Can Neighborhood Social and Environmental Context Account for Race/Ethnic Disparities in Childhood Asthma? Using Geocoded Medical Records to Explore an Ecological Model of Asthma

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Race and ethnic disparities in child asthma persist and are unexplained by a multitude of individual and family-level factors. Existing work which incorporates neighborhood structural and environmental conditions suffers from small sample sizes, especially of children clustered within the same neighborhood; and poor measures of environmental quality. We improve on existing work by utilizing children's clinical records linked with innovative techniques to assign environmental data to the children's Census tracts. Utilizing a clinical sample of 14,331 pediatric patients aged 2-17 who visited a University of Texas Health clinic in 2011-2012 in Houston, TX, including more than 118,000 emergency room (14%), outpatient (53%), and inpatient (33%) visits, we are able to identify children who have physician-diagnosed asthma and other respiratory conditions and link them to their residential neighborhoods. We find robust associations between particulate matter (PM) concentrations and the likelihood a child has asthma, after accounting for neighborhood poverty and a limited set of individual covariates.

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Extended Abstract

Asthma prevalence among children in the U.S. has grown rapidly over the past few decades, increasing from just 3.6% of all children in 1980 to 10% of all children by 2010 (Akinbami et al. 2009). While the etiology of asthma is complex and remains largely unknown, research has identified key risk factors at the individual and environmental level (Lovasi et al. 2008). Specifically, asthma prevalence is especially high in low-income urban communities (Lovasi et al. 2008; Busse and Mitchell 2007) and varies substantially across race/ethnic groups (Gupta et al. 2006; Holt et. al 2009; Black et. al 2012), such that 22% of non-Hispanic black children were ever diagnosed with asthma in 2012 compared to 14% of Hispanic and 12% of non-Hispanic white children (National Center for Health Statistics 2012). Because racial minorities, including non-Hispanic Black children, are more likely concentrated in low-income neighborhoods compared to non-Hispanic whites (Williams and Collins 2001; ?), research has yet to fully disentangle the influence of race/ethnic status from environmental factors that put children at a high-risk for asthma prevalence (Liu et. al 2009).

An ecological approach assumes that neighborhood context impacts pediatric asthma through direct and indirect pathways. For example, exposure to various types of outdoor air pollution, like traffic-pollution or high levels of PM_{10} (particulate matter) can increase asthma incidents and worsen respiratory symptoms in asthmatic children (Jarrett et. al 2008; Wilhelm et. al 2009). Wilhelm et al. (2009) find using survey data that children residing in high ozone (0_3,), particulate matter (PM_{10}) and carbon monoxide (CO) areas in Las Angeles had increased asthma morbidity, even after adjusting for various family- and neighborhood-level characteristics. While this study finds only marginal associations between air pollution and neighborhood quality, some evidence suggests that low-income and minority neighborhoods have

disproportionately higher levels of air pollutants (Young et al. 2012; Houston et al. 2004), raising largely unexplored questions about the role of air quality in worsening health disparities among vulnerable populations.

Neighborhood-level factors may also influence pediatric asthma outcomes through indirect pathways, including exposure to chronic stress or via the health behaviors of residents (Wilhelm et al. 2009; Gold and Wright 2005). Residents in disadvantaged neighborhood are exposed to a number of community-level chronic stressors such as poverty, high crime, violence and unemployment, and research finds that children who are exposed to acute and chronic stress experience worse asthma outcomes (e.g. more vulnerable to inflammation, less responsive to asthma medications) (Chen et al. 2006; Miller, Chen and Cole 2009). Disadvantaged neighborhoods may also influence health behaviors of residents, like increased smoking rates as a stress coping mechanism, which in turn, worsen children's asthma. At least one study finds that neighborhood-level education (measured as percent with at least a high school degree) is a protective factor for childhood asthma, even after adjusting for key family and neighborhood characteristic like parental education level (Holt et al. 2013). Neighborhood education may thus capture an aspect of social capital important for increasing knowledge and access to collective resources, like local health care services and medical care (Holt et al. 2013; Gale et al. 2011).

However, in general research on the importance of neighborhood context for the prevalence and severity of childhood asthma is mixed, especially in terms of which aspects of the environment are key risk factors (Juhn et .al 2005; Beck et al. 2012; Holt 2013). It is even less clear whether significant race/ethnic disparities in childhood asthma remain once key individual and environmental factors (i.e. neighborhood characteristics and exposure to air pollution) are considered. This is likely because among the studies that consider race/ethnic status,

neighborhood context and childhood asthma, there is substantial variation in which neighborhood measures are included (e.g. Wilhelm et. al 2009; Liu and Pearlman 2009), whether the sample size of children is sufficiently large enough to capture race/ethnic differences (e.g. Chen et. al 2003), and how asthma prevalence and severity is reported (e.g. parent-reported or physician-diagnosed) and measured (Wilhelm et. al 2009; Holt et. al 2013; *Black et. al 2012). For instance, although some studies find little to no association between neighborhood context and childhood asthma (Chen et al. 2003; Holt et. al 2013), none of these studies account for exposure to air pollution (for an exception see Wilhelm et. al 2009), which has been identified as a risk factor for asthma prevalence and morbidity. At least one study finds that race/ethnic disparities in childhood asthma remain after accounting for various neighborhood factors, although this study uses a crude measure of neighborhood context (i.e. zip codes) and is unable to account for exposure to air pollution (Pearlman et. al 2006). To address these issues and build upon prior research, we utilize clinical records from a diverse group of children in Houston, TX and link this medical data with the children's social and environmental neighborhood characteristics at the Census tract level.

Data and Methods

To assemble our clinical sample, we extract the medical records of approximately 22,500 pediatric patients aged 0-17 who visited a University of Texas Health clinic in 2011-2012 in Houston, TX. Using these medical records, which constitute over 118,000 separate visits, including emergency room (14%), outpatient (53%), and inpatient (33%) visits, we are able to identify children who have physician-diagnosed asthma and other respiratory conditions. The ability to incorporate ER, inpatient *and* outpatient visits enables us to generate a more complete picture of the association of environmental correlates with child health in Houston than studies

which rely upon ER or inpatient data alone (cites), or those which rely on parent-report data from surveys. Our final analytical sample is 14,331 children ages 2-17 who are not missing on race/ethnicity.

Using a careful procedure of de-identification and subsequent geocoding using ArcGIS, we match children's home addresses with their Census 2010 tracts. These tract identifiers are then merged with Census 2010 and ACS 2006-2011 data to create a rich portrait of the social and economic conditions in each neighborhood. Next, tract identifiers are used to merge air quality data characterizing particulate matter concentrations for each child's neighborhood. Air quality data comes from the Texas Commission of Environmental Quality (TCEQ). We generate 3-year $PM_{2.5}$ concentration averages centered on the year of patient visit (i.e. 2010-2012 for 2011 patients and 2011-2013 for 2012 patients) using daily data from 11 monitoring stations in Houston-Galveston area, TX. Using interpolation (a method of constructing new data point within the range of discrete known data points), and established best practices, we can predict values of PM concentration around a 50 kilometer radius of these stations. The underlying premise of interpolation using ordinary kriging is that things closer together will be more similar than those that are farther apart. Thus, the certainty of the interpolation declines as distances exceed the 50 km radius. This is problematic for the west side of Houston, as the city sprawls considerably and contains very few monitoring stations. Thus, our original models performed poorly on the west side. We experimented with adding additional PM data from cities such as San Antonio and Austin to increase the amount of data to the west of Houston and our models performed better on various diagnostic tests. Ultimately, this impacts 14 census tracts where patients reside and we have elected to present these results but will conduct sensitivity tests which preclude patients in the tracts. From these interpolations we develop a PM Index

classifying census tracts as high, medium, and low PM exposure based on tertiles of PM for the entire Houston area. The map included in the appendix shows the distribution of PM throughout the Houston metropolitan area from our interpolation models.

Measures

Children who visited UTHealth clinics in 2011-2012 and who had received a diagnosis of asthma (ICD9 codes 493.0-493.92) or a respiratory disease more broadly (ICD9 codes 460.0-519.9; includes asthma and also acute respiratory infections, pneumonia, and chronic lung conditions) are coded dichotomously as having that condition. Because our individual level data comes from medical records, we are extremely limited in the individual-level covariates we can include in our models. Patient's gender (1 = male), age (in years), race/ethnicity (Non-Hispanic White (reference), Non-Hispanic Black, Hispanic, and Other), and insurance status (No insurance, public insurance, and private insurance (reference)) are the only available covariates.

At the neighborhood level, we plan to account for a concentrated disadvantage index which incorporates neighborhood poverty, percent black, unemployment, female-headed households, and public assistance receipt. For now, we include a measure of neighborhood poverty, with neighborhoods divided into tertiles (low (reference), medium, and high poverty). Our PM index is likewise categorized as low (reference), medium, and high PM.

Analysis

We assess our research questions using multi-level logistic regression models (Rabe-Hesketh and Skrondal 2008). Multilevel models treat level-1 individuals as nested within level-2 census tracts (which include both the air quality score and the sociodemographic data).

All models utilize maximum likelihood estimation with adaptive quadrature (Rabe-Hesketh and Skrondal 2008), adjusting for clustering by neighborhood, different sample sizes for

level-1 and level-2 units, heteroscedastic error terms, and varying numbers of cases within level-2 units – all problems that otherwise downwardly bias estimated standard errors (Raudenbush and Bryk 2002). The multilevel model for binary outcomes adds to a traditional logit model with the inclusion of a neighborhood-level error component (u_j). For example, the following equation represents the probability of heart attack, allowing odds to vary across neighborhoods and includes individual-level (x_{ij}) and neighborhood-level (z_j) explanatory variables:

$$\log [P_{ij} / (1 - P_{ij})] = \beta_0 + \beta_1 x_{ij} + \beta_2 z_j + u_j \qquad (1)$$

The probability (P_{ij}) that the *i*th patient in the *j*th neighborhood has asthma is determined in equation 1, where β_0 is the model intercept, $\beta_1 x_{ij}$ is a level 1 (individual) predictor, $\beta_2 z_j$ is a level 2 sociodemographic predictor (i.e. poverty) or a level 2 air quality predictor, and u_j is the random effect of neighborhoods on asthma risk. Error across neighborhoods is captured by a level-2 residual term with a mean of zero and an unknown variance, σ_u^2 (McCulloch and Searle 2001). This level-2 residual can be used to estimate the extent to which residual variation in the log-odds of asthma is situated within or between neighborhoods. To address our first objective, we will assess the independent effects of the neighborhood air quality index on the odds of asthma and other respiratory conditions while holding other measures of the patient's individual and neighborhood sociodemographic characteristics constant. Next, we will account for neighborhood poverty and determine whether any observed impact of PM at the neighborhood level on the risk of asthma and other respiratory conditions is sustained. Next, we will assess race/ethnic disparities in asthma before and after accounting for these neighborhood social and environmental factors, seeking to answer the question – does accounting for a child's neighborhood social and environmental factors alter race/ethnic disparities in asthma and respiratory conditions?

Preliminary Results (In Brief)

Table 1 presents descriptive statistics for our analysis file of pediatric patients ages 2-17 in Houston, TX. On average, the children are 9.5 years old, and about one-quarter have no insurance. Another 55% have public insurance such as Medicaid or SCHIP, and 21% are privately insured. Insurance status varies widely by race/ethnicity; such that 51% of Non-Hispanic White children have private insurance, relative to 12% of Non-Hispanic Black children, 7% of Hispanic children, and 21% of Other race/ethnicity children. Non-Hispanic Black children are the most likely to receive public insurance, at 73.5%. In terms of our PM measure, 60% of the children in our sample live in a "High" PM neighborhood, while just 16% live in a "Low" PM neighborhood. This varies, however, again by race/ethnicity, such that Non-Hispanic White children are least likely to live in a High PM neighborhood (31.3%), relative to Non-Hispanic Black children (71%), Hispanic children (72.5%), and Other race/ethnicity children (54.1%). We also tested (not shown) how children's insurance status was associated with PM category; and 32% of privately insured children, 68% of publicly-insured children, and 65% of children with no insurance live in High PM neighborhoods. This distribution of PM by insurance status and race/ethnicity indicates that in Houston, as in other major metropolitan areas, PM concentrations may be highest in more disadvantaged neighborhoods. Finally, the poverty status of neighborhoods also varies by race/ethnicity, such that 13% of Non-Hispanic White children live in high poverty neighborhoods, compared to 55% of Non-Hispanic Black children, 48% of Hispanic children, and 36% of Other race/ethnicity children.

Table 2 presents preliminary results of multilevel logistic regression models predicting "child has asthma." In Model 1, we include only the level-2 PM categorical predictor, and see

substantial effects of PM concentration on a child's risk of asthma. Children living in neighborhoods with high PM have 2.27 times the odds of asthma, and children living in neighborhoods with medium PM concentrations have 1.52 times the odds of asthma, relative to children living in low PM neighborhoods. In model 2, we add neighborhood poverty to try to separate the impact of neighborhood-level socioeconomic disadvantage from the impact of PM concentration. The impact of PM on asthma risk is only slightly attenuated by accounting neighborhood poverty, and as expected, children in higher poverty neighborhoods have increased risk of asthma.

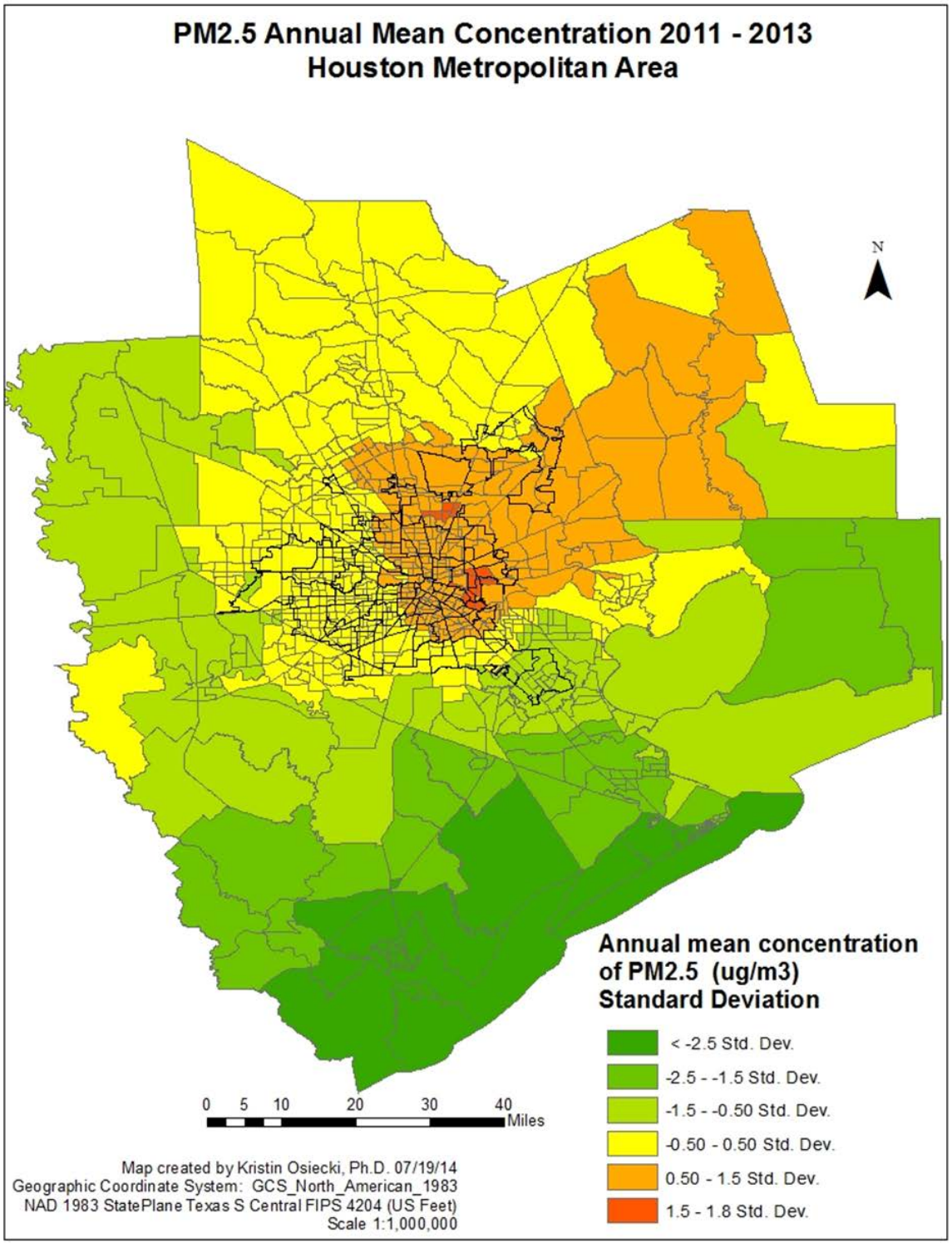
Model 3 includes only the race/ethnicity covariates, and here we see, similar to other studies, that Non-Hispanic Black children have more than three times the odds of asthma relative to White children, and Hispanic children have 1.49 times the odds. In Model 4, we investigate whether accounting for neighborhood PM concentration impacts the race/ethnic disparities in asthma risk, and we see some evidence that it does. Black children's log odds of asthma are reduced by about 14% after accounting for neighborhood PM concentration, and Hispanic children's odds relative to White children's are reduced to marginal significance. Model 5 adds neighborhood poverty to the model, and we see no further attenuation of the race/ethnic disparities in asthma risk, indicating that neighborhood poverty does not do much to explain Black children's increased risk relative to white children. Finally, in Model 6 we add child's gender, age, and insurance status. As expected, children with no or public insurance have elevated odds of asthma relative to children who are privately insured. In addition, adding the individual-level covariates reduces the black-white gap in asthma risk by an additional 10%, but it still remains very stark at almost 2.5 times as high for Black children.

Table 3 presents results from identical sets of models predicting any respiratory condition. Results are quite similar to those for asthma; with the exception that the Black-White gap in respiratory conditions is smaller, and a persistent gap for Hispanics and Whites emerges such that Hispanics have higher odds of respiratory conditions. Across all models, as for asthma, living in neighborhoods of moderate and high PM, relative to low PM, increases the odds of respiratory conditions for children.

Table 1: UT Health Data Sample Descriptives, Geocoded Children Ages 2-17; 2011-2012 (N = 14,113 unique patients)					
	Full Sample	NH White	NH Black	Hispanic	Other
	% or Mean (SD)	% or Mean (SD)	% or Mean (SD)	% or Mean (SD)	% or Mean (SD)
Male	52.4	52.5	52.6	52.0	52.7
Age	9.5 (4.9)	10.1 (4.9)	9.0 (5.0)	9.8 (4.9)	8.7 (4.8)
Insurance Type					
None	24.1	18.0	14.9	39.2	16.5
Public	55.4	31.2	73.5	53.8	62.7
Private	20.5	50.8	11.6	7.0	20.8
Health Outcomes					
Asthma	6.3	3.5	10.6	5.2	4.5
Respiratory Condition	22.5	14.6	28.2	24.0	19.7
Neighborhood Measures					
PM Categories					
Low	15.7	32.8	10.3	7.3	19.6
Mid	24.3	35.9	18.9	20.3	26.3
High	60.0	31.3	70.8	72.5	54.1
Poverty Categories					
Low	26.8	60.2	12.6	14.8	29.9
Mid	33.1	26.8	32.9	37.3	34.6
High	40.1	13.1	54.6	47.9	35.5
N	14,113	3,332	4,168	4,794	1,819
14,113 patients who live in 668 tracts. Average of 21 patients per tract.					

Table 2: Multilevel Logistic Regression Models Predicting "Child Has Asthma Diagnosis" (ages 2-17)												
	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	b		b		b		b		b		b	
Tract-Level PM (3 year average)												
Low PM	(ref)		(ref)				(ref)		(ref)		(ref)	
Mid PM	0.42	**	0.39	**			0.38	**	0.39	**	0.34	*
High PM	0.82	***	0.70	***			0.63	***	0.64	***	0.57	***
Tract-Level Poverty (2006-2011 ACS)												
Low Npov			(ref)						(ref)		(ref)	
Mid Npov			0.31	**					0.11		0.00	
High Npov			0.29	*					0.00		-0.14	
Individual-Level Characteristics												
(White)					(ref)		(ref)		(ref)		(ref)	
Black					1.18	***	1.02	***	1.01	***	0.90	***
Hisp					0.40	***	0.23	+	0.22	+	0.15	
Asian					0.25	+	0.16		0.15		0.05	
Child is male											0.35	***
Child's age (in years)											-0.04	***
Insurance Type												
None											0.35	*
Public											0.47	***
(Private)											(ref)	
_cons	-3.39	***	-3.53	***	-3.319	***	-3.681	***	-3.72	***	-3.68	***
N = 14,113												

Table 3: Multilevel Logistic Regression Models Predicting "Child Has Respiratory Diagnosis" (ages 2-17)												
	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	b		b		b		b		b		b	
Tract-Level PM (3 year average)												
Low PM	(ref)		(ref)				(ref)		(ref)		(ref)	
Mid PM	0.30	***	0.29	***			0.25	**	0.26	**	0.25	**
High PM	0.76	***	0.67	***			0.60	***	0.59	***	0.54	***
Tract-Level Poverty (2006-2011 ACS)												
Low Npov			(ref)						(ref)		(ref)	
Mid Npov			0.30	***					0.17	*	0.09	
High Npov			0.23	**					0.04		-0.08	
Individual-Level Characteristics												
(White)					(ref)		(ref)		(ref)		(ref)	
Black					0.77	***	0.63	***	0.61	***	0.55	***
Hisp					0.53	***	0.39	***	0.37	***	0.32	***
Asian					0.33	***	0.24	**	0.22	**	0.10	
Child is male											-0.02	
Child's age (in years)											-0.07	***
Insurance Type												
None											0.37	***
Public											0.27	***
(Private)											(ref)	
_cons	-3.39	***	-3.53	***	-3.32	***	-3.68	***	-3.72	***	-3.68	***
N = 14,113												


PM2.5 Annual Mean Concentration 2011 - 2013
Houston Metropolitan Area
N
0 5 10 20 30 40
Miles
Annual mean concentration of PM2.5 (ug/m3) Standard Deviation
< -2.5 Std. Dev.
-2.5 - -1.5 Std. Dev.
-1.5 - -0.50 Std. Dev.
-0.50 - 0.50 Std. Dev.
0.50 - 1.5 Std. Dev.
1.5 - 1.8 Std. Dev.
Map created by Kristin Osiecki, Ph.D. 07/19/14
Geographic Coordinate System: GCS_North_American_1983
NAD 1983 StatePlane Texas S Central FIPS 4204 (US Feet)
Scale 1:1,000,000