Journal of Microbiology, Immunology and Infection (2012) 45, 90–95

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ORIGINAL ARTICLE

Patterns of sensitization to peanut allergen components in Taiwanese Preschool children

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Received 8 July 2011; received in revised form 13 July 2011; accepted 13 September 2011

KEYWORDS
Allergy;
Arachis hypogaea;
Asian;
Children;
Component-resolved diagnosis;
Peanut

Background/Purpose: Peanut allergy is very common in Western countries, although it is seldom encountered in Eastern countries. Peanuts are comprised of at least 11 components, but the contribution to clinical symptoms by each component in each individual is not known. This study investigated the distributions of sensitivity to peanut allergen components among Taiwanese children who were sensitized to peanuts and followed the evolution of sensitization patterns to these components.
Methods: We enrolled 29 preschool children (age $= 2.11 \pm 1.36$ years) who were sensitized to peanuts above class 3. Serum was analyzed for specific immunoglobulin E (IgE) antibodies to recombinant Ara h 1, Ara h 2, Ara h 3, Ara h 8, and Ara h 9. Allergen component-specific IgE $\geq$0.35 kU_A/L was defined as positive. Eighteen children were retested 22.64 ± 5.1 months later. Peanut allergy symptoms were recorded from detailed questionnaires.
Results: The percentages of children sensitized to Ara h 1, 2, 3, 8, and 9 were, respectively, 51.8%, 65.5%, 62.1%, 13.8%, and 24.1%. Regarding changing patterns of peanut component sensitization at follow-up, children with clinical symptoms to peanuts had persistent elevations of Ara h 2-specific IgE: 12.6 ± 1.01 up to 34.15 ± 19.4 kU_A/L; $p = 0.144$. In contrast, Ara h 2 concentrations decreased significantly in children without clinical symptoms. Ara h 8 and 9 were nonspecific for children with or without symptoms.
Conclusion: Ara h 1, Ara h 2, and Ara h 3 were major components contributing to peanut sensitization in Taiwanese children. Ara h 2 was probably the most important component that contributed to clinical symptoms and remained steady in children who had peanut allergy.

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1684-1182/$36
doi:10.1016/j.jmii.2011.11.001

Introduction

Allergy to peanuts is one of the leading causes of fatal allergic reactions. The prevalence of peanut allergy has increased dramatically in the last decade. It currently affects about 1.15%—1.5% of these populations, and there is an estimated threefold increase in reported peanut allergies among Westernized countries.[1–4] However, peanut allergy is not common in Asia, and its reported prevalence is about 0.4%—0.6%.[5] Children with allergies to milk or egg whites usually develop tolerance and become asymptomatic as they grow older. In contrast, peanut allergy symptoms are usually lifelong, but a minority of patients may outgrow the reactions over time.[6] Consuming peanut-containing products can lead to shock and even death among those with severe peanut allergies.[7,8] Therefore, it is very important to diagnose peanut allergy at an early stage.

The current clinical gold standard for diagnosing peanut allergy is an oral food challenge test (OFC),[9] but it is time-consuming, expensive, and extremely risky in clinical practice.[10] Therefore, a skin prick test or testing for serum levels of peanut-specific immunoglobulin E (IgE) are currently used as replacements in the clinic.[11,12] However, people who are positive by peanut-specific IgE tests can only be classified as having a peanut sensitization.[13] Evidence is required from a patient's clinical history of peanut contact experience for a diagnosis of peanut allergy. It has also been reported that only a skin prick result larger than 8 mm or a peanut-specific IgE level higher than 15 kU_A/L had a high predictive value for clinical peanut allergy.[14]

A possible reason for the ambiguity between peanut sensitization and peanut allergy is that the allergens used in test kits are derived from crude peanut protein extracts, which contain both allergenic and nonallergenic molecules. In addition, some of these molecules may cross-react with pollens or other allergens in foods.[15,16] Overall, this leads to discrepancies in test results and clinical diagnoses.

At least 11 different allergen components have been found to be associated with peanut allergy. These are designated Ara h 1-11 (*Arachis hypogaea*). Among these, Ara h 1, Ara h 2, and Ara h 3 are the major peanut allergen components based on their protein ratios and cellular activities. These are seed storage proteins comprised of vicilin, conglutin, and glycinin, respectively.[17] It was reported that American patients who became allergic around 1 year of age presented more frequently with IgE antibodies to rAra h 1 to 3 (56.7%—90.0%) than Swedish patients (37.1%—74.3%), followed by Spanish patients (16%—42%).[18] Ara h 4 and Ara h 3 are nearly identical isoforms,[19] and, additionally, Ara h 5 and Ara h 8 are both related to pollen allergy and are not seed storage proteins. Ara h 5 is a profilin protein in peanuts, which presents low quantity in peanuts and leads to a cross-reaction with Bet v 2, the birch pollen profilin[20] and Ara h 6 and Ara h 7 are proteins homologous to Ara h 2.[21] Ara h 8, a homologous protein of Bet v 1, the major allergen in birch pollen, is heat sensitive and labile to digestion, and usually results in clinical symptoms associated with oral allergy syndrome.[22] Ara h 9 is a nonspecific lipid-transfer protein (LTP), with properties of heat and acid resistance. Its clinical symptoms include systemic allergy and oral allergy. People in Mediterranean countries often have LTP allergy due to the high consumption of vegetables and fruits, among which Ara h 9 is the major causative allergen and is more relevant than Ara h 1-3.[23] Ara h 10 and Ara h 11 are both plant protein oleosins that were obtained from oil bodies from peanut and were not realized until their clinical relevance was present.

Peanut allergy has different clinical and immunologic patterns in different areas of the world. We investigated whether the uncommon presentation of peanut allergy in Taiwanese preschool children could be related to a different sensitization pattern to peanut allergen components.

Methods

Study population

From April 2007 to April 2009, 3936 children with allergic diseases had been tested for serum levels of peanut-specific IgE in the Pediatric Allergy and Asthma Center of Chang-Gung Memorial Hospital. Among these children, 215 were tested as positive (peanut-specific IgE levels $\geq$0.35 kU_A/L). There were only 29 preschoolers (age between 6 months to 6 years) out of 215 test-positive children who met our criteria by having peanut-specific IgE levels higher than 3.5 kU_A/L (or ImmunoCAP above class 3); then, their blood samples were tested for peanut component-specific IgE. After 1.5 to 2 years, they returned for follow-up blood tests for peanut component-specific IgE and parents completed a questionnaire about the child's allergic symptoms. The questionnaire included items such as the patient's consumption of peanut-containing products during the follow-up period. If peanut exposure was confirmed, we specifically inquired about the occurrence of allergic symptoms, including skin rash, eyelid swelling, cough, mouth numbness, heart palpitations, and difficulty breathing when the child consumed peanut-containing products. Written informed consent were obtained from the main caregiver for all study patients.

Determination of sensitization

Serum was analyzed for specific IgE antibodies to peanut-recombinant Ara h 1, Ara h 2, Ara h 3, Ara h 8, and Ara h 9 by ImmunoCAP (Phadia, Sweden). An allergen-specific IgE level $\geq$0.35 kU_A/L was defined as positive. Levels between 0.35 and 100 kU_A/L were recorded.

Statistics

A Wilcoxon Signed Rank Test was used to compare changes of specific IgE levels with peanut allergen components between two time points. A p value <0.05 was considered statistically significant.

Results

Baseline results

The average age of the 29 children with peanut sensitization was 2.11 $\pm$ 1.36 years (range: 0.53—5.7 years), and all