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Impact of Aeroallergen Sensitization on Asthma Control in African-American Teens with Persistent Asthma

Allison J. Burbank, MD^{a,*}, Shannon C. Grabich, PhD^{b,*}, Krista Todorich, MD^c, Marcia Frye, MD^d, Ceila Loughlin, MD^e, Kelly Duncan, PhD^f, Carole Robinette, MS^g, Katherine Mills, BA^h, David B. Peden, MD, MSⁱ, David Diaz-Sanchez, PhD^j, Michelle L. Hernandez, MD^k

^aCenter for Environmental Medicine, Asthma, and Lung Biology, University of North Carolina at Chapel Hill, 104 Mason Farm Road, CB #7310, Chapel Hill, NC 27599-7310, USA.

^bU.S. Environmental Protection Agency, National Health and Environmental Effects Research Laboratory, Chapel Hill, NC, USA.

^cAllergy and Asthma Associates of Michigan, 3600 W. 13 Mile Rd, Royal Oak, MI 48073.

^dUniversity of North Carolina at Chapel Hill, Department of Pediatrics, Division of Pediatric Pulmonology.

^eUniversity of North Carolina at Chapel Hill, Department of Pediatrics, Division of Pediatric Pulmonology.

^fMerck Pharmaceuticals

^gCenter for Environmental Medicine, Asthma, and Lung Biology, University of North Carolina at Chapel Hill

^hCenter for Environmental Medicine, Asthma, and Lung Biology, University of North Carolina at Chapel Hill

ⁱCenter for Environmental Medicine, Asthma, and Lung Biology, University of North Carolina at Chapel Hill

^jEnvironmental Public Health Division, National Health and Environmental Effects Research Laboratory, US Environmental Protection Agency, Research Triangle Park, NC, USA.

^kCenter for Environmental Medicine, Asthma, and Lung Biology, University of North Carolina at Chapel Hill

Address for correspondence: Allison J Burbank, MD, Department of Pediatrics, Division of Allergy, Immunology, & Rheumatology, UNC School of Medicine, 104 Mason Farm Road, CB #7310, Chapel Hill, NC 27599-7310, 919-843-5383 (office), 919-966-9863 (fax), allison.burbank@unchealth.unc.edu.

^{*}both authors contributed equally

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Asthma control depends on many factors, including access and adherence to medications and exposure to asthma triggers. Studies of inner-city children with asthma show that exacerbations decrease when household allergen exposure is reduced⁽¹⁾ or with aggressive NAEPP guidelines-based therapy⁽²⁾. African-Americans are at higher risk of poorly controlled asthma than other races⁽³⁾ and experience greater asthma-related morbidity and mortality^(4, 5). We conducted an observational study of African-American adolescents between August 2013 and October 2014 examining factors that influence asthma control. We hypothesized that allergic status influences asthma control despite guidelines-based therapy. In this report, we present the data from a descriptive analysis of 25 African-American teens age 12-17 years with moderate or severe persistent asthma followed in a subspecialty clinic, comparing teens with poorly controlled asthma with age-matched teens with well controlled asthma.

Asthma control was assessed at a baseline visit and five follow-up visits over a two-month period. Study physicians determined asthma control using NHLBI guidelines for management of asthma focusing on degree of impairment by assessing symptom frequency, short-acting β -agonist use, nighttime awakenings, and activity limitation⁽⁶⁾. Spirometry was performed at each visit, and FEV₁ was used in the asthma control assessment. At follow up visits, asthma control determination was based on impairment as well as healthcare utilization and oral corticosteroid (OCS) requirement for asthma since the last study visit.

Asthma medication use was optimized among those classified as poorly controlled at baseline, and inhaler with spacer technique was reviewed. Allergic profile was defined using peripheral blood eosinophil count and allergy skin prick testing (SPT) to indoor and outdoor allergens common to the southeastern U.S. A positive SPT was interpreted as a 3 mm wheal greater than the saline control.

Peripheral blood eosinophil count was modeled as a continuous variable. Allergy exposure was modeled using SPT data as a dichotomous variable for each individual allergen as well as a composite "multiple allergens" variable, as number of allergens continuously, and as ≥ 4 allergens to < 4 allergens. Logistic regression models were run to evaluate each allergic sensitization to asthma control. The asthma control variable was defined as those whose asthma became well controlled by visit 2 and remained controlled versus those whose asthma control was erratic or remained poorly controlled. We secondarily assessed if OCS were prescribed during the study for asthma exacerbation. Results from logistic regression models yielded odds ratios and 95% confidence intervals. Each allergic profile variable and control outcome were assessed for differences by age, BMI, sex, and tobacco smoke exposure to evaluate potential confounding by demographic variables. All analyses were conducted in SAS 9.4 (Cary, NC).

Of the 25 subjects with moderate to severe persistent asthma, the mean age was 14 years, mean BMI was 24.2 kg/m², 36% were female, 14 had well controlled asthma at the baseline

visit, and 11 had poorly controlled asthma. Of the poorly controlled subjects, one became well controlled by visit 2, while 10 remained poorly controlled. Eight of these 10 teens required OCS during the study period for asthma exacerbation. Age, sex, BMI, and tobacco smoke exposure did not vary between those whose asthma was well controlled versus poorly controlled at baseline. Nearly 70% of allergic subjects were studied during a relevant pollen season.

We found significant associations between aeroallergen sensitization patterns and poorly controlled asthma or loss of asthma control, as well as OCS usage for asthma exacerbations (Table 1). For the loss of control variable, we compared those with poorly controlled asthma (n=10) to those who gained control by the second visit and remained well controlled (n=15). The odds of loss of control compared to those who remained well controlled were higher in those with higher eosinophil count [(OR, 95% CI): 1.38, 0.96-2.00], higher number of allergen sensitizations (OR 1.36, 0.98-1.89), and those with ≥ 4 allergens compared to those with < 4 allergens (OR 6.00, 0.93-38.63). Among teens who did not achieve asthma control (n=10), we found significantly increased odds of sensitization to tree mix (OR 18, 1.75-184.68) and weed mix (OR 10.9, 1.6-75.48) excluding ragweed. For OCS requirement, we saw close to significant (p=0.07) association between sensitization to *D. farinae* (OR 5.417, 0.88-33.36) and weed mix (OR 5.5, 0.836-36.196).

Our small study showed a significant association between poorly controlled asthma and sensitization to aeroallergens, particularly seasonal outdoor allergens, despite guidelines-directed therapy in African-American teens with moderate to severe persistent asthma. Our study is limited by small sample size in determining associations, and while power was insufficient to test interactions between the season in which subjects were studied and asthma control, the majority of subjects were studied during a relevant pollen season. Despite these limitations, we found that the likelihood of having poorly controlled asthma was higher in polysensitized individuals, consistent with the findings of several studies suggesting a relationship between degree of atopy and bronchial hyperresponsiveness^(7, 8). We found an 18-fold higher risk in tree pollen-sensitized and 10-fold higher risk in weed pollen-sensitized (excluding ragweed) individuals, suggesting that tree or weed pollen sensitization is an independent risk factor for asthma exacerbation. We also suspect that independent of season, by virtue of being allergic and therefore “primed”, our subjects were more susceptible to exacerbations due to viruses, pollutants, and other triggers. In contrast to pediatric inner city asthma studies, our results did not suggest a significant impact of cockroach sensitization on loss of asthma control, while sensitization to cat was associated with increased likelihood of poorly controlled asthma (Table 1). The effects of global climate change on human disease have become more apparent recently, with longer pollination seasons and rising airborne pollen concentrations occurring alongside a global increase in asthma prevalence and severity⁽⁹⁾. Our findings suggest that in addition to guidelines-directed asthma therapies, targeting the allergic component, particularly tree and weed pollens, is an important strategy for achieving optimal asthma control in this at-risk population. Given these findings, therapies targeting specific IgE such as omalizumab and seasonal allergen-specific immunotherapy may be more relevant than ever for reducing asthma-related morbidity.

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Abbreviations:

(OCS)	Oral corticosteroid
(NAEPP)	National Asthma Education and Prevention Program
(NHLBI)	National Heart, Lung and Blood Institute
(FEV ₁)	Forced expiratory volume in 1 second
(ETS)	environmental tobacco smoke
(AIT)	allergen-specific immunotherapy

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Table 1.

Logistic regression model of allergic profiles by outcome asthma control throughout study.

	Outcome 1: Loss of control during study				Outcome 2: OCS usage				
	Odds Ratio	95% LCL	95% UCL	p-value	Odds Ratio	95% LCL	95% UCL	p-value	
Exposures									
	Peripheral blood eosinophil count	1.38	0.96	2.00	0.08	1.061	0.774	1.455	0.71
	Allergen sensitization (number of)	1.36	0.98	1.89	0.06	1.306	0.935	1.825	0.12
	Sensitization to 4 allergens	6.00	0.93	38.63	0.06	3.375	0.524	21.729	0.20
Allergy (Yes/ no)									
	<i>D. pteromyssinus</i>	1.33	0.25	7.01	0.73	2.400	0.423	13.600	0.32
	<i>D. farinae</i>	1.33	0.25	7.01	0.73	5.417	0.880	33.360	0.07
	2 Cockroach mix	2.75	0.51	14.86	0.24	2.400	0.423	13.600	0.32
	Tree mix 10	18	1.75	184.68	0.02 *	1.481	0.265	8.267	0.65
	Grass mix GS7	3.5	0.55	22.30	0.18	6.222	0.623	62.159	0.12
	National weed mix	10.99	1.60	75.48	0.01 *	5.500	0.836	36.196	0.07
	Mold mix 1	1.5	0.30	7.53	0.62	3.056	0.535	17.462	0.21
	Cat hair extract	6.42	1.09	37.73	0.04 *	3.056	0.535	17.462	0.20
	Dog epithelia	0.72	0.06	9.22	0.80	1.071	0.083	13.896	0.96
	Ragweed mix	0.31	0.03	3.24	0.32	1.556	0.205	11.830	0.67

* P<0.05

UCL = upper control limit

LCL = lower control limit

OCS = oral corticosteroid