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Geographic and social disparities in exposure to air neurotoxicants at U.S. public schools

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ABSTRACT

Children are especially vulnerable to the health and developmental impacts of environmental hazards and they spend significant portions of their days at school. Yet there are no national-level studies examining school-level environmental inequalities and few have examined disparate exposure to neurological air toxicants, even though chronic exposure to air pollution impacts children's brain functioning. We paired information about the geographic locations and demographics of each public school in the US with air neurotoxicant exposure estimates pertaining to 24 known neurotoxicants included in the US Environmental Protection Agency's National Air Toxics Assessment. Using bivariate and multivariate statistics, we tested for environmental injustices in air neurotoxicant exposure at 84,969 US public schools. Metropolitan New York City (EPA Region 2) is the geographic region most burdened by air neurotoxicant exposures at schools since one-third of all schools in that region are in the top 10% (at "high risk") for ambient neurotoxicant exposure among all schools nationwide. Students attending “high risk” public schools nationwide are significantly more likely to be eligible for free/reduced price meals, and to be Hispanic, black, or Asian/Pacific Islander (API). They are significantly less likely to be white or of another race. In a multivariate generalized estimating equation controlling for school district effects, schools with greater proportions of Hispanic, black, and API students, schools with higher enrollment, and schools located in more urban (vs. rural) counties face greater risks. Schools serving the youngest students (e.g., pre-kindergarten) have greater levels of risk than schools serving older students. Across all analyses, this study shows that racial/ethnic minority children are bearing the brunt of air neurotoxicant exposures at school, which may be unequally impacting their school performance and future potential.

1. Introduction

Environmental justice (EJ) research focused on children and schools is currently underdeveloped (Strife and Downey, 2009). This is despite the fact that children are especially vulnerable to the health and developmental impacts of environmental injustice, due to their unique biological vulnerabilities, age-related patterns of exposure, and lack of control over their own environmental circumstances (Landrigan et al., 2010). They also spend significant time at school, approximately 6.6 h per day for 180 days per year for public school students in the US (National Center for Education Statistics, 2008).

A handful of previous EJ studies have examined environmental risks for schoolchildren. EJ studies of the Los Angeles Unified School District, CA (USA) (Pastor et al., 2002; Pastor et al., 2004) and the State of California (Pastor et al., 2006) found that racial/ethnic minority students, especially Hispanic students, were overrepresented as attendees of schools with higher outdoor cancer and respiratory air toxics exposures. Using bivariate correlations, another study examined Sacramento (CA) public schools and found that higher levels of PM2.5 emissions from road traffic were correlated with higher percentages of Black, Hispanic and multi-ethnic students and students eligible for subsidized meals, and a lower percentage of white students (Gaffron and Niemeier, 2015). An individual-level study of schoolchildren in Orange County, FL (USA) examined the locations of students’ homes and schools in relation to major point sources, small area emitters, and major roadways, and found that Hispanic and black children were significantly overrepresented (for both school and home locations) relative to non-Hispanic white children in areas proximate to each type of air pollution source (). Another study focused specifically on the areas surrounding the top 100 point source polluters of developmental and neurological air toxicants in the United States; it found that a significant proportion of the top industrial polluters were located in close proximity to multiple schools and that as the proportion of disadvantaged residents living near the facility went up, so did the number of schools

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(Legot et al., 2010). While that particular study provided an important first step in examining air neurotoxicant exposure at US schools, it was limited by restricting the analyses to only the 1–2 miles around each facility and using only correlations.

Thus, while prior EJ studies of schoolchildren suggest particular city- or state-wide patterns of disparate exposure based on race/ethnicity, knowledge of environmental injustices experienced by this vulnerable population remains limited. No national EJ studies of schools have been undertaken, a gap which our proposed study fills. Additionally, previous distributional studies of children and environmental injustice have focused on cancer or respiratory health risks from air pollution.

Rarely have neurological air toxics been examined, even though emerging evidence indicates that chronic exposure to air pollution may negatively impact children's brain functioning (Brockmeyer and D'Angiulli, 2016; Calderón-Garcidueñas et al., 2016). Children are more vulnerable to air pollution than are adults, due to their higher breathing rate to body size ratio, their lungs' less developed natural barriers, and their propensity to spend more time outside (Brockmeyer and D'Angiulli, 2016). Neurological air toxics include ultrafine particulate matter (PM), polychlorinated biphenyls, mercury, manganese and toluene, among many others (Bandyopadhyay, 2016; Costa et al., 2014; Grandjean and Landrigan, 2014; Loane et al., 2013; World Health Organization, 2010). Neurotoxicants are released into the air through combustion engines, industrial activities, power plants, mining, and refuse incineration. While diet has long been considered the primary vehicle of exposure to neurotoxicants, inhalation exposure is a common and important route of exposure (Costa et al., 2014). Inhalation can even be a more toxic route of exposure than ingestion, as is the case with manganese (Environmental Protection Agency, 2016b).

When air pollutants are inhaled, they damage the body's natural barriers (e.g., nasal and lung epitheliums and the blood brain barrier) and gain entry to the body (Calderón-Garcidueñas et al., 2015). Upon arrival, they generate an innate immune response involving small proteins called cytokines, which are present in blood and cerebrospinal fluids. Cytokines cause tissue swelling and immune cell release of cytotoxic species, while signaling the production of additional cytokines. This induces neuroinflammation in the brain and leads to diffuse loss and damage of neural tissues. Cytokines are also associated with the development of white matter hyperintensities (WMH), which are areas of demyelinated neurons in the brain that negatively impact synaptic capacities. WMH are associated with global congitive deficits. Air pollution thus causes cognitive deficits through neuroinflammation and cell loss, and via damange to myelin and neural functioning (Brockmeyer and D'Angiulli, 2016). In terms of neurotoxicants specifically, they have additional properties which cause neurological harm. Lead, for example, crosses the blood-brain barrier by substituting as calcium ions and wreaks havoc on the prefrontal cortex, hippocampus, and cerebellum by interfering with the regulartory functioning of calcium (Sanders et al., 2009).

The consequences of inhaling air pollution are far-reaching. Autopsies of otherwise clinically healthy youth, for example, revealed that those from the high pollution megacity of Mexico City had brain structures resembling those with early stage Alzheimer's disease; similar damage was not present in the brains of youth living in the two low pollution control cities of Veracruz and Tlaxcala (Calderón-Garcidueñas et al., 2008). Given that low income and racial/ethnic minority populations tend to experience the environmental injustice of disproportionately high exposure to pollution (Mohai et al., 2009; Mohai and Saha, 2015; Pais et al., 2014), it is likely that these detrimental effects on brain functioning are not evenly distributed across society. However, this has not yet been investigated.

To begin to address this important knowledge gap, this study tests for environmental injustices in exposures to air neurotoxicants in an analysis that includes all US public schools. The **first research question** is: which US EPA regions house disproportionate numbers of "high risk" schools? "High risk" schools are those in the top 10% of all schools for neurotoxicant risk. The **second research question** is: are racial/ethnic minority and economically-deprived students disproportionately at "high risk" to ambient neurotoxicants as compared to all US public school students? Finally, the **third research question** is: accounting for geographic clustering and other factors known to influence exposure to toxics, how do the racial/ethnic and economic composition of the schools and the grade level of student served relate to air neurotoxicant risk?

2. Materials and methods

2.1. Unit of Analysis: United States Public Schools

We downloaded the geographic locations and demographics of each public school in the US for 2011 from the National Center for Education Statistics using the ElS*i* Table Generator tool (National Center for Education Statistics, 2017). We selected 2011 since it aligns with the most recent National Air Toxics Assessment. This ElS*i* tool provides users with access to the Common Core of Data (CCD), which is the US Department of Education's primary database on public elementary and secondary schools in the United States. The CCD surveys are conducted annually at all public elementary and secondary schools throughout the United States. CCD contains general descriptive information on schools and on their students and staff. Of relevance to this project, we downloaded the latitude and longitude of each school, information about the racial/ethnic make-up of the student body, eligibility for free and reduced price meals, and the lowest and highest grade offered at the school. Using ArcGIS 10, we plotted all schools based on their latitude and longitude (Fig. 1). We removed schools with fewer than 100 children in 2011 from the analysis (n = 8876) to create more stable proportion variables.

Mapping the schools allowed us to create other variables that were needed for the analysis. Using the spatial join tool in ArcGIS 10, we were able to determine which EPA region and school district each school fell within. To do this, we obtained spatial data for US school districts in 2011 from the National Historical Geographic Information System (NHGIS) portal (IPUMS, 2016) and located each school within its **school district** (n = 11,845).

2.2. Analysis Variables

2.2.1. Dependent Variables

The neurotoxicant measures come from the US Environmental Protection Agency's National Air Toxics Assessment (NATA). The NATA includes all hazardous air pollutants (HAPs) regulated by the US Clean Air Act (except criteria pollutants) that are known or suspected to cause cancer or neurological, developmental, respiratory, immunological, and reproductive damage (Environmental Protection Agency, 2016a). The EPA models the exposure risks of different pollutants additively to estimate an aggregate risk score for each census tract (Environmental Protection Agency, 2016a). Exposure risk scores are based on the concentration of each chemical to which people are exposed and each chemical's reference concentration (i.e., the amount of toxicity below which long-term exposure to the general population is not expected to result in adverse effects). The NATA is currently the best available secondary data source for spatially explicit characterization of toxic air pollution exposure risk in the US and has been used to measure census tract-level pollution in high-tier EJ and environmental health studies (Linder et al., 2008; Roberts et al., 2013).

Air neurotoxicant exposure estimates for the schools were created from the EPA's NATA census tract-level databases from 2011 using ArcGIS 10. The 24 known neurotoxicants included in the NATA [see Appendix H in Environmental Protection Agency (2015)] are: 1,1,1-Trichloroethane, 2,4-Dinitrotoluene, Acrylamide, Allyl Chloride, Benzidine, Calcium Cyanamide, Carbon Disulfide, Carbonyl Sulfide, Cresol/