GIS-Based Retrospective Air Pollution Exposure Estimation and Social-Environmental Interactions in Asthma Etiology

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Abstract

Disproportionate life stress and increased susceptibility to pollution has been proposed as a major pathway linking socioeconomic position, environmental exposures, and health disparities. We consider whether a chronic stressor [lifetime exposure to violence (ETV)] may increase susceptibility to traffic-related air pollution, in the etiology of childhood asthma.

GIS-based models were created to estimate residential exposures to traffic-related air pollution (NO_2) for 413 children in a community-based pregnancy cohort. The model was calibrated using 19 years of monthly NO_2 data from 13 neighborhood sites. Merging pollution estimates with questionnaire data, we explore the independent and multiplicative contributions of social and environmental factors to childhood asthma.

Among children always living in the East Boston area, odds ratios indicate elevated risk of asthma with above-median NO_2 during the year of diagnosis (OR = 2.10 (95% CI = 1.16 to 3.81), which was greater among children with higher ETV (OR = 3.15 (1.69-5.87). Multivariate analyses correcting for maternal asthma, smoking, and education, child's age and sex indicate elevated risk of asthma with increasing NO_2 exposures solely among children with above-median ETV (adj OR = 2.71 (1.67 - 4.39). We find suggestive evidence of greater susceptibility to air pollution among children exposed to violence, with synergistic effects in asthma etiology.

Introduction

The distinct gradient of socioeconomic position (SEP) on health may be explained, in part, by increased exposure to environmental pollution, and greater susceptibility to its effects. Air pollution, for instance, is higher along major roadways, near power plants and industrial sites, where property values are lower, and lower-income populations reside. In addition, higher life stress has been proposed as a primary pathway through which SEP impacts health. Chronic stress is hypothesized to impact health through two immune-related physiological pathways: (1) dysregulation of the hypothalamic-pituitary-adrenocortical (HPA)-axis (and, relatedly, glucocorticoid resistance), and (2) the sympathetic-adrenal-medullary (SAM) axis. Together, these observations suggest that immune-altering social stressors, such as exposure to violence (ETV), may be higher in the same communities where pollution levels are higher, and lower-SEP populations may experience both greater exposures *and* greater susceptibility.

Social and physical environmental exposures are often spatially collinear, and both may impact the same physiological pathways (HPA-axis, SAM dyregulation, glucocorticoid resistance, oxidative stress) and health outcomes (i.e., respiratory and cardiovascular disease), there is a call for stronger methods to disentangle their effects and investigate synergies. Here, we consider one chronic urban stressor, witnessing violence, and investigate its potential to increase susceptibility to traffic-related air pollution in the etiology of childhood asthma.

Methods

This paper is developed in two parts:

(1) GIS-based retrospective model to estimate exposures to primary traffic-related air pollution at cohort homes, using the predictive model:

$$[NO_2]_{ij} = \beta_{1j}*Year_j + \beta_2*(traffic_i) + \beta_3*(land\ use_i) + e_{ij} \quad \text{(Equation 1)}$$

Where $Year_j$ is sampling year, $traffic_i$ is a suite of traffic density characteristics (Table 1), derived from Massachusetts Highway Department (MHD) 1990 traffic data, land use variables from 2000 U.S. Census and aerial photography. Spatial variables were created in ArcGIS 9.1, models in Proc GLM in SAS Version 9.1. Within-site autocorrelation was examined; comparable model results were achieved with random effects.

(2) epidemiologic analyses focusing on multiplicative effects between violence and air pollution exposure in the etiology of asthma.

$$\text{logit [asthma diagnosis]} = \beta_0 + \beta_1*age + \beta_2*sex + \beta_3*maternal\ smoking + \beta_4*maternal\ education + \beta_5*[NO2] + \beta_6*[[NO_2] * High\ RaschETV] + e \quad \text{(Equation 2)}$$

The Rasch ETV score indicates lifetime exposure to violence using the My Child's ETV Scale, which asks, "Has your child witnessed any of the following: Slapping, kicking, punching? A stabbing? A shooting? Heard gun shots? Verbal or physical abuse by your partner?" An Empirical Bayesian model weighed events by frequency, severity (i.e., extent of injury), and child's familiarity with victim/ perpetrator (Franco Suglia 2006).

Figure 1: 19-year monthly NO_2 sampling sites across East Boston area

Figure 2: Annual averages in NO_2 concentrations (ppb) across 13 neighborhood sampling sites.

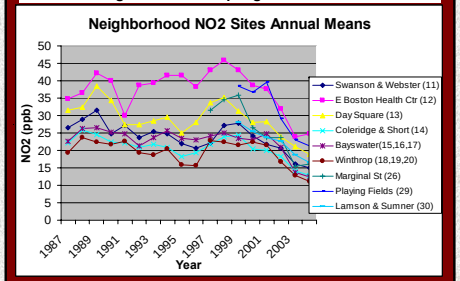

Table 1: Traffic Indicators explored as predictors of monthly NO2 concentrations at sampling sites and cohort homes

Indicator type	Indicator	Units
Cumulative densities:	Unweighted density within: 50, 100, 200, 300, 500m	Vehicle-miles/ day-m^2
	Kernel-weighted density: 50, 100, 200, 300, 500m	Vehicle-miles/ day-m^2
	Density of urban roads (> 8500) within 200m	Vehicle-miles/ day-m^2
Summary measures:	Total roadway length within: 50, 100, 200, 300, 500m	Miles
	Total ADT*Length (VMT) within 200m	Vehicle-miles/day
Distance-based measures	Distance to nearest urban road (>8500 cars/day)	Meters
	To nearest major road (>13,000 cars/day)	Meters
	To nearest highway (>19,000 cars/day)	Meters
	To nearest MHD-designated truck route	Meters
Characteristics of nearest major road:	Average daily traffic (ADT)	Vehicles/day
	ADT/ Distance to major road	(Vehicles/day) /meter
	Diesel fraction	*Percent (%)*
	Trucks per day	*Vehicles/ day*
	Trucks/ Distance to major road	*(Vehicles/day) / meter*

Table 2: NO_2 model for annual average concentration (ppb) at 13 sampling sites. Reference year is 2004.

	Overall (R^2 = 0.83) Estimate (p-value)
Intercept	10.64 (<.0001)
Year (categorical)	-- (<.0001)
1990	13.48
--	--
1997	12.01
Distance to Major Road (>13,000 cars/day)	$-1.27*10^{-3}$ (<.0001)
Kernel traffic density within 500m (VMT/day)	0.0434 (<.0001)
Population Density (persons/ km^2)	$1.086*10^{-4}$ (<.0001)

Figure 3: NO_2 Estimates at cohort homes

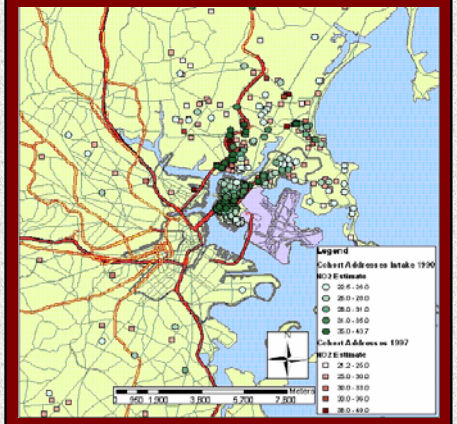

Table 3: NO_2 on Asthma diagnosis, by exposure period

Period of Interest	Measure for Asthmatic child	Measure for non-asthmatic	Odds Ratio (95% CI) (p-val)
Lifecourse, to Diagnosis	Year of birth to diagnosis	Year of birth through follow-up	1.06 (.96, 1.17) (.24)
Year of birth	Year of birth	(same)	0.98 (.90, 1.07) (.65)
First 5 years	First 5 years of life	(same)	0.99 (.91, 1.08) (.80)
Date of ETV questionnaire	Mean exposure 1997	(same)	1.004 (.93, 1.09) (.92)
Between ETV & diagnosis	Year of ETV, to diagnosis	Year of ETV, to end of follow-up	1.08 (.98, 1.18) (.11)
Year of diagnosis	Year of diagnosis	Year of birth + 5 yr (median diag age)	**1.10 (1.02, 1.18) (.01)**
One year prior to diagnosis	One year prior to diagnosis	One year prior to (yr of birth + 5 yr)	1.06 (.96, 1.16) (.25)

Results

Univariate analyses for the full cohort show elevated odds of asthma with above-median NO_2 exposures, but no independent effect of ETV. Both exposures metrics are likely more accurate for children always living in the East Boston area; among these children, we find an independent effect of NO_2 on diagnosis, which was greater with above-median ETV. Multivariate logistic regression accounting for confounders indicates elevated odds of asthma with increasing NO_2 only among children with high ETV, with a significant difference in slopes by ETV (p=.03). For lifetime residents, odds of developing asthma increased with NO_2 only among those with high ETV, also a significant difference.

	Low NO_2	High NO_2
Low ETV	22/ 69 = **0.32**	29/ 80 = **0.36**
High ETV	19/ 93 = **0.20**	34/ 64 = **0.53**

Table 4: Odds of asthma diagnosis by NO_2 and ETV, entire cohort (n=413)

OR (High ETV) = **1.04 (0.67, 1.61)** (p=.87)
OR (High NO_2) = **1.71 (1.09, 2.68)** (p=.02)
OR (High NO_2, High ETV) = **1.84 (1.12, 3.01)** (p=.02)

Table 5: Odds of asthma diagnosis by NO_2 and ETV, among 258 children always living in East Boston/ Winthrop

OR (High ETV) = **1.35 (0.76, 2.40)** (p=.31)
OR (High NO2) = **2.10 (1.16, 3.81)** (p=.01)
OR (High NO2, High ETV) = **3.15 (1.69, 5.87)** (p=.0003)

	Low NO_2	High NO_2
Low ETV	12/ 42 = **0.29**	15/ 57 = **0.26**
High ETV	8/ 58 = **0.14**	26/ 33 = **0.79**

Table 6: Odds ratios from multivariate analysis in Proc Logistic. NO_2 during year of diagnosis is a continuous centered predictor, with SD = 1.0.

	Full cohort (413 children (26% prev)) Adj OR (95% CI) (p-val)	Lifetime residents (255 children (25% prev)) Adj OR (95% CI) (p-val)
Maternal Asthma	1.26 (.59, 2.84) p = .58	0.80 (.25, 1.69) p = .70
In-utero tobacco	1.04 (.43, 2.53) p = .93	1.79 (.51, 6.33) p = .36
Maternal smoking	1.15 (.73, 1.80) p = .56	0.88 (.46, 1.69) p = .69
Education (Less than HS)	1.11 (.70, 1.77) p = .66	1.14 (.61, 2.14) p = .67
Child's Sex (female)	0.87 (.56, 1.38) p = .56	0.62 (.34, 1.14) p = .11
Child's Age (7+ years)	1.23 (.77, 1.98) p = .38	1.10 (.57, 2.10) p = .78
NO_2: Low ETV	**1.08 (.79, 1.47) p = .62**	**0.92 (0.62, 1.37) p = .68**
NO_2: High ETV	**1.82 (1.27, 2.61) p =.001**	**2.71 (1.67, 4.39) p< .0001**

Acknowledgements

Many thanks to Marina Jacobson Canner for programming assistance and to Shakira Franco Suglia for creation of Rasch ETV estimates.