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ORIGINAL PAPER

Local variability in the impacts of residential particulate matter and pest exposure on children's wheezing severity: a geographically weighted regression analysis of environmental health justice

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Abstract Two assumptions have underpinned environmental justice over the past several decades: (1) uneven environmental exposures yield correspondingly unequal health impacts and (2) these effects are stable across space. To test these assumptions, relationships for residential pest and $PM_{2.5}$ exposures with children's wheezing severity are examined using global (ordinary least squares) and local [geographically weighted regression (GWR)] models using cross-sectional observational survey data from El Paso (Texas) children. In the global model, having pests and higher levels of $PM_{2.5}$ were weakly associated with greater wheezing severity. The local model reveals two types of asthmogenic socio-environments, where environmental exposures more powerfully predict greater wheezing severity. The first is a lower-income context where children are disproportionately exposed to pests and $PM_{2.5}$, and the second is a higher-income socio-environment where children are exposed to lower levels of $PM_{2.5}$, yet $PM_{2.5}$ is counterintuitively associated with more severe wheezing. Findings demonstrate that GWR is a powerful tool for understanding relationships between environmental conditions, social characteristics, and health inequalities.

Keywords Environmental health justice · Health disparities · Asthma · Geographically weighted regression · El Paso, Texas

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Introduction

Myriad studies have shown that minority and lower-income children suffer disproportionately from a host of health problems (e.g., Mehta et al. 2013). Minority and lower-income neighborhoods also tend to be burdened by the unequal distribution of hazard exposures (Downey 2006). Studies addressing these two hypothetically linked phenomena have remained largely distinct and a key question remains unanswered: *What is the role of exposure to environmental hazards in health disparities*? Studies investigating this linkage using a spatial approach have relatively recently begun to emerge under the rubric of "Environmental Health Justice" (EHJ) (Chakraborty and Maantay 2011; Corburn 2005; Grineski et al. 2013). This is in response to decades of environmental justice research that has extended from the poorly supported assumption that uneven environmental exposures yield correspondingly unequal health impacts and that these effects are stable across space.

EHJ researchers have reported that environmental degradation plays a role in predicting geographic inequalities in health outcomes (Grineski 2007; Grineski et al. 2013; Jephcote and Chen 2012, 2013; Pearce et al. 2010, 2011; Richardson et al. 2013), but not in the monotonic way that many researchers may have assumed it would. For example, in the UK, Pearce et al. (2010) found that the relationship between environmental deprivation and mortality was strongest in the most affluent areas and weakest in the poorest areas. In California, Hispanics experienced the greatest exposure to ozone and $PM_{2.5}$ as compared to blacks and whites, but they did not have the highest excess attributable risk of hospitalizations due to pollution exposure (Hackbarth et al. 2011). Grineski et al. (2013) found a significant association between air toxics and children's respiratory infections in El Paso neighborhoods after adjusting for relevant controls, but they did not find the same for asthma.

In addition to counterintuitive findings, EHJ work has been characterized by a reliance on secondary data sources. On the health side (see Wheeler and Ben-Shlomo 2005 for an exception), studies have relied on hospitalization data (e.g., Grineski et al. 2013; Jephcote and Chen 2012) and mortality records (e.g., Pearce et al. 2010), which are attributable to the public availability and low cost of these sources of health data. While generating important insights, this work has focused attention on serious health outcomes and has tended to require the analysis of aggregated data for areal units (e.g., neighborhoods). In terms of air quality, modeled criteria air pollution surfaces are publically available in the UK (Jephcote and Chen 2012; Pearce et al. 2010). These types of surfaces are not available from US governmental agencies, and therefore, they are rarely used by EHJ researchers working in the USA (see Grineski 2007 for an exception). This is a critical limitation given the deleterious health effects of these pollutants (Samet and Krewski 2007).

Recent studies raise the possibility that pollution–health linkages might vary widely across space (Pearce et al. 2010; Richardson et al. 2013). This makes geographically weighted regression (GWR) particularly suited to investigating EHJ, even though it is an "underused EJ technique" (Jephcote and Chen 2012, p. 141).

Unlike spatial autoregressive models, which account for spatial autocorrelation in generating parameter estimates (Chakraborty 2011), GWR models how relationships between variables vary across space (Fotheringham et al. 2002). To our knowledge, only three previous EJ studies focused on environmental hazards have used GWR (Gilbert and Chakraborty 2011; Jephcote and Chen 2012; Mennis and Jordan 2005). One study analyzed data from New Jersey and concluded that global models used by many are likely insufficient for modeling environmental injustice (Mennis and Jordan 2005). Most recently, Jephcote and Chen (2012) found that PM_{10} and ethnic minority status were stronger predictors of children's asthma hospitalization rates in the inner city of Leicester (UK) than they were in outlying areas. These findings underscore the point that environmental exposures may not impact people's health the same way in all locations and that the ways in which exposures impact health may be surprising.

While the majority of EJ studies focus on outdoor environments (e.g., factory releases and criteria air pollution), indoor environmental exposures can also be considered environmental injustices (Grineski and Hernandez 2010). Poor home environments have less often been considered in terms of environmental injustice in the past (see Kraft and Scheberle 1995; Landrigan et al. 2010 for exceptions), possibly because there is a tendency to assume that in-home conditions are products of independent household decisions, rather than power-laden products of social processes (Grineski and Hernandez 2010). Certainly, indoor environmental exposures (e.g., roaches, rodents, mold) are more prevalent in substandard housing inhabited by the poor (Matte and Jacobs 2000) and have been linked to respiratory symptoms at the individual (Lanphear et al. 2001) and neighborhood levels (Grineski 2007).

As is the case with air pollution, there is preliminary evidence that in-home exposures may not impact all children the same way. Low-income white children in New York (state) had higher rates of indoor risk factors than did middle class white children, and these risk factors were correlated with elevated overnight epinephrine, norepinephrine, and cortisol, but *only* in the low-income sample (Evans and Marcynyszyn 2004). Pooling five survey datasets, researchers found that the odds of exposure to household pests were significantly associated with asthma only for children born in the USA and not for children born outside the USA (Woodin et al. 2011). Those studies examined variation based on social characteristics—specifically, income and nativity. It remains unclear how the strength of the association between indoor exposures and respiratory health might vary across urban space.

This study makes several advances upon previous studies. First, we utilize individual-level data, which are rare in EJ studies, thus avoiding the problem of the ecological fallacy. Second, as opposed to relying on secondary hospitalization or mortality records, we use a wheezing symptoms severity measure, thus capturing a more common and broadly relevant health problem. This enables us to address Jephcote and Chen's (2012, p. 142) recent call "for future EJ research to develop upon [previous] GWR studies, through applying measurements of actual health events and exploring a wider range of cardiorespiratory conditions influenced by short-term exposures." Asthma hospitalizations, which are more often used in these types of studies, are relatively rare events; for example, the asthma hospitalization rate is 27 per 10,000 children in the USA (Akinbami 2007). Third, we utilize a $PM_{2.5}$ (particulate matter less than 2.5 micrometers in diameter) surface, generated through primary data collection. This allows us to move beyond US EPA-provided data and to analyze an important traffic-associated criteria air pollutant for which data are not currently publicly available in the USA. The associations between $PM_{2.5}$ and respiratory problems have been well-documented, and inhalation of this pollutant has been linked to inflammatory responses and pulmonary oxidative stress (Hansen et al. 2012). Fourth, we consider both indoor (pest exposure) and outdoor ($PM_{2.5}$) environmental conditions through an EHJ framework, which is rarely done.

As per prior EHJ research (Gilbert and Chakraborty 2011), our analysis approach relies on both aspatial and spatial modeling. We answer the following two research questions: (1) What are the global relationships for residential pest exposure and $PM_{2.5}$ with children's wheezing severity adjusting for relevant controls? (2) What is the degree of local spatial variation in the contribution of both residential pest exposure and $PM_{2.5}$ to children's wheezing severity adjusting for the relevant controls?

Data and methods

Study context

The study took place in El Paso County, Texas, which has an estimated population of 830,000 residents. According to the US Bureau of the Census, in 2011, 81 % of its residents were Hispanic (compared with 17 % for the USA and 38 % for TX), while smaller percentages were non-Hispanic white (14 %) and non-Hispanic black (4 %). El Paso County had a lower median household income (2011 US $36,333) than the State of Texas (2011 US $49,391) and the USA (2011 US $50,502) with a poverty rate of 24 %, which was higher than the national rate (16 %). In previous studies in this city, researchers have found relatively modest associations between air pollutants (including $PM_{2.5}$) and respiratory health effects (Grineski et al. 2011; Sarnat et al. 2011; Svendsen et al. 2012; Zora et al. 2013).

Survey data collection

Social and health data were collected through a cross-sectional, observational mail survey that was approved by our university's Institutional Review Board. The closed-ended questionnaire was sent to all primary caretakers (parents and guardians) of fourth and fifth graders attending school in the El Paso Independent School District (EPISD). With more than 64,000 students across 94 campuses, the EPISD is the tenth largest district in Texas and the 61st largest district in the USA (EPISD 2013). Children in the 4th and 5th grade from all 58 elementary schools are represented in the dataset.

Surveys were conducted to obtain the highest achievable response rates by personalizing communication, following up with non-respondents, and offering incentives (Dillman et al. 2009). All survey materials were provided to households in English and Spanish in three waves during May of 2012. Ultimately, 6,295 primary caretakers received surveys at their home address, and 1,904 surveys were returned for a 30 % response rate. Respondents were primarily mothers (82 %), with the next largest shares being fathers (10 %) and grandparents (4 %). Descriptive statistics for the percentages of surveyed children who are male (49.9 vs. 51.4 % in EPISD), Hispanic (82.2 vs. 82.6 % in EPISD), and economically disadvantaged (60.4 vs. 71.1 % in EPISD) indicate that the sample is generally representative of the EPISD student population (EPISD 2013).

Selection criteria

Of the 1,904 children surveyed, 1,736 were selected for inclusion in this study; 162 were excluded due to missing data for the analysis variables, and six were excluded as spatial outliers. We focus on children because of their sensitivity to air pollution. Childhood is a critical time in the development and maturation of the cardiorespiratory system, which is highly susceptible to the absorption of toxins (Jephcote and Chen 2013). A child's lung surface area is significantly larger relative to body mass than an adult's; children can breathe up to 50 % more air per kilogram of body weight. Children also tend to spend more time outdoors participating in activities that increase their breathing rates. When coupled with exposure to air pollutants, these factors create conditions conducive to damaging or stunting the development of children's cardiorespiratory systems, creating health problems which can prevail throughout adulthood (Schwartz 2004).

Dependent variable

The dependent variable is a composite measure of six wheezing measures based on data collected using International Study of Asthma and Allergies in Childhood (ISAAC) (ISAAC Steering Committee 2012) and National Asthma Survey questions (O'Connor et al. 2008). From the ISAAC, we used the following questions: (1) in the last 12 months, has the wheezing ever been severe enough to limit your child's speech to only one or two words at a time between breaths? (1 = yes, 0 = no); (2) in the last 12 months, has the child had wheezing or

Table 1 Component loadings and individual variable means for the six wheezing variables included in the "Current wheezing severity" measure

Variable	PCA component loadings	Individual variable mean
Wheezing limited speech	0.499	0.02
Doctor-diagnosed asthma	0.682	0.16
Asthma symptoms (including wheeze)	0.833	0.19
Night cough	0.514	0.24
Wheezing in sleep	0.785	0.07
Wheezing with no cold	0.821	0.08

$N = 1{,}736$

whistling in the chest when he/she did not have a cold or the flu? (1 = yes, 0 = no); (3) in the last 12 months, has your child's sleep been disturbed due to wheezing? (1 = yes, 0 = no); (4) in the last 12 months, has your child had a dry cough at night apart from cough associated with a cold or chest infection? (1 = yes, 0 = no); and (5) has the child ever been told by a doctor or health professional that he or she has asthma? (1 = yes, 0 = no). From the National Asthma Survey, we used this question: (6) symptoms of asthma include coughing, wheezing, shortness of breath, chest tightness, or phlegm production when someone does not have a cold or respiratory infection. How long has it been since your child had any symptoms of asthma? (1 = less than 1 year ago, 0 = more than 1 year ago). We created a composite measure of current "wheezing severity" using principal components analysis on these six variables (see Table 1 which includes factor loadings), after standardizing all items. The eigenvalue for the one component was 2.96 and it explained 49.4 % of the variance. Due to skewness and kurtosis, we used a natural log transformation on this variable (after first adding 1 to make all values positive). Descriptive statistics are presented in Table 2.

Independent variables

The primary variables of interest are residential pest (indoor) and $PM_{2.5}$ (outdoor) exposure. Pest is a dichotomous variable which is coded 1 if the caretaker reported being troubled by rats (1 %), ants (18 %), mice (2 %), spiders (6 %), cockroaches (14 %), termites (1 %), and/or another pest (3 %) inside the home in the past 12 months, and 0 if she did not (55 %). Biological agents, including allergens from cockroaches and rodents, are among the most prominent environmental factors

Table 2 Descriptive statistics of variables used in analysis

Variable (continuous)	Min.	Max.	Mean.	St. Dev.
Current wheezing severity (ln)[a]	−0.73	1.71	−0.29	0.67
Residential $PM_{2.5}$ (ln)	1.75	2.94	2.10	0.25
Variable (dichotomous)	**Response**	**Frequency**	**Percent**	
Home has pest(s)	Yes (1)	781	45	
	No (0)	955	55	
Child is male	Yes (1)	868	50	
	No (0)	868	50	
Mother has asthma and/or allergies	Yes (1)	573	33	
	No (0)	1,163	67	
Child has allergies	Yes (1)	885	51	
	No (0)	851	49	
Postponed or did not seek health care due to concerns about cost	Yes (1)	417	24	
	No (0)	1,319	76	
Child is Hispanic	Yes (1)	1,424	82	
	No (0)	312	18	

$N = 1{,}736$

[a] 1 was added to the wheezing measure before it was natural logged

implicated in asthma morbidity and poor housing conditions are associated with exposure to these asthma-inducing biological agents. Roaches and rodents are also associated with excess moisture in the home, which can also trigger wheezing and asthma (Derose et al. 2009). Indoor exposures play at least two roles in asthma: (1) as a risk factor for genetically susceptible individuals and (2) as a source of ongoing airway inflammation and hypersensitivity to other irritants (Rauh et al. 2008). In using this variable, we assume that reporting "being troubled" by one or more of the pests maps to a problem with pests in the home, and not just the occasional spider or ant and that it is associated with poor housing conditions more generally. Also, the "pest" variable was more strongly correlated with wheezing severity than was roaches alone.

Residential $PM_{2.5}$ values for outdoor environments at each child's home site were extracted from a 2012 exposure surface created via land use regression (LUR) modeling for this project (as per Jerrett et al. 2007; Olvera et al. 2012). $PM_{2.5}$ measurements were collected via a 26 site monitoring network designed using a location-allocation approach (as per Kanaroglou et al. 2005). The location-allocation method used an existing exposure surface from 2006–2009 to determine an optimal monitoring network (Olvera et al. 2012). We collected one 14-day averaged $PM_{2.5}$ sample at each site per season ($n = 4$) during 2012. Monitoring began in May 2012 to coincide with the survey. $PM_{2.5}$ samples were collected on Teflon filters with multi-stage impactors and concentrations were determined via gravimetric analysis (Olvera et al. 2012). The four seasonal $PM_{2.5}$ concentrations were averaged to produce annual estimates, which are appropriate for use in LUR models (Gerard Hoek et al. 2002). A linear regression model was built with $PM_{2.5}$ at each of the 26 sites as the dependent variable and surrounding land use, traffic, and physical characteristics as predictors (i.e., traffic counts, vehicle miles traveled, land use, property values, population density, distance to the international border, and elevation summarized for circular areas around the monitoring locations).

The relative fit of the model was determined by a R^2 of 0.458, which is good for these types of models (Hoek et al. 2008; Javier et al. 2007; Olvera et al. 2012). The absolute fit represented by the root mean square error was obtained via a leave-one-out bootstrap analysis for 1,000 samples (Isakov et al. 2012; Javier et al. 2007; Rose et al. 2010a, b) and was found to be 0.93 (95 % CI 0.56–1.09). The samples were generated via a "leave-some-out" cross-validation technique. Specifically, we randomly sampled 100 different observation sets from our 26 site observations by leaving out up to three sites each time. Technically, we could have sampled 2,600 different samples from our 26 sites, but for practical purposes, we stopped at 1,000. The samples were used to generate the model and the rest to test it. This validation method has been widely used in LUR studies and has been shown to be adequate and robust for such purposes (Isakov et al. 2012; Johnson et al. 2010; Parenteau and Sawada 2012; Rose et al. 2010a, b). Considering a mean $PM_{2.5}$ concentration of 7.2 $\mu g/m^3$ across the region, the accuracy of the estimates is ± 13 % (95 % CI ± 7.2 to ± 15). Additional details of the monitoring procedures and the LUR modeling technique used can be found elsewhere (Jerrett et al. 2007; Olvera et al. 2012).

This approach generated $PM_{2.5}$ values for 2,193 points on a 500-m grid. We then used inverse distance weighting (IDW) with a distance decay function of 2 to create a continuous $PM_{2.5}$ surface (see Fig. 1) and each child was given the value of $PM_{2.5}$ corresponding to his or her home location. We used 2 as the coefficient because it produced a robust $PM_{2.5}$ surface for the USA in a previous study (Al-Hamdan et al. 2006). Also, the IDW technique was use to interpolate $PM_{2.5}$ values estimated via the LUR model over a grid of 500-m resolution. Hence, the value of 2 ensured that the contributions of more distant observations to the weighted interpolated value were very small. In the analysis, a natural log transformation was used to correct for skewness and kurtosis in $PM_{2.5}$; a standardized version of this variable is used in the models. The pest and $PM_{2.5}$ variables are summarized in Table 2.

Control variables

Five control variables were selected based on relevant literature and contextual relevance to El Paso; we also prioritized selecting variables for which the proportions of missing data were lower being that the GWR tool in ArcGIS cannot handle multiply imputed datasets at this time. These control variables include sex (Wright et al. 2006); whether the biological mother has asthma and/or allergies (Hryhorczuk et al. 2009); and whether the child has allergies or not (Holt et al. 2013; Kocevar et al. 2005). We used an indicator of the family having postponed or avoided seeking health care for the child because of concerns about cost. We do not adjust for household income or parental education due to missing data. This variable

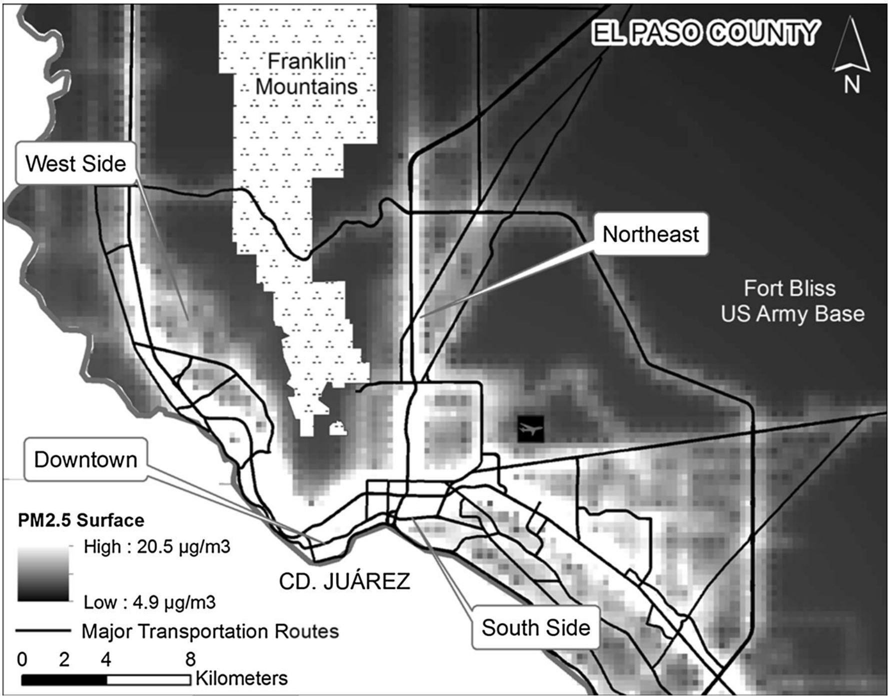

Fig. 1 $PM_{2.5}$ surface and major transportation routes in El Paso County, Texas

represents a SES-related access to care indicator. Given the Hispanic majority context of this study and lower rates of asthma among this population, we include an indicator of Hispanic ethnicity (Padilla et al. 2009); we do not account for membership in any other racial/ethnic groups due to small counts. Descriptive statistics for all variables are included in Table 2.

Analysis methods

These data are analyzed in two steps, following other GWR EHJ studies (Gilbert and Chakraborty 2011; Jephcote and Chen 2012) and in accordance with the two research questions. The starting point for development of a GWR model is the ordinary least squares (OLS) multiple regression equation that expresses the relationship between the dependent variable and a combination of independent variables simultaneously in a single model (Gilbert and Chakraborty 2011). We first used OLS, an aspatial multivariate regression technique, to identify the important overall predictors of wheezing severity in El Paso. This provides what researchers who conduct spatial regression analysis term "global" findings because OLS models assume that the correlations are constant over space. This means that every independent variable has one regression coefficient that maps to the average situation for all the observations in the study area (Tu et al. 2012).

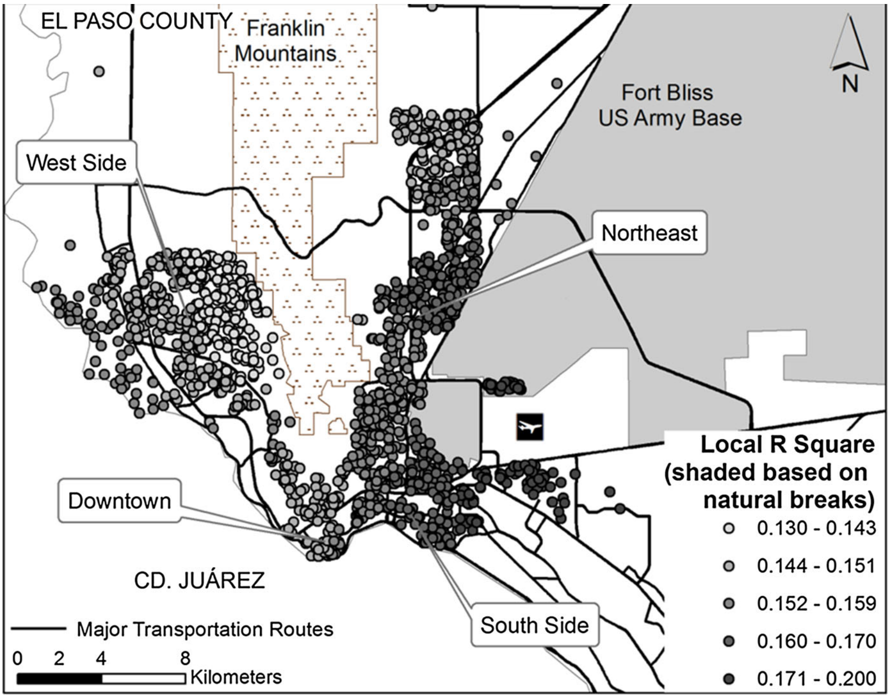

Fig. 2 Study area map including GWR model performance statistics (i.e., local R^2). *Note* "West Side," "Northeast," "South Side," and "Downtown" are used locally to refer to the labeled regions of the city

Second, we used geographically weighted regression (GWR) to uncover "local" spatial relationships. The significance of local spatial relationships is predicated on the concept of spatial non-stationarity, which means that "the measurement of a relationship depends… on where the measurement is taken" (Fotheringham et al. 2002, p. 9). To model non-stationarity, we use GWR in a geographic information system (GIS) to calculate individual regression equations for each data point, using the surrounding points (Mennis and Jordan 2005). Points, in our case, are geocoded addresses of children participating in this study. GWR uses a distance decay function, which assumes that observations closer to a given point will have stronger influences on the local parameter than points further away (Tu et al. 2012).

Either a fixed or adaptive kernel bandwidths can be used to generate the local parameter estimates. Fixed kernels rely on a constant bandwidth for all the observations, while adaptive kernels modify the size of the bandwidth based on spatial variations in the density of observations. With adaptive kernels, longer bandwidths are used in areas where data points are sparser, and shorter bandwidths are employed in areas with a greater density of points (Tu et al. 2012). See Fig. 2 for visual display of the approximate locations of children's home sites (points) under study. Clearly, the density of observations varies over the study area; thus, the adaptive kernel bandwidth was used. Following others (Chalkias et al. 2013; Tu et al. 2012), the optimal bandwidth was determined by minimizing the corrected Akaike Information Criterion (AICc). Additional details regarding GWR (including equations) can be found in the literature (Fotheringham et al. 2002; Mennis and Jordan 2005).

We used the readily available ArcGIS 10 Spatial Statistics tools to run both the global and local models. For OLS models, ArcGIS provides the parameter estimates, standard errors, *t* statistics, and *p* values as well as robust versions of these measures; a measure of multicollinearity, the Koenker statistic (significance indicates robust *p* values should be used and that relationships between some or all

Table 3 OLS and GWR results: predicting children's current wheezing severity

	A. OLS model				B. GWR model		
Adjusted R^2	0.152				0.163		
Variable	β	Robust SE	Robust p value	VIF	Mean β	Min β	Max β
Intercept	−0.647	0.043	0.001	–	0.605	−0.692	−0.512
Male (vs. female)	0.091	0.029	0.002	1.011	0.084	0.033	0.135
Mom has asthma a/o allergies	0.130	0.037	0.001	1.147	0.125	0.009	0.207
Child has allergies	0.439	0.031	0.001	1.140	0.449	0.400	0.495
Postpone health care/cost concerns	0.120	0.037	0.002	1.020	0.110	0.047	0.167
Child is Hispanic (vs. not Hispanic)	−0.004	0.041	0.922	1.070	−0.002	−0.070	0.060
Home has pest(s)	0.040	0.030	0.177	1.014	0.030	−0.049	0.096
Residential PM (ln, Z)	0.012	0.015	0.411	1.073	0.017	−0.053	0.042

$N = 1{,}736$

of the independent variables and the dependent variable are non-stationary); model fit statistics (e.g., R^2), and the ability to test model standardized residuals for spatial autocorrelation. In our case, these OLS diagnostics revealed the absence of a multicollinearity problem, non-normally distributed residuals, and a significant Koenker statistic. For that reason, robust p values are presented in Table 3. The residuals did not exhibit significant autocorrelation based on a global univariate Moran's I test ($I = -0.008$, $p = 0.866$) indicating that the OLS model was appropriate for these data. However, because of our theoretical interest in exploring non-stationarity (i.e., how relationships vary over space) as per research question 2, we ran a GWR model as the next step.

For the GWR results, ArcGIS produces a local parameter estimate, a local R^2 value, and a local standardized residual for each point in the dataset; model fit statistics (e.g., R^2) are also provided for the model as a whole. In our model, a Moran's I test of the residuals revealed insignificant clustering ($I = -0.003$, $p = 0.959$) meaning that there were no spatial dependencies in the residuals. A notable aspect of running GWR in ArcGIS is the fact that no p values are calculated for the individual parameter estimates. This contrasts with OLS where it is conventional to test whether parameter estimates are different from 0 using a t test. Utilizing such tests in GWR raises the problem of multiplicity (Charlton and Fotheringham 2009). It would be inappropriate to carry out 1,736 individual significance tests for each of the seven variables in the model since, at a 95 % significance level, 5 % ($n = 608$) would hypothetically be significant at random. Fotheringham et al. (2002) have suggested a Bonferroni correction to the significance level, but Charlton and Fotheringham (2009) report that Bonferroni is overly conservative and argue that the answer to the multiplicity problem is an avenue for continued research. This issue is beyond the scope of this study.

Setting aside the issue of statistical significance, we analyzed the GWR-generated parameter estimates for the pest and $PM_{2.5}$ variables in two ways. First, we used a standard deviation (SD) break (0.04196 for pest and 0.0198 for $PM_{2.5}$) to map where the parameter estimates were relatively high (1 or more SD above the mean and positive) and low (1 or more SD below the mean and negative). This allowed us to visualize where these two predictors were more closely related to wheezing in the district. We then overlaid these data on a neighborhood map of mean household income from American Community Survey 2006–2011 block group data.

Second, to better understand the local conditions that give rise to our findings, following an approach taken by Chalkias et al. (2013), we characterized the attributes of children for whom $PM_{2.5}$ or pest was a relatively important positive predictor (1 or more SD above mean) and those for whom $PM_{2.5}$/pest was a relatively important negative predictor (1 or more SD below mean) of wheezing severity. Chalkias et al. (2013) used this to determine the range of income, green space, and population density that led to an education level coefficient (scaled so that high values matched lower levels of education) that was negative, slightly positive, and positive in relation to obesity.

To better systematize this type of comparison, we employed two sets of independent samples t tests (one for pest and one for $PM_{2.5}$), grouping the cases based on whether the local parameter was 1 or more SD above mean or 1 or more SD below mean. This provides us with the ability to compare mean scores for the original variables for the two groups. We also conducted a post hoc independent samples *t* test within the “1 or more SD above the mean for $PM_{2.5}$” group because $PM_{2.5}$ was a relatively important positive predictor in two distinct geographic areas of the district. Therefore, we compared these two $PM_{2.5}$ risk groups to each other in terms of mean scores for the original variables.

Results

Global model

In the OLS model (see Table 3A), the variables of interest—pest and $PM_{2.5}$—exhibit modest (insignificant) positive relationships with greater wheezing severity.

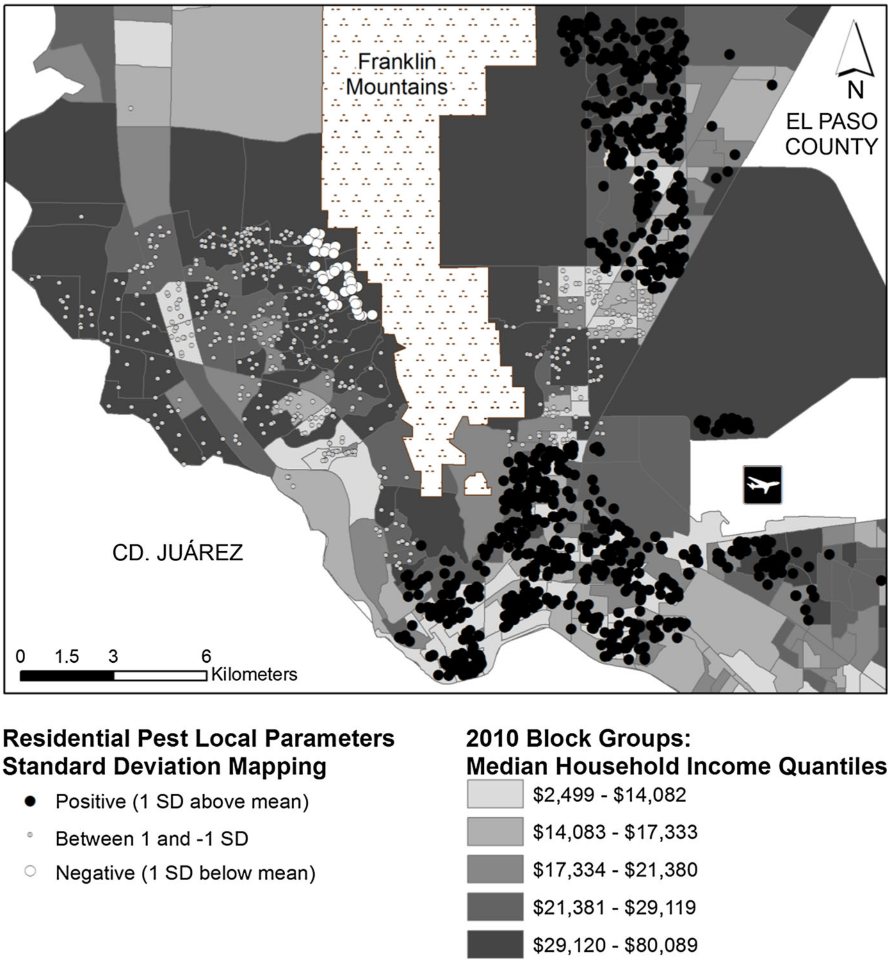

Fig. 3 Standard deviation map of the local pest parameters when predicting wheezing severity

Being male, having postponed or not sought health care due to concerns about cost, having a mother with asthma and/or allergies, and having allergies were statistically significant risk factors.

Local model

Parameter statistics for the GRW model are presented in Table 3B. We found that 55 % of cases had a local R^2 that was higher than the global R^2 for the OLS model, showing that for over half of children, the GWR model showed improved performance over the OLS model. Overall, the model fit for the OLS and GWR models was quite similar (see Table 3, "Model Fit"). Considering the map of the local R^2 values (see Fig. 2), we can see that the children for whom the GWR model

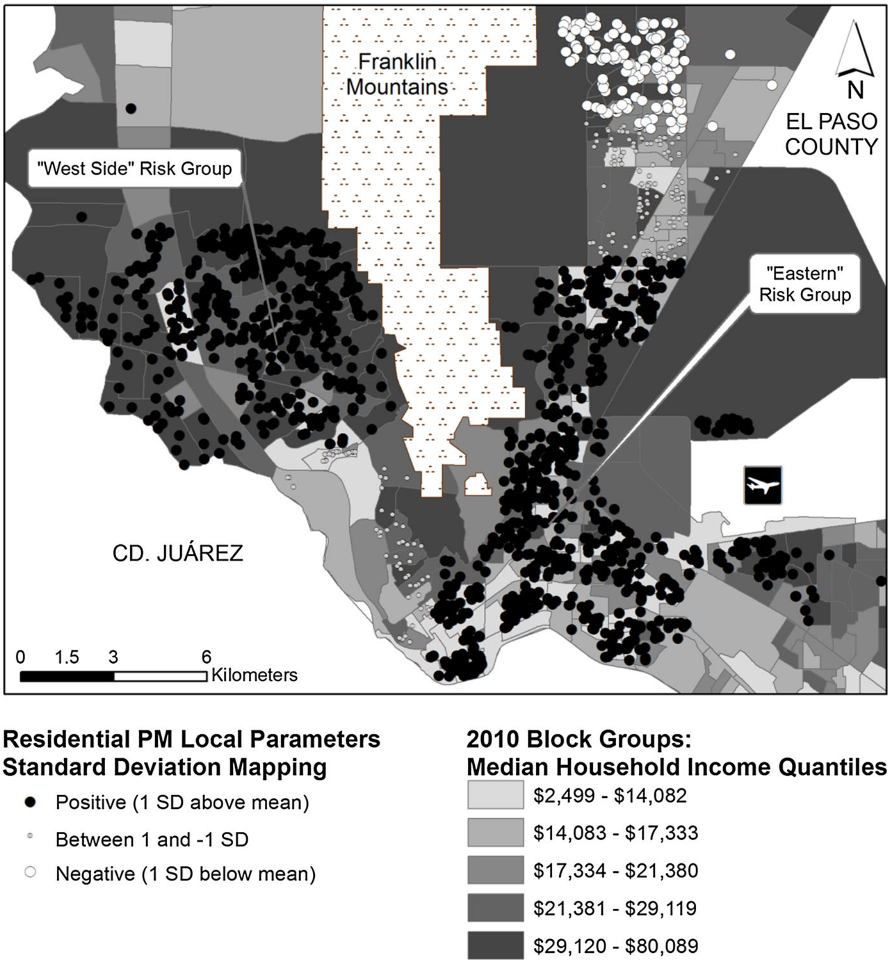

Fig. 4 Standard deviation map of the local $PM_{2.5}$ parameters when predicting wheezing severity

fits best are in the “south side” and “northeast.” Model fit is worst among “west side” children, especially those closest to the mountains.

To visually isolate where pest and residential $PM_{2.5}$ are important predictors, standard deviation maps for the GWR parameter estimates are presented in Figs. 3 and 4. In Fig. 3, the black dots represent children for whom pest was a relatively important *positive* predictor of wheezing; in other words, black dots indicate that pest was a *risk* factor. White dots represent children for whom pest was a relatively important negative predictor of wheezing; in this case, white dots illustrate that pest was a *protective* factor. In Fig. 4, the black dots represent children for whom $PM_{2.5}$ was a relatively important positive predictor of wheezing; in this case, black dots indicate that $PM_{2.5}$ was a *protective* factor. White dots represent children for whom $PM_{2.5}$ was a relatively important *negative* predictor of wheezing; in other words, white dots illustrate that $PM_{2.5}$ was a *protective* factor. Looking at these patterns overlaid on a neighborhood map of mean household income reveals that there is correspondence between children with a pest parameter that is 1 or more SD above the mean and the poorer areas of town: the “downtown,” “south side,” and the “northeast”. The pest variable is actually “protective” (increased odds of pest, less wheezing) among some children living in the wealthier “west side.” $PM_{2.5}$ is an important positive predictor among children on the “west side,” which is relatively affluent, and the “south side,” which is one of the poorer parts of the district. $PM_{2.5}$ is “protective” in the farthest reaches of the “northeast,” where neighborhood incomes are moderate to high.

Table 4 *T* test results characterizing children for whom their local GWR parameters for pest and $PM_{2.5}$ were 1 or more standard deviations above versus below the mean

	GWR pest parameter group	*N*	Mean for original variable		GWR $PM_{2.5}$ parameter group	*N*	Mean for original variable	*p*
Male	1 SD above	943	0.500		1 SD above	1,141	0.520	
	1 SD below	50	0.480		1 SD below	181	0.490	
Mother has asthma and/or allergies	1 SD above	943	0.314		1 SD above	1,141	0.334	
	1 SD below	50	0.260		1 SD below	181	0.403	
Child has allergies	1 SD above	943	0.520		1 SD above	1,141	0.510	*
	1 SD below	50	0.540		1 SD below	181	0.610	
Problems with cost when seeking health care	1 SD above	943	0.249		1 SD above	1,141	0.231	
	1 SD below	50	0.140		1 SD below	181	0.232	
Child is Hispanic	1 SD above	943	0.840	*	1 SD above	1,141	0.813	*
	1 SD below	50	0.460		1 SD below	181	0.680	
Pest	1 SD above	943	0.480		1 SD above	1,141	0.461	*
	1 SD below	50	0.380		1 SD below	181	0.343	
Residential $PM_{2.5}$ (ln, Z)	1 SD above	943	0.245	*	1 SD above	1,141	0.099	*
	1 SD below	50	−1.239		1 SD below	181	−0.949	

$N = 1{,}736$; * $p < 0.05$

Table 4 shows results of the *t* test analyses characterizing the attributes of children for whom their pest or $PM_{2.5}$ parameter was 1 or more SD above the mean versus 1 or more SD below the mean. Children for whom pest was a relatively important positive predictor had significantly higher levels of $PM_{2.5}$ and a significantly greater likelihood of being Hispanic (81 vs. 68 %). They also had a greater likelihood of having pests (although this finding was not quite significant: 49 % of children in the positive group vs. 38 % of children in the negative group). Children for whom residential $PM_{2.5}$ was a relatively important positive predictor had significantly higher levels of $PM_{2.5}$ as well as greater odds of pest exposure (46 % in the positive group as compared to 34 % in the negative group). They were significantly more likely to be Hispanic (84 % in the positive groups as compared to 46 % in the negative group) and not to have allergies (51 % in positive group vs. 61 % in the negative group) than children for whom residential $PM_{2.5}$ was a negative predictor.

An examination of Fig. 4 reveals that there are two areas in which $PM_{2.5}$ was important and positive: a "western" risk group, where neighborhood incomes are relatively high, and an "eastern" risk group encompassing the "south side" and the adjacent southern portion of the "northeast," where neighborhood incomes are low. Figure 3 does not reveal a similar pattern for pest exposure, as all children for whom pest was a relatively important positive predictor are located in the eastern half of the district. Considering the two distinct zones wherein $PM_{2.5}$ was a relatively powerful predictor of more severe wheezing, Table 5 presents results for a *t* test comparing children in the two groups. Children in the "west side" group were significantly more likely to be non-Hispanic (78 % Hispanic vs. 85 % Hispanic), to

Table 5 *T* test results characterizing the two spatial groupings of positive local GWR parameters for $PM_{2.5}$

	GWR $PM_{2.5}$ parameter group (1 + SD above mean)	*N*	Mean for original variable	*p*
Male	"West"	527	0.500	
	"Eastern"	639	0.540	
Mother has asthma and/or allergies	"West"	527	0.347	
	"Eastern"	639	0.327	
Child has allergies	"West"	527	0.520	
	"Eastern"	639	0.500	
Problems with cost when seeking healthcare	"West"	527	0.228	
	"Eastern"	639	0.239	
Child is Hispanic	"West"	527	0.776	*
	"Eastern"	639	0.845	
Pest	"West"	527	0.360	*
	"Eastern"	639	0.540	
Residential $PM_{2.5}$ (ln, Z)	"West"	527	−0.454	*
	"Eastern"	639	0.541	

$N = 1{,}736$; * $p < 0.05$

have lower odds of having pests (36 vs. 54 %), and to have lower levels of $PM_{2.5}$ (below the mean vs. above the mean) as compared to children in the "eastern" group (see Table 5).

Discussion

In the global model, the pest and $PM_{2.5}$ variables were positively associated with wheezing severity, but not significantly. Being male, postponing health care due to concerns about cost, having allergies, and having a mother with asthma and/or allergies were significant risk factors for wheezing severity, which closely aligns with the literature (Carlson and Stroebel 2001; Holt et al. 2013; Wright et al. 2006). In terms of situating the pest exposure findings within the extant literature, an analysis which pooled cross-sectional data from five studies revealed that exposure to any home pests was relatively weakly associated with asthma (Woodin et al. 2011). In comparing our $PM_{2.5}$ findings to other El Paso studies, which used different methodological approaches, we found similar associations. A case-crossover study found that a 10 unit increase in $PM_{2.5}$ was associated with 1–2 % (nonsignificant) daily increase in hospitalizations from asthma and bronchitis (Grineski et al. 2011). An insignificant but positive relationship between higher levels of $PM_{2.5}$ and worse asthma control was also found among a sample of 36 asthmatic children at two elementary schools (Zora et al. 2013). At the same two El Paso schools two years earlier, researchers found that an interquartile increase in $PM_{2.5}$ was associated with an insignificant <1 % increase in airway inflammation for the 30 asthmatic children under study (Sarnat et al. 2011).

However, just because results in this study indicate that social and biological factors—such as postponing care due to concerns about cost or having a mother with asthma—were more important predictors of wheezing severity in the global model than indoor and outdoor environmental factors does not mean that environmental conditions are unrelated to health outcomes. Interpreting results from EHJ analyses must be done carefully. Insignificant and significant results can illuminate the social and environmental structure of health disparities in different contexts (Grineski et al. 2013). Insignificant regression findings do not necessarily mean that environmental exposures or social inequalities have no influence on health. In this case, insignificant findings for pest and $PM_{2.5}$ overall concealed heterogeneity in effects across the school district.

Local results suggest the working hypothesis that the association between air pollution and respiratory problems may vary based on socio-environmental context. This study reveals two types of asthmogenic socio-environments. The first is a lower-income context where children are disproportionately exposed to pests and $PM_{2.5}$ and where both exposures are positively associated with wheezing, especially for Hispanic children. In this situation, pests and $PM_{2.5}$ appear to synergistically amplify wheezing severity. This maps to a classic multiple jeopardies/environmental injustice model whereby mutually reinforcing social, environmental, and health disadvantages can be observed to co-locate in urban space. Something similar was also found in Leicester, where pollution and racial/ethnic minority status were stronger predictors of asthma hospitalization in the inner city than in the surrounding areas (Jephcote and Chen 2012). While not focused on pests, there is evidence that air pollution can combine with other indoor exposures—such as in-home endotoxin levels in Cincinnati (Ryan et al. 2009) and smoking in Beijing (Xu and Wang 1998)—to have synergistic effects on health.

The second is a higher-income socio-environment where children are less likely to have in-home pests or to be Hispanic; children in this situation are exposed to lower levels of $PM_{2.5}$, yet air pollution exposure is counterintuitively associated with more severe wheezing. We hypothesize that, in this context, the air pollution–wheezing linkage is attributable to residents' inability to control exposure to outdoor air pollution relative to other asthma triggers, including indoor pests. This runs counter to the multiple jeopardy model and, from an environmental injustice perspective, is unexpected. However, others have found positive relationships between environmental hazards and health effects in affluent/less polluted areas (Pearce et al. 2010; Tu et al. 2012). In this type of socially advantaged context, air pollution may be a relatively important correlate of respiratory problems, even though levels of exposure are typically lower, because the relationship between pollution and health is not complicated by social deprivation and the challenges of poverty. Qualitative inquiry with parents of asthmatic children revealed that while wealthier parents demonstrated a much greater ability to control their children's home environments than poor parents, they found it quite difficult to protect their children from outdoor air pollution (Grineski 2009). The emergence of two distinct types of asthmogenic socio-environments where outdoor air pollution exposure exerts a more powerful influence highlights the value of GWR for understanding fine-scale spatial variability in contextual determinants of health problems.

Limitations

While the GWR model can reveal spatial variations in the influence of variables on an outcome, a limitation of the technique is that it does not suggest the source of the variation (Wheeler and Páez 2010). Interpretation must be done carefully, using contextual knowledge of the study area (Chalkias et al. 2013). The study relies only on a $PM_{2.5}$ exposure surface; we did not have access to other pollutant surfaces which limit our ability to generalize to other pollutants. Our inability to consider other environmental exposure surfaces (e.g., PM_{10}) may be reflected in the negative $PM_{2.5}$ parameters in the far "northeast" (see the white dots in Fig. 4). In this newly developing, relatively affluent area of the city, air quality could hypothetically be worse further from roadways (the primary sources of $PM_{2.5}$) due to disturbed desert crust. The predictive ability of LUR models to generate pollution surfaces is impacted by the number of monitoring sites employed in its identification (Javier et al. 2007). In this case, the small number of sites might have resulted in an overestimated predictive ability of the LUR model and thus affected its accuracy. Using only residential environmental indicators resulted in an incomplete exposure assessment, given children's space–time geographies. However, we did not have information about school-based pest exposure to include. The study uses a cross-sectional approach, which limits our ability to assess causal relationships. We also relied only on parental reports of children's symptoms using validated ISAAC items. No measures of lung function were collected as part of this study. Lastly, the survey sample is slightly less economically disadvantaged than the EPISD population, which could affect the results.

Conclusion

The answer to the first research question is that having pests and higher levels of $PM_{2.5}$ at a child's residence were modestly associated with greater wheezing severity. The answer to the second research question is more complex and includes two asthmogenic socio-environments. One is characterized by a situation of multiple jeopardy: pest and $PM_{2.5}$ exposures were important predictors of wheezing severity among children living in lower-income neighborhoods, among those who were more likely to be Hispanic, and among those with higher levels of $PM_{2.5}$ exposure at their home sites. The second ran counter to a multiple jeopardy model: $PM_{2.5}$ was an important positive predictor of wheezing severity in some wealthier neighborhoods, among non-Hispanic children, and among those for whom home site levels of outdoor $PM_{2.5}$ and odds of exposure to pests were lower. In more general terms, these results suggest that the assumption underpinning a good deal of research—that uneven environmental exposures produce correspondingly unequal health impacts and that these effects are stable across space—is not tenable.

Because of their capacity to identify specific areas at risk, GWR models lend themselves to locally relevant interventions. From a policy perspective, this research demonstrates particular areas in the city of El Paso where particulate matter and pests are important correlates of children's wheezing severity. This information could be used to aid local and state environmental and public health agencies to design targeted interventions in areas where they are most needed. In this case, GWR results suggest pest remediation as a tool to improve children's respiratory health in the "downtown," "south side," and "northeast" sides of El Paso. Pollution reduction efforts would benefit all children, given what is known about the deleterious health effects of air pollution and the positive coefficient in our global model. Increasing awareness of air pollution might be particularly important on the "west side," given that comparatively low levels of $PM_{2.5}$ play an important role in children's wheezing severity there. Given the apparently synergistic effects of exposure to pests and $PM_{2.5}$ in parts of the study area, addressing even just one of these factors would be advantageous to children's health. Results also suggest that GWR is a powerful tool that should be more widely used by researchers in their quest to understand complex relationships between environmental conditions, social characteristics, and health inequalities.

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