manifestations of the allergy

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Food and drug
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anaphylaxis

Abbreviations used

AUC:	Area under the ROC curve
CI:	Confidence interval
DBPCFC:	Double-blind, placebo-controlled food challenge
NPV:	Negative predictive value
PPV:	Positive predictive value
ROC:	Receiver operating characteristic
SPT:	Skin prick test

and might vary with geographic location. We believe that it is indispensable to set discriminatory threshold values that provide 100% PPV and negative predictive value (NPV), with precise confidence intervals (CIs), to avoid both false-positive results (and the associated lifelong effect) and false-negative results (and the risk of anaphylactic reactions caused by the accidental ingestion of peanut-containing food products). The aim of this study was to develop a new diagnostic strategy combining SPTs and specific IgE assays to provide high PPV and NPV for peanut allergy while reducing the need for double-blind, placebo-controlled food challenges (DBPCFCs).

METHODS

Study population

We conducted a cross-sectional study with 363 children referred for an evaluation of suspected food hypersensitivity. They had not been given a diagnosis of peanut allergy at the time of inclusion, but we systematically tested for this allergy because peanut is the most common food allergen in France. The children were all subjected to the same testing procedure, which was always performed in the same order and at the same time: clinical history, SPT with commercial and fresh (raw) extracts, specific IgE assay, and DBPCFC. All the tests were performed independently on each child, with conditions blind to the results of the other tests.

The children were defined as being allergic to peanut on the basis of a positive DBPCFC result with a standard protocol.^{11-14,18} The children who had a negative DBPCFC result were classified as nonallergic. Children receiving antihistamines or corticosteroids (local or systemic) were excluded because these medications could interfere with the results of the tests. Children with unstable asthma and those whose parents refused to authorize the oral food challenge were also excluded. In total, we excluded 3 children, 2 because of poorly controlled asthma and 1 because the parents refused the DBPCFC. We did not observe children with an unequivocal history of anaphylactic shock induced by ingestion of food. The children were consecutively recruited at the Children's Hospital (CHU de Toulouse) allergology unit between January 1, 1999, and January 1, 2001. They were representative of children who consulted for suspected food hypersensitivity.

Questionnaire

A standardized questionnaire completed by a trained physician was used to gather information on family and personal histories of atopy (asthma, allergic rhinitis, eczema, food allergy, and peanut allergy) and the symptoms that brought the children to the allergology unit.

Skin prick tests

The SPTs were performed on all the children according to a recommended protocol^{12,19-21} with a standardized commercial peanut extract (100 IR/mL, Allerbio), raw peanut crushed and mixed 1:10 (wt/vol) in PBS, a positive control (10 mg/mL histamine), and a

negative control (glycerosaline solution). Other SPTs were routinely performed on children who consulted the allergology unit.

The SPTs were performed on healthy skin on the volar surface of the forearm (on the back for nursing children) at 3-cm intervals along a grid, with the pattern following a corresponding list of the allergens used for each identification. A lancet (Dome-Hollister-Stier) was used to prick the skin at a 90° angle through a drop of test solution on the skin. The raw peanut SPT was performed with the prick-plus-prick technique. Briefly, the prick-plus-prick technique described by Dreborg and Foucard²² consists of crushing raw peanut in PBS to form a paste. The tip of a lancet is then dipped in the peanut paste and used to prick the skin. The same pressure was used for all the SPTs to obtain the same degree of allergen penetration. Duplicate SPTs were performed on children 2 years of age and older. The SPTs were administered by a technician trained in the technique, and the results were read 20 minutes later by the same person to ensure uniformity. The dimensions of the reactions were drawn on transparent paper placed over the reaction sites to measure the diameters of the wheals (in millimeters). An SPT response was scored as positive if the largest wheal diameter was 3 mm or greater than that produced by the negative control and at least 50% greater than that produced by the positive control.

Laboratory studies

Blood samples were drawn from all the children for peanut-specific IgE assays, which were all performed at the same immunology laboratory (CHU Rangueil, Toulouse) with the standardized CAP System FEIA technique (Pharmacia & Upjohn Diagnostics).^{12,23} Children were considered sensitized if their specific IgE concentrations exceeded 0.35 kU_A/L.

Oral food challenge

DBPCFCs were performed according to international recommendations under the supervision of the study doctor and specialized medical and paramedical staff trained in the diagnosis and treatment of anaphylactic reactions. Peanut was eliminated from the diet at least 2 weeks before the oral food challenge. The monitoring process before and after each increase in dose included cardiopulmonary examinations (pulse and auscultation), blood pressure measurements, mucocutaneous assessments, palpations for abdominal pain, and peak expiratory flow or FEV₁ (spirometer) measurements. Intravenous catheters were installed before beginning the DBPCFC to be able to quickly treat allergic reactions that occurred during the DBPCFC. The doses were increased every 15 to 20 minutes until symptoms were observed. The doses of raw peanut extract were increased as follows: 1 mg, 5 mg, 10 mg, 50 mg, 200 mg, 500 mg, 1000 mg, 2000 mg, and 5000 mg. The raw peanut extract was mixed with rice, chocolate, and mint²⁴ to disguise the taste and meet the criteria for a double-blind study. The subject, the nurse performing the test, and the study doctor were unaware of the mixture being used (with or without peanut). The placebo (rice, chocolate, and mint) was administered by using the same progression, with a 4-hour interval between the 2 tests. The same number of doses was administered. The order of the challenge (placebo, peanut) was randomized by a member of the staff not involved in evaluating the study subject. The following objective symptoms were noted for various organs: skin (erythema, pruritus, appearance or reactivation of eczema, urticaria scored as a percentage of body surface affected, and edema); nose (obstruction, sneezing, and rhinorrhea); lungs (dry cough, wheezing, dyspnea, and 20% drop in peak expiratory flow); gastrointestinal (diarrhea and vomiting); and cardiopulmonary (20 mm Hg drop in arterial pressure and tachycardia >20%). Abdominal pain was only taken into consideration as a subjective symptom. In the event of an adverse reaction, the test was stopped, and the first treatment was initiated.

Patient consent

Legal guardians received detailed information on the procedure and the inherent risks and signed an informed consent form before their children were included in the study. The study was approved by the local ethics committee, and the results were recorded in anonymous files.

Statistics

All variables were subject to quality control. Quantitative variables were calculated as means with SDs when the data had a normal distribution. For data without a normal distribution, the medians and ranges were calculated. Normality was verified graphically. Specific IgE values were scored as 0 for those less than 0.35 kU_A/L because 0.35 was the cutoff limit for detectable antigen-specific IgE measured by the assay. The performance characteristics of the 2 SPTs were estimated by comparing the areas under the receiver operating characteristic (ROC) curves by using the Wilcoxon test. A perfect diagnostic test would have perfect sensitivity and specificity. In such cases the ROC curve would have a line straight up the vertical axis to a sensitivity of 1 and then straight across parallel to the horizontal axis. This perfect test would have an area under the ROC curve (AUC) of 1. Thus AUCs close to 1 generally indicate good diagnostic tests. Inversely, a fully noninformative test generates an AUC of 0.5.²⁵ We estimated threshold values that result in 100% sensitivity or specificity of the tests. In a nonparametric ROC curve analysis, each point on the curve represents a 2 × 2 decision matrix at one criterion, where each patient is classified either as negative (ie, below the selected cutoff value) or positive (ie, above the cutoff value). Therefore we estimated 95% CIs for sensitivity, specificity, PPV, and NPV by using exact CIs on the basis of binomial probability distributions. Percentage comparisons were performed with the Pearson χ^2 test. Mean difference testing was performed with the Student *t* test. If the test assumptions were not met, a Mann-Whitney test was performed to compare distributions between the allergic and nonallergic groups. All statistical tests were 2 sided, and comparisons were considered significant at a *P* value of less than .05. The statistical analyses were performed with the Stata software package (Stata Corp 1999, Stata Statistical Software: Release 6.0).

RESULTS

Study population

Three hundred sixty-three children were included in the study. On the basis of the DBPCFC results, 177 children were allergic to peanut, and 186 were not. The median age was 4.0 years (range, 0.1-15.9 years), the boy/girl sex ratio was 1.8, and 67.5% (245/363) had a family history of atopic disease. All 186 children with a negative DBPCFC result had one or more allergies to foods other than peanut: allergies confirmed by DBPCFCs performed with the same procedure as the peanut oral food challenge were egg (44%); cow's milk (33.5%); mustard (7.2%); fish (3.2%); wheat (2.6%); hazelnut (1.9%); shrimp, chicken, and kiwi (1.3%); and sesame, other nuts, soy, vanilla, and banana (0.6%). A comparison of groups with and without peanut allergy is given in Table I. The children with peanut allergy were significantly older, the median age at the time of diagnosis being 4.4 years (range, 1.0-15.0 years) versus 3.7 years (range, 0.1-15.9 years) for children without peanut allergy (*P* < .001). Symptoms triggered by DBPCFCs in

TABLE I. Characteristics of 177 children with peanut allergy and 186 children without peanut allergy who consulted the Children's Hospital (CHU Toulouse) allergology unit between January 1999 and January 2001

	Allergic (n = 177)	Nonallergic (n = 186)	<i>P</i> value
Age, y (median [range])	4.4 (1.0-15.0)	3.7 (0.1-15.9)	.001
Boys (%)	62.2	65.6	.50
Atopic family disease (%)	72.9	62.4	.03
Initial symptoms (%)			
Gastrointestinal	8.5	4.8	.36
Cutaneous	83.6	84.4	
Pulmonary	4.5	7.6	
Anaphylaxis	3.4	3.2	

the 177 children with peanut allergy and 282 events (sometimes in association) were as follows: gastrointestinal, 26.6% (95% CI, 20.2-33.7); oral allergy syndrom, 11.3% (95% CI, 7.0-16.9); urticaria, 72.3% (95% CI, 65.1-78.8); eczema exacerbation, 5.6% (95% CI, 2.7-10.2); edema, 16.9% (95% CI, 11.7-23.3); rhinoconjunctivitis, 9.0% (95% CI, 5.3-14.3); asthma attack, 12.4% (95% CI, 7.9-18.2); laryngeal edema, 1.1% (95% CI, 0.1-4.0); and anaphylactic shock defined by hypotension and collapse, 4.0% (95% CI, 1.7-8.0).

SPTs: Commercial and raw extracts

The median skin reaction (measured at the largest SPT wheal diameter) to the commercial extract was 3 mm (range, 0-16 mm), and the median skin reaction to the raw extract was 6 mm (range, 0-25 mm; *P* = .02). SPT responses were negative when commercial extracts were used in 32 subjects with proved peanut allergy (32/177 [18%]; sensitivity, 82%), and SPT responses were always positive when raw extracts were used (sensitivity, 100%). The AUCs were 0.90 (95% CI, 0.87-0.93) for the raw extract versus 0.79 (9% CI, 0.74-0.83) for the commercial extract (*P* < 10⁻⁴). The ROC curves are shown in Fig 1. The cutoff values required to exclude false-negative and false-positive results were 3 and 16 mm, respectively. The performance characteristics of the raw SPTs for those decision points are summarized in Table II. No significant association was found between the wheal diameters of the SPTs and the symptoms that led the children to consult for food allergy (*P* = .69) or the symptoms triggered by the DBPCFC (*P* = .9).

Specific IgE concentrations that allowed subjects with peanut allergy to be distinguished from subjects without peanut allergy

The specific IgE concentrations of the children with peanut allergy and those without had different distributions. The median was 0 kU_A/L (range, 0-56 kU_A/L) for nonallergic patients versus 10 kU_A/L (range, 0-100 kU_A/L) for patients with peanut allergy (*P* < 10⁻⁴). As

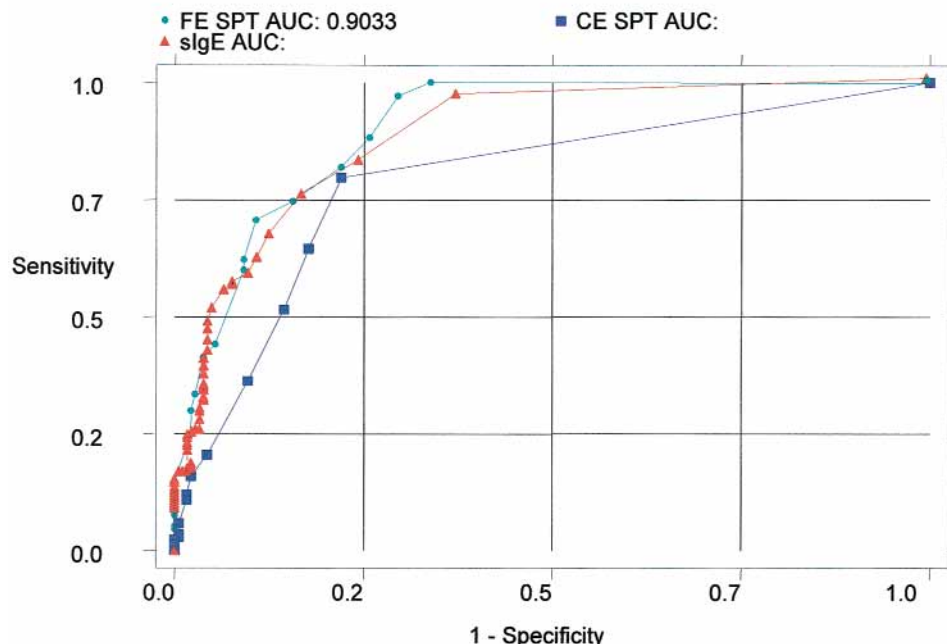


FIG 1. Comparison of ROC curves for SPTs with commercial extracts, SPTs with raw extracts, and specific IgE assays in the diagnosis of peanut allergy in 177 children with proved peanut allergy and 186 children without peanut allergy. *FE SPT AUC*, AUC for SPTs with fresh extracts; *CE SPT AUC*, AUC for SPTs with commercial extracts; *sIgE AUC*, AUC for specific IgE assays.

shown in Fig 1, the performance characteristics of specific IgE concentrations for diagnosing peanut allergy were good because the AUC was 0.87 (95% CI, 0.84-0.91). In our study a threshold value greater than or equal to 57 kU_A/L resulted in a specificity and a PPV of 100% (Table II). This decision point would have avoided the need for DBPCFCs in 27 children. On the other hand, no threshold value provided 100% sensitivity. Taking into account the lower detectable value for the specific IgE concentration as a decision point would result in a sensitivity of 96.6%. The threshold value giving a specificity of 95% was 15 kU_A/L. However, the percentage of undetected allergic subjects was 56%. There was no association between specific IgE concentrations and the initial severity of the food allergy or the symptoms triggered by the DBPCFCs in subjects with peanut allergy.

Combined use of SPTs and specific IgE assays

In an attempt to develop a diagnostic strategy that reduced the number of DBPCFCs to be performed, we intentionally used the threshold required to exclude false-positive and false-negative results to generate the combined test. The combined use of SPTs and specific IgE assays in patients with peanut allergy would result in a positive diagnosis if at least one of the 2 test results was positive (≥ 16 mm, ≥ 57 kU_A/L). It would also provide a sensitivity of 27.8% and a specificity of 100% and would have avoided the need for DBPCFCs in 47 of the 177 allergic subjects (26 with SPT result ≥ 16 mm and 27 with IgE levels ≥ 57 kU_A/L). Therefore when the combined

test result was positive, the subject was classified with nearly 100% certainty as being allergic because the PPV was 100% (Table II).

When the values were between 3 and less than 16 mm for the SPTs and less than 57 kU_A/L for the specific IgE assays, there were always false-positive and false-negative results that made it necessary to perform DBPCFCs to avoid prescribing strict peanut-free diets for nonallergic children and letting children with peanut allergy remain without a diagnosis. In addition, when the raw SPT diameter was less than 3 mm and the specific IgE value was less than 57 kU_A/L, the NPV was 100%, and the subject was classified with nearly 100% certainty as being nonallergic.

DISCUSSION

The DBPCFC is universally recognized as the gold standard for the diagnosis of peanut allergy because of its 100% sensitivity and specificity. Clinical histories, SPTs, and specific IgE assays can indicate a possible diagnosis of peanut allergy. Nevertheless, suggestive clinical histories are validated by DBPCFCs in less than 50% of subjects.²⁶ The effect of a diagnosis of peanut allergy on an individual's lifestyle in terms of school or work performance, leisure activities, and financial burden²⁷ make it essential to avoid wrongly prescribing a strict, lifelong, peanut-free diet for nonallergic subjects. It is also essential to avoid letting children with true peanut allergy remain without a diagnosis, exposing them to the risk of accidentally ingesting foods containing peanut. DBPCFCs require specialized facilities and trained personnel²⁸ and are cost-

TABLE II. Performance characteristics for a diagnosis of peanut allergy by using the SPT with raw peanut extract, specific IgE assay, and combined use of the 2 tests in 177 children with peanut allergy and 186 children without peanut allergy

	SPTs (mm)		Specific IgE (kU _A /L)		Combined use*
	≥3	≥16	≥0.35	≥57	
Sensitivity (%)	100	14.7	96.6	15.3	27.7
95% CI	97.9-100	9.8-20.8	92.7-99.0	10.3-21.4	20.2-33.7
Specificity (%)	66.1	100	62.4	100	100
95% CI	58.8-72.9	98.0-100	55.0-69.3	98-100	98.0-100
PPV† (%)	73.7	100	71.0	100	100
95% CI	67.7-79.2	86.8-100	64.8-76.6	87.2-100	92.5-100
NPV‡ (%)	100	55.2	95.1	55.4	58.9
95% CI	97.5-100	49.7-60.6	89.6-98.2	49.9-60.8	53.2-64.3

*This test would result in a positive diagnosis if at least one of the 2 test results was positive (ie, SPT ≥16 mm or specific IgE ≥57 kU_A/L).

†PPV estimated for a pretest probability of 48.8%.

‡NPV estimated for a pretest probability of 48.8%.

ly and time consuming. They are also dangerous because they can trigger severe reactions. It is thus logical to develop new diagnostic strategies to reduce the need for prescribing them. Our study is valid in that it corresponds to the first diagnostic step for children with suspected food allergies. In the event of an unequivocal clinical history of anaphylactic shock triggered by the ingestion of peanut, peanut consumption must be forbidden, no matter what the values of the SPT and specific IgE assay. However, when in doubt, a DBPCFC must be performed. A strong suspicion of peanut allergy despite negative test results should lead to a DBPCFC under medical supervision and not a return home with no specific dietary advice.

Study population

Our study population was made up mainly of boys, reflecting the normal distribution of peanut allergy in children, and 67.5% had a family history of atopy, which is also typical of children with food allergies.^{16,29-32} The children with peanut allergy were significantly older than those who were not (median age, 4.4 years [range, 1-15 years] vs 3.7 years [range, 0.1-15.9 years]). However, the average reported age at the time of the diagnosis is 24 months.^{30,32-34} Three fourths of the children with peanut allergy have their first symptoms between 12 and 24 months.³⁴ The possibility that the children in the nonallergic group were too young to have had peanut allergy can thus be eliminated. Our study population was made up of 48.8% (177/363) children with peanut allergy. This would reflect the recruitment bias resulting from the inclusion of children who had consulted for suspected food hypersensitivity. Food hypersensitivity is often overestimated, which in turn results in an overestimation of the PPV. Our population had a wide range of symptoms (atopic dermatitis, urticaria, angioedema, asthma, and anaphylactic reactions) typical of those seen in people with peanut allergy. Nevertheless, we did not observe an association between the screening tests and the symptoms. Our study was based on a large number of children with and without peanut allergy subjected to the same testing procedure that was performed independently on each child blindly to the

results of the others tests. Our study allowed us to make more accurate estimations of sensitivity and specificity for the various thresholds to be made.

Skin prick test

A negative SPT response is very useful for eliminating a diagnosis of peanut allergy because of the high NPV (>95%).¹⁵ Nevertheless, the test depends greatly on the quality of the peanut extract used.^{25,35,36} Our results demonstrated the superior effectiveness of raw extracts over commercial extracts. The difference is probably the result of the industrial processing of the peanut to produce a standardized commercial extract. Removing the peanut oil also eliminates hydrophobic allergens from the commercial extract, which is not the case for the raw extract. Another issue is the concentration of proteins (presumably higher in the raw extract for the prick-plus-prick test). We thus propose that patients suspected of peanut allergy be first tested with raw extract to improve the sensitivity and specificity of the SPT.

In a previous study we also demonstrated the superiority of raw extracts.³⁷ In a recent study involving 142 patients (adults and children) with peanut allergy, SPTs with a raw extract achieved 97% sensitivity, with an average skin reaction diameter of 9 mm versus 4.9 mm with a commercial extract.³⁸ The double-blind study was conducted with children given a new diagnosis with peanut allergy, and the observers who measured the skin reactions had no knowledge of the final diagnosis, which enhanced its validity. Sporik et al³⁹ set the threshold value for the SPT at 8 mm (4 mm before the age of 2 years), with a specificity of 100% when using commercial extracts with open food challenges. Our results provide additional information in that they reduced the need for DBPCFCs on the basis of the diameter of the skin reaction. A skin reaction of less than 3 mm avoided false-negative results because the NPV was 100% (95% CI, 97.5-100.0). We could be quite certain that the child was not allergic. On the other hand, if the SPT response was positive, we could only be 74% certain that the child was allergic. A wheal diameter of greater than 16 mm avoided

false-positive results, meaning that the probability of the SPT accurately predicting allergy was 100% (95% CI, 87.0-100.0). In summary, DBPCFCs were indispensable for an unequivocal diagnosis of peanut allergy if the skin reactions with raw extracts were 3 mm or greater and less than 16 mm.

Specific IgE assays

Sampson and Ho¹⁵ set the threshold value for peanut-specific IgE concentrations at 15 kU_A/L for a PPV of 95%, a sensitivity of 73%, and a specificity of 92%. The authors estimated that 40% of oral food challenges could be eliminated when peanut-specific IgE concentrations were 15 kU_A/L or greater. They recommended DBPCFCs only when IgE levels were less than 15 kU_A/L, whereas concentrations over this threshold value were sufficient to establish a diagnosis of peanut allergy without requiring an oral food challenge. However, this study was limited to a small number of patients with atopic dermatitis, resulting in a great variability of the estimations made. In a recent prospective study by Sampson¹⁶ involving children with atopic dermatitis (61%) and asthma (approximately 50%), the prevalence of peanut allergy was reported to be 81%. A diagnosis of peanut allergy was made for 13% of 68 children on the basis of suggestive histories (food allergies that did not require emergency treatment), for 85% on the basis of convincing histories (allergic reactions in the preceding 2 years that required emergency treatment), and only for 2% on the basis of oral food challenges. At the threshold value of 14 kU_A/L, the sensitivity was 57%, and the specificity was 100%.¹⁶ We obtained different results. At the same threshold value, the sensitivity was 44%, and the specificity was 95.2%. In our study if specific IgE concentrations were 57 kU_A/L or greater, we could be quite certain that the child was allergic to peanut. As with the SPTs, we noted no association between specific IgE concentrations and the severity of the initial clinical symptoms or those triggered by the DBPCFCs.

Combined use of SPTs and IgE assays

To our knowledge, no study has yet looked at the effectiveness of the combined use of SPTs and IgE assays to diagnose peanut allergy. The DBPCFC is the gold standard for diagnosing peanut allergy because it has a sensitivity and a specificity of 100%. No other screening tests, not even the combined use of SPTs and specific IgE assays, provide this level of sensitivity and specificity. Nevertheless, the need for prescribing DBPCFCs can be reduced. In our study the combined use of the 2 tests made it quite certain that children had peanut allergy when one of the 2 test results was positive (≥ 16 mm for the raw SPT and ≥ 57 kU_A/L for the specific IgE assay) with a sensitivity of 27.7% and a specificity of 100%. Moreover, a skin reaction diameter of less than 3 mm combined with a specific IgE assay result of less than 57 kU_A/L totally eliminated the possibility of peanut allergy (100% NPV). For other raw SPT and specific IgE assay values, there were always false-positive and false-nega-

tive results that made it necessary to perform an oral food challenge. The objective of any diagnostic strategy is to avoid wrongly prescribing a lifetime peanut-free diet for people who are not allergic because of the devastating effect on their quality of life and social integration and on their family and friends.^{20,27} Furthermore, children with peanut allergy must not be given an incorrect diagnosis of not having allergy because of the dangers posed by accidental exposure to peanut. The prevalence of peanut allergy has an influence on the PPVs and NPVs of the tests. Our study was conducted among a population of children with a prevalence of peanut allergy of 48.8%.

The clinical history must be taken into consideration, and a DBPCFC should be undertaken when the history is suspicious for a reaction. Our study corresponds to the first diagnostic step for children with suspected food allergies. We did not observe children with unequivocal histories of anaphylactic shock induced by ingestion of peanut. This proposed diagnostic strategy must be confirmed in another population with peanut allergy.

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