

Food allergy as a risk factor for life-threatening asthma in childhood: A case-controlled study

Graham Roberts, MSc, MRCPCH,^a Neeta Patel, MRCPCH,^a
Francesca Levi-Schaffer, PhD,^b Parviz Habibi, PhD, FRCPCH,^a and
Gideon Lack, FRCPCH^a London, United Kingdom, and Jerusalem, Israel

Background: No objective clinical risk factors exist for pediatric life-threatening asthma.

Objectives: In this study, we address whether persistent food allergy and degree of atopy are risk factors for life-threatening asthma.

Methods: By use of a case-controlled design, children (1-16 years) ventilated for an exacerbation of asthma were enrolled. Each case was matched by sex, age, and ethnicity, with 2 controls who had attended with a non-life-threatening exacerbation. All subjects were assessed by means of a questionnaire, spirometry, and skin prick or RAST testing. The data were analyzed by conditional logistic regression.

Results: Nineteen cases and 38 controls were enrolled. Compared with controls, cases were found to have the following risk factors: food allergy (odds ratio, 8.58; 95% CI, 1.85-39.71), multiple allergic diagnoses (4.42; 1.17-16.71), early onset of asthma (6.48; 1.36-30.85), and frequent admissions (14.2; 1.77-113.59). After regression analysis, only frequent admission with asthma (9.85; 1.04-93.27) and food allergy (5.89; 1.06-32.61) were independently associated with life-threatening asthma. Half the cases had food allergy compared with only 10% of controls.

Conclusion: This study demonstrates that poorly controlled asthma and food allergy are significant risk factors for life-threatening asthma. More intensive management of this high-risk group of children might help to reduce future morbidity and mortality. (*J Allergy Clin Immunol* 2003;112:168-74.)

Key words: Food allergy, life-threatening asthma, lung function

Exacerbations of asthma are the most common reason for hospital admission in the pediatric age group. Despite significant strides in therapy seen over the past 2 decades, there has been no reduction in mortality, with approximately 50 childhood deaths caused by asthma occurring every year in the United Kingdom¹ and more

Abbreviations used

OR: Odds ratio

PICU: Pediatric intensive care unit

SPT: Skin prick test

than 200 deaths every year in the United States.² A number of risk factors have been highlighted for life-threatening asthma, including markers of asthma severity, the frequent use of bronchodilators, poor compliance, low socioeconomic status, psychological problems, exposure to cigarette smoke, and previous life-threatening exacerbations.³⁻⁷ However, no objective risk factors have been found for life-threatening asthma in the pediatric age group, and half of those who experience a life-threatening exacerbation have previously had only mild or moderately severe asthma.⁸

The role of allergic inflammation in pediatric asthma has now been clearly established.⁹ However, few studies have looked at atopy as a risk factor for life-threatening asthma in childhood. In eczema, increasing sensitization to aeroallergens correlates with disease severity.¹⁰ A direct correlation exists between the number of allergens a child is sensitized to and the degree of bronchial hyper-reactivity¹¹ and impaired lung function.¹² In adults, sensitization to the mold *Alternaria* has been found to be a risk factor in life-threatening asthma.^{13,14} This might provide an explanation for the seasonal variation in asthma mortality.^{15,16} One adult case-controlled study has shown that a history of a previous asthma attack being precipitated by food is the most significant risk factor for life-threatening asthma (odds ratio [OR], 5.1).¹⁷ However, the authors did not define food allergy by history, skin testing, specific IgE testing, or challenge, because they were primarily interested in assessing frequent β -agonist use as a risk factor. Sensitization to inhalants and foods has also been shown to be a risk factor for brittle asthma in adults.¹⁸ It has been estimated that 4% to 8% of children and teenagers with asthma have coexistent food allergy, making food allergy a potentially significant risk factor for life-threatening asthma.^{19,20} In this study, we have investigated whether the degree of atopy or the presence of food allergy is associated with an increased risk for life-threatening asthma. These questions are addressed using a case-controlled design with controls matched for age, sex, and ethnicity.

From the Department of ^aPaediatric Allergy and Clinical Immunology, St Mary's Hospital, London; and the Department of ^bPharmacology, The Hebrew University of Jerusalem.

Dr Lack is supported by the Aimwell Foundation.

Received for publication January 29, 2003; revised March 23, 2003; accepted for publication March 28, 2003.

Reprint requests: Gideon Lack, FRCPCH, Paediatric Allergy and Clinical Immunology, Ground Floor Salton House, St Mary's Hospital, Praed Street, London W2 1NY, United Kingdom.

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0091-6749/2003 \$30.00 + 0

doi:10.1067/mai.2003.1569

METHODS

Setting

St Mary's Hospital is situated in a socioeconomically and ethnically diverse area of London. The pediatric intensive care unit (PICU) at St Mary's Hospital serves as a tertiary referral center for secondary pediatric units in Northwest London. The unit admits approximately 400 children and teenagers annually.

Design

A case-controlled design was used to address the study questions. For each case, 2 controls were recruited. The local research ethics committee approved the study. Informed consent was obtained from parents and, when appropriate, the subjects.

Subject definition and recruitment

For this study, life-threatening asthma was defined as an exacerbation requiring ventilation. Cases were recruited from consecutive children ventilated for an acute exacerbation of asthma within the PICU at St Mary's between January 1995 and December 2000. They were 1 to 16 years of age. All had a history of chronic asthma symptoms as described by the International Study of Asthma and Allergies in Childhood questionnaire.²¹ Children with transient symptoms in association with viral infections without symptoms between viral infections were excluded. We chose to exclude children younger than 1 year of age, because most have wheeze in association with bronchiolitic illnesses caused by viruses such as respiratory syncytial virus. All subjects had airway secretions sent for immunofluorescence for respiratory viruses to further exclude the possibility of including subjects with bronchiolitis and viral pneumonia. All subjects were asked about exposure to relevant food allergens in the 24 hours before exacerbation of their asthma. Children who were initially seen with a history of bronchospasm in association with exposure to a known or suspected allergen were excluded. Subjects were also asked about other symptoms of anaphylaxis (urticaria, angioedema, oral symptoms, abdominal pain) in the 24 hours before their exacerbation. If a history of type I hypersensitivity symptoms was elicited in the 24 hours before exacerbation, subjects were also excluded. Subjects were excluded before their allergic status was known to the investigators. Their parents were approached by letter and reviewed at St Mary's.

Controls were selected for each case from all the patients attending St Mary's emergency department with an exacerbation of asthma in the same year that the case had been admitted for intensive care. Hospital admission was not an inclusion requirement for the control subjects. Controls were matched to the cases by sex, ethnicity, and age; these are all known to affect atopy.²² Parents of the next best-matched child were approached by letter until 2 controls were enrolled for each case. The same inclusion and exclusion criteria were used as for the cases, except that they did not require ventilation to manage the exacerbations. Controls were assessed in the same way as the cases.

Data collection and outcome measures

Cases and controls were assessed between January 2000 and June 2001. All were interviewed by use of a structured questionnaire with predefined answers to each question. The questionnaire included questions on demographic details, usual asthma precipitants, previous admissions, and symptoms and treatment in the 3 months before the index presentation.

Food allergy was diagnosed on the basis of a history of immediate hypersensitivity symptoms or signs (eg, urticaria, angioedema, wheeze, rhinitis, conjunctivitis) within an hour of exposure to a food and a positive skin prick test (SPT) (≥ 3 mm with a negative

saline control and a histamine control [10 mg/mL] of ≥ 3 mm) or serum-specific IgE (CAP RAST class ≥ 2). Subjective symptoms alone were insufficient to establish a diagnosis. A food challenge was performed in cases of clinical doubt. In addition, subjects with serum-specific IgE or SPT results greater than the threshold level that predicts clinical allergy with a 95% positive predictive value^{23,24} were identified. The presence of eczema and allergic rhinitis was determined by use of questions from the International Study of Asthma and Allergies in Childhood questionnaire.²¹

The degree of allergy was defined as the number of allergic diagnoses (asthma, eczema, rhinoconjunctivitis, food allergy, or pet allergy), and the degree of atopy was defined as the total number of positive SPTs to inhalant and food allergens.

The FEV₁ and forced vital capacity were recorded before and 15 minutes after twice their usual dose of bronchodilator (MasterScreen, Jaeger, Germany). All spirometry was performed when the subjects had recovered clinically from their exacerbation.

Allergy testing

SPT was performed as previously described²⁵ to the following: grass pollens, tree pollens, *Alternaria*, *Aspergillus*, *Cladosporium*, dog, cat, *Dermatophagoides pteronyssinus*, *Dermatophagoides fariniae*, saline negative control, and histamine positive control (ALK, Horsholm, Denmark). Cases and controls were all asked whether they "had had any reaction to a food?" They were also asked whether there was any food that they disliked or avoided. They were all asked specifically about allergic reactions to cow's milk, soy, hen's egg, fish, peanuts, or tree nuts. When a subject had never eaten 1 of these commonly allergenic foods regularly in normal portions or had experienced typical allergic symptoms to any food, SPT was also performed for that food. A wheal diameter of at least 3 mm was taken to be positive if there was a positive histamine (≥ 3 mm) and negative saline result. When the subject had taken any antihistamine medication in the previous 5 days or the specific allergen solution was not available, blood was taken for specific IgE testing through use of the Pharmacia CAP system (Pharmacia, Uppsala, Sweden).

Data analysis

A univariate analysis was initially performed to look at the effect of the degree of atopy and food allergy on the risk of life-threatening asthma. Each variable was transformed into a binary parameter. A model was then constructed using any risk factor with a univariate *P* value of .1 or less. The results are presented as OR with 95% CI. Conditional logistic regression was used to perform most of the analyses. A paired analysis was used to compare continuous variables such as the age of onset of asthma or lung function. In this analysis, the average for each pair of controls was compared with the matched case using a paired *t* test or Wilcoxon sign-rank test as appropriate. A 5% level of significance was used for all analyses. All analyses were completed through use of STATA (version 6; Stata Corporation, College Station, Tex).

RESULTS

Subjects

Between January 1995 and December 2000, 26 children aged 1 to 16 years with a diagnosis of asthma were ventilated in the PICU. Of these, 1 did not wish to participate in the study, and 6 were excluded because on further evaluation they only had viral-induced wheeze. We therefore included 19 cases. We approached 76 subjects, 38 (50.0%) of which were enrolled as controls; 32 (42.1%) did not respond to the letter of invitation; 4

TABLE I. Details of cases and controls

	Cases	Control
Male (%)	13 (68.4)	26 (68.4)
Ethnicity (%)		
Caucasian	11 (57.9)	22 (57.9)
Black	3 (15.8)	5 (13.2)
Indian subcontinent	5 (26.3)	11 (28.9)
Average age at exacerbation (mo)	118 (range, 22-192)	110 (range, 24-192)
Parental occupation (%)		
Professional or managerial	6 (35.3)	6 (17.1)
Skilled	7 (41.2)	8 (22.9)
Semi-skilled/unskilled	1 (5.9)	10 (28.6)
No income	3 (17.7)	11 (31.4)
Mean interval between exacerbation and assessment for study (mo)	20.3 (range, 9-45)	18.0 (range, 5-41)

No significant differences were seen between cases and controls for sex, ethnicity, age, parental occupation, or interval between exacerbation and review.

TABLE II. Asthma history

Item	Cases: n = 19 (%)	Controls: n = 38 (%)	Odds ratio	95% CI	P value
Asthma developed in first year of life	14 (73.7)	11 (34.4)	6.48	1.36-30.85	.019
No. with frequent (4 or more) previous admissions with asthma	11 (57.9)	7 (18.4)	14.20	1.77-113.59	.012
No. previously ventilated for asthma	3 (15.8)	0 (0.0)	—	—	—
3 months before presentation					
Wheeze more than 3 times/wk	10 (52.6)	8 (21.0)	12.56	1.53-103.13	.019
Use of reliever more than twice/wk	12 (64.2)	14 (63.2)	2.45	0.78-7.76	.127
Daily use of inhaled steroids	13 (68.4)	9 (23.7)	6.15	1.70-22.30	.006
400 µg or more daily beclomethasone equivalent	9 (47.4)	8 (21.1)	3.791	0.980-14.674	.054
Frequent wheeze or cough with exercise	11 (57.9)	14 (36.8)	3.17	0.80-12.64	.102
Long-acting bronchodilator	7 (36.8)	4 (10.5)	4.22	1.08-16.54	.039

Univariate analysis examining the association between the asthma history and life-threatening asthma. Associations described in terms of the odds ratio, 95% CI, and associated *P* value.

(5.3%) only had viral-induced wheeze; and 2 (2.6%) did not wish to participate. No potential cases or controls were excluded as possible cases of anaphylaxis. Twenty-four (63.2%) of the controls were admitted to the hospital with their index exacerbations. Demographic details of the cases and controls enrolled in the study are listed in Table I. None of the subjects had a history consistent with type 1 hypersensitivity symptoms in the 24 hours before their exacerbation. The time between the index asthma exacerbation and date of assessment was identical for cases and controls (Table I).

Asthma history

The onset of asthma in the first year of life was significantly associated with life-threatening asthma (OR, 6.48; 95% CI, 1.36-30.85; *P* = .019); cases had asthma develop at 10.8 compared with 28.8 months for the controls (*P* < .001). Furthermore, cases had been more frequently admitted with asthma than controls (*P* = .012). Three of the cases (15.8%) had been previously ventilated for asthma compared with none of the controls. Cases were significantly more likely to have indicators of severe asthma (Table II) and were receiving significantly larger doses of inhaled steroid (geometric mean, 337 versus 197 µg/day of the controls; *P* = .043). There were no differences in exposure to damp, mold, dust, or smoke among the cases and controls (data not shown).

Allergen sensitization

Cases and controls were sensitized to an average of 3.9 and 1.9 allergens, respectively (Wilcoxon signed rank test; *P* = .031). The presence of sensitization to 4 or more allergens was found to be a risk factor for life-threatening asthma (*P* = .041). In the univariate analysis, only sensitization to dog or foods was significantly associated with life-threatening asthma (Table III).

Food allergy

Food allergy was found to be a significant risk factor for life-threatening asthma; 52.6% (*n* = 10) of cases had food allergy compared with only 10.5% (*n* = 4) of the controls (*P* = .006) (Table IV). All these subjects had a convincing clinical history of reaction to food, supported by evidence of specific IgE or SPT. Nine subjects (6 cases, 3 controls) had a skin prick wheal diameter or a Pharmacia CAP result greater than the threshold level that predicts clinical allergy to the common allergenic foods with a 95% positive predictive value.^{23,24} These subjects were allergic to peanuts (6 cases, 1 control), walnuts (1 control), and shrimp (1 control).

Other allergic diagnoses

The average number of allergic diagnoses for cases and controls were 3.58 and 2.73, respectively (*P* = .042).

TABLE III. Allergen sensitization

Sensitization to	Cases (%)	Controls (%)	Odds ratio	95% CI	P value
Grass pollen	10 (52.6)	12 (31.6)	4.00	0.80-20.02	.092
Tree pollen	4 (21.1)	5 (13.2)	2.17	0.36-12.94	.395
<i>Alternaria</i>	1 (5.6)	1 (2.6)	—	—	—
<i>Cladosporium</i>	1 (5.6)	1 (2.6)	2.00	0.13-31.98	.624
<i>Aspergillus</i>	1 (5.6)	2 (5.3)	1.00	0.053-18.92	1.000
Dog	7 (38.9)	4 (10.5)	6.31	1.29-30.74	.023
Cat	7 (41.2)	8 (21.1)	2.56	0.72-9.12	.147
<i>D pteronyssinus</i>	10 (52.6)	19 (50.0)	1.12	0.35-3.57	.845
<i>D fariniae</i>	5 (27.8)	9 (23.7)	1.12	0.36-3.49	.845
Any aeroallergens	14 (73.7)	24 (63.2)	1.90	0.46-7.85	.377
Any food allergens	10 (52.6)	5 (13.2)	6.90	1.45-32.78	.015
4 or more allergens	8 (42.1)	6 (15.8)	5.26	1.07-25.86	.041

Univariate analysis examining the association between the sensitization to different allergens and life-threatening asthma. Associations described in terms of the odds ratio, 95% CI, and associated *P* value.

TABLE IV. Food allergy and other allergic diagnoses

Item	Cases (%)	Controls (%)	Odds ratio	95% CI	P value
Food allergy	10 (52.6)	4 (10.5)	8.58	1.85-39.71	.006
Rhinitis	12 (66.7)	23 (60.5)	1.72	0.55-5.41	.350
Eczema	14 (73.7)	29 (76.3)	0.86	0.23-3.19	.823
Pet allergy	9 (50.0)	8 (21.6)	2.82	0.97-8.19	.056
More than 3 allergic diagnoses	10 (52.6)	8 (21.1)	4.42	1.17-16.71	.028

Univariate analysis examining the association between different forms of atopic disease and life-threatening asthma. Associations described in terms of the odds ratio, 95% CI, and associated *P* value. Cases were allergic to peanuts or tree nuts (*n* = 7), egg (*n* = 3), fish or shellfish (*n* = 2), banana (*n* = 1), milk (*n* = 1), sesame (*n* = 1), beans (*n* = 2), lentils (*n* = 1), or (kiwi (*n* = 1). Controls were allergic to peanuts or tree nuts (*n* = 3), egg (*n* = 1), fish or shellfish (*n* = 2), or sesame (*n* = 1).

(Table IV). The presence of more than 3 allergic diagnoses was found to be a risk factor for life-threatening asthma (*P* = .028).

Lung function at follow-up

At follow-up, the percent predicted FEV₁ for cases was significantly reduced (81.0%; 95% CI, 68.6%-93.4%) despite therapy with oral and inhaled steroids. This contrasted with the normal values seen in the control group (93.4%; 95% CI, 84.1%-102.7%). Despite a lower FEV₁ before bronchodilator therapy, there was a trend toward reduced bronchodilator reversibility in cases (3.58%; 95% CI, 1.67%-8.66%) compared with controls (6.63%; 95% CI, 0.0%-18.22%). The postbronchodilator FEV₁ was significantly lower (*P* < .05) in the cases (83.6%; 95% CI, 69.8%-97.4%) than the controls (97.1%; 95% CI, 90.0%-104.3%), reflecting both the lower initial FEV₁ and the reduced bronchodilator response.

Multivariate analysis

The parameters considered for inclusion in the multivariate analysis are listed in Table V. With multivariate, conditional logistic regression analysis, the best model for predicting a life-threatening exacerbation was found to include frequent previous admissions and clinical food allergy. The adjusted ORs (95% CI) for these were 9.85 (1.04-93.27) and 5.89 (1.06-32.61), respectively. Each of these 2 factors explain a quarter of the variance seen in

this model (adjusted *R*²). None of the other factors were found to be significantly associated with life-threatening asthma in this model.

DISCUSSION

Several risk factors for life-threatening asthma have been described, but there are no objective clinical markers in the pediatric population. This study compared 19 children ventilated for an exacerbation of asthma, in a general PICU, with 38 matched controls with milder exacerbations. Subjects and controls were matched for age, sex, and ethnicity and were also identical in terms of socioeconomic factors. This design thereby eliminated 4 important confounders. The factors that were found to be associated with life-threatening exacerbations were developing asthma in the first year of life, frequent wheeze, daily use of inhaled steroids or a long-acting bronchodilator, frequent previous admissions, food allergy, sensitization to dog, and multiple atopic diagnoses or sensitization to multiple allergens (Table V). After logistic regression, only food allergy and frequent admissions to the hospital with asthma were found to be independent risk factors for life-threatening asthma.

This study demonstrates that increasing atopy is a risk factor for life-threatening asthma; this is primarily reflected in sensitization to food rather than inhalant allergens (Table III). Food allergy is an independent risk factor for life-threatening asthma with an adjusted OR of

TABLE V. Risk factors for life-threatening asthma

Parameter	Univariate analysis		Multivariate analysis	
	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value
4 or more previous admissions with asthma	14.20 (1.77-113.59)	.012	9.85 (1.04-93.27)	.046
Frequent episodes of wheeze	12.56 (1.53-103.13)	.019	—	—
Food allergy	8.58 (1.85-39.71)	.006	5.89 (1.06-32.61)	.042
Developing asthma in the first year of life	6.48 (1.36-30.85)	.019	—	—
Sensitization to dogs	6.31 (1.29-30.74)	.023	—	—
Daily use of inhaled steroids	6.15 (1.70-22.30)	.006	—	—
Sensitization to 4 or more allergens	5.26 (1.07-25.86)	.041	—	—
More than 3 allergic diagnoses	4.42 (1.17-16.71)	.028	—	—
Sensitization to grass pollens	4.00 (0.80-20.02)	.092	—	—

The following parameters were excluded from the multivariate analysis, because they were closely correlated with other parameters that were stronger risk factors for life-threatening asthma: a daily dose of 400 µg or more of inhaled steroids (beclomethasone equivalent), use of a long-acting bronchodilator, grass pollen as a precipitant for asthma, pet allergy, and sensitization to grasses or foods.

5.89 (95% CI, 1.06-32.61) (Table V). Similar results have been found in the 1 adult study of life-threatening asthma in which the investigators found that a history of food-provoking asthma was the strongest risk factor (OR, 5.1).¹⁷ The association with sensitization to food has been shown in a study of subjects with brittle asthma.¹⁸ Coexisting food allergy and asthma is a significant problem in the pediatric population. More than a third of children with food allergies have asthma, and up to 8% of asthmatic children have a food allergy.^{19,20,26} An opportunity exists to target these groups with more intensive treatment with a possibility to reduce their risk for a life-threatening event. In 1 cohort of peanut-allergic children, 4 of 46 subjects died of asthma over the 2- to 14-year follow-up period.²⁷ This is an elevated mortality rate for asthma and much higher than one would expect in any high-risk population. These results suggest that subjects with food allergy might have a greater risk of dying from asthma than from anaphylaxis.

A number of explanations exist for the association between food allergy and life-threatening asthma. The first possibility is that anaphylaxis is misdiagnosed as asthma. This is plausible, because food-induced bronchospasm is often seen in anaphylaxis, and there is often a delay between allergen exposure and the development of respiratory symptoms.^{28,29} Studies have documented life-threatening asthma symptoms within an hour of the onset of the exacerbation,⁸ a short time frame that is consistent with anaphylaxis. In this study, no subject had a history that indicated anaphylaxis as a cause of their asthma exacerbation. However, we cannot rule out the possibility of exposure to trace contaminants of food allergen causing a severe early-phase or late-phase response consisting primarily of bronchospasm. The second explanation for the observed association is that food allergy might be a marker for severe asthma. The mean age of children in this study was 9.4 years (range, 1.8-16.0 years). Most food allergies, particularly to egg and milk, are outgrown well before the age of 5 years. The persistence of food allergy in this group of children suggests an increased atopic state. A skewed host immune response, manifested by an increase in pro-inflammatory allergic

cytokines (eg, IL-13), could account for persistent IgE responses, bronchial inflammation, and airway fibrosis.³⁰⁻³² Third, chronic inhalation of aerosolized food might worsen asthma in subjects with food allergy; both early-phase and late-phase responses have been demonstrated.³³ During the Barcelona asthma epidemic, there was a substantial increase in hospital admissions and deaths caused by asthma after exposure to aerosolized soy flour in subjects with specific IgE to soy.³⁴

Previous studies have shown that markers of severe asthma, such as frequent symptoms, were associated with life-threatening asthma.³⁵⁻³⁹ Our study confirms this association and additionally finds that children with life-threatening asthma had an earlier onset of asthma and partially irreversible obstructive airway disease (Table II). It is possible that the early presentation of asthma in a group of children with heightened atopic responses might lead to chronic airway inflammation, poor asthma control, airway remodeling, and abnormal lung function.⁴⁰ One case had evidence of subepithelial membrane thickening and airway remodeling on an endobronchial biopsy. Conceivably, exposure to food or inhalant allergens could lead to a life-threatening event in a child with severe airway inflammation and poor bronchodilator responses.

There are 2 potential limitations to this study: possible selection bias and the diagnosis of food allergy in the absence of double-blinded, placebo-controlled food challenges. It is unlikely that recruiting consecutive cases from a general PICU has resulted in a biased case mix toward allergy. All cases were referred for intensive care from other hospitals in Northwest London. The rate of food allergy in the control group was 10.5%, this is slightly higher than the rate of food allergy reported in a series of subjects with asthma (4%-8%).^{19,20} This might have actually led us to underestimate the association between life-threatening asthma and food allergy. Only 1 of the subjects required a food challenge for diagnostic uncertainty. In this case the history was not as clinically convincing for immediate hypersensitivity to food. He therefore had an open graded challenge to wheat and reacted with objective signs of immediate hypersensitiv-

ity. The other subjects were not challenged, because they all had convincing histories of food allergy corroborated by positive SPTs, serum-specific IgE, or both. All these children had histories of reacting to the food allergen within an hour of exposure on at least 2 occasions. All were carefully avoiding the food in their diet. Given the clinical conviction that these patients were food allergic, the history of a life-threatening exacerbation of asthma and the ongoing poor lung function in most cases, it was considered that it would be unethical to undertake randomized double-blind food challenges in this group of subjects. In addition, 9 of these subjects (6 cases and 3 controls) had an SPT or specific IgE levels result, which has been shown to predict clinical allergy with a positive predictive value of at least 95%.^{23,24} When the analysis was repeated with only these 9 subjects being labeled as food allergic, food allergy was still found to be a significant risk factor for life-threatening asthma (OR, 5.31; 95% CI, 1.05-26.77).

This study demonstrates that frequent previous admissions and clinical food allergy are independent risk factors for life-threatening asthma. This is the first time that clinical food allergy has been shown to be statistically associated with life-threatening asthma in children. It would be important to replicate these findings, particularly in adult patients with asthma. Our findings have important implications for children with coexistent asthma and food allergies. Food allergy is seen in the first few years of life and is potentially a useful marker that would allow increased supervision of this group of high-risk children with asthma to reduce subsequent asthma morbidity and mortality. Excellent control of coexistent asthma is an integral part of the management of food allergy in children. Similarly, the accurate diagnosis and management of food allergy must comprise an essential part of the management of childhood asthma.

We acknowledge a number of our colleagues whose help has been invaluable in completing this study: Catriona Hurley for performing many of the skin prick tests and spirometric assessments; Paul Bassett and Ian Grace for their advice on the statistical analysis of the data; and Norman Noah for all his constructive and useful criticism of the project. Last, we thank all the subjects and their families, without whom this study would have been impossible to undertake.

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