

Letter to the Editor

Underdiagnosis of allergic rhinitis in underserved children*To the Editor:*

Allergic rhinitis (AR) is a common asthma comorbidity,¹ and untreated rhinitis is often a contributory factor to asthma morbidity.² AR can significantly affect the quality of life for affected children.³ Although Puerto Rican (PR) children are disproportionately affected by asthma,⁴ access to specialized care is limited. PR allergists currently provide less than 4% of asthma care.⁵ We hypothesized that AR may be underdiagnosed in PR children and that identifying major risk factors would allow us to devise a diagnostic approach that could easily be used by primary care providers in underserved areas.

Children aged 6 to 14 years in San Juan, Puerto Rico, were chosen from randomly selected households using a multistage probability sample design, resulting in 678 children being chosen. Only those with nonmissing data on allergy skin testing (n = 547) were included. Study protocol included questionnaires, allergy skin testing, and blood sample collection. Written parental consent and assent from participating children were obtained. The study was approved by the institutional review boards of the

University of Puerto Rico (San Jan, Puerto Rico), Brigham and Women's Hospital (Boston, Mass), and the University of Pittsburgh (Pittsburgh, Pa).

AR was defined by (1) rhinitis symptoms (in the last 12 months) apart from colds and (2) skin test reactivity (STR) to 1 or more allergens, consistent with Allergic Rhinitis and its Impact on Asthma 2008 guidelines⁶ (henceforth referred to as STR-positive AR). Physician-diagnosed AR (PD-AR) was defined by (1) rhinitis symptoms (as above) and (2) physician's diagnosis (see this article's [Methods](#) section in the Online Repository at www.jacionline.org).

A comparison of the characteristics of participants who were (n = 547) and were not (n = 131) included is given in [Table E1](#) in the Online Repository at www.jacionline.org. The characteristics of participants who did and did not have STR-positive AR are listed in [Table E2](#) in the Online Repository at www.jacionline.org. Asthma was present in 288 (52.7%) participants. STR-positive AR was present in 192 (66.7%) and 73 (28.2%) children with and without asthma, respectively. Variables significantly associated with STR-positive AR included having symptoms triggered by house dust, pollen, mold, and cat; history of an eczematous rash; serum total IgE level; having positive IgE to dust

TABLE I. Sensitivity, specificity, PPV, negative predictive value (NPV),* and area under the receiver operating characteristics curve (AUC) for PD-AR and various predictive models

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	AUC (95% CI)
PD-AR—Model A					
Asthma	22.9	94.8	89.8	38.1	0.59 (0.55-0.63)
No asthma	12.3	98.9	81.8	74.2	0.56 (0.52-0.59)
All	20.0	97.5	88.3	56.5	0.59 (0.56-0.61)
Rhinitis symptoms only—Model B					
Asthma	100.0	54.2	81.4	100.0	†
No asthma	100.0	86.6	74.5	100.0	†
All	100.0	75.5	79.3	100.0	†
Rhinitis symptoms and dust mite-IgE—Model C					
Asthma	72.6	75.5	85.7	57.7	0.74 (0.69-0.79)
No asthma	67.1	95.1	83.9	88.3	0.81 (0.75-0.87)
All	71.2	88.5	85.3	76.6	0.80 (0.76-0.83)
Rhinitis symptoms and (dust mite-IgE and/or cockroach IgE)—Model D					
Asthma	75.8	73.4	85.2	60.0	0.75 (0.69-0.80)
No asthma	68.6	94.5	82.8	88.7	0.82 (0.76-0.87)
All	73.9	87.4	84.6	78.1	0.81 (0.77-0.84)
Rhinitis symptoms and (dust mite-IgE and/or house dust trigger)—Model E					
Asthma	96.8	53.2	80.7	89.3	0.75 (0.70-0.80)
No asthma	88.6	89.1	75.6	95.3	0.89 (0.84-0.93)
All	94.6	76.9	79.4	93.8	0.86 (0.83-0.89)
Rhinitis and total IgE ≥ 100 kU/L—Model F					
Asthma	83.3	70.8	85.1	68.0	0.77 (0.72-0.82)
No asthma	76.7	91.4	77.8	90.9	0.84 (0.79-0.89)
All	81.5	84.4	83.1	82.9	0.83 (0.80-0.86)
Rhinitis and (total IgE ≥ 100 kU/L and/or house dust trigger)—Model G					
Asthma	97.9	55.2	81.4	93.0	0.77 (0.71-0.82)
No asthma	91.8	87.6	74.4	96.5	0.90 (0.86-0.94)
All	96.2	76.6	79.4	95.6	0.86 (0.84-0.89)
Rhinitis and house dust trigger—Model H					
Asthma	90.1	57.3	80.8	74.3	0.74 (0.68-0.79)
No asthma	82.2	90.3	76.9	92.8	0.86 (0.81-0.91)
All	87.9	79.1	79.8	87.5	0.84 (0.80-0.87)

*Gold standard = STR-positive AR.

†AUC unable to be calculated.

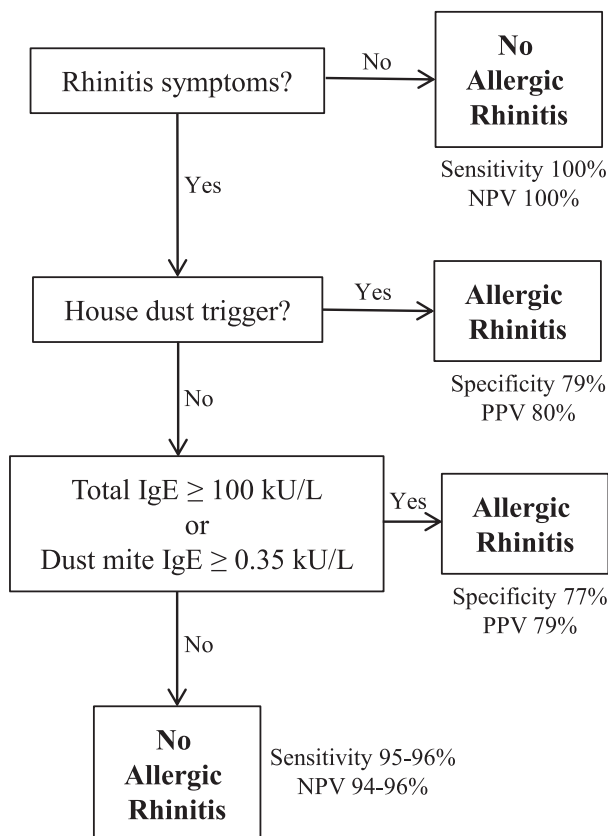


FIG 1. Schema for the diagnosis of AR. Sensitivity, specificity, PPV, and negative predictive value (NPV) presented are for both children with and without asthma.

mite or cockroach; and STR to dust mite, *Blomia tropicalis*, cockroach, Alternaria, mouse, mixed trees, Mugwort sage, and ragweed. Parental history of AR was significantly associated with STR-positive AR only in children without asthma.

Results of the unadjusted and adjusted analysis of STR-positive AR are presented in Table E3 in the Online Repository at www.jacionline.org. After adjustment for age and sex (Table E3), private/employer-based health insurance, symptoms triggered by house dust, eczematous rash, and either total IgE level or positive IgE to dust mite were significantly associated with increased odds of STR-positive AR in children with asthma. Asthmatic children who shared their bedroom had reduced odds of STR-positive AR. In the multivariate analysis in nonasthmatic children (Table E3), having symptoms triggered by house dust, having symptoms triggered by pollen, and positive IgE to dust mite were significantly associated with STR-positive AR.

Compared with STR-positive AR, PD-AR was reported in lower proportions of children with (49 or 17.0%) and without (11 or 4.3%) asthma, respectively. Physicians diagnosed AR correctly in only 44 (15.3%) and 9 (3.5%) children with and without asthma, respectively. Table E4 in the Online Repository at www.jacionline.org shows the multivariate analysis of PD-AR. Having private/employer-based health insurance, parental history of AR, symptoms triggered by cats, a dog at home, and a positive IgE to *Derma-tophagoides Pteronyssinus* were significantly associated with increased odds of PD-AR in asthmatic children. In nonasthmatic children, parental history of AR and having symptoms triggered by pollen were significantly associated with PD-AR.

Table 1 presents the sensitivity, specificity, positive predictive value (PPV), negative predictive value, and area under the curve of various predictive models for STR-positive AR. PD-AR (model A) had excellent specificity and PPV but poor sensitivity. Rhinitis symptoms (model B) had 100% sensitivity (by definition) and acceptable PPV, but reduced specificity, particularly in asthmatic children. By adding positive dust mite-IgE (model C), specificity and PPV were improved, though sensitivity decreased, particularly in children without asthma. A combination of rhinitis symptoms, positive IgE to dust mite, and having symptoms triggered by house dust (model E) markedly increased sensitivity (88.6% to 96.8%), while maintaining a high PPV (75.6% to 80.7%). Substituting total IgE ≥ 100 kU/L for positive dust mite-IgE (model G) yielded similar results (and AUC).

Diagnosing AR requires a careful history and physical examination, as well as targeted testing for allergic sensitization. Because skin testing or a large panel of specific IgE is usually not easily accessible to primary care physicians in underserved areas, we formulated an efficient algorithm to accurately diagnose AR (Fig 1). We recommend that physicians inquire about (1) recent naso-ocular symptoms apart from colds, and (if yes), (2) whether house dust triggers such symptoms. If both answers are affirmative, AR can be diagnosed. If the answer is no to house dust triggers, then physicians should measure IgE to dust mite or total IgE. AR can be diagnosed if dust mite-IgE level is more than 0.35 kU/L or total IgE is 100 kU/L or more. This approach was highly sensitive and maintained a high PPV. Because of potential detrimental effects from undertreating AR on asthma control, and relatively low risks of overtreatment (due to the safety of most AR medications), this would be particularly attractive in asthmatic children.

This is the first study of the prevalence and risk factors for AR using objective markers of allergic sensitization (instead of relying on self-report) in Puerto Ricans. Limitations to our findings include the cross-sectional design, which preclude assessment of temporal relationships. We focused on PR children because of their disproportionate burden of asthma and atopy,⁴ but our findings and proposed diagnostic approach to AR should be generalizable to other underserved children. In a previous study, the total IgE level was often elevated in ethnic minority women in Boston, most of whom were not diagnosed with allergic diseases.⁷ We previously showed that allergic sensitization is underdiagnosed in Puerto Rican and Black children with asthma living in Hartford, Connecticut.⁸ Moreover, we found that AR is often not diagnosed in children with asthma in Costa Rica, a Latin American country with universal health care.⁹

In summary, AR is markedly underdiagnosed in PR children. Physicians missed more than 75% and more than 85% of AR in children with and without asthma, respectively. Physicians could accurately diagnose AR by inquiring about the presence of rhinitis symptoms, triggers, and measuring dust mite-IgE or total IgE level.

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Tammy S. Jacobs, MD, MA^a
Erick Forno, MD, MPH^a
John M. Brehm, MD, MPH^a
Edna Acosta-Pérez, PhD^b
Yueh-Ying Han, PhD, MS^a
Joshua Blatter, MD, MPH^a
Angel Colón-Semidey, MD^b

María Alvarez, MD^b
Gloria Canino, PhD^b
Juan C. Celedón, MD, DrPH^d

From ^athe Division of Pediatric Pulmonary Medicine, Allergy and Immunology, Children's Hospital of Pittsburgh of UPMC, University of Pittsburgh, Pittsburgh, Pa; and ^bthe Behavioral Sciences Research Institute, University of Puerto Rico, San Juan, Puerto Rico. E-mail: juan.celedon@chp.edu.

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METHODS

Study population

From March 2009 to June 2010, children in San Juan (Puerto Rico) were chosen from randomly selected households using a scheme similar to that of a previous study.^{E1} In brief, households in San Juan were selected by a multistage probability sample design. Primary sampling units were randomly selected neighborhood clusters based on the 2000 US census, and secondary sampling units were randomly selected households within each primary sampling unit. A household was eligible if 1 or more resident was a child aged 6 to 14 years. In households with more than 1 eligible child, 1 child was randomly selected for screening.^{E1} On the basis of the sampling design, 7073 households were selected and 6401 (90.5%) were contacted. Of these 6401 households, 1111 had 1 or more children who were within the age range of the study and had 4 Puerto Rican grandparents. In an effort to reach a target sample size of approximately 700 children, we attempted to enroll a random sample ($n = 783$) of these 1111 children. Parents of 105 of these 783 eligible households refused to participate or could not be reached. There were no significant differences in age, sex, or area of residence between eligible children who did ($n = 678$, ~87%) and did not ($n = 105$, ~13%) agree to participate. Because we focused on AR, only those with nonmissing data on allergy skin testing ($n = 547$) were included in this analysis.

Study procedures

Study participants completed a protocol that included questionnaires, allergy skin testing, and collection of blood samples (for DNA extraction, and measurement of serum total and allergen-specific IgE level, and eosinophil count). The child's parents completed 2 questionnaires used in the Genetics of Asthma in Costa Rica Study.^{E2} These questionnaires were used to obtain information about the child's general and respiratory health, family history, sociodemographic characteristics, *in utero* smoke exposure, family history, household characteristics, use of asthma medications, and asthma morbidity (including information on severe asthma exacerbations^{E3} and core measures of asthma exacerbations^{E4}).

Subjects were genotyped using the HumanOmni2.5 BeadChip (Illumina, Inc, San Diego, Calif). As previously described,^{E5} 85,059 single-nucleotide polymorphisms were selected to estimate the racial ancestry of study participants using the Local Ancestry in admixed Populations method, using estimated ancestral proportions in Puerto Ricans and external reference panels.

STR to aeroallergens, histamine, and diluents was assessed using a Multi Test device (Lincoln Diagnostics, Decatur, Ill) on the skin of the forearm (in a site free of eczema). Aeroallergens tested included house dust mites (*D pteronyssinus* and *D farinae*), *B tropicalis*, German cockroach (*B germanica*), mouse pelt, dog dander, cat dander, mold mix, *Alternaria tenuis*, mixed tree pollen, mixed grass pollen, mugwort sage, and ragweed (Alk-Abello, Round Rock, Texas). A skin test result was considered positive if the maximum wheal diameter exceeded the diluent wheal diameter by 3 mm or more. Serum levels of total IgE and IgE specific to common allergens (dust mite [Der p 1], cockroach [Bla g 2], mouse urinary protein [Mus m 1], dog dander [Can f 1], and cat dander [Fel d 1]) were measured using the UniCAP 100 system (Pharmacia & Upjohn, Kalamazoo, Mich). For each allergen, an IgE level of 0.35 kU/L or more was considered positive.

Written parental consent was obtained for participating children, from whom written assent was also obtained. The study was approved by the institutional review boards of the University of Puerto Rico (San Juan, Puerto Rico), Brigham and Women's Hospital (Boston, Mass), and the University of Pittsburgh (Pittsburgh, Pa).

Outcome and case definitions

A child was considered to have current symptoms suggestive of AR if there were affirmative answers to 2 questions (taken from the International Study of Asthma and Allergies in Childhood questionnaire^{E6}): (1) Has your child ever had hay fever or a runny or stuffy nose accompanied by sneezing and itching at

a time when he or she did not have a cold or flu? and (2) Has your child had these symptoms in the last 12 months? AR was defined by (1) current symptoms suggestive of AR and (2) STR to 1 or more allergens. This definition, henceforth referred to as STR-positive AR, is consistent with Allergic Rhinitis and its Impact on Asthma 2008 guidelines.^{E7} PD-AR was defined by (1) current symptoms suggestive of AR (as above) and (2) a physician's diagnosis. Asthma was defined as physician-diagnosed asthma and wheeze in the previous year.

Statistical analysis

All analyses were conducted separately in children with and without asthma. Bivariate analyses of the covariates of interest and either STR-positive AR or PD-AR were conducted using Fisher exact tests for categorical variables and 2-sample 2-sided *t* tests for continuous variables. Logistic regression was used for multivariable analyses. Because of their known relation to AR, all models included age, sex, type of health insurance (private/employer-based vs others), and parental (paternal or maternal) history of AR. Other covariates, including STR or a positive IgE to the allergens tested (see Table E2), were also included in the initial multivariate models if $P \leq .20$ in bivariate analyses. These additional covariates remained in the final models if they were associated with AR at $P < .05$ or if they changed the parameter estimate (β) by 10% or more.

The covariates used in Table E2 included baseline demographic characteristics, symptom triggers, asthma medications and exacerbations, atopic history, family history, home environment, laboratory values for total and allergen-specific IgE, and STR to common allergens (see Table E2). Parental education was dichotomized into at least 1 parent completed high school versus none. Household income was dichotomized into above or below \$15,000/year, as that was the approximate median income in Puerto Rico during the study. Eczematous rash referred to ever having a prolonged, itchy, scaly, or weepy skin rash. For children with asthma, more than or equal to 1 severe exacerbation in the preceding year was defined by any systemic steroids, emergency department or urgent care visit, or hospitalization in the last year.

We then assessed the predictive characteristics of PD-AR for the diagnosis of STR-positive AR, before and after adding covariates associated with AR in our multivariate analysis (see above). In particular, we examined the sensitivity, specificity, and PPV of PD-AR in various predictive models. All analyses were done using Stata/SE 12.1 for Windows (College Station, Tex).

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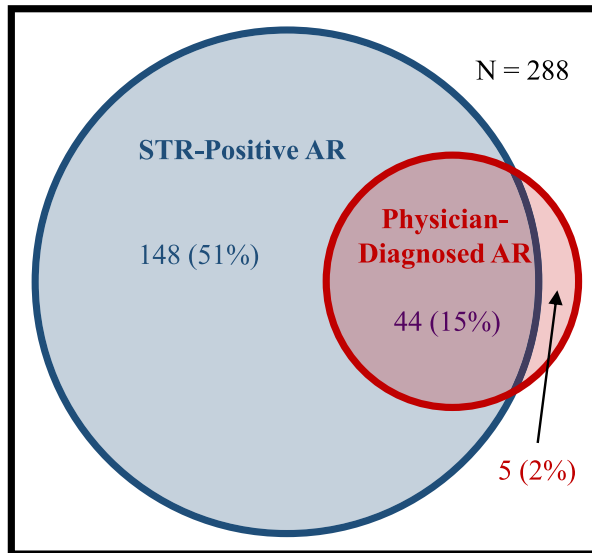
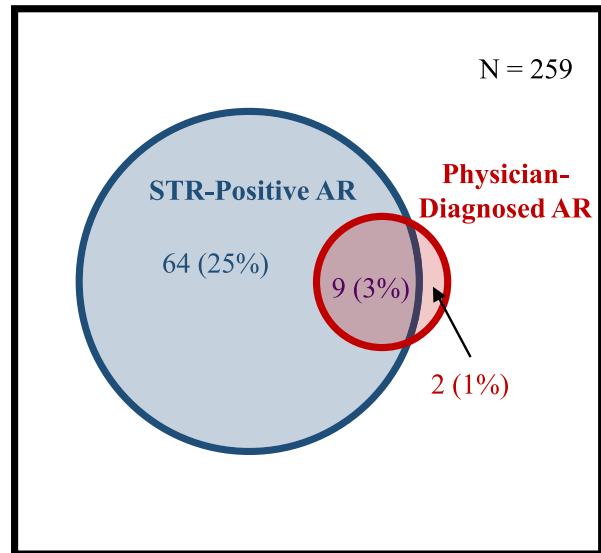
A Children with Asthma**B Children without Asthma**

FIG E1. Venn diagram of STR-positive AR and PD-AR in children with (A) and without (B) asthma.

TABLE E1. Comparison of children who were and were not included in this analysis

Characteristic	Asthma (n = 351)		No asthma (n = 327)	
	Included participants (n = 288)	Excluded participants (n = 63)	Included participants (n = 259)	Excluded participants (n = 68)
Baseline characteristics				
Female sex	120 (41.7)	30 (47.6)	138 (53.3)	30 (44.1)
Age (y)	10.1 (9.8-10.4)	10.0 (9.3-10.6)	10.5 (10.2-10.9)	10.1 (9.4-10.8)
African ancestry (%)	25.5 (24.0-26.9)	22.2 (17.2-27.1)	25.3 (23.7-26.9)	21.3 (17.2-25.4)
Parental education (>high school)	233 (80.9)	55 (87.3)	206 (79.5)	57 (83.8)
Higher household income (>\$15K/y)	90 (32.0)	29 (46.0)*	93 (37.2)	23 (37.1)
Private/employer-based health insurance	89 (30.9)	23 (36.5)	97 (37.5)	25 (36.8)
Symptom triggers				
Pollen	145 (51.2)	26 (42.6)	38 (14.7)	9 (13.2)
House dust	238 (82.6)	51 (81.0)	114 (44.0)	28 (41.2)
Mold	57 (22.0)	16 (23.5)	199 (69.8)	44 (69.8)
Dog	77 (26.7)	21 (33.3)	10 (3.9)	4 (5.9)
Cat	92 (32.1)	15 (24.2)	18 (7.0)	1 (1.5)
Asthma				
Inhaled steroids in previous 6 mo	93 (32.3)	21 (33.3)		
Leukotriene inhibitor in previous 6 mo	70 (24.3)	16 (25.4)		
≥1 severe exacerbation in the last year†	152 (52.8)	31 (49.2)		
Atopic history				
Eczematous rash, ever	110 (38.2)	26 (41.3)	68 (26.3)	12 (17.7)
Family history				
Parental history of AR	69 (24.1)	11 (17.5)	36 (13.9)	11 (16.9)
Parental history of asthma	193 (67.3)	39 (61.9)	81 (31.3)	22 (33.3)
Parental history of eczema	11 (3.9)	3 (4.8)	3 (1.2)	1 (1.5)
Home environment				
Number of older siblings	6 (2.2)	1 (1.7)	1 (0.4)	1 (1.6)
Shared bedroom	179 (62.2)	38 (60.3)	119 (46.0)	34 (50.0)
History of daycare	68 (23.9)	19 (30.2)	50 (19.5)	12 (18.5)
Signs of mold/mildew	130 (45.1)	25 (39.7)	91 (35.3)	21 (30.9)
Dog at home	155 (53.8)	38 (61.3)	129 (50.2)	37 (54.4)
Cat at home	22 (7.6)	10 (16.1)*	37 (14.5)	9 (13.2)
Laboratory findings				
Log total IgE (kU/L)	2.5 (2.4-2.5)	2.5 (2.2-2.8)	2.2 (2.1-2.3)	2.2 (2.0-2.5)
Any positive IgE	197 (69.4)	13 (61.9)	124 (49.0)	19 (55.9)
Positive IgE to				
Dust mite	184 (64.8)	13 (61.9)	108 (42.7)	19 (55.9)
Cockroach	113 (39.8)	11 (52.4)	66 (26.1)	12 (35.3)
Mouse	11 (3.9)	1 (4.8)	3 (1.2)	2 (5.9)
Dog	60 (21.1)	4 (19.1)	36 (14.2)	9 (26.5)
Cat	31 (11.1)	1 (4.8)	16 (6.3)	3 (9.1)

Note. Values are presented as number (%) or mean (95% CI).

** $P < .005$ for Fisher exact test for categorical variables, and 2-sample (2-sided) t test with unequal variances for continuous variables.

*Difference between subjects with and without STR-positive AR on univariate analysis at $P < .05$.

†Any systemic steroids, emergency department or urgent care visit, or hospitalization in the last year.

TABLE E2. Characteristics of study participants (n = 547)

Characteristic	Asthma (n = 288)		No asthma (n = 259)	
	No STR-positive AR (n = 96)	STR-positive AR (n = 192)	No STR-positive AR (n = 186)	STR-positive AR (n = 73)
Baseline characteristics				
Female sex	39 (40.6)	81 (42.2)	95 (51.1)	43 (58.9)
Age (y)	9.8 (9.3-10.3)	10.2 (9.8-10.6)	10.3 (10.0-10.7)	11.1 (10.4-11.7)
African ancestry (%)	25.6 (23.1-28.2)	25.4 (23.8-27.1)	25.0 (23.2-26.9)	24.7 (21.9-27.5)
Parental education (>high school)	74 (77.1)	159 (82.8)	146 (78.5)	60 (82.2)
Higher household income (>\$15K/y)	25 (26.6)	65 (34.8)	61 (34.5)	32 (43.8)
Private/employer-based health insurance	18 (18.8)	71 (37.0)*	66 (35.5)	31 (42.5)
Symptoms triggers				
Pollen	36 (37.9)	109 (58.0)*	13 (7.0)	25 (34.3)*
House dust	65 (67.7)	173 (90.1)*	54 (29.0)	60 (82.2)*
Mold	55 (57.3)	144 (76.2)*	22 (11.8)	35 (48.0)*
Dog	19 (19.8)	58 (30.2)	3 (1.6)	7 (9.7)†
Cat	22 (22.9)	70 (36.7)†	7 (3.8)	11 (15.1)*
Asthma				
Inhaled steroids in previous 6 mo	25 (26.0)	68 (35.4)		
Leukotriene inhibitor in previous 6 mo	18 (18.8)	52 (27.1)	1 (0.54)	1 (1.4)
≥1 severe exacerbation in the last year‡	46 (47.9)	106 (55.2)		
Atopic history				
Eczematous rash, ever	24 (25.0)	86 (44.8)*	41 (22.0)	27 (37.0)†
Family history				
Parental history of AR	23 (24.2)	46 (24.1)	19 (10.2)	17 (23.3)†
Parental history of asthma	64 (67.4)	129 (67.2)	54 (29.0)	27 (37.0)
Parental history of eczema	2 (2.1)	9 (4.7)	2 (1.1)	1 (1.4)
Home environment				
Number of older siblings	1.9 (1.6-2.3)	1.6 (1.3-1.8)	1.7 (1.4-1.9)	1.6 (1.2-1.9)
Shared bedroom	75 (78.1)	104 (54.2)*	90 (48.4)	29 (39.7)
History of daycare	17 (17.7)	51 (27.0)	33 (17.8)	17 (23.6)
Signs of mold/mildew	34 (35.4)	96 (50.0)†	59 (31.9)	32 (43.8)
Dog at home	46 (47.9)	109 (56.8)	94 (51.1)	35 (48.0)
Cat at home	8 (8.3)	14 (7.3)	26 (14.2)	11 (15.1)
Laboratory findings				
Log total IgE (kU/L)	2.2 (2.1-2.4)	2.6 (2.5-2.7)*	2.1 (2.0-2.2)	2.4 (2.2-2.6)*
Any positive IgE	52 (55.3)	145 (76.3)*	75 (41.0)	49 (70.0)*
Positive IgE to				
Dust mite	46 (48.9)	138 (72.6)*	61 (33.3)	47 (67.1)*
Cockroach	28 (29.8)	85 (44.7)†	37 (20.2)	29 (41.4)*
Mouse	4 (4.3)	7 (3.7)	1 (0.6)	2 (2.9)
Dog	11 (11.7)	49 (25.8)†	21 (11.5)	15 (21.4)
Cat	7 (7.6)	24 (12.8)	7 (3.8)	9 (12.9)†
Skin test reactivity				
≥1 allergen	41 (42.7)	192 (100.0)*	111 (59.7)	73 (100.0)*
Dust mite mix	20 (20.8)	85 (44.3)*	31 (16.8)	32 (44.4)*
<i>B. tropicalis</i>	26 (28.0)	138 (73.0)*	59 (32.4)	55 (76.4)*
Cockroach	12 (12.6)	90 (47.4)*	39 (21.3)	25 (35.2)†
Mouse	14 (14.6)	51 (26.6)†	27 (14.5)	24 (32.9)*
Dog	9 (9.4)	67 (34.9)*	35 (18.8)	17 (23.3)
Cat	17 (17.7)	82 (42.7)*	54 (29.0)	30 (41.1)
Mold mix	4 (4.2)	26 (13.5)†	22 (11.8)	12 (16.4)
<i>Alternaria</i>	10 (10.4)	54 (28.1)*	35 (18.8)	25 (34.3)†
Mixed trees	19 (19.8)	59 (30.7)†	44 (23.7)	27 (37.0)†
Mixed grasses	9 (10.5)	40 (22.4)†	23 (14.4)	12 (19.1)
Mugwort sage	7 (8.1)	31 (17.8)†	15 (9.4)	14 (23.0)†
Ragweed	19 (19.8)	103 (53.7)*	56 (30.1)	38 (52.1)*

Note. Values are presented as number (%) or mean (95% CI).

* $P < .005$ for Fisher exact test for categorical variables, and 2-sample (2-sided) t test with unequal variances for continuous variables.†Difference between subjects with and without STR-positive AR on univariate analysis at $P < .05$.

‡Any systemic steroids, emergency department or urgent care visit, or hospitalization in the last year.

TABLE E3. Multivariate analysis of STR-AR in children with and without asthma

Asthma	Unadjusted		Adjusted model 1*		Adjusted model 2*	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Private/employer-based health insurance	2.5 (1.4-4.6)	.002	3.3 (1.6-6.7)	.001	2.7 (1.4-5.5)	.004
Parental history of allergic rhinitis	1.0 (0.6-1.8)	.981	0.6 (0.3-1.3)	.204	0.6 (0.3-1.3)	.201
Symptoms triggered by house dust	4.3 (2.3-8.2)	<.001	4.4 (2.1-9.0)	<.001	4.4 (2.2-9.1)	<.001
Shared bedroom	0.3 (0.2-0.6)	<.001	0.3 (0.2-0.6)	<.001	0.3 (0.2-0.6)	.001
Skin rash suggestive of eczema, ever	2.4 (1.4-4.2)	.001	2.1 (1.1-3.8)	.023	2.2 (1.2-4.0)	.013
Total IgE†	2.1 (1.4-3.1)	<.001	2.3 (1.5-3.5)	<.001		
Positive IgE to <i>D pteronyssinus</i>	2.8 (1.7-4.6)	<.001			2.3 (1.3-4.1)	.004

No asthma	Unadjusted		Adjusted model 1*		Adjusted model 2*	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Private/employer-based health insurance	1.3 (0.8-2.3)	.297	1.2 (0.6-2.3)	.665	1.1 (0.5-2.2)	.774
Parental history of allergic rhinitis	2.7 (1.3-5.5)	.008	1.1 (0.4-2.6)	.908	1.1 (0.4-2.8)	.816
Symptoms triggered by house dust	11.3 (5.7-22.2)	<.001	8.9 (4.2-18.6)	<.001	8.4 (4.0-17.7)	<.001
Symptoms triggered by pollen	6.9 (3.3-14.6)	<.001	2.9 (1.2-6.8)	.015	2.7 (1.2-6.4)	.021
Total IgE†	2.1 (1.4-3.3)	.001	1.6 (0.9-2.8)	.080		
Positive IgE to <i>D pteronyssinus</i>	4.1 (2.3-7.3)	<.001			3.0 (1.5-6.1)	.002

OR, Odds ratio.

*Because total IgE and a positive IgE to dust mite were highly collinear, 2 multivariate models are presented—one including total IgE and no specific IgE (model 1) and another including positive IgE to dust mite but not total IgE (model 2). Adjusted for age, sex, and other variables in the column for the model.

†Presented per each log unit increment.

TABLE E4. Multivariate analysis of PD-AR for children with and without asthma

Asthma	Unadjusted		Adjusted*	
	OR (95% CI)	P value	OR (95% CI)	P value
Private/employer-based health insurance	5.4 (2.8-10.3)	<.001	4.5 (2.1-9.8)	<.001
Parental history of AR	8.0 (4.1-15.7)	<.001	7.5 (3.5-16.2)	<.001
Dog at home	2.2 (1.1-4.3)	.018	2.9 (1.2-6.8)	.014
Symptoms triggered by cat	2.9 (1.6-5.5)	.001	3.9 (1.7-8.9)	.001
Positive IgE to <i>D pteronyssinus</i>	3.3 (1.5-7.3)	.004	3.3 (1.3-8.6)	.015

No asthma	Unadjusted		Adjusted*	
	OR (95% CI)	P value	OR (95% CI)	P value
Private/employer-based health insurance	3.1 (0.9-10.8)	.080	2.2 (0.5-10.0)	.310
Parental history of AR	36.8 (7.6-179.4)	<.001	27.4 (5.0-150.1)	<.001
Symptoms triggered by pollen	12.3 (3.4-44.3)	<.001	8.2 (1.9-35.8)	.005

OR, Odds ratio.

*Adjusted for age, sex, and other variables in the model.