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 (NO2) for 413 children in a community-based pregnancy cohort. The model was calibrated using 19 years of monthly NO2 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 NO2 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 (1.68-6.87)). Multivariate analyses correcting for maternal smoking, asthma, and education, child’s age and sex indicate elevated risk of asthma with increasing NO2 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 sympathetic 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-adrenomedullary (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 colinear, and both may impact the same physiological pathways (HPA-axes, SAM dysregulation, glucocorticoid resistance, oxidative stress) and health outcomes (i.e., respiratory and cardiovascular diseases). 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:

\[ (NO2) = β_0 + β_1 \text{traffic} + β_2 \text{land use} + ε \]  

Where \( (NO2) \) is NO2 concentration, \( β_0 \) is a constant, \( β_1 \) is a suite of traffic density characteristics (Table 1), and \( ε \) is a residual error term. The model was derived from Massachusetts Highway Department (MHD) 1989 traffic data, land use variables were measured in ArcGIS 3.1 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.

Logit [asthma diagnosis] = β0 + β1 age + β2 sex + β3 maternal smoking + β4 maternal education + ε

Where \( \text{sex} = 0 \) for male and \( \text{sex} = 1 \) for female, \( \text{maternal smoking} = 0 \) for nonsmoking and \( \text{maternal smoking} = 1 \) for smoking mother.

The Rasch ETV score indicates lifetime exposure to violence using the My Child’s ETV Scale, which asks: "Has your child ever been experiencing: Stressing for the child's sake?" "A shooting? Heard gun shots? Verbal or physical abuse by your partner?" An Empirical Bayesian model weighted events by frequency, severity (i.e., extent of injury), and child’s familiarity with victim/perpetrator (Franco Suglia 2008).

Figure 1: 19-year monthly NO2 sampling sites across East Boston area

Figure 2: Annual averages in NO2 concentrations (ppb) across 13 neighborhood sampling sites.

Figure 3: NO2 Estimates at cohort homes

Table 1: NO2 model for annual average concentration (ppb) at 13 sampling sites. Reference year is 2004.

Table 2: NO2 model for annual average concentration (ppb) at 13 sampling sites. Reference year is 2004.

Table 3: NO2 on Asthma diagnosis, by exposure period

Table 4: Odds of asthma diagnosis by NO2 and ETV, entire cohort (n=413)

Table 5: Odds of asthma diagnosis by NO2 and ETV, among 258 children always living in East Boston/Winthrop

Table 6: Odds ratios from multivariable analysis in Proc Logistic, NO2 during year of diagnosis is a continuous centered predictor, with SD=1.0

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