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Predictors of Emergency Department Utilization in Children with Persistent Asthma in Metropolitan Atlanta, Georgia

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Keywords

Childhood asthma; Asthma disparities; Healthcare utilization; Asthma exacerbations; Aeroallergen sensitization

Introduction

Ongoing surveillance data clearly indicate racial disparities among children with asthma in the United States. Since 2001, the prevalence of asthma has been consistently higher in non-Hispanic Black versus White children and reached a peak prevalence ratio of 2.1 (95% confidence interval [CI]: 1.7 – 2.4) in 2011.[1] More recent (2014) prevalence estimates demonstrate a similar trend, with 13.4% versus 7.6% of non-Hispanic Black versus White children with current asthma, defined by both a physician diagnosis and current asthma features.[2] The prevalence of asthma is also higher in children with low household income[1] and children residing in metropolitan areas;[3] however, a greater proportion of Black children live in low-income and metropolitan environments than White children..[4] While at-risk rates of asthma attack prevalence (which remove the contribution of prevalence differences between groups) suggest that exacerbations are not more frequent in Black versus White children, Black children do have nearly twice as many emergency department **(ED)** visits and hospitalizations for asthma and 4 times more asthma-related deaths.[5]

Although previous studies have examined risk factors for asthma exacerbations in children in outpatient settings,[6] fewer studies have studied factors associated with asthma-related ED utilization in Black versus White children. While some studies suggest that disparities in ED utilization may be attributed to socioeconomic hardship variables (i.e., limited household income or lower educational attainment)[7, 8] or community characteristics (i.e., population density or limited health care access),[9] other studies have shown that asthma may be intrinsically more severe in Black children[10] and may be associated with differing biological

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features.[11] However, there are limited prospective studies of ED utilization in well-phenotyped children with asthma. Given recent federal efforts to reduce asthma-related racial disparities in U.S. children,[12] the purpose of this study was to compare asthma features in non-Hispanic Black versus White children residing in metropolitan Atlanta and to determine whether differing features predict future ED utilization for asthma after adjustment for socioeconomic status and residential population features. The results demonstrate that Black children with persistent asthma have differing risk factors for ED utilization compared to White children that are possibly related to the home environment and might require tailored management approaches.

Methods

Self-reported non-Hispanic Black and White children 6 though 17 years of age with persistent asthma treated with daily controller medications who resided in Fulton or Dekalb county were recruited year-round from an outpatient asthma clinic in Atlanta, Georgia. Each participant had a history of either ≥ 12% reversibility in the forced expiratory volume in one second **(FEV_1)** after bronchodilator administration or airway hyperresponsiveness to methacholine, evidenced by a provocative concentration of methacholine ≤ 16 mg/mL. Exclusion criteria included residence outside of metropolitan Atlanta (defined as living outside the perimeter of interstate 285), premature birth before 35 weeks gestation or other co-morbid airway disorders such as aspiration or vocal cord dysfunction. Permission to proceed with this study was granted by the Emory University Institutional Review Board. Informed written consent and assent was obtained from caregivers and participants, respectively.

Study design and procedures

Participants completed a single research characterization visit. Informed consent was obtained at the beginning of the visit. Children had no signs of acute respiratory illnesses at the time of characterization. The visit was postponed if an asthma exacerbation treated with systemic corticosteroids was reported within the preceding four weeks. Medical records were reviewed for asthma-related ED visits for 12 months after the initial study visit. Children were also telephoned at 6 and 12 months to assess for ED utilization outside of our facility.

Children withheld bronchodilator medication prior to the study visit (i.e., four or more hours for short-acting bronchodilators and twelve or more hours for long-acting bronchodilators). Spirometry (KoKo® PDS, Ferraris, Louisville, CO) was performed at baseline and after bronchodilator reversibility testing with 4 inhalations of albuterol sulfate (90μg per inhalation). The best of three forced vital capacity **(FVC)** maneuvers was interpreted according to population reference equations.[13] Allergy skin prick testing was performed after a three-day antihistamine withhold using 12 allergen extracts: tree mix (*Quercus alba, Ulmus americana, Platanus acerifolia, Salix caprea, Populus deltoides*), grass mix (*Cynodon dactylon, Lolium perenne, Phleum pratense, Poa pratensis, Sorghum halepense, Paspalum notatum*), weed mix (*Artemisia vulgaris, Chrysanthemum leucanthemum, Taraxacum vulgare, Solidago virgaurea*), common ragweed (*Ambrosia artemisiifolia*), *Alternaria*

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alternata, *Aspergillus fumagatis*, *Cladosporidium herbarum*, dog dander, cat dander, German cockroach (*Blatella germanica*), *Dermatophagoides farinae*, and *Dermatophagoides pteronyssinus* (Greer® Laboratories, Lenoir, NC). Histamine and saline served as positive and negative controls, respectively. Tests were considered positive if a wheal of 3 mm diameter or greater and flare 10 mm or more compared to the control was present 15 minutes after application. Venipuncture was performed for total serum immunoglobulin E (**IgE)** and blood eosinophils, which were quantified by a local laboratory (Children's Healthcare of Atlanta, Atlanta, GA). Medical history questionnaires assessed healthcare utilization over the previous 12 months, symptoms over the previous 4 weeks, current medications, and current demographic features and exposures such as tobacco smoke. Asthma-related quality of life over the preceding two weeks was also assessed with the Asthma Quality of Life Questionnaire **(AQLQ)**, which has established measurement properties.[14, 15] Self-reported asthma characteristics and medical history information such as asthma medications, previous healthcare utilization and payor status were verified by a review of medical records. The geographic coordinators of each participant's residential address were mapped in Geographic Information System software (ArcGIS Online, June 2016 Version Update, Esri, Redlands, CA). The ArcGIS geocoder function was used to determine the exact geographic coordinate based on the residential street and house number. The map containing all residential locations was then overlaid with the U.S. median household income in 2015 and the U.S. total crime index in 2016 available through Esri® U.S. Data, which were derived from the U.S. Census Bureau data and the Applied Geographic Solutions (AGS) national CrimeRisk database, respectively. The total crime index represents the combined risks of rape, murder, assault, robbery, burglary, larceny and vehicle theft compared to the national average of 100. Other features of participant's ZIP code were obtained from U.S. Census Bureau data and the American Community Survey available through the congressional district lookup feature at www.insidegov.com.

Statistical analyses

Data were analyzed with SPSS® Statistics (Version 24, IBM, Armonk, NY). Variables that were not normally distributed were logarithmically transformed prior to analysis. T-tests and Chi-square tests were used to test for differences between Black and White children. Cox proportional hazards regression was used to determine time to ED utilization adjusting for season of enrollment. For predictor analyses, redundant variables such as lung function values and ZIP code features were reduced with orthogonal varimax factor analysis. Cut-points for blood eosinophils and FEV_1 were based on clinical relevance and practice guidelines.[16] Univariate logistic regression was first performed on selected clinical predictors to obtain crude odds ratios to estimate the probability of ED utilization. Race specific models for Black and White children were generated. Univariate predictors with a p-value <0.15 were further reduced with multivariate forward conditional logistic regression. The log likelihood test was used to evaluate whether variables should remain in the model. Final estimates were adjusted for payor status (public versus private insurance) and ZIP code education level (% of adults with Bachelor's degree). Results are presented as odds ratios with 95% confidence intervals.

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Results

Two hundred sixty-five children with persistent asthma treated with daily asthma controller medications (inhaled corticosteroids, n = 239 (90.2%); montelukast, n = 26 (9.4%)) were enrolled from metropolitan Atlanta. Children were enrolled year-round with no significant differences in season of enrollment (January–March: n = 72 (27.2%), April–June: n = 80 (30.2%), July–September: n = 50 (18.9%), October–December: n = 63 (23.8%); p = 0.214). The ZIP codes of enrolled participants had an average population of 37,536 ± 16,450 people and 12.9 ± 5.1% unemployment rates. Additionally, 31.0 ± 15.5% of individuals age 25 and older residing in these ZIP codes had attained a bachelor's degree and 24.4 ± 11.2% of households had incomes less than $25,000/year. However, racial differences in ZIP code features were noted. Black children were more likely than White children to reside in disadvantaged ZIP codes with higher unemployment rates, lower educational attainment, and lower income (Table 1, Figure 1A). Black children were also exposed to more crime (Figure 1B). Other demographic features of participating children are shown in Table 1. Although there were no differences in age or environmental tobacco smoke exposure, Black children were more likely to be male, have public insurance, and have physician-diagnosed atopic dermatitis (Table 1).

Asthma features

Asthma features are shown in Table 2. Compared to White children, Black children had an earlier age of asthma symptom onset and more urgent healthcare utilization for asthma in the preceding year despite treatment with more asthma controller medications. Black children also had higher blood eosinophil percentages, higher total serum IgE concentrations, more prevalent aeroallergen sensitization and increased airflow obstruction with lower baseline FEV_1 and FEV_1/FVC values and higher absolute FEV1 reversibility after bronchodilator administration (Table 2). Although the frequency of self-reported asthma symptoms over the preceding 4 weeks was not different between groups (Figure 2A), asthma-related quality of life was significantly more impaired in Black children as reflected by lower total AQLQ scores (5.24 ± 1.18 vs. 5.74 ± 1.24, p = 0.016) that were largely attributed to the burden of asthma symptoms and the environment (Figure 2B).

ED utilization during the study period

Ninety-one of the 265 children enrolled (34.3%) had an ED visit for an asthma exacerbation within 12 months of enrollment. Twenty-one children (22.6%) were hospitalized. ED utilization was disproportionately higher in Black (38.7%) versus White (26.1%) children (p = 0.039) and children with public (40.4%) versus private (23.4%) insurance (p = 0.005), irrespective of race. A significant interaction between race and payor status was also observed, with the highest odds of an ED visit observed in Black children with public insurance, as compared to White children with private insurance (OR: 2.42, 95% CI: 1.22, 4.81; p = 0.011). ZIP code socioeconomic features were also associated with ED utilization. The odds of ED utilization were significantly greater for children residing in ZIP codes with greater unemployment (OR for each percentage increase: 1.10; 95% CI: 1.04, 1.16; p < 0.001), lower educational attainment (OR for each percent increase in Bachelors degree attainment: 0.98; 95% CI: 0.96, 0.99; p = 0.009), and a higher percentage of families living

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below the poverty threshold (OR for each percentage increase: 1.03; 95% CI: 1.01, 1.05; p = 0.029). The proportion of children who visited an ED during the course of the study, stratified by race and payor status, is also illustrated in Figure 3A–C.

Predictors of ED utilization

Univariate (unadjusted) predictors of ED utilization are shown in Table 3. Whereas ED utilization in the previous year was associated with an ED visit during the study period irrespective of race, other predictors including patterns of specific aeroallergen sensitization differed by race (Table 3). In multivariate analyses, different predictors were identified for Black versus White children even after adjustment for potential confounding effects of payor status and regional (i.e., ZIP code) educational attainment (Table 4). In White children, an ED visit in the previous year and sensitization to dust mites and pets were associated with ED utilization during the study period. White children were also more likely than Black children to report having a cat (27.5% vs. 9.5%, p = 0.001) or dog (58.2% vs. 34.3%, p < 0.001) inside the home. However, in Black children, the variables associated with ED utilization during the study period included an ED visit in the previous year, the number of asthma controller medications, FEV_1 <80% predicted, blood eosinophils >4%, and mold sensitization (Table 4).

Discussion

Although racial disparities in asthma prevalence and outcomes are apparent in U.S. children, the responsible factors are multifactorial and not well understood. In this prospective study of children with persistent asthma residing in metropolitan Atlanta, we identified differences in the geographical residential features (i.e., ZIP code population attributes) and household socioeconomic features (i.e., payor status) of Black versus White children that were each independently associated with future ED visits. After adjustment for these potentially confounding factors, many clinical predictors of ED utilization such as aeroallergen sensitization profiles still differed between Black and White children. For example, whereas dust mite sensitization and pet sensitization (and a higher prevalence of indoor pet ownership) predicted ED utilization in White children, mold sensitization predicted ED utilization in Black children. However, other predictors of ED utilization did not differ by race. Similar to the recent multi-center study of Teach et al.,[6] our results confirm that previous ED visits are a strong predictor of future ED utilization, but additionally demonstrate that this variable is a predictor in both races.

Although the regional and prospective nature of this analysis resulted in limited sample sizes for modeling and a corresponding lack of precision in the adjusted results, the findings confirm and extend other studies of inner-city populations that have demonstrated multifactorial reasons for asthma-related healthcare utilization.[9, 17, 18] In one study of asthma hospitalizations and deaths in New York City, the rate of hospital admission was nearly 5 times higher in Blacks than Whites and was further associated with striking differences in neighborhood features. In that study, nearly 75% of the geographic variation in the asthma hospitalization rate was associated with median household income and the percentage of the population that was Black.[17] A separate study similarly observed 88-fold

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variation in asthma hospital admission rates across a single county that was strongly associated with Black race and neighborhood socioeconomic variables such educational attainment, car access and population density.[9] Financial and social hardship variables such as unemployment, lack of home ownership and single/not married status have also been associated with hospital readmission in regional populations of children.[7, 8] A large, retrospective analysis of the National Health Interview Survey further demonstrated on a national level that living in an inner-city environment *per se* may have less impact on key asthma outcomes than non-Hispanic Black race or individual household variables such as those described above.[19]

Separating biological factors from socioeconomic and residential features in the analysis of asthma-related racial disparities is challenging, but some evidence does exist to support the hypothesis of greater intrinsic disease severity in Blacks. In a relatively large analysis of well-phenotyped adults enrolled in the National Heart, Lung and Blood Institute's Severe Asthma Research Program, serum IgE was significantly associated with asthma severity in Blacks but not in Whites.[11] In that same study, Blacks with severe asthma, compared to Whites with severe asthma, also had lower lung function as reflected by FEV_1 and a stronger family history of asthma. Other genetic studies suggest that Black patients may have unique asthma-associated variants that increase the risk of asthma susceptibility[20–23] and potentially asthma severity as reflected by lung function values.[24] Indeed, studies have shown an inverse relationship between lung function and ancestry, with a decrease in FEV_1 of approximately 50 to 100 mL for each 20% increase in African ancestry.[25, 26] A separate analysis further noted that every 20% increase in African ancestry was associated with a 4.3-fold increased rate of exacerbation in males.[27]

Home environmental assessments were not performed in the study, which is a potential limitation. Therefore the nature and degree of the allergen exposures in our participants is not clear. Our observation of differing aeroallergen sensitization profiles suggests that there may be racial differences in the home environment of Black versus White children that contribute to asthma outcomes. Others have similarly reported a higher of prevalence of cat or dog ownership in White versus Black households that may be attributed to higher income.[28, 29] Although a pooled analysis of birth cohorts found that pet ownership did not modify the risk of asthma onset in children,[30] sensitized children who own pets may be at higher risk for severe asthma exacerbation requiring intensive care.[31] Cat and dog ownership also contributes to higher indoor endotoxin concentrations, which have been associated with wheezing and increased respiratory medication use.[32] Indoor dust mite and mold exposures are other risk factors that have also been linked to exacerbations of asthma in children.[6, 33, 34] However, neither dust mite prevention strategies[35, 36] nor remediation of root causes of home moisture and mold have had a clear impact on asthma outcomes in children, in part due to the limited quality of available evidence.[37]

Although we assessed payer status and ZIP code features in the present study, the lack of detailed information on household income and household education is a potential limitation. In a recent study of 686 Black children with asthma, low household income was the strongest individual socioeconomic predictor of poor asthma control.[38] However, a composite socioeconomic index consisting of maternal education level, annual household

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income and insurance status also predicted worse asthma outcomes in Black children that were not limited to the very poor alone.[38] We also did not collect information on residential relocation during the study period, which could impact aeroallergen exposures or residential geographical features. Access to care is also an important issue that was not adequately addressed with our study design. In a recent study of children presenting to an ED in Baltimore for asthma-related care, nearly one-third of parents reported access to care (i.e., inability to receive a same-day appointment with a pediatrician or work conflicts) as a major factor in their decision to seek emergency care.[39]

Another limitation is the lack of prescription adherence monitoring in our participants, which could have contributed to ED utilization during the study period. A large study of 2,499 children in the National Health and Nutrition Examination survey during three time periods between 1988 and 2008 noted significantly lower preventive asthma medication use in Black versus White children, children without health insurance, and adolescents (i.e., 12 to 19 year olds) versus younger age groups.[40] Other studies have similarly found that Black patients are less likely to adhere to asthma prescriptions than Whites,[41] perhaps due to negative inhaled corticosteroid beliefs and greater endorsement of complementary and alternative medicine,[42] although asthma prescription adherence is still quite low even in insured, predominantly White populations when measured by electronic claims.[43] It is also possible that prescribing practices by healthcare providers may have differed between racial groups. Previous studies have found that Black versus White children enrolled in the Medicaid system may be less likely to receive preventative treatments such as ICS.[44] A separate study of adults with asthma similarly noted that Black versus White patients were less likely to meet eligibility criteria for Medicare prescription drug benefits, in part due to decreased prescription use in Blacks.[45] Similarly, in the present study, Black versus White children had more atopic features and greater disease severity yet there were no differences in the receipt of anti-IgE therapy between groups.

In conclusion, the results of this study demonstrate differences in asthma features and asthma-related ED utilization between Black and White children residing in metropolitan Atlanta. Because exposures and sensitization patterns clearly differ between cities, validation in other populations is needed before the results can be generalized to other areas. Given that poor asthma outcomes are also observed in Black children living in rural areas with more limited access to primary and ED care,[46] other studies in rural populations are also needed. Nonetheless, our findings of racial differences in metropolitan Atlanta children suggest that strategies to eliminate allergen exposure in the home and improve asthma control may require tailoring for different racial groups in this area. However, an in-depth individualized assessment and personalized approach to asthma care is also needed to improve outcomes.

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Abbreviations

AQLQ	Asthma Quality of Life Questionnaire
ED	Emergency department
FEV_1	Forced expiratory volume in one second
FVC	Forced vital capacity
IgE	Immunoglobulin E

References

1. Akinbami LJ, Simon AE, Rossen LM. Changing Trends in Asthma Prevalence Among Children. Pediatrics. 2016:137. [PubMed: 27543009]
2. Centers for Disease Control and Prevention. National Center for Health Statistics. National Health Interview Survey, 2014, Table 4-1: Current asthma prevalence percents.
3. Bloom B, Freeman G. Tables of Summary Health Statistics for U.S. Children: 2014 National Health Interview Survey. 2015
4. Beckles GL, Truman BI, Centers for Disease Control and Prevention. Education and income - United States, 2009 and 2011. MMWR Suppl. 2013; 62:9–19.
5. Akinbami LJ, Moorman JE, Simon AE, et al. Trends in racial disparities for asthma outcomes among children 0 to 17 years, 2001–2010. J Allergy Clin Immunol. 2014; 134:547–53 e5. [PubMed: 25091437]
6. Teach SJ, Gergen PJ, Szefler SJ, et al. Seasonal risk factors for asthma exacerbations among inner-city children. J Allergy Clin Immunol. 2015; 135:1465–73 e5. [PubMed: 25794658]
7. Beck AF, Huang B, Auger KA, et al. Explaining Racial Disparities in Child Asthma Readmission Using a Causal Inference Approach. JAMA Pediatr. 2016; 170:695–703. [PubMed: 27182793]
8. Beck AF, Huang B, Simmons JM, et al. Role of financial and social hardships in asthma racial disparities. Pediatrics. 2014; 133:431–9. [PubMed: 24488745]
9. Beck AF, Moncrief T, Huang B, et al. Inequalities in neighborhood child asthma admission rates and underlying community characteristics in one US county. J Pediatr. 2013; 163:574–80. [PubMed: 23522864]
10. Fitzpatrick AM, Teague WG, Meyers DA, et al. Heterogeneity of severe asthma in childhood: confirmation by cluster analysis of children in the National Institutes of Health/National Heart, Lung, and Blood Institute Severe Asthma Research Program. J Allergy Clin Immunol. 2011; 127:382–9. e1–13. [PubMed: 21195471]
11. Gamble C, Talbott E, Youk A, et al. Racial differences in biologic predictors of severe asthma: Data from the Severe Asthma Research Program. J Allergy Clin Immunol. 2010; 126:1149–56 e1. [PubMed: 21051082]
12. President's Task Force on Environmental Health Risks and Safety Risks to Children. Coordinated federal action plan to reduce racial and ethnic asthma disparities. Washington, D.C.: President's Task Force on Environmental Health Risks and Safety Risks to Children; 2012.
13. Wang X, Dockery DW, Wypij D, et al. Pulmonary function between 6 and 18 years of age. Pediatr Pulmonol. 1993; 15:75–88. [PubMed: 8474788]
14. Juniper EF, Guyatt GH, Ferrie PJ, et al. Measuring quality of life in asthma. Am Rev Respir Dis. 1993; 147:832–8. [PubMed: 8466117]
15. Juniper EF, Guyatt GH, Willan A, et al. Determining a minimal important change in a disease-specific Quality of Life Questionnaire. J Clin Epidemiol. 1994; 47:81–7. [PubMed: 8283197]
16. National Asthma Education and Prevention Panel. Expert Panel Report 3 (EPR-3): Guidelines for the Diagnosis and Management of Asthma-Summary Report 2007. J Allergy Clin Immunol. 2007; 120:S94–138. [PubMed: 17983880]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

17. Carr W, Zeitel L, Weiss K. Variations in asthma hospitalizations and deaths in New York City. Am J Public Health. 1992; 82:59–65. [PubMed: 1536336]
18. Malhotra K, Baltrus P, Zhang S, et al. Geographic and racial variation in asthma prevalence and emergency department use among Medicaid-enrolled children in 14 southern states. J Asthma. 2014; 51:913–21. [PubMed: 24915006]
19. Keet CA, McCormack MC, Pollack CE, et al. Neighborhood poverty, urban residence, race/ethnicity, and asthma: Rethinking the inner-city asthma epidemic. J Allergy Clin Immunol. 2015; 135:655–62. [PubMed: 25617226]
20. Torgerson DG, Capurso D, Ampleford EJ, et al. Genome-wide ancestry association testing identifies a common European variant on 6q14.1 as a risk factor for asthma in African American subjects. J Allergy Clin Immunol. 2012; 130:622–9 e9. [PubMed: 22607992]
21. White MJ, Risse-Adams O, Goddard P, et al. Novel genetic risk factors for asthma in African American children: Precision Medicine and the SAGE II Study. Immunogenetics. 2016; 68:391–400. [PubMed: 27142222]
22. Torgerson DG, Capurso D, Mathias RA, et al. Resequencing candidate genes implicates rare variants in asthma susceptibility. Am J Hum Genet. 2012; 90:273–81. [PubMed: 22325360]
23. Torgerson DG, Ampleford EJ, Chiu GY, et al. Meta-analysis of genome-wide association studies of asthma in ethnically diverse North American populations. Nat Genet. 2011; 43:887–92. [PubMed: 21804549]
24. Li X, Howard TD, Moore WC, et al. Importance of hedgehog interacting protein and other lung function genes in asthma. J Allergy Clin Immunol. 2011; 127:1457–65. [PubMed: 21397937]
25. Brehm JM, Acosta-Perez E, Klei L, et al. African ancestry and lung function in Puerto Rican children. J Allergy Clin Immunol. 2012; 129:1484–90 e6. [PubMed: 22560959]
26. Kumar R, Seibold MA, Aldrich MC, et al. Genetic ancestry in lung-function predictions. N Engl J Med. 2010; 363:321–30. [PubMed: 20647190]
27. Rumpel JA, Ahmedani BK, Peterson EL, et al. Genetic ancestry and its association with asthma exacerbations among African American subjects with asthma. J Allergy Clin Immunol. 2012; 130:1302–6. [PubMed: 23069492]
28. Downes MJ, Roy A, McGinn TG, et al. Factors associated with furry pet ownership among patients with asthma. J Asthma. 2010; 47:742–9. [PubMed: 20684732]
29. Beck AF, Huang B, Kercsmar CM, et al. Allergen sensitization profiles in a population-based cohort of children hospitalized for asthma. Ann Am Thorac Soc. 2015; 12:376–84. [PubMed: 25594255]
30. Lodrup Carlsen KC, Roll S, Carlsen KH, et al. Does pet ownership in infancy lead to asthma or allergy at school age? Pooled analysis of individual participant data from 11 European birth cohorts. PLoS One. 2012; 7:e43214. [PubMed: 22952649]
31. McDowell KM, Kercsmar CM, Huang B, et al. Medical and Social Determinants of Health Associated with Intensive Care Admission for Asthma in Children. Ann Am Thorac Soc. 2016; 13:1081–8. [PubMed: 27144510]
32. Thorne PS, Mendy A, Metwali N, et al. Endotoxin Exposure: Predictors and Prevalence of Associated Asthma Outcomes in the United States. Am J Respir Crit Care Med. 2015; 192:1287–97. [PubMed: 26258643]
33. Kanchongkittiphon W, Mendell MJ, Gaffin JM, et al. Indoor environmental exposures and exacerbation of asthma: an update to the 2000 review by the Institute of Medicine. Environ Health Perspect. 2015; 123:6–20. [PubMed: 25303775]
34. Soto-Quiros M, Avila L, Platts-Mills TA, et al. High titers of IgE antibody to dust mite allergen and risk for wheezing among asthmatic children infected with rhinovirus. J Allergy Clin Immunol. 2012; 129:1499–505 e5. [PubMed: 22560151]
35. DiMango E, Serebrisky D, Narula S, et al. Individualized Household Allergen Intervention Lowers Allergen Level But Not Asthma Medication Use: A Randomized Controlled Trial. J Allergy Clin Immunol Pract. 2016; 4:671–9 e4. [PubMed: 27025297]
36. Gotzsche PC, Johansen HK. House dust mite control measures for asthma: systematic review. Allergy. 2008; 63:646–59. [PubMed: 18445182]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

37. Sauni R, Verbeek JH, Uitti J, et al. Remediating buildings damaged by dampness and mould for preventing or reducing respiratory tract symptoms, infections and asthma. Cochrane Database Syst Rev. 2015:CD007897.

38. Thakur N, Martin M, Castellanos E, et al. Socioeconomic status and asthma control in African American youth in SAGE II. J Asthma. 2014; 51:720–8. [PubMed: 24654704]

39. Mudd SS, Ogborn CJ, Bollinger ME, et al. Parental decision making associated with pediatric emergency department use for asthma. Ann Allergy Asthma Immunol. 2016; 117:490–4. [PubMed: 27788877]

40. Kit BK, Simon AE, Ogden CL, et al. Trends in preventive asthma medication use among children and adolescents, 1988–2008. Pediatrics. 2012; 129:62–9. [PubMed: 22144697]

41. Wells KE, Cajigal S, Peterson EL, et al. Assessing differences in inhaled corticosteroid response by self-reported race-ethnicity and genetic ancestry among asthmatic subjects. J Allergy Clin Immunol. 2016; 137:1364–9 e2. [PubMed: 27016472]

42. George M, Topaz M, Rand C, et al. Inhaled corticosteroid beliefs, complementary and alternative medicine, and uncontrolled asthma in urban minority adults. J Allergy Clin Immunol. 2014; 134:1252–9. [PubMed: 25218286]

43. Wu AC, Butler MG, Li L, et al. Primary adherence to controller medications for asthma is poor. Ann Am Thorac Soc. 2015; 12:161–6. [PubMed: 25569765]

44. Wu CL, Andrews AL, Teufel RJ, et al. Demographic predictors of leukotriene antagonist monotherapy among children with persistent asthma. J Pediatr. 2014; 164:827–31. [PubMed: 24370344]

45. Lu D, Qiao Y, Johnson KC, et al. Racial and ethnic disparities in meeting MTM eligibility criteria among patients with asthma. J Asthma. 2016 Sep 27.:1–10. (Epub ahead of print).

46. Garcia E, Serban N, Swann J, et al. The effect of geographic access on severe health outcomes for pediatric asthma. J Allergy Clin Immunol. 2015; 136:610–8. [PubMed: 25794659]

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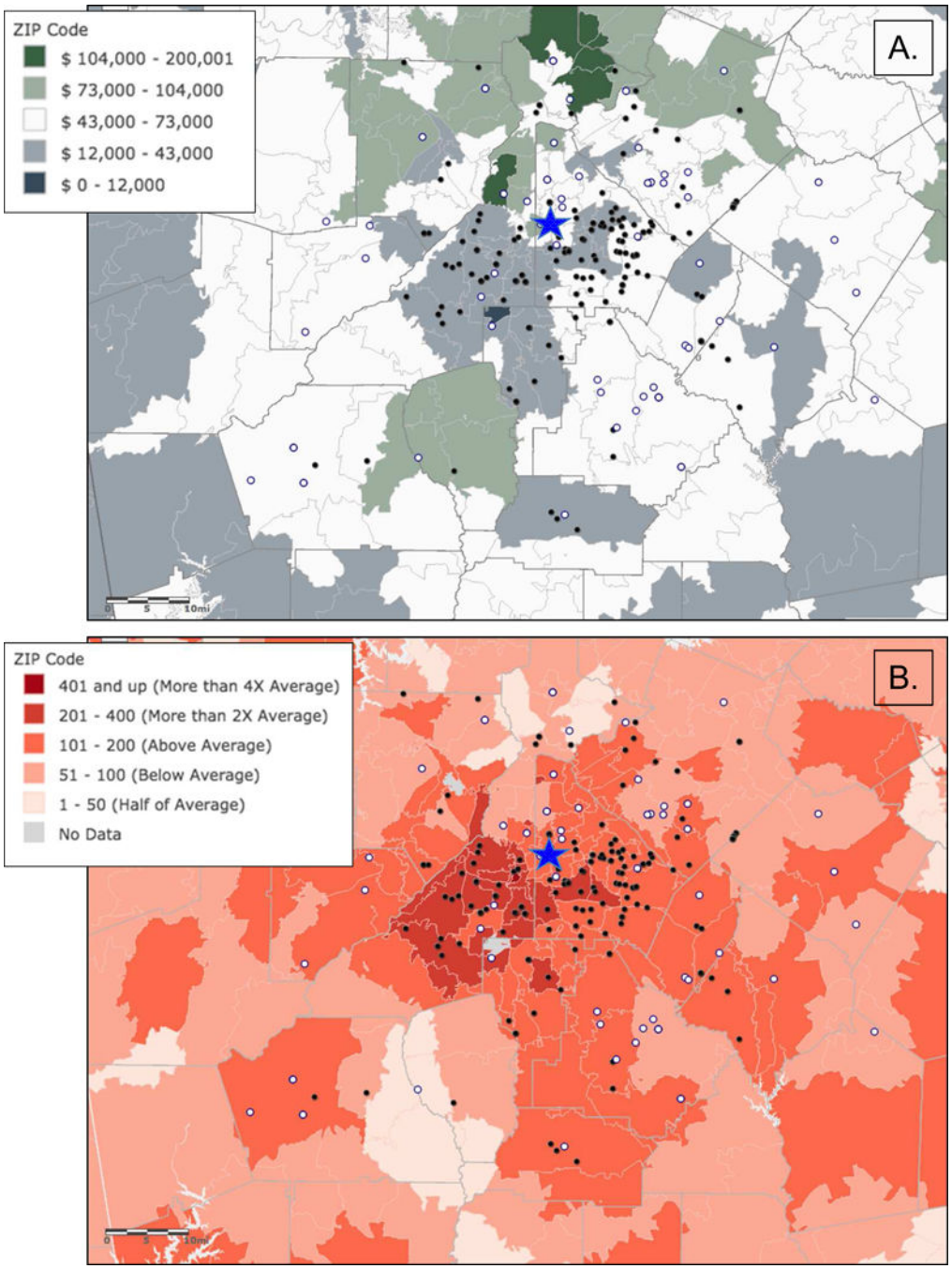

Figure 1.

Residences of children with overlays of **(A)** median household income and **(B)** total crime indices, by ZIP code. The crime index value for the U.S. as a whole is 100; values of 200 indicate twice the national average. Black and white circles represent Black and White children, respectively. The blue star represents the location of the clinic and Emergency Department.

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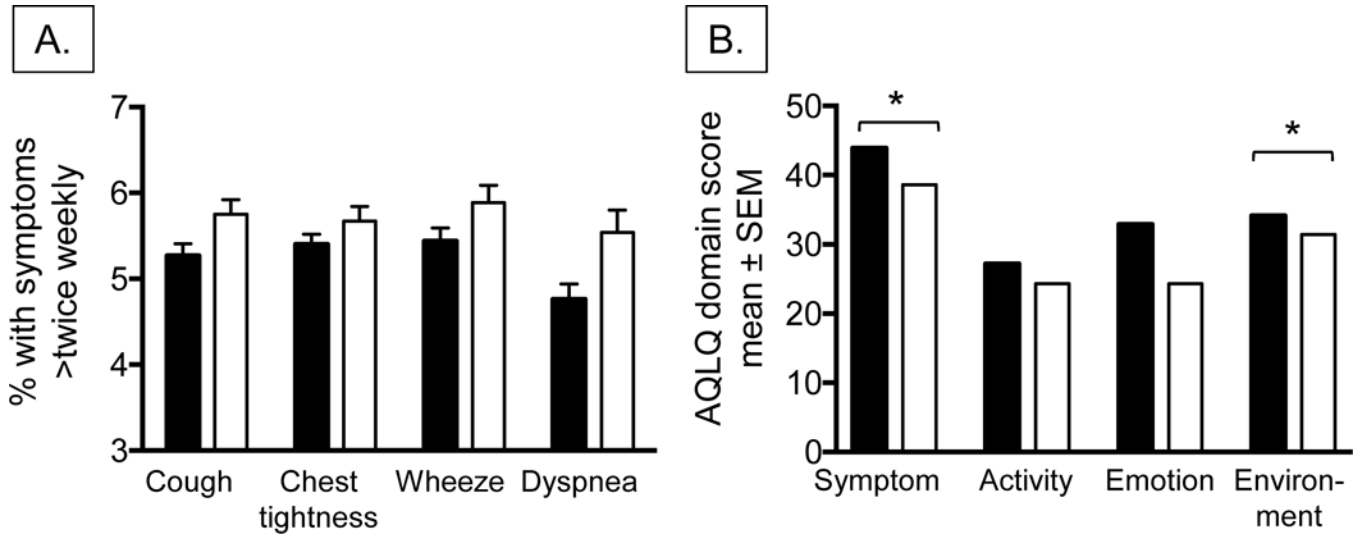

Figure 2.

(A) The percentage of children with symptoms more than twice weekly and **(B)** individual domain scores for the Asthma Quality of Life Questionnaire (AQLQ). Black and white bars represent Black and White children, respectively. *$p < 0.05$

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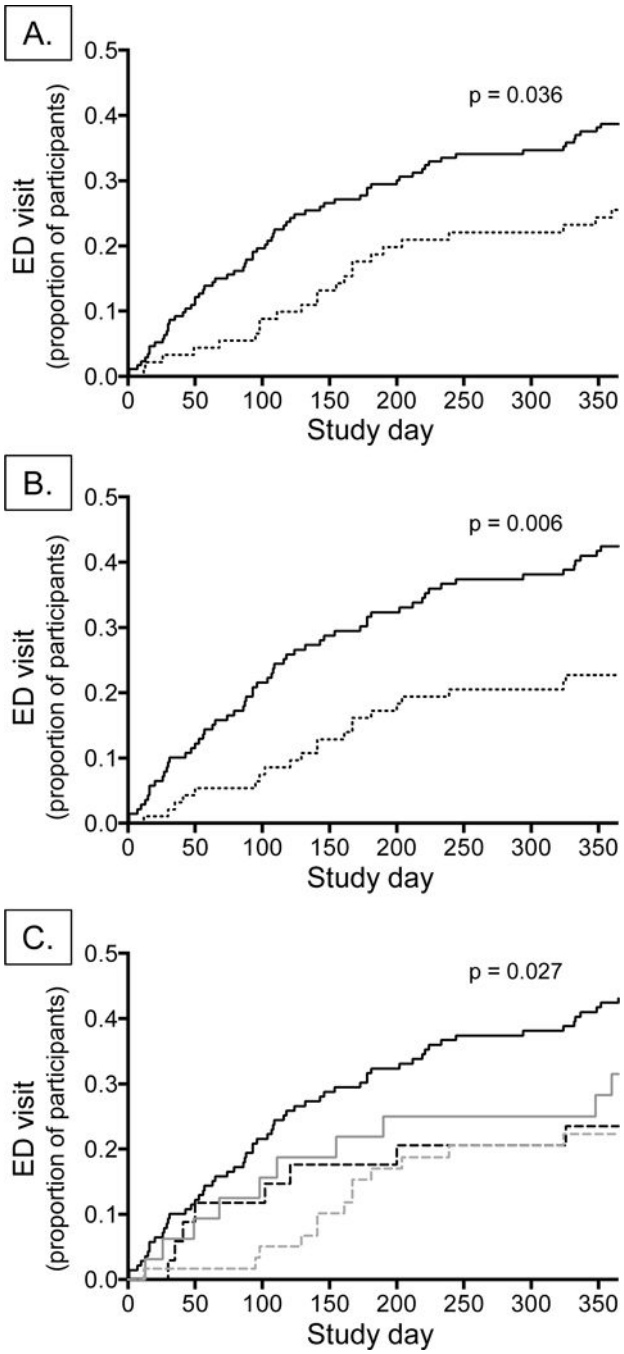

Figure 3.

Proportion of participants with an Emergency Department (ED) visit during the study, stratified by **(A)** Black (solid line) versus White race (dashed line), **(B)** public (solid line) versus private insurance (dashed line), **(C)** and White and Black children with public insurance (gray and black solid lines, respectively) versus White and Black children with private insurance (gray and black dashed lines, respectively).

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Table 1

Demographic features of the participants at enrollment. Values represent the mean ± SD or the number of participants (%).

Feature	All participants N = 265	White N = 92	Black N = 173	p-value for White vs. Black
Age (years)	11.5 ± 2.9	11.5 ± 2.9	11.5 ± 2.9	0.998
Males	152 (57.4)	35 (38.0)	117 (67.6)	< 0.001
Public insurance	171 (64.5)	32 (34.8)	139 (80.3)	< 0.001
Tobacco smoke exposure	48 (18.3)	14 (15.2)	34 (20.0)	0.217
Other conditions				
Obesity (BMI >95th percentile)	67 (25.4)	21 (22.8)	46 (26.7)	0.486
Atopic dermatitis	149 (56.9)	44 (47.8)	105 (61.8)	0.030
Historical recurrent sinusitis	111 (42.4)	41 (44.6)	70 (41.2)	0.596
Historical pneumonia	148 (56.5)	49 (53.3)	99 (58.2)	0.314
Residential (ZIP code) features				
Population (in thousands)	37.5 ± 16.5	34.0 ± 18.1	39.4 ± 15.2	0.009
Unemployment (%)	12.9 ± 5.1	9.3 ± 2.7	14.8 ± 5.1	< 0.001
Bachelor's degree (%)	31.0 ± 15.5	33.9 ± 18.4	29.4 ± 13.6	0.024
Rented homes (%)	37.5 ± 15.7	31.4 ± 13.0	40.8 ± 16.1	< 0.001
Families in poverty (%)	24.4 ± 11.2	18.8 ± 8.0	27.4 ± 11.5	< 0.001

BMI = body mass index

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Table 2

Features of asthma in the participants. Values represent the mean ± SD, the median (25^{th}, 75^{th} percentile) or the number of participants (%).

Feature	All participants N = 265	White N = 92	Black N = 173	p-value for White vs. Black
Age of symptom onset (years)	1 (1, 4)	3 (1, 6)	1 (0.7, 3)	< 0.001
Healthcare utilization for asthma				
Emergency department visit (previous year)	153 (57.7)	36 (39.1)	117 (67.6)	< 0.001
Hospitalization (previous year)	90 (34.1)	15 (16.3)	75 (43.6)	< 0.001
Intubation (ever)	61 (23.1)	4 (4.3)	57 (33.1)	< 0.001
Daily asthma controller medications				
Number of controller medications	3 (2, 3)	2 (2, 3)	3 (2, 3)	0.008
High-dose inhaled corticosteroid[1]	150 (56.6)	32 (34.8)	118 (68.2)	< 0.001
Systemic corticosteroid	19 (7.2)	6 (6.5)	13 (7.5)	0.766
Long-acting beta-agonist	179 (67.5)	51 (55.4)	128 (74.0)	0.002
Montelukast	199 (75.1)	61 (66.3)	138 (79.8)	0.016
Omalizumab	8 (3.0)	2 (2.2)	6 (3.5)	0.558
Blood eosinophils (%)[2]	5.0 (2.3, 8.0)	3.5 (1.6, 7.2)	5.2 (3.2, 8.7)	< 0.001
Serum IgE (kU/L)[2]	227 (80, 698)	139 (22, 562)	253 (145, 786)	0.002
Aeroallergen sensitization				
Positive aeroallergen tests (# out of 12)	3 (1, 5)	2 (0, 5)	3 (1, 6)	0.032
Pollen sensitization	93 (35.1)	27 (29.3)	66 (38.2)	0.015
Mold sensitization	76 (28.7)	22 (23.9)	54 (31.2)	0.044
Dust mite sensitization	129 (48.7)	39 (42.4)	90 (52.0)	0.003
Dog or cat sensitization	73 (27.5)	28 (30.4)	45 (26.0)	0.974
Cockroach sensitization	57 (21.5)	16 (17.4)	41 (23.7)	0.065
Pre-bronchodilator lung function				
FVC (% predicted)	102 ± 16	104 ± 15	101 ± 17	0.292
FEV_1 (% predicted)	91 ± 18	94 ± 17	89 ± 19	0.027
FEV_1/FVC (% predicted)	89 ± 12	92 ± 12	88 ± 12	0.029
FEV_1/FVC (actual ratio)	0.78 ± 0.10	0.80 ± 0.10	0.77 ± 0.10	0.004
Post-bronchodilator lung function				
FVC (% predicted)	108 ± 16	107 ± 14	110 ± 17	0.170
FEV_1 (% predicted)	103 ± 16	104 ± 15	103 ± 17	0.862
FEV_1/FVC (% predicted)	95 ± 11	97 ± 13	94 ± 10	0.101
FEV_1/FVC (actual ratio)	0.83 ± 0.08	0.85 ± 0.07	0.82 ± 0.08	0.003
FEV_1 reversibility (% change from baseline)	16.0 ± 15.7	11 ± 10	18 ± 18	0.001

[1] High-dose inhaled corticosteroid is defined as a daily dose of >440 or >880 micrograms of fluticasone priopionate equivalent for children 6–11 years and children 12–17 years, respectively

[2] p-values obtained after logarithmic transformation

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Table 3

Univariate (unadjusted) predictors of emergency department (ED) utilization. Data represent the odds ratio (OR) and the 95% confidence interval.

	White children N = 92		Black children N = 173	
Predictor	**OR (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**
Age of symptom onset (years)	0.75 (0.60, 0.92)	0.006	0.89 (0.77, 1.02)	0.0.087
Emergency department visit for asthma (previous year)	8.33 (2.86, 24.28)	< 0.001	3.25 (1.56, 6.78)	0.002
Intubation for asthma (ever)	0.96 (0.09, 9.51)	0.960	1.36 (0.71, 2.59)	0.354
Number of controller medications	1.37 (0.86, 2.19)	0.184	2.05 (1.38, 3.02)	< 0.001
Blood eosinophils > 4%	1.68 (0.63, 4.49)	0.301	2.20 (1.07, 4.50)	0.031
Serum IgE > 150 kU/L	1.69 (0.61, 4.62)	0.305	1.48 (0.71, 3.05)	0.287
Pollen sensitization	1.75 (0.63, 4.88)	0.285	1.99 (0.98, 4.07)	0.059
Mold sensitization	1.98 (0.68, 5.74)	0.210	2.21 (1.08, 4.53)	0.031
Dust mite sensitization	3.75 (1.28, 11.03)	0.016	2.18 (0.97, 4.89)	0.058
Dog or cat sensitization	3.67 (1.30, 10.34)	0.014	1.63 (0.78, 3.41)	0.191
Cockroach sensitization	0.93 (0.27, 3.32)	0.925	2.10 (0.99, 4.46)	0.053
FEV_1 < 80% of predicted value	3.44 (0.98, 11.99)	0.052	2.98 (1.49, 5.97)	0.002

BMI = body mass index

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Table 4

Multivariate (adjusted)[1] predictors of emergency department (ED) utilization. Data are shown as the odds ratio (OR) and the 95% confidence interval.

Predictor	Adjusted OR (95% CI)	p-value
White children		
ED visit (previous year)	7.03 (2.36, 20.91)	< 0.001
Dust mite sensitization	5.04 (1.45, 17.51)	0.011
Dog or cat sensitization	3.09 (1.08, 8.88)	0.036
Black children		
ED visit (previous year)	2.99 (1.42, 6.29)	0.004
Number of controller medications	2.02 (1.36, 3.02)	< 0.001
FEV_1 < 80% of predicted value	2.91 (1.43, 5.92)	0.003
Blood eosinophils >4%	2.25 (1.09, 4.62)	0.028
Mold sensitization	2.32 (1.07, 4.65)	0.032

[1] Adjusted for payor status (public versus private insurance) and ZIP code education level (percentage of adults with a Bachelor's degree).