2-8-2017

Comprehensive Neighborhood Portraits and Child Asthma Disparities Introduction

Ashley W. Kranjac
Chapman University, kranjac@chapman.edu

Rachel T. Kimbro
Rice University

Justin T. Denney
Rice University

Kristin M. Osiecki
University of Illinois at Springfield

Brady S. Moffett
Baylor College of Medicine

See next page for additional authors

Recommended Citation

Kranjac AW, RT. Kimbro, Denney, JT, et al. Comprehensive neighborhood portraits of child asthma disparities. *Maternal and Child Health Journal* 2017; 21(7): 1552–1562. doi: 10.1007/s10995-017-2286-z

Comprehensive Neighborhood Portraits and Child Asthma Disparities

Introduction

The prevalence and severity of asthma in children is a significant public health concern both globally and in the United States. Even though scholars have documented a downward trend of asthma among children in recent years [1], increasing disparities of asthma by child race/ethnicity and socioeconomic status (SES) threaten potential gains from this decline [1,2]. Researchers cite complex individual and structural factors including genetic predisposition [3], aberrant immune response [4], and environmental triggers [5] that interdependently influence children's asthma risk in an attempt to explain differences in asthma risk.

The etiology of asthma is further complicated by neighborhood social patterning that closely follows racial/ethnic and socioeconomic lines [6,7,8]. The neighborhoods in which children live may play a critical role in explaining asthma disparities [9,10]. An underlying challenge, however, is identifying which, and to what extent, individual-level risk factors combine across distinctive neighborhood contexts to influence asthma outcomes.

Previous studies that assess the causes of asthma in children highlight the roles of social, economic, and environmental factors [5,6,7,8,9,10] but they do not distinguish between these mechanisms of difference, nor do they quantify their relative strength. We examine asthma prevalence in children and the social and structural conditions of neighborhoods in conjunction with demographic and familial characteristics to determine the relative importance of these determinants based on disparate neighborhood characteristics.

Background

Asthma is a multifactorial chronic inflammatory disease associated with airway obstruction [2], wheezing [3], episodic cough [11], and shortness of breath [2] that can have lasting effects on children and their families [12]. Despite advances in preventative treatment, asthma prevalence continues to increase for some in the United States [12]. This increase, however, is highly variable across children [13,14,15]. Race/ethnicity and socioeconomic characteristics underlie differential asthma prevalence [16,17,18]. Asthma hospitalization and mortality rates are higher for African American compared to white children [17], and children living in poverty are at a significantly higher asthma risk, relative to more affluent children [18,19].

Despite the important role of characteristics such as child race and family poverty, researchers have increasingly attended to neighborhood factors that may trigger or exacerbate asthma symptoms [20,21]. Environmental concerns are often at the forefront of these investigations. Ambient air pollutants such as particulate matter (PM2.5) and ozone (O3) [3,14], much like asthma prevalence, are more highly concentrated in low-income areas than in places with higher income residents [5]. Consequently, children in less affluent communities are exposed to higher asthma risks from air pollutants [14]. These data imply that children's neighborhood conditions affect asthma through mutually dependent mechanisms embedded within structural processes of racial and socioeconomic stratification, such as the concentration of racial/ethnic minorities, areas of deprivation, and differential levels of air pollution [16,17,18,19,20].

One pathway through which this occurs is differential pathogenic factors in the physical environment that influence several other factors that alter asthma risk [9,17, 21]. For example, children in disadvantaged communities are disproportionately racial/ethnic minorities [6,7] who are exposed to higher levels of pollution [14, 22], which can exacerbate asthma symptoms. Further, the availability of social services in these types of neighborhoods can impact access to health care resources, and facilitate or impede a parent's ability to weaken the effect of poor air quality on children's lung functioning through treatment or medication, which may then influence asthma outcomes. Residential segregation may also contribute to variation in asthma risk among children. Research indicates that segregation leads to crowding, which may predispose children to viral illnesses [7]. Moreover, housing deterioration including water damage, cockroach infestation, mold, and chipped paint may increase child exposure to indoor air pollutants and allergens as parents keep their children indoors due to neighborhood violence and safety concerns [10].

Differences in social and economic characteristics that correlate with health and neighborhood disparities among children can lead to an accumulation of advantage or disadvantage. These inequalities may explain, in part, why asthma differences exist by neighborhood type. For example, individual measures of disadvantage, including racial/ethnic minority status and low socioeconomic position, are associated with higher asthma risk [5,7,16]. Moreover, neighborhood measures of concentrated disadvantage, including high rates of poverty, unemployment, female-headed households, and low levels of education and income, are also associated with higher asthma prevalence [5,7,8,9,10]. It is possible that these social environmental factors combine with physical environmental factors to influence and alter asthma risk [9,22]. For example, children in socioeconomically disadvantaged communities are also exposed to higher levels of pollution [23,24], which can increase the likelihood of asthma diagnosis. Despite growing evidence that racial/ethnic minority children living in areas of deprivation are disproportionately exposed to higher environmental risks and are more likely to be diagnosed and even die from asthma [18,24], less is known about the degree to which social and environmental characteristics influence asthma disparities across distinctive neighborhood contexts.

Objectives and Significance

Prior studies account for a range of proximate and distal factors associated with childhood asthma, yet researchers have not been able to identify the relative importance of social and environmental risk factors or completely account for asthma disparities across groups. In fact, relatively little is known about which factors within distinctive neighborhood contexts contribute to asthma disparities in children. To fill these gaps, we link unique data sources and use latent profile modeling techniques to characterize neighborhoods into areas of distinctive racial and socioeconomic contexts. We then use a modified version of the Blinder-Oaxaca regression decomposition method to examine the difference in asthma diagnoses for children in Disadvantaged, Middle-class, and Advantaged neighborhoods to determine the relative importance of individual- and neighborhood-level characteristics for asthma diagnoses.

Our approach affords the opportunity to identify to what degree demographic, familial, and air pollutant factors within distinctive neighborhood contexts contribute to asthma disparities, and then quantify the extent to which differences in these social, economic, and environmental characteristics lead to variation in asthma outcomes. We expect that children living in Disadvantaged communities will have higher rates of asthma relative to children in more affluent communities, as previously demonstrated [5,6]. Our approach, however, lets us go further and assess whether asthma disparities are driven by differences in the *population composition* across neighborhoods or by variation in the *associations between* child, familial, and air quality characteristics. For example, is the risk of asthma higher for children who live in low-income, minority neighborhoods simply because the concentration of people there are more likely to have asthma? Or is it because the relationships between individual-level factors, such as insurance status, and asthma risk differ in magnitude across neighborhoods?

Methods

Data Sources

Our child-level data comes from a compilation of electronic medical and administrative records from a network of pediatric clinics and emergency room visits in Houston, TX (n = 206,974 children in 1,076 Census tracts or neighborhoods). Medical records include inpatient and emergency room pediatric visits to Texas Children's Hospital (TCH) as well as outpatient visits to one of 50 Texas Children's Pediatric Associates (TCPA) clinics throughout the Houston metropolitan area. Children who were 2-12 years old in 2011 and 2012 were included and we randomly selected one child per family to eliminate bias at the household level. Each child record was geocoded based on the physical residential address included in the medical record and linked to the matching census tract, or neighborhood-level, social and economic indicators generated using the 2010 decennial census files and 2009-2013 American Community Survey (ACS) data.

Historical air quality data were collected from the Texas Commission on Environmental Quality (TCEQ) Texas Air Monitoring Information System (TAMIS) (http://www17.tceq.texas.gov/tamis/) from the years 2010 – 2012. We replicated the approach of the California Communities Environmental Health Screening Tool, Version 2.0 (2014) wherein concentrations for particulate matter 2.5 micrometers or less (PM2.5) and daily 8-hour averages of ozone (O3) were estimated at the centroid of each census tract using interpolation prediction methods [25].

Variables

The key outcome measure is a dichotomous variable indicating whether or not the child carried a diagnosis of asthma. Children were coded as having asthma if they received a diagnosis for asthma consistent with ICD-9 diagnostic codes (those that begin with '493') or if the word "asthma" appeared in any of the first five diagnosis fields in the billing record for any visit between 2011 and 2012. Although it is possible that asthmatic children in our data are not coded as having asthma if they visited the doctor or were seen as an inpatient but did not receive a billing code for asthma, we assessed potential bias of selection into an asthma diagnosis by verifying that our data are consistent with asthma hospitalization and prevalence rates provided by the Texas Department of State Health Services [26]. We acknowledge that while billing data are not perfect, the ability to use physician diagnoses of asthma instead of parental reports significantly reduces potential reporting bias [27,28].

We include covariates from the medical record to represent child and familial characteristics. Child characteristics include: age at time of visit (mean-centered), gender, race/ethnicity, and insurance type as a proxy for SES. Age is a continuous measure and represents the age of the child when he/ she visited the clinic or hospital, centered on the mean. Gender is a dichotomous variable and represents whether or not the child is male, with female as the reference. Race/ethnicity is a categorical measure representing the parent-reported race/ethnicity of the child categorized as non-Hispanic White, non-Hispanic Black, Hispanic, and Asian/Other Race, with non-Hispanic White as the reference. We also include an indicator to represent whether the child was missing information on race/ethnicity as a covariate due to the large number of missing data for this variable in clinical record data (approximately 18%). Insurance type is a categorical measure indicating the type of medical insurance held by the child at the time of the clinical visit, and is categorized as private provider, public provider, and other or missing insurance provider, with private provider as the reference. Similar to race/ethnicity, approximately 24% of children were missing on insurance status so we also include a “missing” category in our models for this measure. Sensitivity analyses which excluded children missing on race/ethnicity and insurance status revealed substantively similar results (available upon request).

Social and economic indicators of the child’s neighborhood of residence come from the Census and ACS, and include: community-level education, unemployment rate, median income, median year the house was built, percent of female-headed households, percent foreign born, percent receiving public assistance, percent in poverty, percent of homes that are vacant in the tract, and racial and ethnic composition measured by percent of major racialized categories. Air pollutants include $PM^{2.5}$ and O^3 exposure centering them at each respective mean. To estimate the air quality measures, we used ordinary kriging, a spatial interpolation method, to calculate $PM^{2.5}$ and ozone concentrations. The quarterly mean is estimated at the geographic center of a census tract to create an annual mean which is then calculated into a three year average to find a PM2.5 concentration value for each census tract. The same steps are taken using daily maximum 8-hour average ozone concentrations to come-up with three-year averages of ozone for each census tract. All 1,076 Census tracts in the greater Houston metropolitan area are represented.

Statistical Analysis

Consistent with our research objectives, we ultimately aim to illuminate the sources of asthma disparities in an effort to inform whether population compositional differences in residential neighborhoods drive asthma inequities or whether differentials in resources alter families' ability to minimize risks of asthma. Thus, we use a maximum-likelihood latent profile analysis (LPA) [29] to characterize neighborhoods into classes on the basis of the sociodemographic community-level variables described above. We first estimated a 1-class model and fit successive models with an increasing number of classes. We used Bayesian information criterion (BIC), p-value-based likelihood ratio tests, entropy R^2, bootstrap p-value, and theoretically-driven evidence to select the most parsimonious model. Analyses indicated that neighborhoods are most appropriately captured by a 3-class solution, which we label Advantaged, Middle-Class, and Disadvantaged based on the neighborhood characteristics (Table 1).

To explain the difference in asthma diagnoses between neighborhoods we use a step-wise process, and stratification is necessary in each [30,31]. First, we estimate the sample means for the child, familial, and air pollution covariates to identify differences across neighborhoods. Then, we estimate separate regression equations by neighborhood type to assess the associations between these characteristics and asthma within neighborhoods. Finally, to partition the difference estimated in steps 1 and 2 into two components, we use a modified version of the standard Oaxaca-Blinder regression decomposition technique [30,31]. The recently proposed version produces robust estimates of the contributions of individual variables [32].

The components estimated by the Oaxaca-Blinder technique are two counterfactuals based on the differences in the sample means and differences in the coefficients between neighborhoods. The value of the first counterfactual is the contribution of child, familial, and environmental variation in the means of the covariates (i.e., differences in *population composition or sample means*). This addresses whether differences in asthma exist between neighborhoods because children in more disadvantaged communities are, for example, more likely to be exposed to higher levels of air pollutants. The value of the second counterfactual is the contribution of differences in the regression coefficients and intercepts (i.e., differences in *associations or magnitude of determinants*). This addresses whether asthma differences exist between neighborhoods because children's parents, for example, are less able to minimize risks of the deleterious effects of air pollution due to a lack of resources. The decomposition is estimated from the "perspective" of children in more advantaged neighborhoods. That is, if children in Middle-class or Advantaged communities have the same social, economic, and environmental exposure characteristics as children living in Disadvantaged neighborhoods, how would asthma diagnoses differ (e.g., lack of private insurance), and if children in more advantaged communities have the same magnitude of coefficients as children in Disadvantaged neighborhoods how would asthma diagnoses differ (e.g., parents lack knowledge needed to maximize insurance benefits)? We then identify how much each individual characteristic included in the analysis contributes to the overall difference in asthma diagnoses between neighborhoods.

With this method it is necessary to make comparisons between two neighborhood types at a time in a three-step process, as the Oaxaca-Blinder approach only permits decomposition between two groups. We estimate the expected values for each comparison, using ordinary least squares (OLS) regression. We use OLS because our conservative estimates from the resulting linear probability model can be interpreted in terms of a difference in the probability of being diagnosed with asthma (i.e., β represents the difference in the probability of an asthma diagnosis with a one unit change in the independent variable *X*). Estimates from a model using logistic regression are similar to those from the linear probability model. The research was conducted in accord with prevailing ethical principles and reviewed by the Rice University and Baylor College of Medicine Institutional Review Boards.

RESULTS

Table 1 displays social, economic, and demographic characteristics of neighborhoods in Houston from the Census and ACS data. Middle-class and Advantaged neighborhoods have smaller proportions of racial and ethnic minorities living in the community relative to Disadvantaged neighborhoods. Advantaged neighborhoods have, on average, 8% non-Hispanic Blacks and 15% Hispanics relative to 26% and 60% in Disadvantaged communities, respectively. Compared to Disadvantaged, in Middle-class and Advantaged communities, a significantly smaller proportion of the population is unemployed (13% vs. 8% vs. 5%), receives public assistance (3% vs. 2% vs. 1%), and lives in poverty (29% vs. 11% vs. 4%).

[Table 1 about here]

Differences in Asthma between Neighborhoods. — Table 2 displays means and standard errors for asthma diagnoses overall and by neighborhood. Asthma significantly differs between neighborhoods. Children in Disadvantaged neighborhoods have higher asthma diagnoses relative to children living in Middle-Class and Advantaged neighborhoods (8% vs. 6% vs. 4%, respectively).

[Table 2 about here]

Compositional Differences between Neighborhoods— Table 2 also displays means and standard errors overall and by neighborhood type for key covariates. The average age of sampled children at time of visit is about 6.04 years old. There are significantly higher proportions of African American and Hispanic children living in Disadvantaged neighborhoods (25% vs. 15% vs. 6% African American; 51% vs. 23% vs, 11% Hispanic). In general, children living in Middle-class and Advantaged neighborhoods have advantages over those in Disadvantaged neighborhoods. More children in affluent neighborhoods have private insurance providers (71% vs. 54% vs. 23%), indicating higher SES. Affluent children in Middle-class and Advantaged neighborhoods are also exposed to significantly lower levels of PM2.5 (10.30 and 10.24, respectively), relative to Disadvantaged (10.45) communities. Although these differences may appear small, the range of effects for PM2.5 exposure on respiratory health varies considerably with slight deviations of mean concentration [23].

Associational Differences between Neighborhoods— Asthma diagnoses may be different across neighborhoods because the associations between selected characteristics and childhood asthma differ. For example, asthma disparities may exist partially because children in advantaged communities have parents who are more knowledgeable about the harmful effects associated with the air quality. If these parents take precautionary measures so that their children play inside on ozone action days, then the effect of ozone will appear to be reduced relative to the impact in other neighborhood types. Table 3 displays the estimated coefficients from OLS models regressing a binary indicator of asthma diagnoses on selected factors by neighborhood type. The coefficients represent the increment or decrement to the probability of having asthma attributable to that particular characteristic.

[Table 3 about here]

Asthma is associated with a child's age at visit, but the magnitude of this association is significantly smaller in more advantaged communities. For children living in Disadvantaged neighborhoods, the probability of having asthma increases by about 0.005 for each 1-year increase in age at visit, compared to 0.003 for those in Middleclass and 0.002 for children in Advantaged communities.

The probability of asthma for African American children is 0.088 higher in Disadvantaged, 0.067 higher in Middle-class, and 0.058 higher in Advantaged communities, compared to White children in the same community. That is, African American children, compared to White children, experienced a higher probability of asthma diagnosis across neighborhood types but the more disadvantaged the neighborhood the larger the difference. The association between child air quality and asthma diagnoses significantly differs between neighborhoods. For children living in Disadvantaged communities, the probability of having asthma is associated with a statistically insignificant increase of less than 0.01 for each 1-unit increase in PM2.5. In Middle-class and Advantaged neighborhoods, the probability of being diagnosed with asthma decreases for each 1-unit increase in PM2.5 (Middle-class: -0.008; Advantaged: - 0.010). We speculate that this result may be due to the ability of families in more affluent areas to buffer the harmful effects of pollution, rather than higher air pollution actually decreasing asthma incidence. For children living in Advantaged, but not Disadvantaged or Middleclass, communities the association between O3 and asthma increases with higher-than average exposure levels (0.006).

Regression Decomposition

The decomposition results are shown in Table 4. The estimated contribution of each factor to compositional difference and difference in associations is also displayed. If a factor is associated with a diagnosis of asthma, the resulting estimate is positive. If a factor counteracts a diagnosis of asthma, the estimate is negative.

[Table 4 about here]

Overall Difference.— We isolated the sources of the asthma gap identified for children in Disadvantaged and Advantaged (Δ4.00), Disadvantaged and Middle-class (Δ2.00), and Middle-class and Advantaged (Δ2.00) communities. Compositional differences in ambient air pollutants explain a significant proportion of the overall asthma gap between Disadvantaged and Middle-class neighborhoods (O3 = 1.1%, PM2.5

= 2.8%). For Disadvantaged and Advantaged communities, on the other hand, associational differences of selected child (Non-Hispanic White: 1.1%) and family (private provider insurance: 0.3%) characteristics explain a significant proportion of the asthma gap. Among Middle-class and Advantaged areas, the compositional difference in age at visit explains 0.7% of the asthma difference between neighborhoods, whereas differences in the associations in race, SES, and air quality explain a larger proportion of the asthma gap (Non-Hispanic White: 0.4%, Private Provider 0.2%, PM2.5 = 1%).

Compositional Difference. — The principal factors responsible for asthma disparities between Disadvantaged and Middle-class are the observed differences in pollutant exposure composition for the population of children. Differences in PM2.5 (0.011*100 = 1.1%) and O3 (0.028 = 2.8%) exposure contribute to the total difference in asthma between these neighborhoods.[1] Another 0.4% (0.004*100 = 0.4%) of the explained portion of the 2% asthma gap between Disadvantaged and Middle-class communities is attributable to compositional differences in children's age at visit. That is, because children in Disadvantaged communities are generally younger when visiting the doctor, asthma diagnoses differences are present between Disadvantaged and Middle-class and Middle-class and Advantaged communities. The decompositions between Disadvantaged and Advantaged and Middle-class and Advantaged neighborhoods reveal that observed differences in race/ethnicity, health insurance, and air quality contributes to the narrowing of the asthma gap.

Associational Difference. — Asthma diagnoses are lower in Advantaged compared to Disadvantaged communities, partially because the magnitude of the association attributable to child and family characteristics is higher in Disadvantaged communities. More specifically, 1.1% (0.011*100 = 1.1%) of the 4% asthma gap between Disadvantaged and Advantaged is explained by the variation in asthma diagnosis between Non-Hispanic White children and those of other racial and ethnic backgrounds. Similarly, the differential effect among private and all other insurance holders in Disadvantaged neighborhoods explains 0.3% (0.003*100 = 0.3%) of this 4% asthma gap. In fact, as shown in Table 4, net of other factors, some of which counteract the positive effect of difference in asthma, variation in the coefficients for race/ethnicity and economic characteristics explains a proportion of children's disparities between Disadvantaged and Advantaged (race: 0.5% and health insurance: 0.2% of the 4% gap), Disadvantaged and Middle-class (race: 0.2% and health insurance: 0.0% of the 2% gap), and Middle-class and Advantaged (race: 0.1% and health insurance: 0.1% of the 2% gap) communities.[2] A part of the positive or counteracting effect in the coefficients is due to differences in the intercepts or the expected value of asthma diagnoses when all covariates equal zero (Disadvantaged and Advantaged: 0.014; Disadvantaged and Middle-class: 0.023, and Middle-class and Advantaged: 0.008).

[1] Difference sums to more than total difference because of the negative estimates for other factors such as race/ ethnicity that offset asthma diagnoses differences.
[2] Some researchers suggest this unexplained portion reflects discrimination (e.g., [32]).

Discussion

Asthma prevalence continues to increase among many children in the U.S. [1,22], a phenomenon that is disproportionately linked to racial/ethnic minorities and areas of deprivation [16,1718,19]. Methodologically, the mechanisms of disparity in asthma imply two different sources or components of difference. We used LPA to classify neighborhoods into distinctive communities of racial and sociodemographic characteristics, and decomposition techniques to identify the sources of, and relative contribution to, children's asthma disparities across different neighborhood contexts.

Both the compositional and associational differences between demographic, familial, and air quality characteristics within distinctive neighborhood contexts influence asthma outcomes. Unequal exposure to PM2.5 and O3 among children in Disadvantaged and Middle-class neighborhoods contributes to asthma disparities. Our results are consistent with previous studies which indicate that low income communities have disproportionately higher levels of ambient air pollutants. We add to this literature by disaggregating associational and compositional effects and showing that, in Houston, the concentration of environmental exposures in areas of deprivation heightens risks for children who already face many disadvantages in health.

For children in Disadvantaged and Advantaged communities, associational differences between racial/ethnic and socioeconomic characteristics and asthma diagnoses explain a significant proportion of the gap. The associations between Non-Hispanic whites and children with private insurance and asthma are lower relative to other racial/ethnic minorities and among those with public insurance providers. These results provide evidence that white children and children from affluent families may be better able to buffer the harmful effects of factors which exacerbate asthma risk in other groups, although we are unable to explain why. We speculate that more advantaged children may live in families with higher levels of education and other types of family and community resources which function as protective factors for asthma risk. It is also likely that more affluent children have central heat and air, spend more time outside of their home neighborhood in extracurricular activities, and/or attend schools where the air quality is better. Even if they are exposed to air pollution at home, these differences may buffer the effects of pollution on asthma outcomes.

Our results show that differential exposure to pollution and difference in returns to racial/ethnic and socioeconomic characteristics among children in more affluent areas, relative to those who share similar attributes but live in more disadvantaged communities, contribute to asthma disparities across neighborhoods. Given the magnitude of our estimates, however, results ultimately indicate that neighborhoods matter less than we expected for asthma disparities. Rather, it is the difference in returns to racial and socioeconomic characteristics within distinctive social contexts that comparatively contributes more to disparities in asthma.

Strengths and Limitations

The cross-sectional nature of our data, our focus on ambient air pollution without inclusion of indoor air quality, and our limited indicators of child and family characteristics limit the scope of our analysis. However, researchers generally lack access to data that explicitly link social determinants of health to children's asthma disparities. It is important to better understand how ambient air quality issues combine with other environmental risks influencing asthma. This does not negate the importance of studying the links between indoor air quality and asthma [33]. We also use insurance type as a proxy measure for SES. While this is not ideal, publicly-provided health care coverage such as Medicaid is only available to children who meet stringent income criteria, with the exception of some that suffer from limited medical conditions [34]. In addition, insurance type is widely used as a marker for individual-level SES with reasonable validity and reliability [35,36,37,38]. Further, we link more than 200,000 medical records from a diverse group of children residing in Houston, TX, to demographic and environmental data based on their residential census tract. Although it is unclear whether the same relationships between neighborhood context and asthma diagnoses exist in the broader U.S. population, leveraging medical records, neighborhood, and environmental contextual data in the most diverse city in the United States provides a rich comprehensive account of the communities in which children live, and allows deeper insight into the association between context and asthma prevalence.

Conclusion

Our findings have important implications for research on asthma disparities. Despite finding that children living in areas of deprivation are exposed to higher levels of environmental toxics, and are more likely to be diagnosed with asthma, these differences do not drive asthma disparities across neighborhoods. Instead, inequalities across communities appear to create an environment wherein a child diagnosed with asthma in a disadvantaged neighborhood may be less able to buffer the harmful effects, as evidenced by the associational variation by race/ethnicity and SES. This difference may be due to lowered access to appropriate and timely treatment or decreased awareness of the severity of health risks. Future researchers should consider social and racial inequalities as more proximate drivers of asthma disparities in children, not merely as associated with asthma disparities in children.

References

[1] Akinbami LJ, Simon AE, Rossen LM. (2016). “Changing Trends in Asthma Prevalence Among Children.” *Pediatrics* 137(1):e20152354

[2] CDC - National Center for Health Statistics - Homepage. http://www.cdc.gov/nchs/. Accessed February 17, 2016.

[2] Eder, W. Ege, M. J., and E. Von Mutius. (2006). “The asthma epidemic,” *New England Journal of Medicine,* 355, 21 2226–2235.

[3] Esposito S, Tenconi R, Lelii M, Preti V, Nazzari E, Consolo S, Patria MF. (2014). “Possible molecular mechanisms linking air pollution and asthma in children.” *BMC Pulmonary Medicine,* 14, 31–40.

[4] von Hertzen LC. (2002). “Maternal stress and T-cell differentiation of the developing immune system: possible implications for the development of asthma and atopy.” *Journal of Allergy and Clinical Immunology* 109:923–8.

[5] Ash and Fetter. (2004). “Who lives on the wrong side of the environmental tracks? Evidence from the EPAs risk-screening environmental indicators model.” *Social Science Quarterly,* 85(2), 441-462.

[6] Crowder, Kyle, and Liam Downey. (2010). "Inter-neighborhood migration, race, and environmental hazards: modeling micro-level processes of environmental inequality." *American Journal of Sociology, 115*(4), 1110-1149.

[7] Pais, Jeremy, Kyle Crowder, and Liam Downey. (2013). "Unequal Trajectories: Racial and Class Differences in Residential Exposure to Industrial Hazard." *Social Forces* doi:10.1093/sf/sot099.

[8] Brisbon N, Plumb J, Brawer R, Paxman D. (2005). “The asthma and obesity epidemics: the role played by the built environment: a public health perspective.” *Journal of Allergy and Clinical Immunology* 115(5): 1024–1028.

[9] Wright RJ, Fisher EB. (2003). Putting asthma into context: community influences on risk, behavior, and intervention. In: Kawachi I, Berkman LF, eds. *Neighborhoods and Health*. New York, NY: Oxford University Press 233–262.

[10] Wright RJ, Mitchell H, Visness CM, et al. (2004). Community violence and asthma morbidity: the Inner-City Asthma Study. *American Journal of Public Health,* 94(4), 625–632.

[11] Groneberg-Kloft B, Kraus T, van Mark A, Wagner U, Fischer A. (2006). Analyzing the causes of chronic cough: relation to diesel exhaust, ozone, nitrogen oxides, sulfur oxides and other environmental factors. *Journal of Occupational Medical Toxicology,* 1, 6–16.

[12] Brandt, S. J., Perez, L., Künzli, N., Lurmann, F., & McConnell, R. (2012). Costs of Childhood Asthma due to Traffice-related Pollution in Two California Communities. *The European Respiratory Journal*, 40(2), 363–370.

[13] Pride NB. (1992). Asthma. Definition and clinical spectrum. *British Medical Bulletin,* 48(1), 1–9.

[14] Pope CA, Dockery DW. (2006). Health effects of particulate air pollution: Lines that connect. *Journal of Air and Waste Management,* 56, 709-742.

[15] Goldizen, F. C., Sly, P. D. and Knibbs, L. D. (2015). Respiratory effects of air pollution on children. *Pediatric Pulmonology*, doi: 10.1002/ppul.23262.

[16] Weitzman M, Gortmaker S, Sobol A. (1990). Racial, social, and environmental risks of childhood asthma. *American Journal of Diseases of Children,* 144, 1189–1194.

[17] Wright, Rosalind and S.V. Subramanian. (2007). Advancing a multilevel framework for epidemiologic research on asthma disparities. *CHEST*, 132, 757S-769S.

[18] Mitchell E, Stewart A, Pattermore P, et al. (1989). Socioeconomic status in childhood asthma. *International Journal of Epidemiology,* 18, 888–890.

[19] Williams, DR., Strenthal, M., and Rosalind Wright. (2009). Social determinates: Taking the social context of asthma seriously. *Pediatrics,* 123(3), S174-S184.

[20] Sampson, Robert J. (2008). Moving to Inequality: Neighborhood Effects and Experiments Meet Social Structure. *American Journal of Sociology,* 114 (1), 189–231.

[21] Sandel M, Wright RJ. (2006). When home is where the stress is: expanding the dimensions of housing that influence asthma morbidity. *Archives of Disease in Childhood,* 91(11), 942–948.

[22] Byrd RS, Joad JP. (2006). Urban asthma." *Current Opinion Pulmonary Medicine,* 12(1), 68–74.

[23] Schwartz, J. (2004). Air pollution and children's health. *Pediatrics,* 113(4 suppl), 1037–1043.

[24] Weiss KB, Wagener DK. (1990). Changing patterns of asthma mortality: identifying target populations at high risk. *JAMA,* 264,1683–1687.

[25] Rodriquez, M., and Alexeeff, G. V. (2014). California communities environmental health screening tool, version 2.0. http://oehha.ca.gov/media/CES20FinalReportUpdateOct2014.pdf

[26] Wickerham and Bhakta. (2013). “Asthma Hospitalization Rates and Prevalence Among Children in the Greater Houston Area, Texas, 2011”. Texas Department of State Health Services. Available at https://www.dshs.texas.gov/asthma/data.shtm.

[27] Akinbami, Lara J., Jeanne E. Moorman, Alan E. Simon, and Kenneth C. Schoendorf.(2014). "Trends in racial disparities for asthma outcomes among children 0 to 17 years, 2001-2010." *Journal of Allergy and Clinical Immunology* 134(3): 547-553.

[28] Akinbami, Lara J., Julia C. Rhodes, and Marielena Lara. (2005) "Racial and ethnic differences in asthma diagnosis among children who wheeze." *Pediatrics* 115(5): 1254-1260.

[29] Horton NJ, Roberts K, Ryan L, Franco Suglia S, Wright RJ. (2008). A maximum likelihood latent variable regression model for multiple informants. *Statistics in Medicine,* 27(24), 4992–5004.

[30] Blinder AS. (1973). “Wage discrimination: reduced form and structural estimates.” *Journal of Human Resources* 8(4):436-455.

[31] Oaxaca R. (1973). “Male-female wage differentials in urban labor markets.” International Economic Review;14(3):693-709.

[32] Kim, CH. (2013). “Detailed wage decompositions: Revisiting the identification problem.” Composition, Context, and Structure 43(1): 346-363.

[33] Breysse, Patrick N, Gregory B Diette, Elizabeth C Matsui, Arlene M Butz, Nadia N Hansel, and Meredith C McCormack. 2010. "Indoor air pollution and asthma in children." Proceedings of the American Thoracic Society 7(2):102-06.

[34] Rosenbaum S. Medicaid. New England Journal of Medicine. 2002;346:635–640. doi: 10.1056/NEJM200202213460825.

[35] Ayanian JZ, Kohler BA, Abe T, Epstein AM. 1993. The Relation between Health Insurance Coverage and Clinical Outcomes among Women with Breast Cancer. *New England Journal of Medicine* 329:326–331. doi: 10.1056/NEJM199307293290507.

[36] Harnick DJ, Cohen JL, Schechter CB, Fuster V, Smith DA. 1998. Effects of practice setting on quality of lipid-lowering management in patients with coronary artery disease. *American Journal of Cardiology* 81:1416–1420. doi: 10.1016/S0002-9149(98)00209-4.

[37] Shen JJ, Wan TT, Perlin JB. 2001. An exploration of the complex relationship of socioecologic factors in the treatment and outcomes of acute myocardial infarction in disadvantaged populations. *Health Services Research* 36:711–732.

[38] Foraker, R. E., Rose, K. M., Whitsel, E. A., Suchindran, C. M., Wood, J. L., & Rosamond, W. D. 2010. Neighborhood socioeconomic status, Medicaid coverage and medical management of myocardial infarction: Atherosclerosis risk in communities (ARIC) community surveillance. *BMC Public Health* 10, 632. doi.org/10.1186/1471-2458-10-632

Comprehensive Neighborhood Portraits

Table 1. Neighborhood-level Descriptive Statistics Overall and by Neighborhood Type

	Overall		Disadvantaged		Middle-Class		Advantaged	
	Mean or %	SE	Mean or %	SE	Mean or %	SE	Mean or %	SD/ SE
	Mean	(SD)	Mean	(SD)	(SD)	SE	Mean	(SD)
Race/ Ethnicity Proportions								
% Non-Hispanic White	0.39	(0.28)	0.11	(0.09)	0.45	(0.23)	0.65	(0.16)
% Non-Hispanic Black	0.18	(0.21)	0.26	(0.26)	0.19	(0.19)	0.08	(0.07)
% Hispanic	0.36	(0.24)	0.60	(0.24)	0.30	(0.13)	0.15	(0.07)
Socioeconomic Proportions								
Median Income (in $10K)	6.10	(0.01)	3.39	(1.14)	5.81	(1.56)	10.06	(3.68)
Median Year House Built	1980	(3.37)	1986	(0.01)	1981	(0.01)	1970	(0.01)
% Adults < 12 years of Education	0.21	(0.16)	0.40	(0.11)	0.16	(0.07)	0.05	(0.04)
% Adults = 12 years of Education	0.24	(0.10)	0.30	(0.07)	0.27	(0.07)	0.12	(0.06)
% Adults > 12 and < 16 years of Education	0.27	(0.09)	0.21	(0.06)	0.33	(0.06)	0.25	(0.08)
% Adults = 16 years of Education	0.18	(0.12)	0.07	(0.01)	0.16	(0.07)	0.34	(0.06)
% Adults = 18 years of Education	0.07	(0.06)	0.02	(0.02)	0.05	(0.03)	0.15	(0.05)
% Adults > 18 and < 21 years of Education	0.02	(0.03)	0.00	(0.01)	0.01	(0.01)	0.05	(0.04)
% Adults = 21 years of Education	0.01	(0.02)	0.00	(0.01)	0.01	(0.01)	0.03	(0.02)
% Unemployed	0.09	(0.05)	0.13	(0.06)	0.08	(0.04)	0.05	(0.02)
% Foreign-born Residents	0.22	(0.13)	0.31	(0.14)	0.17	(0.10)	0.19	(0.10)
% Receiving Public Assistance	0.02	(0.04)	0.03	(0.02)	0.02	(0.04)	0.01	(0.01)
% Female-Headed Households	0.15	(0.09)	0.22	(0.08)	0.15	(0.06)	0.07	(0.04)
% of Residents in Poverty	0.15	(0.15)	0.29	(0.11)	0.11	(0.06)	0.04	(0.03)
% of Vacant Homes	0.12	(0.09)	0.15	(0.08)	0.11	(0.08)	0.09	(0.08)
***N* =**	1,076		355		444		277	

Source: Data are from Cenus & American Community Survey (ACS).

Table 2. Child, Family, and Environmental Exposure Descriptive Statistics Overall and by Neighborhood Type									
	Overall		Disadvantaged		Middle-Class		Advantaged		Sig.
	Mean or %	SE	Mean or %	SE	Mean or %	SE	Mean or %	SD/ SE	
Health Outcome									
Asthma Diagnosis	0.06	(0.01)	0.08	(0.04)	0.06	(0.05)	0.04	(0.03)	< 0.001
Individual Characteristics									
Age at Visit	6.04	(0.01)	5.89	(0.02)	6.04	(0.01)	6.11	(0.01)	< 0.001
Gender									
Male	0.51	(0.01)	0.51	(0.02)	0.51	(0.01)	0.51	(0.01)	< 0.001
Female	0.49	(0.01)	0.49	(0.02)	0.49	(0.01)	0.49	(0.01)	0.43
Race/ Ethnicity									
Non-Hispanic White	0.40	(0.01)	0.09	(0.01)	0.39	(0.01)	0.55	(0.01)	< 0.001
Non-Hispanic Black	0.13	(0.01)	0.25	(0.01)	0.15	(0.01)	0.06	(0.01)	< 0.001
Hispanic	0.24	(0.01)	0.51	(0.01)	0.23	(0.01)	0.11	(0.01)	< 0.001
Asian/ Other Race	0.05	(0.01)	0.02	(0.01)	0.04	(0.01)	0.09	(0.01)	< 0.001
Missing	0.18	(0.01)	0.14	(0.01)	0.18	(0.01)	0.19	(0.01)	< 0.001
Health Insurance									
Private Provider	0.55	(0.01)	0.23	(0.01)	0.54	(0.01)	0.71	(0.01)	< 0.001
Public Provider	0.21	(0.01)	0.51	(0.01)	0.22	(0.01)	0.06	(0.01)	< 0.001
Other/ Missing	0.24	(0.01)	0.26	(0.01)	0.25	(0.01)	0.23	(0.01)	< 0.002
Exposure									
Particulate Matter (PM)	10.36	(0.01)	10.45	(0.01)	10.30	(0.01)	10.24	(0.01)	< 0.001
Ozone (O3)	25.92	(0.01)	24.86	(0.01)	26.27	(0.01)	25.95	(0.01)	< 0.001
***N* =**	206,974		38,919		84,872		83,183		

Source: Data are from Pediatric Health Records & Texas Commission of Environmental Quality (TCEQ).

Note: Significance is evaluated using two-tailed independent means t test, simple linear regression, or ANOVA

Table 3. Estimated Coeffecients from OLS Predicting Asthma Outcomes

	Disdvantaged		**Middle-class**		**Advantaged**		**p-value**
	Coef.	Std. Error	Coef.	Std. Error	Coef.	Std. Error	**for diff.**
Intercept	0.060***	(0.00)	0.048***	(0.00)	0.043***	(0.00)	0.000***
Individual Characteristics							
Age at Visit	0.005***	(0.00)	0.003***	(0.00)	0.002***	(0.00)	0.000***
Gender (Female, ref)							
Male	-0.020***	(0.00)	-0.019***	(0.00)	-0.016***	(0.00)	0.30
Race/ Ethnicity (non-Hispanic white, ref)							
Non-Hispanic Black	0.088***	(0.00)	0.067***	(0.00)	0.058***	(0.00)	0.000***
Hispanic	0.010*	(0.00)	0.009***	(0.00)	0.016***	(0.00)	0.136
Asian/ Other Race	0.005	(0.01)	0.010**	(0.00)	0.011***	(0.00)	0.785
Missing	0.011	(0.01)	0.003	(0.00)	0.001	(0.00)	0.123
Health Insurance (Private Provider, ref)							
Public Provider	0.024***	(0.01)	0.031***	(0.00)	0.030***	(0.00)	0.249
Other/ Missing	-0.006	(0.01)	-0.013***	(0.00)	-0.016***	(0.00)	0.036*
Exposure							
Particulate Matter ($PM^{2.5}$)	0.003	(0.01)	-0.002***	(0.00)	-0.010***	(0.00)	0.015*
Ozone (O^3)	0.004	(0.01)	-0.000	(0.00)	0.006***	(0.00)	0.000***
n =	38,919		84,872		83,183		

Source: Data are from Census, American Community Survey (ACS) , Texas Commission of Environmental Quality (TCEQ), and Pediatric Health Records.

*** p <0.001, **p<0.01, *p<0.05

Note: Reference for p-value for difference is Disadvantaged.

Table 4. Regression Decomposition of Childhood Asthma, by Neighborhood Type

	Disadvantaged vs. Advantaged		**Disadvantaged vs. Middle-class**		**Middle-class vs. Advantaged**	
	Composition	**Association**	**Composition**	**Association**	**Composition**	**Association**
Variable						
Age at Visit	-0.001	0.000	0.004**	-0.001	0.007**	-0.001
Gender						
Female	0.000	-0.001	0.000	0.000	0.000	-0.001
Male	0.000	0.001	0.000	0.000	0.000	0.001
[∑ gender effect]	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]
Race/ Ethnicity						
Non-Hispanic White	-0.022**	0.011***	-0.017**	0.006**	-0.005*	0.004**
Non-Hispanic Black	-0.005	-0.001	-0.002	0.000	-0.002	-0.001
Hispanic	-0.013**	-0.002	-0.007	-0.002	-0.003	0.000
Asian/ Other Race	0.003	-0.001	0.001	0.000	0.002	0.000
Missing	0.002	-0.002	0.001	-0.001	0.000	-0.001
[∑ race effect]	[-0.035]	[0.005]	[-0.024]	[0.002]	[-0.008]	[0.001]
Health Insurance						
Private Provider	-0.010**	0.003*	-0.009*	0.001	-0.003	0.002*
Public Provider	-0.016**	0.000	-0.009*	0.000	-0.006*	0.000
Other/ Missing	-0.001	-0.001	0.000	-0.001	0.000	-0.001
[∑ economic effect]	[-0.028]	[0.002]	[-0.018]	[0.000]	[-0.009]	[0.001]
Exposure						
Particulate Matter ($PM^{2.5}$)	0.001	-0.000	0.011**	-0.010*	-0.001	0.010**
Ozone (O^{3})	-0.008	-0.000	0.028**	-0.017*	-0.007*	-0.014*
Intercept		0.014***		0.023***		0.008***

Source: Data are from Census, American Community Survey (ACS) , Texas Commission of Environmental Quality (TCEQ), and Pediatric Health Records.

*** p <0.001, **p<0.01, *p<0.05