

Association Between Peanut Allergy and Asthma Morbidity

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Objective To evaluate the relationship between peanut allergy and asthma morbidity in school-age children.

Study design The study involved a medical chart review to assess the association of peanut allergy with asthma morbidity in children beyond age 3 years. Peanut allergy was assessed by specific and validated criteria. A Poisson regression model was used to compare the frequency of systemic steroid use and of hospitalization for asthma beyond age 3 years in children with asthma with and without peanut allergy.

Results Children with peanut allergy had a 2.32-times greater rate of hospitalization ($P = .03$) and a 1.59-times greater rate of systemic steroid use ($P < .001$) after controlling for covariates.

Conclusions Peanut allergy serves as an early marker for asthma morbidity. Early prevention and intervention can improve quality of care. (*J Pediatr* 2010; ■: ■-■).

Asthma is a highly prevalent disease, affecting approximately 9 million children under age 18.^{1,2} Asthma exacerbations are the leading cause of childhood inpatient admissions, causing 2 million emergency department visits and 500 000 hospitalizations among all patients with asthma in 2005.³ Asthma also is a major risk factor for fatal and near-fatal food-induced anaphylaxis.⁴⁻⁶ More than one-third of children with food allergies are currently living with asthma.^{7,8}

Morbidity from asthma has been increasing in the United States. Reducing asthma morbidity is a key focus of research and public health initiatives. A better understanding of the underlying mechanisms of asthma and risk factors for poor asthma control is therefore essential to improve the outcomes of these patients.

Several risk factors for increased asthma morbidity have been identified, including the presence of food allergy.^{4,9-11} In a previous study, we established a link between allergy to milk and peanut and asthma morbidity in children in the early childhood years.¹¹ As more children are entering school with a dual diagnosis of peanut allergy and asthma,¹²⁻¹⁴ we now need to focus on the effect of peanut allergy on asthma symptoms beyond age 3 years. Because children often present with food allergy in early childhood, peanut allergy could serve as an early marker for asthma morbidity. In the present study, we assess the effect of peanut allergy on asthma morbidity beyond age 3 years.

Methods

The study population comprised 410 children age 5-18 years with asthma and a food allergy who received care at a large tertiary care children's hospital at any time between their third birthday up to September 2008. A central computerized patient database was used to identify potential subjects. Information in the database included hospitalizations, discharge summaries, outpatient and emergency department records, prescribed medications (including systemic steroids), laboratory tests, and medical imaging.

A list of patients with *International Classification of Diseases, Ninth Revision* codes 493.90 (asthma, unspecified), 493.91 (asthma with status asthmaticus), and/or 493.92 (asthma with acute exacerbation), was obtained. The medical records of 410 patients age 5-18 years with a diagnosis of asthma were reviewed. Study subjects were identified by collecting data on those patients age ≥ 5 years who had developed persistent asthma symptoms by age 3 years. The study protocol was approved by the hospital's Institutional Review Board.

Definition and Characteristics of Children With Asthma with and without Peanut Allergy

Asthma was diagnosed based on the following criteria: (1) symptoms of recurrent (ie, more than 2) episodes of wheezing, cough, shortness of breath, or a combination of these; and (2) documented reversibility with bronchodilators.¹⁵ All of the patients were prescribed inhaled corticosteroids. Patients in the peanut allergy group were diagnosed based on the following criteria: (1) adverse reaction to food;¹⁶ (2) food-specific IgE level $>95\%$ positive predictive value (PPV) to peanut (IgE ≥ 15)

CI	Confidence interval
ICAP RAST	ImmunoCAP radioallergosorbent test
NAEPP	National Asthma Education and Prevention Program
PPV	Positive predictive value
RR	Risk ratio
SPT	Skin prick test

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(Table I; available at www.jpeds.com);^{17,18} (3) and a skin prick test (SPT) 3 mm greater than negative control.¹⁹⁻²¹

Exclusion Criteria

Patients were excluded from the study if: (1) their clinical history did not support the diagnosis of asthma; (2) were not being treated according to National Asthma Education and Prevention Program (NAEPP) guidelines;¹⁵ (3) did not attend >75% of their scheduled outpatient visits, as documented in our computerized outpatient database; (4) were lost to follow-up before September 2008; (5) were followed for <2 years beyond age 3 years; or (6) had cystic fibrosis or immunodeficiency. Patients with peanut allergy were excluded if they had a clinical history suggestive of peanut allergy but did not have a confirmatory ImmunoCAP radioallergosorbent test (ICAP RAST; Pharmacia Diagnostics AB, Portage, Michigan) value >15, the 95% PPV to peanut,¹⁶⁻¹⁸ and an SPT 3 mm greater than negative control.¹⁹⁻²¹ Children diagnosed with egg, milk, and shellfish allergy also fulfilled the strict inclusion criteria for food allergy, including a suggestive clinical history, IgE >95% PPV for each specific food,¹⁶⁻¹⁸ and SPT 3 mm greater than negative control.¹⁹⁻²¹

Predictors

Demographic information was gathered, including race, sex, age (documented in months), history of atopic dermatitis, family history of asthma, gastroesophageal reflux, history of food allergy, history of aeroallergen sensitivity, and passive smoke exposure in the home. Atopic dermatitis was defined according to the diagnostic criteria of Hannifin and Rajka.²² Gastroesophageal reflux was defined based on clinical signs and symptoms as well as diagnostic test results (ie, pH probe, gastric emptying scan, endoscopy with biopsy, or upper gastrointestinal series). Most of the covariates were organized into categorical variables, including sex (male or female), race (Caucasian, African American, or others), and the presence or absence of the following: atopic dermatitis; family history of asthma; gastroesophageal reflux; adenoidal hypertrophy; passive smoke exposure in the home; allergy to milk, egg, or shellfish; and the presence or absence of sensitization to dust mite, aspergillus, alternaria, grass, weed, cat, dog, and tree. Age was recorded at 3 points in time: at the first visit to the food allergy clinic, at the onset of persistent asthma symptoms, and at the end of the review period in September 2008. Chronologic age was included in the regression as a continuous variable.

IgE Level and Skin Testing

The results of previous skin tests and food-specific IgE levels (ICAP RAST) to peanuts, egg, milk, and shellfish were obtained in addition to the results of skin testing to aeroallergens. All SPTs were performed using the GreerPick method (Greer Lab, Lenoir, North Carolina) and allergen extract (provided by Hollister-Stier Laboratories, Spokane, Washington). A SPT was considered positive when the wheal diameter was 3 mm and 50% larger than negative control, and the negative control remained negative.

Table II. Univariate Poisson regression model of the effect of peanut allergy and other factors/covariates on the hospitalization of pediatric patients with asthma in a sample from a large tertiary hospital

Variables	RR	95% CI	P*
Peanut allergy			
No	1.0	Reference	Reference
Yes	2.88	1.86-4.47	<.001
Race			
Caucasian	1.0	Reference	Reference
African American	2.36	1.47-3.81	<.001
Others	3.24	1.69-6.22	<.001
Sex			
Female	1.0	Reference	Reference
Male	1.73	1.03-2.89	.037
Family history of atopy			
No	1.0	Reference	Reference
Yes	2.55	0.35-18.32	.353
Smokers at home			
No	1.0	Reference	Reference
Yes	2.01	1.29-3.13	.002
Atopic dermatitis			
No	1.0	Reference	Reference
Yes	1.56	0.98-2.47	.058
Gastroesophageal reflux			
No	1.0	Reference	Reference
Yes	1.79	1.16-2.78	.009
Adenoidal hypertrophy			
No	1.0	Reference	Reference
Yes	0.47	0.26-0.83	.01
Aspergillus sensitization			
No	1.0	Reference	Reference
Yes	2.93	1.86-4.62	<.001
Alternaria sensitization			
No	1.0	Reference	Reference
Yes	1.43	0.91-2.23	.12
Dust mite sensitization			
No	1.0	Reference	Reference
Yes	2.91	1.78-4.76	<.001
Milk allergy			
No	1.0	Reference	Reference
Yes	0.92	0.53-1.62	.780
Egg allergy			
No	1.0	Reference	Reference
Yes	1.53	0.97-2.41	.064
Shellfish allergy			
No	1.0	Reference	Reference
Yes	3.41	2.09-5.58	<.001
Grass sensitization			
No	1.0	Reference	Reference
Yes	1.57	1.01-2.44	.043
Weed sensitization			
No	1.0	Reference	Reference
Yes	0.73	0.42-1.26	.251
Tree sensitization			
No	1.0	Reference	Reference
Yes	2.02	1.31-3.15	.002
Cat sensitization			
No	1.0	Reference	Reference
Yes	2.61	1.61-4.23	<.001
Dog sensitization			
No	1.0	Reference	Reference
Yes	1.20	0.75-1.92	.453
Age, months	1.01	1.00-1.01	.120

*P < .05.

All children with a history suggestive of allergic rhinitis or atopic dermatitis underwent skin testing with the same panel of aeroallergens (Table II). Data were recorded in a Microsoft

Excel file and later transferred to an SPSS data set (SPSS version 17.0; SPSS Inc, Chicago, Illinois).

Response Variables

The primary outcome for the study was the frequency of systemic steroid use for an asthma exacerbation, and the secondary outcome was the frequency of hospitalizations for an asthma exacerbation. Systemic steroids and hospital admissions for systemic food or other allergic reactions were excluded. All courses of systemic steroids and hospitalizations administered at our institution and at any other outside facility, including a primary care physician, between age 3 years to September 2008 were accounted for and documented in the medical record. Records from outside primary care offices and hospitals were scanned into our system documenting clinical findings, hospital admissions, and medications, including the use of systemic steroids.

Statistical Analysis

Categorical variables were described using frequency and percentages, and continuous variables were summarized using mean and standard deviation. Pearson's χ^2 test and the 2-sample *t* test were used to examine the balance of the distribution of peanut allergic pediatric patients with respect to the distribution of other covariates and 2 outcome variables of asthma morbidity. The associations between the outcome variables and the main predictor variable—peanut allergy—as well as other covariates were examined using univariate Poisson regression model. A multivariate Poisson regression model was used to adjust for the mixing effects of the potential confounding variables on the association between asthma morbidity (outcome variable) and peanut allergy. To enter into the multiple regression model, a variable must have been significant at $P < .10$. Due to their biological/clinical relevance, atopic dermatitis, gastroesophageal reflux, and adenoidal hypertrophy were used in the multivariate Poisson regression without consideration of their significance in the univariate model. An interaction effect was not tested for because of the small sample size. All analyses were 2-tailed at a .05 significance level. All analyses were performed using SPSS version 17.0 (SPSS Inc., Chicago, Illinois).

Results

A total of 160 patients met the inclusion criteria, 46 (28.75%) with asthma and peanut allergy and 114 (71.25%) with asthma but without peanut allergy. The patients ranged in age from 5 years to 18 years. **Table I** presents the distribution of covariates between the children with peanut allergy and those without peanut allergy. The 2 groups differed significantly ($P < .05$) in terms of mean chronologic age, mean age at onset of persistent asthma symptoms, and mean age at first presentation to the allergy practice. The 2 groups were similar in terms of sex and race distributions ($P = .06$ and .768, respectively). The cohort was predominately male (65%) and Caucasian (68.8%).

There were significant differences in the distribution of atopic features between the children with and without peanut allergy. Those with peanut allergy were more likely to have atopic dermatitis ($P = .001$) and to be sensitive to specific aeroallergens, including dust mite ($P = .012$), aspergillus ($P = .032$), grass ($P = .013$), weed ($P = .001$), and cat ($P = .022$). They also were more likely to have another food allergy, including milk ($P = .011$), egg ($P < .001$), and shellfish ($P = .048$). Differences between the 2 groups in the distributions of gastroesophageal reflux ($P = .103$), adenoidal hypertrophy ($P = .099$), and smoke exposure at home ($P = .299$) were not statistically significant (**Table I**).

Table II illustrates the association between the frequency of hospitalization and peanut allergy, as well as other covariates. There was a statistically significant association between the frequency of hospitalization and peanut allergy (risk ratio [RR] = 2.88; 95% confidence interval [CI] = 1.86–4.47). Race, sex, smoke exposure at home, gastroesophageal reflux, adenoidal hypertrophy, shellfish allergy, and sensitization to aspergillus, dust mite, grass, tree, and cat were significantly ($P < .05$) related to the frequency of hospitalization, but family history of atopy, age, milk allergy, and sensitization to alternaria, weed, and dog were not related ($P > .05$). Although not quite reaching the level of significance, atopic dermatitis ($P = .058$) and egg allergy ($P = .064$) also had a considerable association with the frequency of pediatric hospitalizations. Compared with Caucasian subjects, African-American subjects were 2.36-times more likely and Asian, Hispanic, and American Indian subjects were 3.24-times more likely to have an increased rate of hospitalization.

Table III illustrates the association between the frequency of systemic steroid use and peanut allergy, as well as other covariates. There was a statistically significant association between the frequency of systemic steroid use and peanut allergy (RR = 1.72; 95% CI = 1.41–2.10). Children with peanut allergy were 1.72-times more likely to require more courses of oral steroids compared with those without peanut allergy. African-American race; age; sex; smoke exposure at home; gastroesophageal reflux; sensitivity to aspergillus, dust mite, tree, grass, and cat; and allergy to egg and shellfish also were significantly ($P < .05$) related to the frequency of hospitalization, but family history of atopy, atopic dermatitis, adenoidal hypertrophy, milk allergy, and sensitization to alternaria, weed, and dog were not ($P > .05$). Compared with Caucasian subjects, African-American subjects were 2.21-times more likely to receive more courses of systemic steroids.

After adjusting for all other significant covariates, children with peanut allergy were 1.59 times more likely to have an increased rate of systemic steroid use for asthma beyond age 3 years ($P < .001$; 95% CI = 1.24–2.04). Other covariates significant in the regression included African-American race ($P < .001$), other minority ($P = .015$), male sex ($P < .001$), atopic dermatitis ($P = .005$), gastroesophageal reflux ($P < .001$), sensitization to aspergillus ($P < .001$), and shellfish allergy ($P = .032$). Smoke exposure at home, adenoidal hypertrophy, age, egg allergy, and sensitization to dust, grass, tree, and cat were not significant predictors (data available on request).

Table III. Univariate Poisson regression model of the effect of peanut allergy and other factors/covariates on the use of number of oral steroids of pediatric patients with asthma in a sample from a large tertiary hospital

Variables	RR	95% CI	P*
Peanut allergy			
No	1.0	Reference	Reference
Yes	1.72	1.41-2.10	<.001
Race			
Caucasian	1.0	Reference	Reference
African American	2.21	1.80-2.72	<.001
Others	1.42	0.96-2.10	.76
Sex			
Female	1.0	Reference	Reference
Male	2.28	1.78-2.94	<.001
Family history of atopy			
No	1.0	Reference	Reference
Yes	1.78	0.84-3.76	.13
Smokers at home			
No	1.0	Reference	Reference
Yes	1.74	1.42-2.13	<.001
Atopic dermatitis			
No	1.0	Reference	Reference
Yes	1.04	0.85-1.26	.74
Gastroesophageal reflux			
No	1.0	Reference	Reference
Yes	1.55	1.27-1.89	<.001
Adenoidal hypertrophy			
No	1.0	Reference	Reference
Yes	1.01	0.82-1.26	.897
Aspergillus sensitization			
No	1.0	Reference	Reference
Yes	1.71	1.36-2.15	<.001
Alternaria sensitization			
No	1.0	Reference	Reference
Yes	1.18	0.96-1.45	.111
Dust mite sensitization			
No	1.0	Reference	Reference
Yes	1.44	1.18-1.75	<.001
Milk allergy			
No	1.0	Reference	Reference
Yes	1.21	0.95-1.52	.118
Egg allergy			
No	1.0	Reference	Reference
Yes	1.25	1.01-1.54	.039
Shellfish allergy			
No	1.0	Reference	Reference
Yes	2.48	1.95-3.16	<.001
Grass sensitization			
No	1.0	Reference	Reference
Yes	1.52	1.25-1.86	<.001
Weed sensitization			
No	1.0	Reference	Reference
Yes	1.14	0.92-1.42	.235
Tree sensitization			
No	1.0	Reference	Reference
Yes	1.50	1.22-1.84	<.001
Cat sensitization			
No	1.0	Reference	Reference
Yes	1.53	1.25-1.87	<.001
Dog sensitization			
No	1.0	Reference	Reference
Yes	1.08	0.87-1.34	.503
Age, months	1.003	1.00-1.01	.048

*P < .05.

After adjusting for all other significant covariates, children with peanut allergy were 2.32-times more likely to have an increased hospitalization rate for asthma beyond age 3 years ($P = .003$; 95% CI = 1.33-4.05). Other significant factors in

the regression included African-American race ($P = .024$), other minority ($P < .001$), male sex ($P = .03$), gastroesophageal reflux ($P = .003$), egg allergy ($P = .029$), shellfish allergy ($P = .025$), and sensitization to aspergillus ($P < .001$). Smoke exposure at home, atopic dermatitis, and sensitization to dust mite, grass, tree, and cat were not significant covariates (data available on request).

Discussion

Coexistent peanut allergy and asthma is a growing problem in school-age children, with the prevalence of both disorders on the rise. We found an association between peanut allergy and asthma morbidity after controlling for known risk factors for asthma morbidity.²³⁻²⁵ We chose the use of systemic steroids as our primary outcome because previous studies have accepted worsening asthma symptoms requiring the intervention of systemic steroids by a medical professional as a practical working definition for an asthma exacerbation.²³ We accounted for many of the known risk factors associated with asthma morbidity, including sex; age; race; family history of atopy; exposure to smoke in the home; atopic dermatitis; gastroesophageal reflux; adenoidal hypertrophy; milk, egg, or shellfish allergy; and sensitization to grass, tree, cat, or aspergillus.²³⁻²⁶ Children with peanut allergy were 2.32-times more likely to have an increased rate of hospitalization ($P = .03$) and 1.59 times more likely to require more courses of oral steroids ($P < .001$). Many of the young school-age children included in our study presented with peanut allergy before tree nut exposure.²⁷⁻³⁰ We advised these high-risk children to avoid tree nuts, because peanut and tree nut allergies are highly coreactive (20%-50%).²⁷⁻³⁰ We did not include tree nut allergy, because many of the subjects did not fulfill the strict clinical criteria for our study. Shellfish allergy also was associated with asthma morbidity, which may reflect the persistence of both peanut and shellfish allergies throughout school age and adolescence.

Previously known risk factors for asthma morbidity²³⁻²⁶ also were associated with increased asthma and reflect the recent trends in racial disparities.³¹ Sensitization to aspergillus ($P < .001$ for both), gastroesophageal reflux ($P = .003$ and $< .001$, respectively), and African-American race ($P < .001$ and $.015$) were associated with increased hospitalization rate and increased systemic steroid use. Surprisingly, we did not find a significant association between asthma morbidity and the following variables: atopic dermatitis, egg allergy, and milk allergy. This may reflect the early anticipatory guidance given to these children.

The strengths of our study include the provision of treatment for persistent asthma according to NAEPP guidelines,¹⁵ good compliance and access to health care, and the strict inclusion criteria for food allergy and persistent asthma. All children with food allergy had a suggestive clinical history supported by an IgE level >95% PPV¹⁶⁻¹⁸ and a SPT 3 mm greater than negative control.¹⁹⁻²¹ This distinguishes our study from those that have examined only the relationship between food sensitivity and asthma morbidity. Although

a double-blinded food challenge is the gold standard for diagnosing peanut allergy, this type of study would be unethical in such a high-risk patient population. All children with persistent asthma continued to wheeze and to need treatment with inhaled corticosteroids according to the NAEPP guidelines beyond age 5, which helps exclude children with bronchiolitis.

Our study has some limitations. The study's retrospective nature makes it susceptible to bias, including different thresholds for prescribing systemic steroids among primary care or emergency physicians. The study also excluded children who may have had peanut allergy but did not fulfill our strict inclusion criteria,¹⁶⁻¹⁸ and it did not include a significant number of adolescents. Thus, our findings may not apply to all children with peanut allergy presenting in the outpatient practice.

In conclusion, our findings suggest that peanut allergy may be a risk factor for increased asthma morbidity in school-age children. Early prevention and intervention can improve the quality of care. Instructing parents about tangible risk factors for an exacerbation may help improve compliance and reduce the need for oral steroids. Further studies are needed to validate our findings in large populations using prospective methods. ■

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Table I. Demographic and other study variable characterizations by group

Variable	Peanut allergy	No peanut allergy	Total	P value
Patients, n (%)	46 (28.75)	114 (71.25)	160 (100)	
Age, months				
Mean (SD)	111.33 (33.33)	126.26 (37.3)	121.97 (36.7)	.019
Median	104.5	124	115	
Minimum-maximum	67-194	59-222	59-222	
Mean age of first allergy visit, months	40.28	51.41	48.21	.018
Mean age of persistent asthma symptoms, months	27.78	41.68	37.68	.002
Sex				.062
Male	35 (76.1)	69 (60.5)	104 (65.0)	
Female	11 (23.9)	45 (39.5)	56 (35.0)	
Race				.768
Caucasian	30 (65.2)	80 (70.2)	110 (68.8)	
African American	13 (28.3)	26 (22.8)	39 (24.4)	
Other (Asian, Hispanic, Indian)	3 (6.5)	8 (7.0)	11 (6.8)	
Presence of atopic features				
Atopic dermatitis				<.001
Yes	37 (80.4)	50 (43.9)	87 (54.4)	
No	9 (19.6)	64 (56.1)	73 (45.6)	
Family history				.149*
Yes	46 (100.0)	109 (95.6)	155 (96.9)	
No	0 (0.0)	5 (4.4)	5 (3.1)	
Sensitization to aeroallergens				
Dust mite				.012
Yes	29 (63.0)	47 (41.2)	76 (47.5)	
No	17 (37.0)	67 (58.8)	84 (52.5)	
Aspergillus				.032
Yes	12 (26.1)	14 (12.3)	26 (16.3)	
No	34 (73.9)	100 (87.7)	134 (83.7)	
Alternaria				.104
Yes	19 (41.3)	32 (28.1)	51 (31.9)	
No	27 (58.7)	82 (71.9)	109 (68.1)	
Grass				.013
Yes	26 (56.5)	40 (35.1)	66 (41.2)	
No	20 (43.5)	74 (64.9)	94 (58.8)	
Weed				.001
Yes	20 (43.5)	20 (18.4)	41 (25.6)	
No	26 (56.5)	93 (81.6)	119 (74.4)	
Cat				.022
Yes	29 (63.0)	49 (43.0)	78 (48.8)	
No	17 (37.0)	65 (57.0)	82 (51.2)	
Dog				.597
Yes	14 (30.4)	30 (26.3)	44 (27.5)	
No	32 (69.6)	84 (73.7)	116 (72.5)	
Tree allergy				<.001
Yes	21 (45.7)	25 (21.9)	46 (28.8)	
No	25 (54.3)	89 (78.1)	114 (71.2)	
Risk factors for asthma morbidity				
Gastroesophageal reflux				.103
Yes	13 (28.3)	48 (42.1)	61 (38.1)	
No	33 (71.7)	66 (57.9)	99 (61.9)	
Adenoidal hypertrophy				.099
Yes	10 (21.7)	40 (35.1)	50 (31.2)	
No	36 (78.3)	74 (64.9)	110 (68.8)	
Smoke exposure at home				.299
Yes	15 (32.6)	28 (24.6)	43 (26.9)	
No	31 (67.4)	86 (75.4)	117 (73.1)	
Associated food allergies				
Milk				.011
Yes	15 (32.6)	17 (14.9)	32 (20.0)	
No	31 (67.4)	97 (85.1)	128 (80.0)	
Egg				<.001
Yes	26 (56.5)	19 (16.7)	45 (28.1)	
No	20 (43.5)	95 (83.3)	115 (71.9)	
Shellfish				.048
Yes	8 (17.4)	8 (7.0)	16 (10.0)	
No	38 (82.6)	106 (93.0)	144 (90.0)	

P values are generated from either the χ^2 statistic or a 2-sample *t* test.

*P value from Fisher's exact test.